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DYNAMIC OF AN SIR EPIDEMIC MODEL WITH A SATURATED TREATMENT RATE UNDER STOCHASTIC INFLUENCE

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ABSTACT:
Adding a saturating treatment logistic growth rate under both deterministic and stochastic frameworks is suggested as an addition to the conventional SIR (Susceptible-Infected-Recovered) epidemic model in this research. Treatment efficacy is constrained in real-world situations, especially when the number of infected persons is considerable, contrary to the idealized linear treatment rate assumed by the classic SIR model. We propose a continuously differentiable treatment function to account for this, which characterizes the sluggish reaction of medical therapy when the healthcare system is overloaded. In this model, the saturation effect is represented by a function that shows how the treatment rate grows at first, but then approaches a maximum limit when resources are limited. To account for the dynamic between susceptible and infected people, the model uses a bilinear incidence rate. To mimic the declining rewards of medical treatments as the illness spreads, the availability of therapy modifies this rate, reaching saturation at higher infection levels. Derivation of the fundamental reproduction number (R_0) provides a crucial starting point for comprehending the epidemic's spread. The likelihood of the illness spreading increases when $R_0 > 1$, and it decreases with decreasing values, suggesting that the sickness will ultimately die out. First, for the deterministic model, we study the local and global stability of the disease-free and endemic equilibrium points. By examining the effects of changing the treatment function and other model parameters on epidemic control, the stability analysis sheds light on this topic.
We include the inherent uncertainties and random fluctuations in real-world epidemic dynamics into the stochastic model. Variations in treatment efficacy, healthcare capacity, and external variables like environmental changes or governmental initiatives are all examples of what might cause these oscillations. We demonstrate that, subject to certain constraints relating to the strength of the stochastic perturbations, the endemic equilibrium is stable on a global scale. By shedding light on how the system acts when faced with ambiguity, our study demonstrates that the epidemic might stabilise into an endemic state, even in the face of random oscillations. At last, numerical examples are given to back up the analytical results. By simulating the effects of treatment saturation and stochastic disease dynamics on epidemic development, the simulations reveal the model's practical consequences. More accurate forecasts of epidemic outcomes, especially in resource-limited situations, are produced by combining treatment saturation with stochastic impacts, as shown by the numerical findings. In conclusion, our research offers a more refined paradigm for simulating epidemic breakouts in the face of constrained healthcare resources and random disruptions. When it comes to dealing with large-scale epidemics in real-world situations, where healthcare facilities are often overloaded, the results have significant implications for public health strategy.

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KEYWORDS: SIR epidemic model, Logistic growth, Stochastic stability, Lyapunov function, saturated treatment, reproduction number, global stability.

