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Dynamics of an SEIR epidemic model with saturated incidence rate including stochastic influence

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Abstract

This paper aims to develop a stochastic perturbation into SEIR (Susceptible-Exposed-Infected-Removed) epidemic model including a saturated estimated incidence. A set of stochastic differential equations is used to study its behavior, with the assumption that each population's exposure to environmental unpredictability is represented by noise terms. This kind of randomness is considerably more reasonable and realistic in the proposed model. The current study has been viewed as strengthening the body of literature because there is less research on the dynamics of this kind of model. We discussed the structure of all equilibriums' existence and the dynamical behavior of all the steady states. The fundamental replication number for the proposed method was used to discuss the stability of every equilibrium point; if $R_0 < 1$, the infected free equilibrium is resilient, and if $R_0 > 1$, the endemic equilibrium is resilient. The system's value is primarily described by its ambient stochasticity, which takes the form of Gaussian white noise. Additionally, the suggested model can offer helpful data for comprehending, forecasting, and controlling the spread of various epidemics globally. Numerical simulations are run for a hypothetical set of parameter values to back up our analytical conclusions.

Keywords. SEIR model, Basic reproduction number, Stochastic stability, White noise.

2010 Mathematics Subject Classification. 93E15, 60H40, 34D20.

1. Introduction

Communicable diseases have a detrimental effect on people's lives and constitute a risk to human health. Many academics have used mathematical models to investigate the transmission of infectious diseases and their control techniques. Kermack and McKendrick [10–12] introduced some current compartment models. Many scholars have developed alternative infectious disease models based on these models, which incorporate modern computational techniques, since then. It is common as individuals of society may be able to infect a huge number of people, there is clearly diversity in human nature and interpersonal interactions. As a result, it is more appropriate to represent disease transmission via complicated networks. In addition, certain diseases have a time of incubation. Susceptible, infected, and recovered populations, as well as undiagnosed infectious populations, can be divided. Individuals with diseases such as measles and pertussis who go undiagnosed are known as undiagnosed infectious populations. Populations without a diagnosis significantly contribute to the transmission of infectious diseases. Modeling incubation periods for many diseases is a strong suit for the susceptible-exposed-infectious-recovered (SEIR) approach.

In the real world, environmental factors like precipitation, temperature, and specific humidity invariably have an impact on biological populations. It is required to inject randomness into the deterministic model in order for it to be more realistic. The significance of stochasticity in epidemic dynamics has long been recognized. Because the deterministic predator–prey model has some limitations in accurately predicting future dynamics, many researchers

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have begun to investigate how noise affects the dynamics of prey–predator systems (see, for example, [6–8] and [15]). The stochastic SEIR epidemic approach too has interesting findings. For instance, Zhang and Wang developed stochastic SEIR schemes using discontinuities for simulate its extensive transmission of infectious diseases brought on by negligence in the medical field [28].

Witbooi established that the disease-free equilibrium has almost certain exponential stability in an SEIR epidemic approach through separate stochastic disturbances [16]. Liu et al. explored the asymptotic behaviour of the disease-free equilibrium and the endemic equilibrium about a stochastic delayed SEIR epidemic model using nonlinear occurrence[26]. A two-group stochastic SEIR system exhibiting infinite delay was developed by Liu et al., who also discovered the prerequisites of endemic equilibrium asymptotic stability [16]. In China, where human rabies is one of the biggest public health issues, the SEIR model has also been utilized to investigate efficient preventative and control approaches [29]. About the influenza A(H1N1), the traditional SEIR model has been combined with statistical approaches to estimate the prevalence of A(H1N1) in Singapore [20, 24]. It has been used to predict the infected individuals, hospital bed shortages, and effectiveness of vaccination in a city of Japan.

The dynamic behaviour of a nonlinear stochastic SEIR epidemic approach using various population sizes was examined by Han et al. in [3]. Considering it enables us to include disturbance within deterministic biological systems to highlight the effects of ecological fluctuation, whether it is a random disturbance on differential setups or ecological vacillations in borders [13, 14, 25–27, 30]. Numerous researchers [2, 4, 5, 17–19] recently concentrated on parts of stochastic populations that are annoyed with recurring sound (Brownian movement).

