

A Mathematical Analysis of Dynamical Behaviour of Epidemiological Models with Nonlinear Incidence Rates

Manisha Halder^{1*}, Dr. D.S. Sharma²

¹Research Scholar, Department of Mathematics, St. John's College, Agra, U.P. (India)

²Professor, Department of Mathematics, St. John's College, Agra, U.P. (India)

*Email: manishahalder05@gmail.com

Abstract:

An updated model of an epidemic is discussed, one in which incidence has plateaued but treatment has not been fully implemented. All equilibrium points are checked for existence. In this research, we examine how shifts from the SIR (susceptible-infectious-resistant) to the SIS (susceptible-infectious-susceptible) paradigm manifest in epidemiological models. These models hypothesize that the irresistible power is a nonlinear capability of the populace thickness of contaminated individuals. At last, this model might be utilized to research the elements of infection spread, provided that the two phenomena follow consistent patterns.

Keywords: Dynamical behaviour, Epidemiological models

1. Introduction:

Tudor developed the SIRI epidemiological model, which contains of a scheme with three sections: susceptible, communicable, and detached people, denoted by S, I, and R, respectively. According to this hypothesis, susceptible individuals contract an infection, are temporarily protected against it, and later redevelop the infection. The SIRI paradigm is applicable for various diseases, including herpes, bovine and human tuberculosis, and herpes, where recovered patients may return to the transferable class as a result of buried infection reactivation or inadequate therapy. Peirce et al. (2008) provided a review of the many mathematical and computational modelling techniques that have been used to investigate angiogenesis, and they summed up a variety of published models developed to address various issues about this phenomenon. Some of the difficulties of developing and deploying mathematical and computational models of angiogenesis were also explored.

To investigate the impact of Adriamycin dosage on the development of avascular tumours, Dixit et al. (2012) created a modelling system. In this numerical analysis, we focused on the impact of a dimensionless parameter that captures the drug's depth of penetration on the survival of tumour cells. They employed tumour cell density in relation to Adriamycin's action to characterise the migration of tumour cells, having previously established a tumour cell energy model. 2020 Vaghi, C., et al. In spite of their widespread usage

over the last several decades, most mathematical models of tumour development kinetics have been fitted to average or single cases. Here we use a population-based method that takes into account natural variation between individual animals to evaluate three traditional models: the exponential, the logistic, and the Gompertz. The experimental data did not match the exponential or logistic models, however the Gompertz model was quite descriptive. To comprehend the theory of illness transmissions, some scholars have researched this kind of SIRS wide-ranging model. By an straightforward investigation of Liénard's calculation and Lyapunov's through technique, Moreira and Wang analyses a SIRS model with a generic saturation frequency rate $\phi(S)$ and created satisfactory standards for the absolute asymptotic strength of the sickness free likewise endemic balances of the run of the mill. By studying how the Bari cholera outbreak spread, Capasso announced a frequency rate $KI/(1+\alpha I)$ in 1978. In their study of a rampant model through a detailed nonlinear episode rate $KI^2 S/\alpha I^2$, Ruan and Wang also offered a thorough qualitative examination of the copy's Hopf and Bogdanov-Takens bifurcations. In addition, a person with many infectious diseases must go through a period of latent infection before they can spread the disease to others after becoming sick. This mathematically reflects the infection's delay effect. Investigating the nonlinear rate rates and the concealed passé is therefore intriguing. Investigating a SIRS epidemic typical by means of a latent period, nonlinear frequency rate, and transitory immunity is our goal. The construction of this paper is as per the following. In Partion, we depict the SIRS classical and talk about whether or not the model's equilibrium can be achieved with the essential multiplication number R_0 . The edge elements on unflinching quality focused on the model denied of the torpid period ($\tau = 0$) are obtained in Section. Additionally, for the model with $\tau > 0$, we investigate stability and Hopf bifurcation. These results demonstrate how the idle period and nonlinear frequency rate impact the dynamical ways of behaving of the SIRS classical. We provide some inferences, talk about the influence, and also provide some illustrated instances and simulations.

