Stability and Hopf Bifurcation Analysis of a SIS Epidemic Model with Time Delay

N. Ramesh[1],Prof. B. RavindraReddy[2]

[1]Department of Mathematics, AVN Institute of Engineering &Technology, Hyderabad, India, Email:neeradiramesh27@gmail.com
[2]Department of Mathematics, Jntuceh, Kukatpally, Hyderabad, India, Email:rbollareddy@gmail.com

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ABSTRACT
This research is being carried out within the framework of a SIS pandemic with a saturating incidence rate and a latent latency. Immediate and urgent attention is given to ensuring the stability of the model's disease-free and endemic equilibrium. To determine whether Hopf bifurcation takes place, one must first analyse the situation and, using the elapsed time as a metric, derive the bifurcation parameter. In order to help shed light on the results that were achieved, examples and simulations are provided. This paper investigates the dynamics of an SIS (Susceptible-Infectious-Susceptible) epidemic model incorporating a time delay to represent the period between infection and recovery. The analysis focuses on understanding how the time delay influences the system's stability and leads to the emergence of Hopf bifurcations. This study highlights the critical role of time delay in shaping the spread of infectious diseases and provides insights for developing effective control strategies.

Keywords: SIS epidemic model, time delay, stability analysis, basic reproduction number, disease dynamics Hopf bifurcation.

1. INTRODUCTION

In recent years, mathematical models have emerged as powerful resources for studying and managing the transmission of infectious illnesses. Much of the classic mathematical literature on epidemiology makes use of continuous models, often expressed as simple non-linear differential equations. To combat the spread of illnesses, researchers in recent years have developed a variety of epidemiological models (e.g., SI, SIS, SIR, SIER, SIERS, etc.) that include various treatment options. The fundamental assumptions of these model state that the whole population is quantitatively classified into different groups based on their epidemiological condition and that the transmission of the virus is represented by incidence terms. Since there is no way to prevent reinfection in a SIS model, infected individuals revert to the susceptible class after they recover. [1] With a delay and a population size that may vary, H.W. Hethcote and P. Van den Driessche developed a SIS epidemic model. [2] G. Ranjith Kumar, K. Lakshmi Narayan, and B. Ravindra Reddy conducted a stability study of an epidemic model that included immigration and nonlinear incidence rates. [3] In their work, H.W. Hethcote and Vanden Driessche provide two delayed SIS epidemiologic models. [4] M.Sridevi, B.Ravindra Reddy, Dynamics of an SIS Epidemic Model with Double Epidemic Hypothesis. [5] V.N. Afanasev,V.B. Kolmanowski,and V.R. Nosov,MathematicalTheoryofControlSystem Design. P. Das, D. Mukherjee, and A. K. Sarkar, Study of an S-I epidemic model with nonlinearincidence rate [6]. S. Ruan, D. Xiao, Examining a non-monotone pandemic model on a global scale [7]. Authors: W.O. Kermack and A.G. Munro Enhancement of mathematical theory pertaining to epidemics [8]. Chen L.S., Chen J, Biological System with Nonlinear Dynamics [9]. In their work on epidemiological models for SIR, SIRS, and SIS, A. Korobeinikov and G.C. Wake addressed Lyapunov functions as well as global stability [10]. For Classical SIS, SIR, and SIRS Epidemic Models with Variable Population Size, C. Vargas-De-Le'on constructs Lyapunov Functions [11]. Vargas-De León C. On the Global Stability of SIS, SIR and SIRS Epidemic Models with Standard Incidence [12]. J. Zhou and H. W. Hethcote [13] state that in models for non-immune diseases, the incidence is proportional to the population size. Raid kamel Naji and Ashraf Adnan Thirthar released their book in 2018. The SIS epidemic model presented in [14] incorporates stability and bifurcation into its treatment function, as well as a saturated incidence rate. Bnerjee, S.K. [15] studied a fractional-order SIS pandemic where the population size is variable and the recruitment rate is constant. Zhang, J. Sun, Physicists tested the robustness of an epidemic model in SIS that included a feedback mechanism for networks [16]. J.S. Y. Muroya, T. Kuniya, and Zhou's SIS disease transmission model based on recruitment-birth-death

emographics [17] Keeping the world's population movement model consistent with a multi-group SIS pandemic [18]. Yasuo Muroya and Taiyo Kuniya A worldwide pandemic model that incorporates many populations and their resiliency [19]. In order to account for non-linear epidemic rates, K. Madhusudhan Reddy, K. Lakshmi Narayan, and B. Ravindra Reddy created the delayed SIS epidemic model [20].
In this research, we examine a SIS-type epidemic model and use it to find the fundamental reproduction number, stable states, and stability.