To account for unpredictability in the model, epidemic models with random perturbations have been extensively investigated. Recent research has shown evidence of a possible mechanism by which the development of COVID-19 may have been directly influenced by environmental change. The paper emphasizes, in particular, the stochastic stability of the endemic equilibrium.

The breakup of an article's organization is as described in the following: With the presumption that exposed as well as infected people come into contact with susceptible people at the same rate, the model formulation is explored in section 2 of the article. In section 3, we explored a qualitative analysis of the model: In section 3.1, we compute the proposed model's fundamental reproduction number; In section 3.2, we evaluate the local stability of the disease-free and endemic equilibrium points in terms of R_0. Section 4 discuss the proposed system's stochastic stability. In section 5 of the numerical simulations, the utility of our approach is demonstrated. Section 6 of our findings brings us to a close.

2. Mathematical Model

The SIR model, a compartmental model, divides the population into three categories at time t: the susceptible $S(t)$, the infected $I(t)$, and the recovered $R(t)$ individuals. We can better understand how the presence of infectious persons affects the risk of infection among susceptible individuals by using the transmission-dynamic epidemic models. A person who has contracted an infection recovers after receiving therapy. The differential equation system now looks like this:

$$\begin{aligned} \frac{dS}{dt} &= -\theta SI, \\ \frac{dI}{dt} &= \theta SI - \eta I, \\ \frac{dR}{dt} &= \eta I. \end{aligned} \tag{2.1}$$

where θ, η denote the rate of infection, rate of infection recovery respectively, and N is the total population quantity

such that $S + I + R = N$ for all t. These older models, however, did not take into consideration the likelihood of immigration and emigration. We used a demographics-based model in which the emigration rate ν and the immigration rate r were taken into consideration.

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \theta SI - \delta S,\\
\frac{dI}{dt} &= \theta SI - \eta I - \delta I,\\
\frac{dR}{dt} &= \eta I - \delta R.
\end{aligned} \tag{2.2}$$

This model includes a bilinear incidence rate, and in the suggested model, disease transmission between S and I is taken into account using a nonlinear incidence rate $\dfrac{\theta SI}{1+\phi_1 S+\phi_2 I}$, which represents the effect of behavioral changes in the susceptible population caused by an increase in the infective population on disease transmission. Now that the population of type SEIR has been specified, the differential equations that control this model are as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \delta S - \frac{\theta SI}{1+\phi_1 S+\phi_2 I},\\
\frac{dE}{dt} &= \frac{\theta SI}{1+\phi_1 S+\phi_2 I} - (\delta+\zeta)E,\\
\frac{dI}{dt} &= \zeta E - (\delta+\eta)I,\\
\frac{dR}{dt} &= \eta I - \delta R.
\end{aligned} \tag{2.3}$$

Where E is the exposed individuals, π is the population's frequency of recruiting, δ is the rate of natural death, η is the infected people's rate of recovery, θ is the rate of exposure, and is the rate at which exposed people get an infection. Thus $1/\zeta$ presents the mean latent period. ϕ_1, ϕ_2 are the metrics used to assess how social, psychological, or other systems have an impact. Notably, the first three equations under discussion may be solved independently of the fourth, indicating that the fourth equation is essentially redundant.

3. Qualitative analysis of the model

The basic reproduction number is one of the most important thresholds to consider when analyzing infectious disease models that quantify disease invasion or extinction in a population. In this section, we calculate our model's basic reproduction number (2.3) and investigate the disease-free equilibrium's locally asymptotically stability.

3.1. **Equilibrium points and their stability.** An infection-free steady state $\Omega_1(\pi/\delta, 0, 0)$ and an endemic steady state $\Omega_2(S^\star, E^\star, I^\star)$ are present in the system (2.3) at all times.

where $S^\star = \dfrac{\pi\left(\pi\zeta\phi_2 + (\delta+\eta)(\zeta+\delta)\right)}{\pi\zeta\phi_2\delta + (\delta+\eta)(\zeta+\delta)\left[(\delta+\pi\phi_1)(R_0-1)+\delta\right]}$, $E^\star = \dfrac{(\delta+\eta)\,I^\star}{\zeta}$,

$I^\star = \dfrac{\zeta\pi(\delta+\pi\phi_1)(R_0-1)}{\pi\zeta\phi_2\delta + (\delta+\eta)(\zeta+\delta)\left[(\delta+\pi\phi_1)(R_0-1)+\delta\right]}$ and R_0 (basic reproduction rate) is defined as

$R_0 = \dfrac{\theta\pi\zeta}{(\delta+\eta)(\zeta+\delta)(\delta+\pi\phi_1)}$.