2 Model description

This research follows a long-standing methodological convention in epidemiology to analyse the total frequency rate.

$$g(I)S = \frac{KIS}{1+\alpha I^h}$$

where KI embodies the taint energy of the ailment and $\frac{1}{1+\alpha I^h}$ epitomizes the hang-up effect after the social modification of the vulnerable folks once their figure increases or after the flocking effect of the catching folks. Still, shoulder that the hidden old-fashioned is a continuous τ , Thus, a susceptible individual becomes contagious if he has already spread the illness via his communication. Thus, nonlinear incidence purpose as shadows may be coupled with delay jerry:

$$g(I(t))S = \frac{KI(t-\tau)S}{1+(t-\tau)\alpha I^h}$$

Before we recommend the subsequent SIRI perfect through over-all occurrence rate and hidden dated:

$$\begin{cases} \frac{dS(t)}{dt} = b - dS(t) - \frac{Kl(t-\tau)S}{1+(t-\tau)\alpha I^h} \\ \frac{dl(t)}{dt} = \frac{Kl(t-\tau)S}{1+(t-\tau)\alpha I^h} - (d + \mu)l(t) + \gamma R(t) \\ \frac{dR(t)}{dt} = \mu l(t) - (d + \gamma)R(t) \end{cases} \quad (1)$$

someplace b be the organic part of the populace, d be the commonplace passing pace of the populace, k be the proportionality consistent, μ be the negligible portion of tainted individuals who become briefly safe, is the fraction of healthy people who regress to the infectious session, is the soaked limit, and, $\gamma, b, d, \alpha, \mu, k, h$ are all optimistic parameters. Converting Eq. (1) from three dimensions to a two-dimensional coordinate system is a first step towards this goal. After summing the three results in Eq. (1), we may represent

$$N(t) = S(t) + R(t) + I(t) \text{ we require}$$

$$\frac{dN(t)}{dt} = b - dN(t)$$

Clearly,

$$N(t) = \frac{1}{d}(b - (b - dN(t_0))e^{-d(t-t_0)}) \text{ and } \lim_{t \rightarrow \infty} N(t) = \frac{b}{d} \quad (2)$$

This leads us to the following limit computations in Eq. (1):

$$\begin{cases} \frac{dl(t)}{dt} = \frac{Kl(t-\tau)}{1+(t-\tau)\alpha I^h} \left(\frac{b}{d} - I - R\right) - (d + \mu)l(t) + \gamma R(t) \\ \frac{dR(t)}{dt} = \mu l(t) - (d + \gamma)R(t) \end{cases} \quad (3)$$

To regulate the subtleties of Equation (3), we determination emphasis on the existence, individuality, and constancy of the symmetry as well as the Hopf fork of the limit Equation (3). When understanding Eq. (1) biological implication, we continuously assume that the early value is nonnegative then takes the method of

$$I(s) = \phi(s) \geq 0, R(s) = \psi(s) \geq 0, \quad (4)$$

Everyplace $(\phi_1, \phi_2) \in D; D = D([- \tau, 0], \mathbb{R}^2)$ signifies the unceasing purpose planetary on $[- \tau, 0]$ by the average $\|\phi\| = \sup_{-\tau \leq \varphi \leq 0} |\phi(\varphi)|, \phi \in D$. After the \dagger prose on underdeveloped DE or practical DE in, there is a sole native answer of Eq. (3) aimed at all allowable optimistic parameters.

Originally, we obligate the subsequent assumptions.

Lemma 1: The district $S_\Delta = \{(I, C) \mid I \geq 0, C \geq 0, I + C \leq b/d\}$ is an invariant laid out and furthermore an arresting arrangement of Eq (3) in the 1st quadrant.