2.MATHEMATICAL MODEL

Immunity cannot be achieved against all infectious diseases. There is no way to recover from these illnesses; in fact, once infected, people are always at risk of contracting them again.
The SIS type may be used to simulate this illness. Classes (S) represent the susceptible members of the population, whereas classes (I) represent the infectious members (I).In order to put the SIS model into differential equations form,

$$\frac{dS}{dt} = b + rS - \frac{\beta SI}{1+\alpha I} + \delta I - \gamma S \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha I} - (\gamma + c + \delta) I$$

With initial conditions S (0)> 0, I (0)> 0 Where

S (t) and I (t) represent the number of susceptible and infected populations, respectively

b is the recruitment rate of the population, γ is the natural death rate, c is the death rate due to disease.

β is the infection coefficient, δ is the recovery rate of the infective individuals, r is the maximal per capita birth rate,

the parameters are positive constants and variables are non-negative.

In the nonlinear incidence rate $\frac{\beta SI}{1+\alpha I}$ used by Capasso and Serio. In their modeling of cholera, βSI measures theinfection force of the disease and $\frac{1}{1+\alpha I}$ measure the inhibition effect from the behavioral change of the susceptible individuals when their number increases.

3. EQUILIBRUIM ANALYSIS

There are two equilibrium for system (1)

i) Disease-Free-Equilibrium

$$P^0 = \left(\frac{b}{\gamma - r}, 0 \right)$$

ii) Endemic equilibrium

$P^* = (S^*, I^*)$ with coordinates

$$S^* = \frac{(1+\alpha I)(\gamma + c + \delta)}{\beta}, \quad I^* = \frac{b\beta + (r-\gamma)(\gamma + c + \delta)}{\alpha(\gamma + c + \delta)(\gamma - r) + \beta(\gamma + c)} \tag{2}$$

The parameter $R_0 = \frac{b\beta}{(\gamma - r)(\gamma + c + \delta)}$ is the Basic reproductive number

4. LOCALSTABILITY ANALYSIS

In this section, we investigate the stability analysis of Disease-Free-Equilibrium P^0 and Epidemic-Equilibrium P^*. The Jacobian matrix of system (1)

$$J = \begin{pmatrix} r-\gamma-\frac{\beta I^*}{1+\alpha I^*} & -\frac{\beta S^*}{(1+\alpha I^*)^2}+\delta \\ \frac{\beta I^*}{1+\alpha I^*} & \frac{\beta S^*}{(1+\alpha I^*)^2}-(\gamma+c+\delta) \end{pmatrix} \quad (3)$$

4.1 Stability of disease-free equilibrium

Theorem1: The disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: For the disease-free equilibrium at the point P^0 the system (3) reduces to

$$J(P^0) = \begin{pmatrix} (r-\gamma) & \frac{-\beta b}{(\gamma-r)}+\delta \\ 0 & \frac{b\beta}{(\gamma-r)}-(\gamma+c+\delta) \end{pmatrix} (4)$$

With characteristic equation

$$(r-\gamma-\lambda)\left(\frac{b\beta}{(\gamma-r)}-(\gamma+c+\delta)\right)-\lambda=0$$

The characteristic roots are given by

$\lambda_1 = r-\gamma, \lambda_2 = \frac{b\beta}{(\gamma-r)}-(\gamma+c+\delta)$ then the system is stable if $\frac{b\beta}{(\gamma-r)}-(\gamma+c+\delta)<0$

$$\frac{b\beta}{(\gamma-r)(\gamma+c+\delta)}<1$$

$$\therefore R_0 < 1$$

Hence the given system is stable if $R_0 < 1$ and unstable if $R_0 > 1$.