Hence, an endemic steady state Ω_2 exists when $R_0 > 1$.

3.2. **Stability of the steady state solutions.** The linearized system of the model (2.3) Jacobian matrix is

$$J = \begin{pmatrix} -\delta - \dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} & 0 & -\dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} \\ \dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} & -(\delta+\zeta) & \dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} \\ 0 & \zeta & -(\delta+\eta) \end{pmatrix}. \tag{3.1}$$

We define the above Jacobian matrix at to examine the stability of infected free equilibrium Ω_1.

$$J_{\Omega_1} = \begin{pmatrix} -\delta & 0 & -\dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star)^2} \\ 0 & -(\delta+\zeta) & \dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star)^2} \\ 0 & \zeta & -(\delta+\eta) \end{pmatrix}. \tag{3.2}$$

The characteristic equation of (3.2) is given by

$$(\lambda+\delta)(\lambda^2+p_1\lambda+p_2)=0,$$

the coefficients are given by $p_1=(\delta+\eta)+(\delta+\zeta)$, $p_2=(\delta+\eta)(\delta+\zeta)-\dfrac{\theta\zeta S^\star}{(1+\phi_1 S^\star)}$, on using $S^\star$ in this, we obtain $p_2=(\delta+\eta)(\delta+\zeta)\,[1-R_0]$. Therefore, we have $\lambda=-\delta$ and $\lambda^2+p_1\lambda+p_2=0$ that is quadratic equation in λ. If two negative roots exists in this equation, then Ω_1 is stable; otherwise, it is unstable. If and only if $p_1>0$ and $p_2>0$, the aforementioned quadratic equation meets Routh Hurwitz condition for having two negative roots. Thus p_1 is indeed positive and $p_2>0$ if $R_0<1$. Therefore, infected free steady state is stable for$R_0<1$.

The Jacobian matrix of (2.3) at endemic equilibrium can be obtained as follows.

$$J_{\Omega_2} = \begin{pmatrix} -\delta - \dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} & 0 & -\dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} \\ \dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} & -(\delta+\zeta) & \dfrac{\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2} \\ 0 & \zeta & -(\delta+\eta) \end{pmatrix}. \tag{3.3}$$

The characteristic equation of (3.3) is given by

$$\lambda^3+L_1\lambda^2+L_2\lambda+L_3=0. \tag{3.4}$$

The coefficients are $L_1=\delta+(\delta+\eta)+(\delta+\zeta)+\dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}$, $L_2=[(\delta+\eta)+(\delta+\zeta)]\left[\delta+\dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}\right]+(\delta+\eta)(\delta+\zeta)-\dfrac{\zeta\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}$, $L_3=\delta\left[(\delta+\eta)(\delta+\zeta)-\dfrac{\zeta\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}\right]+(\delta+\zeta)(\delta+\eta)\dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}$.

The equilibrium point is locally asymptotically stable when the equation (3.4) does not have positive roots or a set of complex roots having a real portion that is negative.

By Routh Hurwitz criteria (3.4) has negative roots if $L_1>0, L_3>0$, and $L_1L_2-L_3>0$. Thus L_1 is always positive and if $R_0>1$, we have $(\delta+\zeta)(\delta+\eta)-\dfrac{\zeta\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}=\dfrac{\phi_2(\delta+\zeta)(\delta+\eta)I^\star}{(1+\phi_1 S^\star+\phi_2 I^\star)}>0$, $L_1L_2-L_3=[2\delta+\eta+\theta_1][2\delta+\eta+\zeta](\delta+\theta_1)+[3\delta+\eta+\zeta+\theta_1]\,((\delta+\zeta)(\delta+\eta)-\theta_2)+\delta\theta_2+(\delta+\zeta)^2[\delta+\theta_1]>0$.