INTRODUCTION: To comprehend the dynamics of infectious illnesses and to create efficient methods for controlling them, the study of epidemic models is essential. Disease transmission in populations may be anticipated with the use of mathematical models, such as the SIR (Susceptible-Infected-Recovered) model. But, the limits of real-world healthcare systems are rarely reflected in the traditional SIR model, which often assumes constant treatment rates. Medical resources, healthcare infrastructure, and response times all have a role in limiting therapy efficacy. In order to better depict the dynamics of disease propagation, particularly in cases of resource overload, it is crucial to include saturation effects in treatment rates. In most cases, the number of sick people is used to simulate the treatment rate, with the idea being that the rate of recovery grows as the number of infected people does, but that there is a maximum threshold that healthcare systems can handle. The epidemic model becomes nonlinear due to this saturation treatment rate; this represents the reality that the healthcare system is overwhelmed by the increasing number of sick people, and treatment efficacy decreases as a result. More accurate epidemic dynamics modelling is possible with the addition of a saturation treatment rate, which is particularly useful in settings with limited resources. The deterministic SIR model can provide light on how an epidemic might unfold in a perfect environment, but it can't handle the uncertainties and random fluctuations that are inevitable in the actual world. Conditions in the environment, variations in healthcare accessibility, and changes in human behaviour are examples of external variables that might impact the onset of epidemics. It is required to add stochastic impacts into the model in order to account for these uncertainties. To better understand how epidemics really play out in the real world, stochastic models include random perturbations to represent the unpredictable dynamics of epidemics. Adding stochastic effects and a saturation treatment rate to the standard SIR epidemic model is what we suggest in this study. To account for the fact that the saturation of the treatment rate is proportional to the number of infected persons, the model is constructed by include a logistic function. As the number of infected individuals grows, this function shows how healthcare services are limited and how treatment effectiveness diminishes. To account for the unpredictability of the actual world, we include random fluctuations in treatment efficacy, environmental variables, and other stochastic components as white noise perturbations in the model. Adding the saturation treatment rate makes the model more grounded in reality by considering the fact that healthcare systems have a maximum capacity to serve patients simultaneously. To comprehend how random changes in treatment or environmental factors might impact the development of the illness, it is essential to account for the unpredictable character of epidemic management, and the stochastic component does just that. This research aims to determine the model's fundamental reproduction number (R_0), examine the stability of the disease-free and endemic equilibrium points, and evaluate the global stability of the system when random perturbations are applied. In epidemiology, the basic reproduction number is an important threshold since it indicates the spread or extinction of an epidemic. The propensity for the illness to spread is indicated by a $R_0 > 1$, however its ultimate eradication is predicted when $R_0 < 1$. Both the deterministic and stochastic versions of the model's stability of the equilibrium points are examined. We start with the deterministic model, which describes the system's behaviour in an idealized setting, and then we go on to the stochastic

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model, which incorporates random disturbances to represent the unpredictability in the actual world. We verify our analytical results with numerical simulations and investigate the consequences of adding stochastic factors and treatment saturation to epidemic models. Public health planning and epidemic management may benefit greatly from this study's findings. The evolution of epidemics in resource-constrained situations may be better understood by taking treatment restrictions and stochastic impacts into account. Policies and tactics for epidemic control may be informed by this knowledge, especially in settings when healthcare resources are limited or unpredictable. In light of our results, it is clear that dynamic models are required to capture the realities of treatment capacity saturation and the intrinsic unpredictability of epidemic dynamics in the actual world. In assumption, this work offers a thorough method for simulating the dynamics of epidemic outbreaks in settings with limited treatment capacity and substantial influence of random fluctuations. The suggested model sheds light on the ways in which treatment saturation and random factors impact the transmission of infectious illnesses, and it also gives a more practical framework for comprehending the development of epidemics. Future epidemics may be better controlled and managed with the use of public health initiatives that are based on the findings of this research.

1. MODEL FRAME WORK: In this section, we formulate an epidemic model for the spread of a general infectious disease. We split the total population N(t), into three distinct subclasses which are susceptible S(t), infectious I(t), and recovered R(t). The model can be represented by the following system of differential equations.

$$\begin{aligned}\frac{dS}{dt} &= rS\left(1-\frac{S}{C}\right)-\frac{\theta SI}{1+\delta I}-\varepsilon S\\ \frac{dI}{dt} &= \frac{\theta SI}{1+\delta I}-(\varepsilon+\psi+\lambda)I-\frac{\zeta I}{1+\chi I}\\ \frac{dI}{dt} &= \psi I+\frac{\zeta I}{1+\chi I}-\varepsilon R\end{aligned} \qquad (1)$$

With the initial conditions

$$S(0)\geq 0, I(0)\geq 0, R(0)\geq 0.$$

Here r is the intrinsic growth rate of susceptible population, c denotes the carrying capacity of the country ignoring the infection and recovered persons. θ is the transmission rate, δ is the saturation factor, ϵ is the natural death rate, λ is the disease – induced death rate, ψ represents the recovered rate, ζ is the Treatment rate, $\boldsymbol{\chi}$ is the Saturation in treatment. The saturated incidence rate $\frac{\theta SI}{1+\delta I}$ was employed by caspase. where θSI measures the infection force of the disease and $\frac{I}{1+\delta I}$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or as a result of the crowding effect of the infective individual. It is assumed in this paper that ζ is negative constant and other parameters are positive constant.