Lemma 2: (i) If $S_0 \leq 1$, before Eq. (3) takes a sole evenness $E_0 = (0, 0)$ in the 1st quadrant.

(ii) If $S_0 > 1$, then Equation (3) consumes 2 symmetry in the 1st quadrant, which remain E_0 plus $E_* = (I_*, S_*)$, wherever $I_*, S_* > 0$.

Threshold dynamics aimed at the case $\tau = 0$

In this fragment, we will amendment the steadiness and topological development of the DFE then EE of Eq. (3) denied of the undercover retro, that is,

$$\begin{cases} \frac{dl}{dt} = \frac{Kl}{1+\alpha l^h} \left(\frac{b}{d} - I - R \right) - (d + \mu)l(t) + \gamma R(t) \\ \frac{dR(t)}{dt} = \mu l - (d + \gamma)R \end{cases} \quad (5)$$

We first bounce the indigenous steadiness and topological building of the equilibrium

Lemma 3: (i) If $S_0 < 1$, before the DFE E_0 of Eq. (5) is a nearby asymptotically constant hyperbolic node.

(ii) If $S_0 = 1$, formerly DFE E_0 of Eq. (5) is a saddle-node besides is nearby asymptotically constant in the 1st quadrant.

(iii) If $S_0 > 1$, now the DFE E_0 of Eq. (5) is a shaky timber and the EE E^* is a distant asymptotically fixed exaggerated protuberance.

Lemma 4: Calculation (5) has neither a nontrivial intervallic orbit nor an extraordinary bolted course plus a finite integer of steadiness in S_Δ .

Lemma 5: The dual stable manifolds of burden E_0 stand not in S_Δ if $R > 1$.

Theorem 1: (i) If $S_0 < 1$, before Equation (5) consumes a single DFE E_0 , which stands a worldwide asymptotically steady hyperbolic swelling in the 1st quadrant.

(ii) If $S_0 = 1$, before Eq. (5) consumes a single DFE E_0 , which is a seat projection and is generally asymptotically steady in the 1st quadrant.

(iii) If $S_0 > 1$, then Eq. (5) consumes one exclusive DFE E_0 and a single EE E_* , and E_0 is a wobbly encumber and E_* is a generally asymptotically perpetual exaggerated knock in the 1st quadrant.

Dynamical behaviors aimed at the case $\tau > 0$

In this segment, we'll take a gander at the dependability and Hopf disparity of the steady state by the hidden period $\tau > 0$ in Eq. (3).

The following results show that R_0 is a benchmark an incentive for the DFE.

Theorem 2: If $R_0 \leq 1$, the DFE E_0 of Eq. (3) is universally asymptotically unchanging. If $S_0 > 1$, the DFE is uneven.

Theorem 3: If $S_0 > 1$ to $0 < g \leq 2$, Eq. (3) consumes a nearby asymptotically even EE $E_* = (I_*, R_*)$ aimed at several $\tau > 0$.

Theorem 4: *Shoulder that $S_0 > 1$, $g > 2$, plus $G < 0$, then to hand is a optimistic real integer*

T_0 s.t. the subsequent assumptions hold.

- (i) *If $0 < \tau < \tau_0$, Eq. (3) takes an EE E_* which stands close by asymptotically even;*
- (ii) *Equation (3) can endure a Hopf junction if $\tau > \tau_{0s}$, and a intermittent circle occurs in the minor locality of the EE E_* .*

Examples

The consequences on the undercurrents of the SIRI model (1) by one of its breaking point conditions are consistent with the aforementioned theorems (3). To demonstrate the utility of these findings, we will provide a few instances and their simulations in this section. We also demonstrate how the latent period and nonlinear incidence rate affect the dynamical activities of the SIRI model.

Take into account the SIRI exemplary (1) in the interest of the accompanying instances with various parameter values.