Where $\theta_1=\dfrac{\theta I^\star(1+\phi_2 I^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}$, and $\theta_2=\dfrac{\zeta\theta S^\star(1+\phi_1 S^\star)}{(1+\phi_1 S^\star+\phi_2 I^\star)^2}$.

Hence, by Routh-Hurwitz criteria the endemic steady state of the scheme (2.3) is locally asymptotically stable when $R_0 > 1$.

4. Stochastic Stability

The effects of external disturbances on the system (2.3) were presented in this part using white noise theory. At endemic equilibrium, these findings are discussed. We consider the linearized model with perturbations x_1 and x_2 to discuss the stochastic system's stability. Mean-square variations were used to assess the scheme's stochastic stability. The delayed stochastic disturbed scheme is provided by

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \delta S - \frac{\theta SI}{1+\phi_1 S+\phi_2 I} + q_1\xi_1(t),\\
\frac{dE}{dt} &= \frac{\theta SI}{1+\phi_1 S+\phi_2 I} - (\delta+\zeta)E + q_2\xi_2(t),\\
\frac{dI}{dt} &= \zeta E - (\delta+\eta)I + q_3\xi_3(t).
\end{aligned} \tag{4.1}$$

Linearising the scheme (4.1) by taking perturbations $S = S_1 + S^\star, E = E_1 + E^\star$ and $I = I_1 + I^\star$ then

$$\begin{aligned}
\frac{dS_1(t)}{dt} &= -\theta S^\star I_1 + q_1\xi_1(t),\\
\frac{dE_1(t)}{dt} &= q_2\xi_2(t),\\
\frac{dI_1}{dt} &= q_3\xi_3(t).
\end{aligned} \tag{4.2}$$

We can obtain the following on using Fourier transforms both sides,
$q_1\xi_1(t) = i\omega S_1(\omega) + \theta S^\star I_1(\omega)$,
$q_2\xi_2(t) = i\omega E_1(\omega)$,
$q_3\xi_3(t) = i\omega I_1(\omega)$.
From above equations, we can write the matrix form as

$$\overline{\xi}(\omega) = L(\omega)V(\omega), \tag{4.3}$$

where $L(\omega) = \begin{pmatrix} l_{11} & l_{12} & l_{13}\\ l_{21} & l_{22} & l_{23}\\ l_{31} & l_{32} & l_{33}\end{pmatrix}$, $\overline{\xi}(\omega) = \begin{pmatrix} q_1\xi_1(t)\\ q_2\xi_2(t)\\ q_3\xi_3(t)\end{pmatrix}$, $V(\omega) = \begin{pmatrix} S_1(\omega)\\ E_1(\omega)\\ I_1(\omega)\end{pmatrix}$, and the elements (row wise) of $L(\omega)$are given by
$l_{11} = i\omega$; $l_{12} = 0$; $l_{13} = \theta S^\star$;
$l_{21} = 0$; $l_{22} = i\omega$; $l_{23} = 0$; $l_{31} = 0$; $l_{32} = 0$; $l_{33} = i\omega$.
Since $|L(\omega)| \neq 0$;we can write (4.3) as

$$V(\omega) = L^{-1}(\omega)\overline{\xi}(\omega) = O(\omega)\overline{\xi}(\omega), \tag{4.4}$$

where $O(\omega) = L^{-1}(\omega) = \dfrac{AdjL(\omega)}{|L(\omega)|}$.

Now from the spectral density, we define $S_g(\omega)d\omega = \lim_{T\to\infty}\dfrac{\overline{|\overline{g}(\omega)|^2}}{T}$,

where $g(t)$ an arbitrary function with mean is zero and $S_g(\omega)$ represents the variance of the elements of $g(t)$ within the interval $[\omega, \omega + d\omega]$.
The inverse transform of $S_g(\omega)$ is the auto covariance function is given by

$$C_g(\tau') = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_g(\omega)e^{i\omega\tau'}\,d\omega,$$

and the variance function $g(t)$ is given by

$$\sigma_g^2 = C_g(0) = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_g\, d\omega.$$

From(4.4), the average value of the population is $\overline{V_i} = \sum_{j=1}^{3} o_{ij}\xi_j(\omega)$ with $i = 1, 2, 3$. Therefore, $S_{v_i} = \sum_{j=1}^{3} q_j |o_{ij}(\omega)|^2$ $(i = 1, , 2, 3)$.