2. EQUILIBRIUM POINTS AND THEIR STABILITY

In this section we show the stability analysis. The disease – free equilibrium (DFE) point is

$$E_0 = \left(C\left(1-\frac{\varepsilon}{r}\right),\ 0\right)$$

To find the endemic equilibrium (EE) point $E^* = (S^*, I^*)$ we set the right-hand side of the system (1) equal to zero to get

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$S^* = \frac{(1+\delta I^*)\left[(\varepsilon+\psi+\lambda)+\frac{\zeta}{1+\chi I^*}\right]}{\theta}$, where I^* is the positive solution of

$A_1 I*^3 + A_2 I*^2 + A_3 I* + A_4 = 0$

Here $A_1 = -r\,C\,\delta^2(\varepsilon+\psi+\lambda+\zeta)$,

$A_2 = r\,\delta(\varepsilon+\psi+\lambda+\zeta) - \delta\varepsilon\theta - r\,C\,\delta(\varepsilon+\psi+\lambda+\zeta) - \theta$

$A_3 = r(\varepsilon+\psi+\lambda+\zeta) - \varepsilon\theta - r\,C(\varepsilon+\psi+\lambda+\zeta)$,

$A_4 = r(\varepsilon+\psi+\lambda) - \varepsilon\theta - r\,C(\varepsilon+\psi+\lambda)$,

3. BASIC REPRODUCTION NUMBER

To find the reproduction number using next generation method.

Let system (1) can be written as

New infection term $F = \frac{\theta\,\delta\,I}{1+\delta\,I}$

Transform $V = (\varepsilon+\psi+\lambda)I + \frac{\zeta\,I}{1+\chi\,I}$

The Jacobian matrix of F and V at the DFE $E_0 = \left(C\left(1-\frac{\varepsilon}{r}\right),\ 0\right)$ are respectively,

$F_{DFE} = \theta C\left(1-\frac{\varepsilon}{r}\right)$, $V_{DFE} = (\varepsilon+\psi+\lambda+\zeta)$

The Next generation matrix is $K = \frac{F_{DFE}}{V_{DFE}} = \frac{\theta C\left(1-\frac{\varepsilon}{r}\right)}{(\varepsilon+\psi+\lambda+\zeta)}$ which is the spectral radius and it is equal to model's basic reproduction number is

$$R_0 = \frac{\theta C\left(1-\frac{\varepsilon}{r}\right)}{(\varepsilon+\psi+\lambda+\zeta)} \qquad (2)$$

Theorem1. The system (1) has

(I) Disease – free equilibrium, if $R_0 \le 1$ and

(II) If $R_0 > 1$, then there is a unique positive equilibrium called the endemic equilibrium.

New we shall study the local stability of each equilibrium.

The Jacobian matrix of (1) at E_0 is

$$J_{E_0} = \begin{bmatrix} \frac{-r\,S^*}{C} & -\theta\,S^* \\ 0 & \theta\,S^* - (\varepsilon+\psi+\lambda+\zeta) \end{bmatrix}$$

The characteristic equation of above matrix is

$\left(\lambda+\frac{r\,S^*}{C}\right)(\lambda - \theta\,S^* + (\varepsilon+\psi+\lambda+\zeta) = 0$

The roots are $\lambda_1 = -(r-\varepsilon)$, $\lambda_2 = \theta S^* - (\varepsilon+\psi+\lambda+\zeta)$ then the system is stable if $\theta S^* - (\varepsilon+\psi+\lambda+\zeta) < 0$

therefore $\frac{\theta C\left(1-\frac{\varepsilon}{r}\right)}{(\varepsilon+\psi+\lambda+\zeta)} < 1$

clearly, if $R_0 < 0$, then the disease- free equilibrium is a locally asymptotically stable.