Example 1: Consider the values $b = 0.3$, $k = 0.3$, $d = 0.3$, $h = 1$, $\beta = 0.5$, $\gamma = 0.2$, and $\alpha = 10$. As a result of Theorems 1 and 2, it can be deduced that the sickness free equity is overall asymptotically in any event, for one idle time since $R_0 = 0.85 < 1$. Fig 1 depicts the universal asymptotical firmness of the evenness on behalf of the model (1) thru $\tau = 0$, $\tau = 20$, correspondingly.

Example 2: Consider the following values: $b = 0.3$; $k = 0.375$; $d = 0.3$; $h = 1$; $\beta = 0.5$; $\gamma = 0.2$; $\alpha = 10$. Later $R_0 = 1$, it tails since Theorem 1 and 2 that the uninfected evenness is universally asymptotically constant aimed at any latent period. The comprehensive asymptotic solidity of the equilibrium on behalf of the archetypal (1) with $\tau = 0$ and $\tau = 20$ is depicted in Figure 2.

Example 3: Consider the values $b = 1.5$, $k = 0.3$, $d = 0.3$, $h = 1$, $\beta = 0.5$, $\gamma = 0.2$, and $\alpha = 10$. It follows from Theorems 1 and 2 that the endemic evenness is universally (far away) asymptotically constant aimed at $\tau = 0$ (> 0), as $R_0 = 3.25 > 1$ and $0 < h < 2$. The equilibrium's asymptotic stability for the model (1) with $\tau = 0$ and $\tau = 30$ is depicted in Figure 3 similarly.

Example 4: Take $\gamma = 0.5$; $b = 1$; $k = 0.5$; $d = 0.3$; $h = 3$; $\mu = 0.2$; $\alpha = 1,000$.

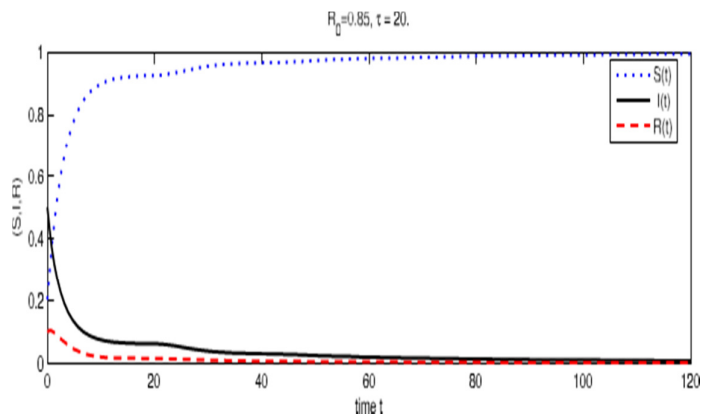


Fig. 1: Asymptotically globally stable sickness-free equilibrium exists if and only if $R_0 < 1$ $\tau = 20$ similarly $\tau = 0$.

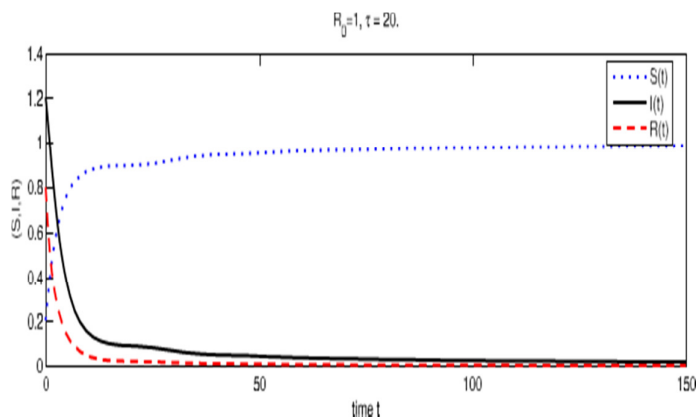


Fig. 2: There is an asymptotic global constancy in the absence of disease if $R_0 = 1$ as $\tau = 20$ similarly $\tau = 0$.