The deviations of $v_i (i = 1, 2, 3)$ as indicated by

$$\sigma_{v_i}^2 = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_{v_i}, d\omega = \frac{1}{2\pi}\sum_{j=1}^{3}\int_{-\infty}^{\infty} q_j |n_{ij}(\omega)|^2 d\omega.$$

As a result, using aforementioned deviations and scheme (4.1), we can determine

$$\sigma_{v_1}^2 = \frac{1}{2\pi}\left[q_1\int_{-\infty}^{\infty}\left|\frac{A_1}{|L(\omega)|}\right|^2 d\omega + q_2\int_{-\infty}^{\infty}\left|\frac{A_2}{|L(\omega)|}\right|^2 d\omega + q_3\int_{-\infty}^{\infty}\left|\frac{A_3}{|L(\omega)|}\right|^2 d\omega\right],$$

$$\sigma_{v_2}^2 = \frac{1}{2\pi}\left[q_1\int_{-\infty}^{\infty}\left|\frac{B_1}{|L(\omega)|}\right|^2 d\omega + q_2\int_{-\infty}^{\infty}\left|\frac{B_2}{|L(\omega)|}\right|^2 d\omega + q_3\int_{-\infty}^{\infty}\left|\frac{B_3}{|L(\omega)|}\right|^2 d\omega\right], \tag{4.5}$$

$$\sigma_{v_3}^2 = \frac{1}{2\pi}\left[q_1\int_{-\infty}^{\infty}\left|\frac{C_1}{|L(\omega)|}\right|^2 d\omega + q_2\int_{-\infty}^{\infty}\left|\frac{C_2}{|L(\omega)|}\right|^2 d\omega + q_3\int_{-\infty}^{\infty}\left|\frac{C_3}{|L(\omega)|}\right|^2 d\omega\right],$$

where $|L(\omega)| = L_1(\omega) + iL_2(\omega)$ and $L_1(\omega) = 0$ and $L_2(\omega) = -\omega^3$, where $|A_1|^2 = |B_2|^2 = |C_3|^2 = \omega^4; |A_2|^2 = |B_1|^2 = |B_3|^2 = |C_1|^2 = |C_2|^2 = 0; |A_3|^2 = (\theta S^\star\omega)^2$.

The deviations of the scheme's (4.1) populations x and y are provided by the results in (4.5). These integrals are extremely tough to come by in general. We can simply explain these results using numerical simulations without sacrificing generality. We may determine variance by using alternative parameter values and a time lag. If the variance is modest, the corresponding population is stable; otherwise, it is unstable.

5. Numerical Simulations

We'll consider throughout this section that every variable is provided as in proper units.

Illustration 1. Given scheme (2.3), which is asymptotically stable for different sigma estimates using parameter values as $\pi = 5.5, \delta = 0.01, \theta = 0.814, \phi_1 = 1.08, \phi_2 = 0.3, \eta = 0.621$. System (2.3) has an infected free steady state for $\zeta = 0.04$, and is asymptotically stable when $R_0 < 1$. If $\zeta = 0.052$, an endemic equilibrium is exists and it is asymptotically stable when $R_0 > 1$.

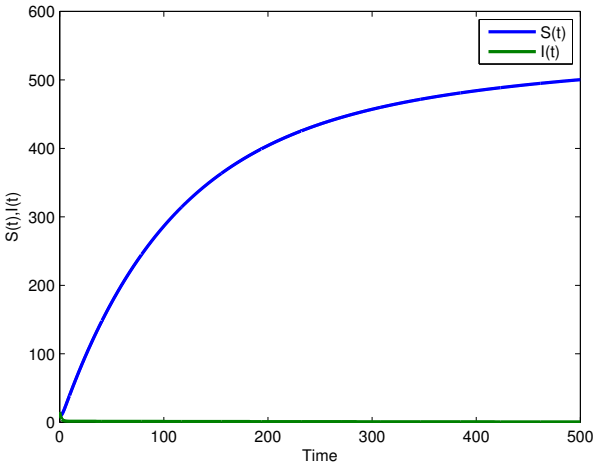

FIGURE 1. System (2.3) predictive direction at disease-free equilibrium when $\zeta = 0.04$.