4.2 Stability of Endemic-Equilibrium

Theorem2: The Endemic steady state P^* of (1) is locally asymptotically stable if $R_0 > 1$

Proof: The Jacobian matrix for system (1) evaluated at the endemic steady state P^* is

$$J\left(P^*\right) = \begin{pmatrix} (r-\gamma)-\frac{\beta I^*}{\left(1+\alpha I^*\right)^2} & \frac{-\beta S^*}{\left(1+\alpha I^*\right)^2}+\delta \\ \frac{\beta I^*}{\left(1+\alpha I^*\right)^2} & \frac{\beta S^*}{\left(1+\alpha I^*\right)^2}-(\gamma+c+\delta) \end{pmatrix}$$

that can be rewritten as

$$J\left(P^*\right) = \begin{pmatrix} (r-\gamma)-\frac{\beta I^*}{\left(1+\alpha I^*\right)^2} & \frac{-\beta S^*}{\left(1+\alpha I^*\right)^2}+\delta \\ \frac{\beta I^*}{\left(1+\alpha I^*\right)^2} & \frac{-\beta S^* I^* \alpha}{\left(1+\alpha I^*\right)^2} \end{pmatrix} \quad (5)$$

when we consider the identify

$$\frac{\beta S^*}{1+\alpha I^*} = (\gamma+c+\delta)$$

which is obtained by the endemic steady state the trace of $J(P^*)$ is

$$trJ\left(P^*\right) = -\left[\left(\gamma - r\right) + \frac{\beta I^*(1+\alpha S^*)}{\left(1+\alpha I^*\right)^2}\right] < 0$$

Thus tr $J(P^*) < 0$

Also, we obtain

$$\det(J(P^*)) = \left[\left(\gamma - r\right) + \frac{\beta I^*}{\left(1+\alpha I^*\right)^2}\right]\left[\frac{\beta S^* I^* \alpha}{(1+\alpha I^*)^2}\right] + \left[\frac{\beta I^*}{\left(1+\alpha I^*\right)^2}\right]\left[\frac{\beta I^*}{\left(1+\alpha I^*\right)^2} - \delta\right] > 0$$

thus det $J(P^*) > 0$

Here, the eigen value of the Jacobian matrix $J(P^*)$ have negative real parts. This means that P^* is asymptotically stable whenever it exists.

5. DELAYED SISMODEL

This part of the paper is devoted to construction the dynamical model for our proposed problem.The process dynamical model can be described as

$$\begin{aligned}\frac{dS}{dt} &= b + rs - \frac{\beta SI(t-\tau)}{1+\alpha I(t-\tau)} + \delta I - \gamma S \\ \frac{dI}{dt} &= \frac{\beta SI(t-\tau)}{1+\alpha I(t-\tau)} - \gamma I - cI - \delta I\end{aligned} \tag{6}$$

6. LOCAL STABILITY ANALYSIS

In this section, we investigate the stability analysis of Disease-Free-Equilibrium P^0 and Endemic-Equilibrium P^*

The Jacobian matrix of system (6)

$$J = \begin{pmatrix} r - \frac{\beta I^*}{(1+\alpha I^*)} - \gamma & \frac{-\beta S^* e^{-\lambda\tau}}{\left(1+\alpha I^*\right)^2} + \delta \\ \frac{\beta I^*}{(1+\alpha I^*)} & \frac{\beta S^* e^{-\lambda\tau}}{(1+\alpha I^*)^2} - (\gamma + c + \delta) \end{pmatrix} \tag{7}$$

6.1 Stability of Disease-Free-Equilibrium

The Jacobian matrix of the linearized of model (6) at $P^0 = (S^*, 0)$ is given by

$$J(P^0) = \begin{bmatrix} (r-\gamma) & -\beta S^* e^{-\lambda\tau} + \delta \\ 0 & \beta S^* e^{-\lambda\tau} - (\gamma + c + \delta) \end{bmatrix} \tag{8}$$

With the characteristic equation

$$\left[\left(r - \gamma - \lambda\right)\right]\left[\beta S^* e^{-\lambda\tau} - (\gamma + c + \delta) - \lambda\right] = 0$$

In case of τ =0 in above equation

$$\lambda_1 = r - \gamma, \lambda_2 = \beta S^* - (\gamma + c + \delta) < 0$$

$$\frac{\beta S^*}{(\gamma + c + \delta)} < 1$$

$$\frac{\beta b}{(\gamma - r)(\gamma + c + \delta)} < 1,$$

$R_0 < 1$

Hence the given system is locally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$