We analyze $h > 2$, $R_0 = 3.5733 > 1$, $H = -0.2723 < 0$. Subsequently Theorem 3, prevalent regularity is neighboring asymptotically steady on behalf of a slight covert dated, whereas the SIRI typical can smart a Hopf junction and food an intervallic trajectory on behalf of a large buried retro. Figure 4 shows the homogeneity asymptotically becoming solid at $\tau = 8$, , while Figure 5 shows the irregular route at $\tau = 30$. After the upstairs consequences and mathematical instances, we container attraction the next deductions

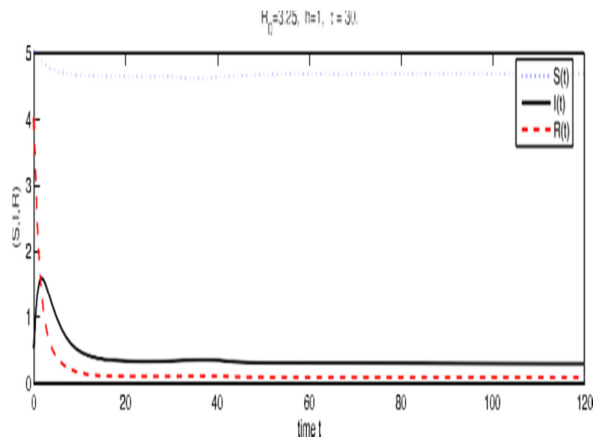


Figure 3: There exists a locally asymptotically stable endemic equilibrium if $R_0 > 1$ and $0 < h < 2$ and $\tau = 30$ similarly $\tau = 0$

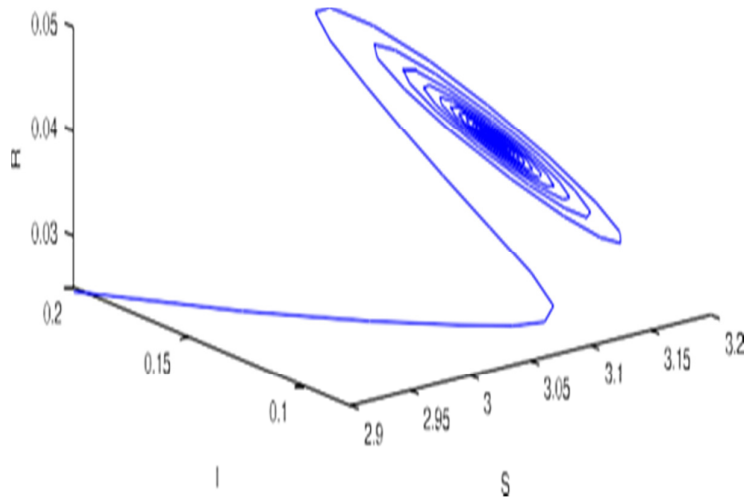


Figure 4: The prevalent equilibrium is locally asymptotically constant if $R_0 > 1$ and $h > 2$ as $\tau = 8$.

The rudimentary generative quantity R_0 controls the being of the symmetry. When $R_0 \leq 1$, perfect (1) has one sole sickness free balance. After $R_0 > 1$, great (1) consumes a sickness free balance and one novel prevalent symmetry.

On behalf of classical (1) lacking latent historical, its verge undercurrents is resolute by R_0 . Specifically, if $R_0 > 1$, then the illness free balance is worldwide asymptotically steady, and on the off chance that $R_0 \leq 1$, the select broad equality is universally asymptotically steady. $R_0 > 0$.