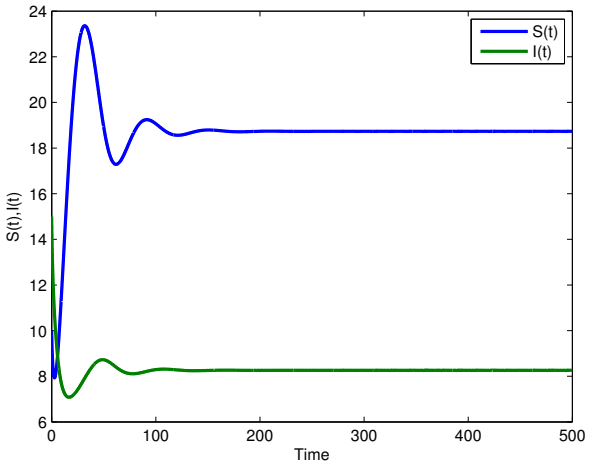

FIGURE 2. Deterministic trajectories of system (2.3) at endemic equilibrium when $\zeta = 0.52$.

Illustration 2. By taking the above parameter values as $\pi = 5.5, \delta = 0.001, \theta = 0.814, \phi_1 = 1.08, \phi_2 = 0.3, \eta = 0.621, \zeta = 0.8$, and for different values of white noise intensities, we got the following graphs. We can see in the next pictures (Figures 3, 4, and 5) that as the white noise intensities increase, the populations converge on the equilibrium point with substantially less oscillations.

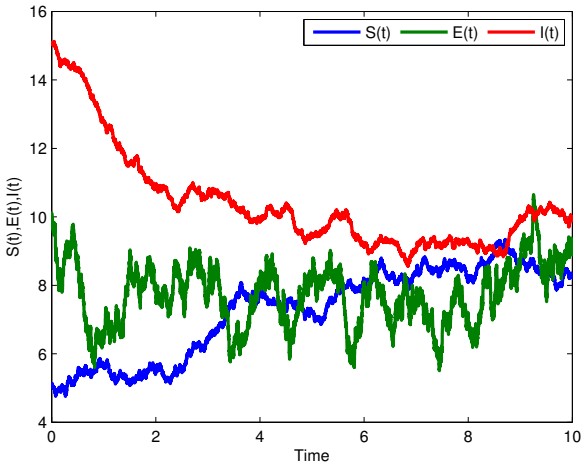

FIGURE 3. Shows deterministic system (2.3)'s time series assessment with random fluctuations around the equilibrium point with $q_1 = 0.03; q_2 = 0.07; q_3 = 0.08$.

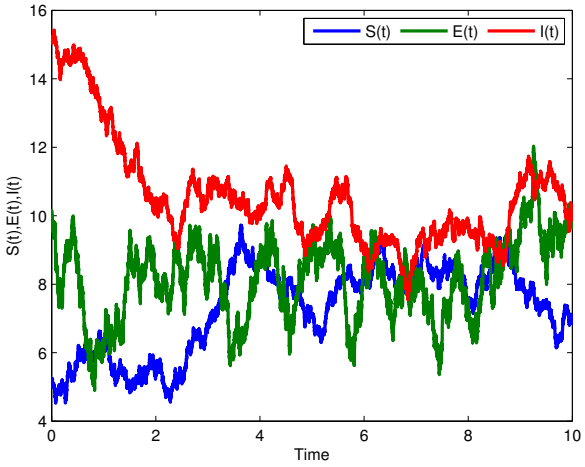

FIGURE 4. Shows deterministic system (2.3)'s time series assessment with random fluctuations around the equilibrium point with $q_1 = 0.06; q_2 = 0.09; q_3 = 0.2$.