The Jacobian matrix of (1) at $E^* = (S^*, I^*)$ is

$$J_{E^*} = \begin{bmatrix} \frac{-r\,S^*}{C} & \frac{-\theta\,S^*}{(1+\delta\,I^*)^2} \\ \frac{\theta\,I^*}{(1+\delta\,I^*)} & \frac{\theta\,S^*}{(1+\delta\,I^*)^2} - (\varepsilon+\psi+\lambda) - \frac{\zeta}{(1+\chi\,I^*)^2} \end{bmatrix}$$

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that can be rewritten as

$$J_{E^*} = \begin{bmatrix} \frac{-r\,S^*}{C} & \frac{-\theta\,S^*}{(1+\delta\,I^*)^2} \\ \frac{\theta\,I^*}{(1+\delta\,I^*)} & \frac{\zeta\,I^*}{(1+\chi\,I^*)^2} - \frac{\theta\,\delta\,I^*S^*}{(1+\delta\,I^*)^2} \end{bmatrix}$$

the characteristic equation of above matrix is

$$\lambda^2 + P_1\lambda + P_2 = 0 \qquad (3)$$

where

$$P_1 = (\varepsilon - r) + \frac{2r\,S^*}{C} + \frac{\theta\,S^*}{1+\delta\,I^*} - \frac{\zeta\,I^*}{(1+\chi\,I^*)^2} + \frac{\theta\,\delta\,I^*S^*}{(1+\delta\,I^*)^2}$$

$$P_2 = \left(\frac{2r\,S^*}{C} + \frac{\theta\,S^*}{1+\delta\,I^*}\,\varepsilon - r\right)\left(-\frac{\zeta\,I^*}{(1+\chi\,I^*)^2} + \frac{\theta\,\delta\,I^*S^*}{(1+\delta\,I^*)^2}\right) + \frac{\theta^2 I^*S^*}{(1+\delta\,I^*)^3}$$

Since all coefficients P_1, P_2 will be positive only if $R_0 > 1$, therefore by Routh- Hurwitz criteria all roots of the equation (3) have negative real part. Hence the endemic equilibrium point $E^* = (S^*, I^*)$ is a locally asymptotically stable.

4. GLOBAL STABILITY

To show that the proposed system is globally asymptotically stable, we use the Lyapunov function theory for the disease – free equilibrium. we present the global stability of the disease-free equilibrium.

Theorem 2. If $R_0 \leq 1$, then the disease- free equilibrium E_0 of the system is globally asymptotically stable.

Proof: To establish the global stability of the disease- free equilibrium E_0, we construct the following Lyapunov function.

$$L(S, I) = I(t)$$

calculating the time derivative of V along the solution of the proposed system, we obtain

$$\frac{dL}{dt} = \frac{dI}{dt}$$

$$= \frac{\theta SI}{1+\delta I} - (\varepsilon + \psi + \lambda)I - \frac{\zeta\,I}{1+\chi\,I}$$

$$= (\theta C\left(1 - \frac{\varepsilon}{r}\right) - (\varepsilon + \psi + \lambda))I$$

$$= \frac{\theta C\left(1-\frac{\varepsilon}{r}\right)}{(\varepsilon+\psi+\lambda+\zeta)} - 1$$

$$= (R_0 - 1)I \leq 0$$

therefore $R_0 < 0$

If $R_0 < 0$ *then* $L' = 0$, I = 0

Hence by LaSalle's invariance principle, the disease- free equilibrium E_0, is globally asymptotically stable.