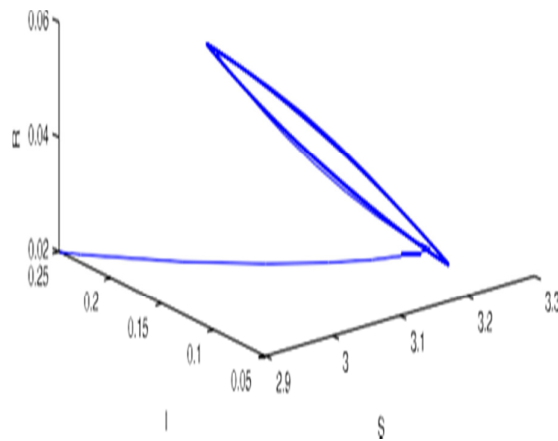


Figure 5: The emergence of a periodic orbit $R_0 > 1$ and $h > 2$ as $\tau = 30$.

The dynamical acts of the SIRS classical (1) convert composite when the SIRS perfect becomes covertly dated; these actions depend on the ethics of R_0 , and h . If $R_0 > 1$, the asymptotic universality of the disease-free steadiness holds true even for a single hidden date, hence the hidden date has no effect on the stability of the disease-free steadiness. If $R_0 > 1$, maintaining the wide-spread uniformity still requires a limit h in the nonlinear occurrence percentage and a sleeping dated. At the point when $h > 2$ and $R_0 > 1$, the SIRS model might go through a Hofstadter fork, producing an intervallic path for a fantastic latent history.

The global evenness is locally asymptotically even when $0 < h < 2$ notwithstanding $R_0 > 1$.

To that end, we are giving the green light to two quite puzzling bugs in this week's planned work.

(i) If $0 < h < 2$ too $R_0 > 1$, does the prevailing symmetry remain asymptotically constant everywhere?

Based on some quick maths, we think it's optimistic..

- (i) Ensures the model experience a Hopf bifurcation and harvest a periodic orbit if $R_0 > 1$, $h > 2$, then $H < 0$ is not satisfied?

References:

- [1]. Ruan, S, Wang, W: Dynamical behavior of an epidemic model with a nonlinear incidence rate. *J. Differ. Equ.* 188, 135-163 (2003)
- [2]. Xiao, D, Ruan, S: Global analysis of an epidemic model with nonmonotone incidence rate. *Math. Biosci.* 208, 419-429 (2007)
- [3]. Yang, Y, Xiao, D: Influence of latent period and nonlinear incidence rate on the dynamics of SIRS epidemiological models. *Discrete Contin. Dyn. Syst., Ser. B* 13, 195-211 (2010)
- [4]. Liu, WM, Levin, SA, Iwasa, Y: Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models. *J. Math. Biol.* 23, 187-204 (1986)
- [5]. Derrick, WR, van der Driessche, P: A disease transmission model in a nonconstant population. *J. Math. Biol.* 31, 495-512 (1993)
- [6]. Hethcote, HW, Lewis, MA, van der Driessche, P: An epidemiological model with a delay and a nonlinear incidence rate. *J. Math. Biol.* 27, 49-64 (1989)
- [7]. Imran, M.; Usman, M.; Dur-e-Ahmad, M.; Khan, A. Transmission dynamics of Zika Fever: A SEIR based model. *Differ. Equ. Dyn. Syst.* 2021, 29, 463–486.
- [8]. Dantas, E.; Tosin, M.; Cunha, A., Jr. Calibration of a SEIR–SEI epidemic model to describe the Zika virus outbreak in Brazil. *Appl. Math. Comp.* 2018,
- [9]. Rahman, M.U.; Arfan, M.; Shah, Z.; Kumam, P.; Shutaywi, M. Nonlinear fractional mathematical model of tuberculosis (TB) disease with incomplete treatment under Atangana-Baleanu derivative. *Alex. Eng. J.* **2021**, *60*, 2845–2856.

Cite this Article:

Manisha Halder, Dr. D.S. Sharma" **A mathematical analysis of Dynamical behaviour of Epidemiological models with nonlinear incidence rates**", *International Journal of Scientific Research in Modern Science and Technology (IJSRMST)*, ISSN: 2583-7605 (Online), Volume 2, Issue 5, pp. 33-40, May 2023.

Journal URL: <https://ijrmst.com/>