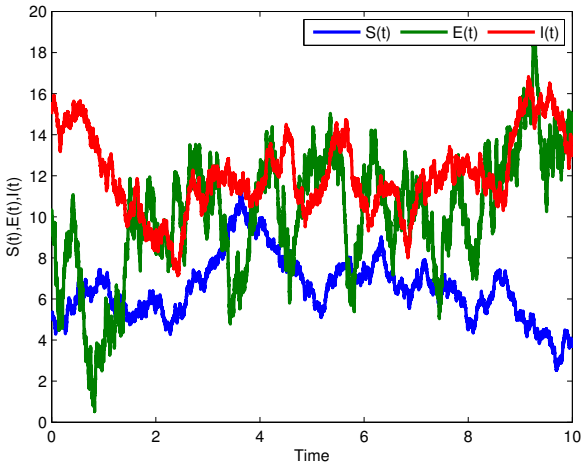

FIGURE 5. Shows deterministic system (2.3)'s time series assessment with random fluctuations around the equilibrium point with $q_1 = 0.09; q_2 = 0.2; q_3 = 0.4$.

6. Conclusion

In this study, mathematical analysis was used to examine the evolution of the SEIR scheme having saturation relative risk with a stochastic effect. We identified two equilibrium points: the infected free equilibrium and the endemic equilibrium, then we found that these two points are stable if R_0 is less than one and more than one respectively. The incidence of transmission and the rate of disease detection are significantly influenced by a number of extraneous factors in the actual world; as a result, this unpredictability must be accounted for in the model. We examined stochastic perturbation of model (2.3), which causes considerable changes in population intensity due to low, medium, and high oscillation variances. As a result, we infer that the incorporation of stochastic perturbation in our considered dynamical system generates a major change in system behavior for a little change in white noise intensities, resulting in enormous environmental fluctuations. We discovered that environmental disruptions had an impact on disease propagation in the population using numerical simulation.

Studying the epidemic's spread after the implementation of control measures like vaccination or isolation could be a potential follow-up to this paper. A different option is to use the BSDE approach [1] to study the spread of infections for non-homogeneous Markov-modulated models. The BSDE approach yields efficient computation of system performance and aids in lowering the computational costs resulting from the large size of the matrices involved in models based on Markovian arrival process. Additionally, by adopting fractional order, we can extend these models [9, 21, 23].

Conflict of Interest: The authors declare no conflict of interest.

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References

[1] JR. Artalejo and A. Gomez-Corral, *A state-dependent Markov-modulated mechanism for generating events and stochastic models*, Mathematical Methods in the Applied Sciences., *33* (2010), 1342-1349.

[2] A. Bahar and X. Mao, *Stochastic delay Lotka–Volterra model*, J. Math. Anal. Appl., *292* (2004), 364–380.

[3] X. Han, F. Li, and X. Meng, *Dynamics analysis of a nonlinear stochastic SEIR epidemic system with varying population size*, Entropy., *20* (2018), 376-20.

[4] S. Hariprasad, M. A. S. Srinivas, and N. Phanikumar, *Three Species Epidemiological Model with Holling Type Functional Responses.International journal of mathematical models and methods in applied sciences*, Int. J. Math. Models and Methods in App. Sci., *14* (2020), 62–75.

[5] S. Hariprasad, M. A. S. Srinivas, and N. Phanikumar,*Mathematical Study of Plant-Herbivore-Carnivore System with Holling Type-II Functional Responses*, J. Adv. Research in Dynamical and Control Systems., *11* (2019).

[6] C. Ji, D. Jiang, and X. Li, *Qualitative analysis of a stochastic ratio-dependent predator–prey system*, J. Comput. Appl. Math., *235* (2011), 1326–1341.

[7] C. Ji, D. Jiang, and N. Jiang, *Analysis of a predator–prey model with modified Leslie-Gower and Holling-type II schemes with stochasticperturbation*, J. Math. Anal. Appl., *359* (2009), 482–498.

[8] D. Jiang, N. Shi, and X. Li, *Global stability and stochastic permanence of a non-autonomous logistic equation with random perturbation*, J. Math. Anal. Appl., *340* (2008), 588–597.

[9] K. Jothimani, N. Valliammal, and S. Alsaeed, *Controllability Results of Hilfer Fractional Derivative Through Integral Contractors*, Qual. Theory Dyn. Syst., *22* (2023), 137.

[10] W. O. Kermack and A. G. McKendrick, *A contribution to the mathematical theory of epidemics," Proceedings of the Royal Society*, A: Mathematical, Physical and Engineering Sciences, *115* (1927), 700–721.