6.2 Stability of Endemic Equilibrium

Now the Jacobian matrix J at endemic equilibrium $P^* = (S^*, I^*)$ is given by

$$J(P^*) = \begin{pmatrix} (r-\gamma) - \dfrac{\beta I^*}{(1+\alpha I^*)} & \dfrac{-\beta S^* e^{-\lambda\tau}}{(1+\alpha I^*)^2} + \delta \\ \dfrac{\beta I^*}{(1+\alpha I^*)} & \dfrac{\beta S^* e^{-\lambda\tau}}{(1+\alpha I)^2} - (\gamma + c + \delta) \end{pmatrix} \quad (9)$$

The characteristic equation of (9) is given by

$\lambda^2 + P_1\lambda + P_2 + e^{-\lambda\tau}(Q_1\lambda + Q_2) = 0$ (10)

Where $P_1 = 2\gamma + c + \delta - r + \dfrac{\beta I^*}{1+\alpha I^*}$, $P_2 = (\gamma - r)(\gamma + c + \delta) + \dfrac{\beta I^*(\gamma + c)}{1+\alpha I^*}$

$Q_1 = \dfrac{-\beta S^*}{(1+\alpha I^*)^2}$, $Q_2 = \dfrac{\beta S^*(r-\gamma)}{(1+\alpha I^*)^2}$

we need to find the necessary and sufficient condition for every root of the characteristic equation (10)

Case 1: If $\tau = 0$ equation (10) becomes

$\lambda^2 + P_1\lambda + P_2 + Q_1\lambda + Q_2 = 0$

$\lambda^2 + (P_1 + Q_1)\lambda + (P_2 + Q_2) = 0$ (11)

where $R_0 > 1$, we have $(P_1 + Q_1) > 0 \,\&\, (P_2 + Q_2) > 0$

By Routh-Hurwitz criteria, all roots of (11) are real and negative, or complex conjugate with the negative real part

Hence, the system (6) without delay is locally asymptotically stable when $R > 1$.

Case 2: If $\tau > 0$

Suppose that there is a positive τ_0 such that equation (10) has pair of purely imaginary roots $\pm\omega i$,

$\omega > 0 \, put \lambda = i\omega$ in (10), we get

$-\omega^2 + P_1\omega i + P_2 + (Q_1\omega i + Q_2)(\cos\omega t - i\sin\omega t) = 0$ (12)

Separating the real and imaginary parts,

we have

$\omega^2 - P_2 = Q_1\omega s\text{in}\,\omega t + Q_2\cos\omega t$

$-P_1\omega = -Q_2 s\text{in}\,\omega t + Q_1\omega\cos\omega t$ (13)

Which is equivalent to

$\omega^4 + (P_1^2 - Q_1^2 - 2P_2)\omega^2 + (P_2^2 - Q_2^2) = 0$ (14)

If $(P_1^2 - Q_1^2 - 2P_2) > 0$ and $(P_2^2 - Q_2^2) > 0$

Then, there is no ω such that $i\omega$ is an Eigen value of the characteristic equation (10)

i.e., λ will never be a purely imaginary root of equation (10). Thus, the real part of all the Eigen value of equation (10) is negative for all $\tau \geq 0$.

Hence, the endemic equilibrium P^* is asymptotically stable for all τ.

If the following conditions hold:

(i) $R_0 > 1$

(ii) $(P_1 + Q_1) > 0, (P_2 + Q_2) > 0$ (15)

(iii) $(P_1^2 - Q_1^2 - 2P_2) > 0, (P_2^2 - Q_2^2) > 0$

If $(P_2^2 - Q_2^2) < 0$. There is a unique positive ω_0 satisfying (14). That is, there is a positive τ_0 such that equation (10) has pair of purely imaginary roots $\pm i\omega_0$ as $\tau = \tau_0$ and all the eigen values with negative real parts as $0 < \tau < \tau_0$.