5. STOCHASTIC STABITY OF THE MODEL AT POSITIVE EQUILIBRIUM

In this section, we investigate the dynamical behavior of the system (1). as follows

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$$\frac{dS}{dt} = rS\left(1-\frac{S}{C}\right) - \frac{\theta SI}{1+\delta I} - \varepsilon S + \sigma_1(S-S^*)d\zeta_1{}^1$$
$$\frac{dI}{dt} = \frac{\theta SI}{1+\delta I} - (\varepsilon + \psi + \lambda)I - \frac{\zeta I}{1+\chi I} + \sigma_1(I-I^*)d\zeta_1{}^2 \quad (4)$$

where σ_1, σ_2 are real constants and known as the Intensities of environmental fluctuations and $\zeta_t{}^i = \zeta_i(t)$, i = 1,2 are independent from each other standard Wiener processes.

The stochastic differential system (4) can be centered at its positive equilibrium E^* with the change of variables

$u_1 = s - s^*, u_2 = I - I^*$

the linearized stochastic differential equations around E^* take the form

$$du(t) = f\big(u(t)\big)dt + g\big(u(t)\big)d\zeta(t) \quad (5)$$

where $u(t) = (u_1(t), u_2(t))^T$

$$f\big(u(t)\big) = \begin{bmatrix} \frac{-r\,S^*}{C} & \frac{-\theta\,S^*}{(1+\delta\,I^*)^2} \\ \frac{\theta\,I^*}{(1+\delta\,I^*)} & \frac{\theta\,S^*}{(1+\delta\,I^*)^2} - (\varepsilon + \psi + \lambda) - \frac{\zeta}{(1+\chi\ I^*)^2} \end{bmatrix} \quad (6)$$

and $g(u) = \begin{bmatrix} \sigma_1 u_1 & 0 \\ 0 & \sigma_2 u_2 \end{bmatrix}$

Let $W(t,u)$ defined on $[0,+\infty) \times R^2$) is a continuously differentiable function with respect to t and twice with respect to u.

We define the differential operator L for a function $W(t,u)$ by

$$LW(t) = \frac{\partial W(t,u)}{\partial t} + f^T(u)\frac{\partial W(t,u)}{\partial u} + \frac{1}{2}Tr\left[g^T(u)\frac{\partial^2 W(t,u)}{\partial u^2}g(u)\right] \quad (7)$$

$\frac{\partial W}{\partial u} = col\left(\frac{\partial W}{\partial u_1}, \frac{\partial W}{\partial u_2}, \frac{\partial W}{\partial u_3}\right)$

$\frac{\partial^2 W(t,u)}{\partial u^2} = \left(\frac{\partial^2 W}{\partial u_j \partial u_i}\right)$ i, j = 1,2 and T means transposition.

It is easy to see that the stability of the endemic point of model (4) is equivalent to the stability of zero solution of (5).

With reference to the book by Afanas' ev et al. [9], the following theorem holds.

Theorem 3. Suppose there exists a function $W(t,u) \in C^{1,2}([0,+\infty) \times R^2, R^+)$ satisfying the following inequalities.

$K_1|u|^p \le W(t,u) \le k_2|u|^p$

$LW(t,u) \le -k_3|u|^p$

where K_1 , K_2 , K_3 and P are positive constant.

Then the trivial solution of (5) is exponentially P- stable for t ≥ 0.

Theorem 4. Assume that $\sigma_1{}^2 \le 2\left(\frac{rS^*}{C}\right)$, $\sigma_2{}^2 \le 2\left(-\frac{\theta\,S^*}{(1+\delta\,I^*)^2} + (\varepsilon + \psi + \lambda) + \frac{\zeta}{(1+\chi\ I^*)^2}\right)$ hold. then, the trivial solution of (5) is asymptotically mean square stable.

proof: Let as consider the Lyapunov function

$$W(u) = \frac{1}{2}[W_1 u_1{}^2 + W_2 u_2{}^2] \quad (8)$$

where W_1 , W_2 are nonnegative constants to be chosen in the following. It is easy to check that inequalities (8) holds with p =2.