[11] W. O. Kermack and A. G. McKendrick, *Contributions to the mathematical theory of epidemics: II. The problem of endemicity," Proceedings of the Royal Society*, A: Mathematical, Physical and Engineering Sciences, *138* (1932), 55–83.

[12] W. O. Kermack and A. G. McKendrick, *Contributions to the mathematical theory of epidemics. III. Further studies of the problem of endemicity*, Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences, *141* (1933), 94–122.

[13] A. Lahrouz and A. Settati, *Necessary and sufficient condition for extinction and persistence of SIRS system with random perturbation*, Appl. Math. Comput., *233* (2014), 10–19.

[14] Q. Liu, D. Jiang, T. Hayat, and A. Alsaedi, *Dynamics of a stochastic tuberculosis model with antibiotic resistance*, Chaos Solitons Fractals., *109* (2018), 223–230.

[15] Y. Li and H. Gao, *Existence, uniqueness and global asymptotic stability of positive solutions of a predator–prey system with Holling IIfunctional response with random perturbation*, Nonlinear Anal., *68* (2008), 1694–1705.

[16] M. Liu, C. Bai, and K. Wang, *Asymptotic stability of a two-group stochastic SEIR model with infinite delays*,Communications in Nonlinear Science and Numerical Simulation., *19* (2014), 3444–3453.

[17] X. Mao, S. Sabanis, and E. Renshaw, *Asymptotic behaviour of the stochastic Lotka–Volterra model*, J. Math. Anal. Appl., *287* (2003), 141–156.

[18] X. Mao, C.Yuan, and J. Zou, *Stochastic differential delay equations of population dynamics*, J. Math. Anal. Appl., *304* (2005), 296–320.

[19] J. Miekisz, J. Poleszczuk, M. Bodnar, and U. Forys, *Stochastic models of gene expression with delayed degradation*, Bulletin of Mathematical Biology., *73* (2011), 2231–2247.

[20] J. Ong, M. Chen, A. Cook, H. Lee, V. Lee, R. Lin , P.Tambyah, and L. Goh, *Real-time epidemic monitoring and forecasting of H1N1-2009 using influenza-like illness from general practice and family doctor clinics in Singapore*, PLoS ONE 2010 *4* (2010), e10036.

[21] C. Ravichandran, K. Logeswari, and F.Jarad, *New results on existence in the framework of Atangana–Baleanu derivative for fractional integrodifferential equations*, Chaos, Solitons.Fractals., *125* (2019), 194–200.

[22] Li. Shangzhi and Guo. Shangjiang, *Asymptotic behaviour of the stochastic Lotka–Volterra model*, Mathematics and Computers in Simulation., *187* (2021), 308–336.

[23] K. Sooppy Nisar, K. Munusamy, and C. Ravichandran, *Results on existence of solutions in nonlocal partial functional integrodifferential equations with finite delay in non-dense domain*, Alexandria Engineering Journal., *73* (2023), 377–384.
[24] S. Takeuchi and Y. Kuroda, *Predicting spread of new pandemic swine-origin influenza A (H1N1) in local mid-size city: evaluation of hospital bed shortage and effectiveness of vaccination*, Nippon Eiseigaku Zasshi., *65* (2010), 48–52.
[25] F. Wei and R. Xue, *Stability and extinction of SEIR epidemic models with generalized nonlinear incidence*, Math. Comput. Simul., *170* (2020), 1–15.
[26] P. J. Witbooi, *Stability of an SEIR epidemic model with independent stochastic perturbations*, Physica A: Statistical Mechanics and its Applications., *392* (2013), 4928–4936.
[27] Q. Yang and X. Mao, *Extinction and recurrence of multi-group SEIR epidemic models with stochastic perturbations*, Nonlinear Anal., Real World Appl., *14* (2013), 1434–1456.
[28] X. Zhang and K. Wang, *Stochastic SEIR model with jumps*, Applied Mathematics and Computation., *239* (2014), 133–143.
[29] J. Zhang, Z. Jin, GQ. Sun, T. Zhou, and S. Ruan, *Analysis of rabies in China: transmission dynamics and control*, PLoS ONE., *7* (2011), e20891.
[30] X. Zhao and Z. Zeng, *Stationary distribution and extinction of a stochastic ratio dependent predator prey system withstage structure for the predator*, Physica A., *14* (2019), 1434–1456.