From (13) τ_k corresponding to ω_0 can be obtained

$$\Delta = \begin{vmatrix} Q_1\omega & Q_2 \\ -Q_2 & Q_1\omega \end{vmatrix} = Q_1^2\omega^2 + Q_2^2$$

$$\cos\omega_0\tau_k = \frac{1}{\Delta}\begin{vmatrix} Q_1\omega & \omega^2 - P_2 \\ -Q_2 & -P_1\omega \end{vmatrix} = \frac{-P_2Q_1\omega^2 + Q_2\omega^2 - P_2Q_2}{(Q_1^2\omega^2 + Q_2^2)}$$

$$\cos\omega_0 = \frac{-P_2Q_1\omega^2 + Q_2\omega^2 - P_2Q_2}{(Q_1^2\omega^2 + Q_2^2)}$$

$$\tau_k = \frac{1}{\omega_0}\cos^{-1}\left[\frac{-P_2Q_1\omega^2 + Q_2\omega^2 - P_2Q_2}{(Q_1^2\omega^2 + Q_2^2)}\right] + \frac{2n\pi}{\omega_0} \text{ n = 0, 1, 2} \quad (16)$$

7. Hopf Bifurcation

Based on the above results, we have the following

Theorem3:AssumethatR₀>1thenthereisapositiveτ₀suchthatthefollowingresultshold.

i) If $0 < \tau < \tau_0$, equation(6)has anendemic equilibrium $P*$ which is locally a symptotically stable.

ii) Equation(6)can undergo a Hopfbifurcation if $\tau > \tau_0$, and a periodic orbit exists in the small neighborhood of the endemic equilibrium.

Proof: To obtain the Hopf bifurcation, we need to check the transversal condition for the complex eigen values of the $P*$ at $\tau = \tau_0$. Then, from equation(10),wehave

$$\frac{d\lambda}{d\tau}\left[2\lambda + P_1 + Q_1e^{-\lambda\tau} - \left(Q_1\lambda + Q_2\right)\tau e^{-\lambda\tau}\right] = \lambda\left(Q_1\lambda + Q_2\right)e^{-\lambda\tau} \quad (17)$$

$$\left[\frac{d\lambda}{d\tau}\right]^{-1} = \left[\frac{2\lambda + P_1 + Q_1e^{-\lambda\tau} - \left(Q_1\lambda + Q_2\right)\tau e^{-\lambda\tau}}{\lambda\left(Q_1\lambda + Q_2\right)e^{-\lambda\tau}}\right]$$

$$\left[\frac{d\lambda}{d\tau}\right]^{-1} = \frac{2\lambda + P_1}{\lambda\left(\lambda^2 + P_1\lambda + P_2\right)} + \frac{Q_1}{\lambda\left(Q_1\lambda + Q_2\right)} - \frac{\tau}{\lambda}$$

$$\left|\frac{d\,\mathrm{Re}(\lambda)}{dt}\right|_{\lambda=i\omega_0} = \left|\mathrm{Re}\left(\frac{d\lambda}{d\tau}\right)^{-1}\right|_{\lambda=i\omega_0}$$

$$= \mathrm{Re}\left[\frac{2i\omega_0 + P_1}{-i\omega_0\left(-\omega_0^{\,2} + P_1i\omega_0 + P_2\right)} + \frac{Q_1}{i\omega_0\left(Q_1i\omega_0 + Q_2\right)} - \frac{\tau}{i\omega_0}\right]$$

$$= \mathrm{Re}\left[\frac{1}{\omega_0}\left(\frac{2i\omega_0 + P_1}{P_1\omega_0 + \left(\omega_0^{\,2} - P_2\right)i} + \frac{Q_1}{\left(-Q_1\omega_0 + Q_2i\right)} - i\tau\right)\right]$$

$$= \frac{1}{\omega_0}\left[\frac{2\omega_0(\omega_0^{\,2} - P_2) + P_1^2\omega_0}{P_1^2\omega_0 + (\omega_0^{\,2} - P_2)^2} - \frac{Q_1^{\,2}}{(Q_1^{\,2}\omega_0^{\,2} + Q_2^{\,2})}\right]$$

$$= \frac{2\omega_0^2 + (P_1^2 - 2P_2 - Q_1^2)}{(Q_1^2\omega_0^2 + Q_2)^2}$$

Under the condition $(P_1^2 - 2P_2 - Q_1^2) > 0$

We have $\left|\frac{dR_e(\lambda)}{dt}\right|_{\lambda=i\omega_0} > 0$

Therefore, the transversality condition holds, and Hopf bifurcation occurs at $\omega = \omega_0, \tau = \tau_0$

8. NUMERICAL SIMULATION

In this section, we substantiate as well as augment our analytical results through numerical simulations considering the following