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$$LW(u) = W_1\left[\left(\frac{-rS*}{C}\right)u_1 - \frac{\theta S*}{(1+\delta I^*)^2}u_2\right]u_1 + W_2\left[\frac{\theta I^*}{(1+\delta I^*)}u_1 + \left(\frac{\theta S^*}{(1+\delta I^*)^2} - (\varepsilon + \psi + \lambda) - \frac{\zeta}{(1+\chi I^*)^2}\right)u_2\right]u_2 + \frac{1}{2}Tr\left[g^T(u)\frac{\partial^2 W(t,u)}{\partial u^2}g(u)\right] \quad (9)$$

with $\frac{1}{2}Tr\left[g^T(u)\frac{\partial^2 W(t,u)}{\partial u^2}g(u)\right] = \frac{1}{2}[W_1\sigma_1{}^2u_1{}^2 + W_2\sigma_2{}^2u_2{}^2]$

If we choose $\frac{\theta S^*}{(1+\delta I^*)^2}W_1 = \frac{\theta I^*}{(1+\delta I^*)}W_2$ then

$$LW(u) = -\left[\frac{rS^*}{C} - \frac{1}{2}\sigma_1{}^2\right]w_1u_1{}^2 - \left[\frac{\zeta}{(1+\chi I^*)^2} + (\varepsilon + \psi + \lambda) - \frac{\theta S^*}{(1+\delta I^*)^2} - \frac{1}{2}\sigma_2{}^2\right]w_2u_2{}^2$$

6. NUMERICAL SIMULATION

In this section, we present the numerical simulations to illustrate our analytical results. we consider the parameter values

r = 0.8, C = 100, $\theta = 0.8, \delta = 0.1, \varepsilon = 0.05, \psi = 0.2, \lambda = 0.1, \xi = 0.3, \chi = 0.1$

In this case R_0 = 115.384615>1, $\sigma_1{}^2 = 0.0001$<1, $\sigma_2{}^2 = 0.0016$<1