Example: We take the following parameters

$b = 0.9, \beta = 0.1, r = 0.4, \alpha = 0.023, \delta = 0.003, \gamma = 0.5, c = 0.071$

system(6) has the unique positive equilibrium P^* (7.0602, 0.5580) and R_0 = 1.5679 > 1

It follows from result (16), that the critical positive time delay τ_0 = 0.45 and we know that when as $0 < \tau < \tau_0$ E_1 is asymptotically stable. From the theorem (2) when τ passes through the critical value τ_0 =0.45 The positive equilibrium loses its stability and a family of periodic solution bifurcate from P*.(Fig. 1-3).

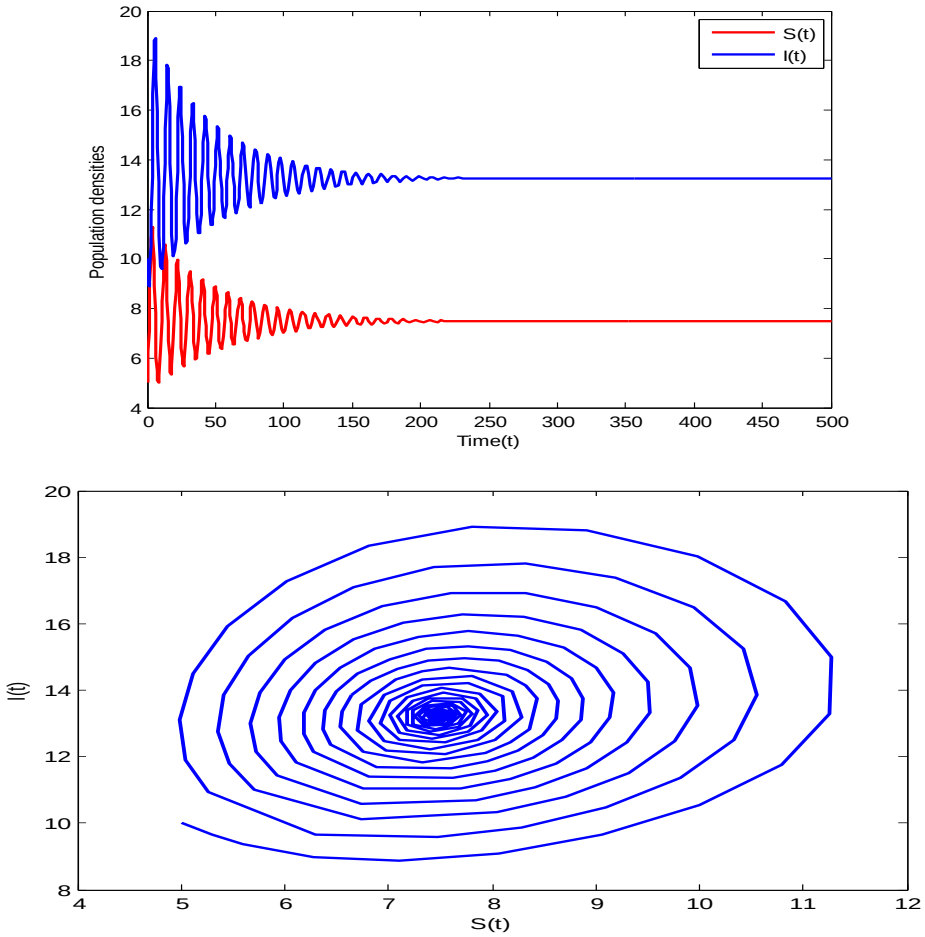

Figure 1: The trajectories and phase graphs of system (6) with $\tau = 0.05 < \tau_0 = 0.45$