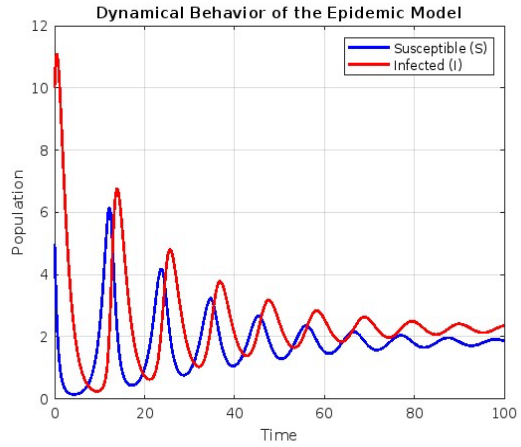

Fig 1

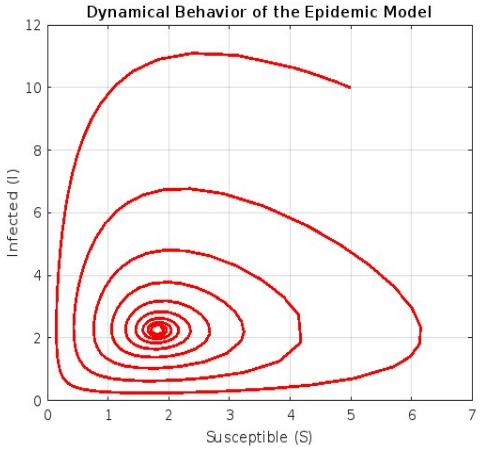

Fig 2

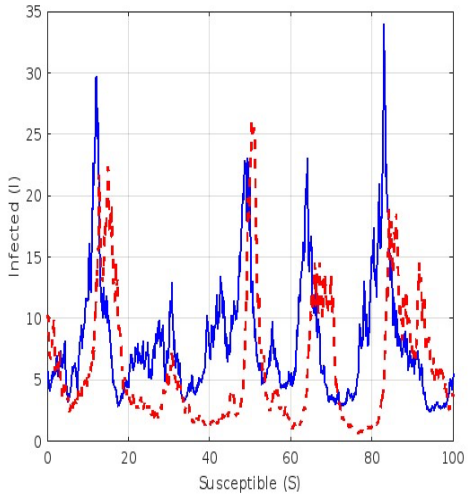

Fig 3

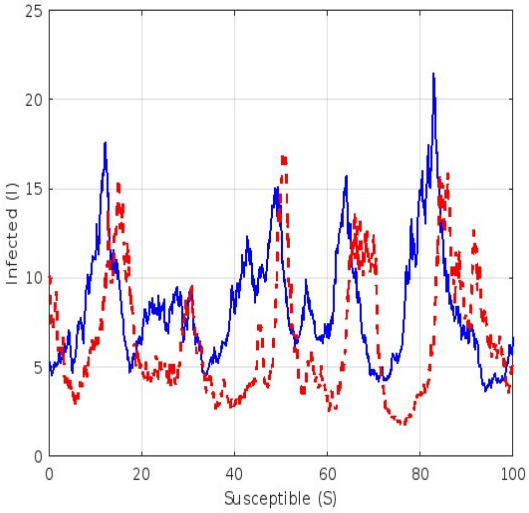

Fig 4

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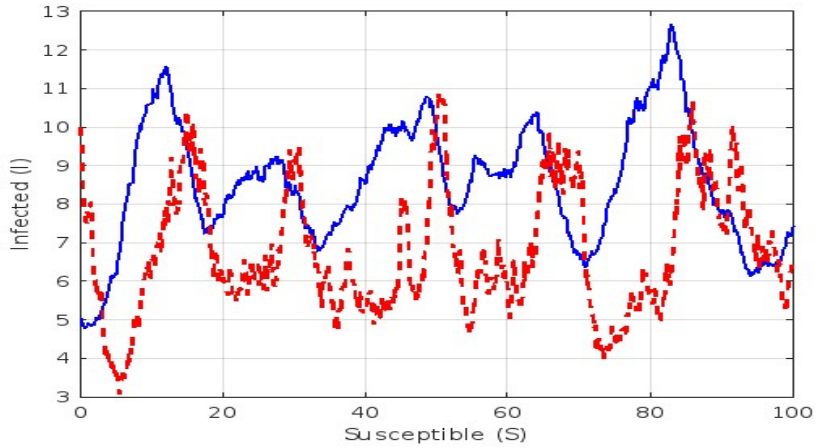

Fig 5

Fig1 and Fig2 represent deterministic and phase portrait of the system (1) for the above parameter values.

Fig. (3-5) represent stochastic trajectories of the system (4).

In Fig 3 the solutions of model (1) will be oscillating slightly around the endemic point E^* of the model (1).

7.Conclusion

Here, we provide a SIR epidemic model with logistic growth and saturation treatment rate integration. A deterministic and a stochastic analysis of the model is carried out, taking into consideration white noise disturbances near the endemic equilibrium state. The fundamental reproduction number (R_0), which changes with the parameter values, forms the basis of the study. The illness will finally disappear when the disease-free equilibrium is determined to be locally stable, which occurs when $R_0 < 1$. On the other hand, there is an endemic equilibrium that is locally stable when $R_0 > 1$, which means that the illness may survive in the population. Even when random disturbances are present, our model shows that the stochastic version is globally asymptotically stable, which means that the system will achieve a stable state given suitable circumstances. Our numerical simulations back up the analytical conclusions, therefore our theoretical findings are validated. The results of these simulations demonstrate that the mathematical model is valid and reliable; they show that the model can properly depict the epidemic's dynamics under both random and predictable conditions.

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