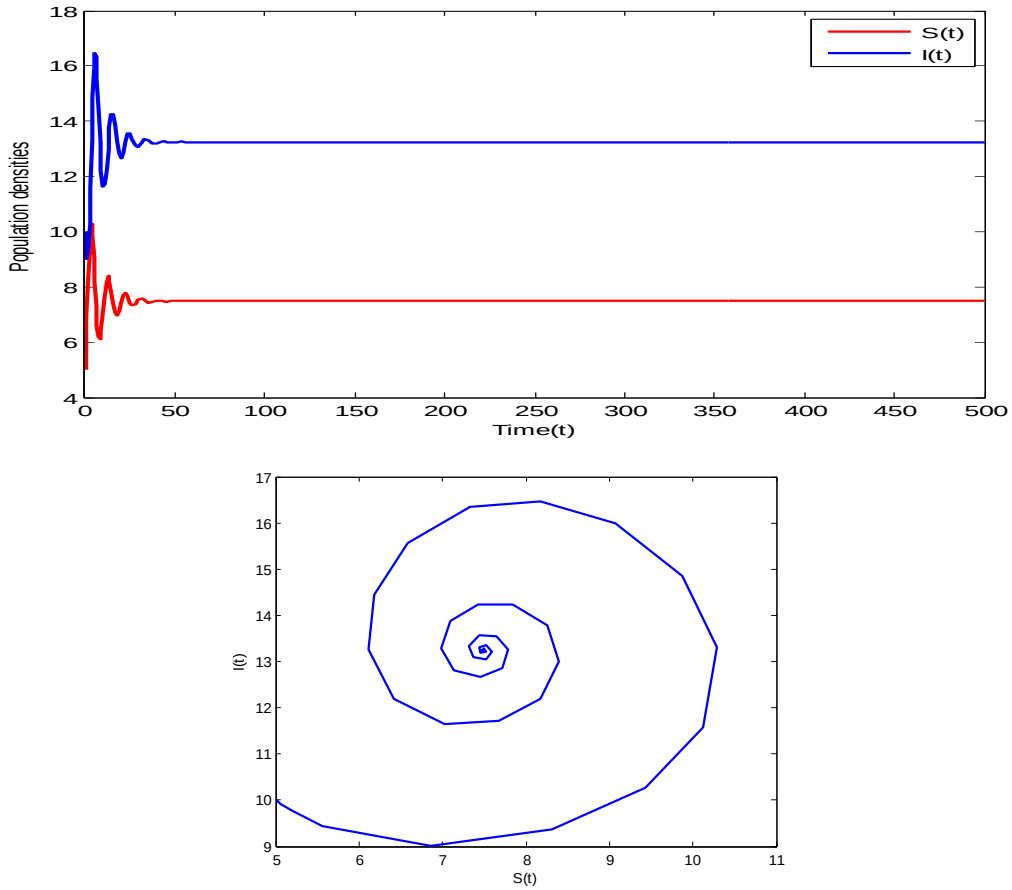

Figure 2: The trajectories and phase graphs of system (6) with $\tau = \tau_0 = 0.45$

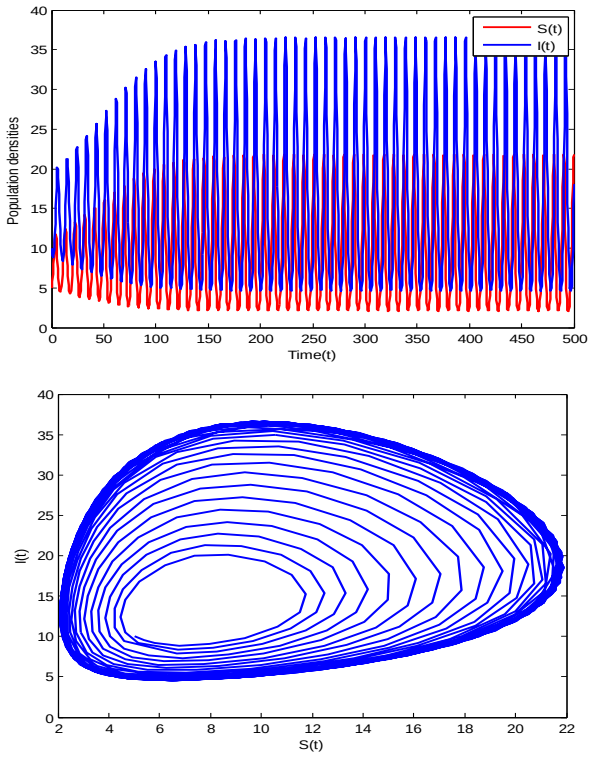

Figure 3: The trajectories and phase graphs of system (6) with $\tau = 0.6 > \tau_0 = 0.45$

Findings

Threshold Dynamics: The system's behaviour is dictated by the fundamental reproduction number (R_0). The sickness will disappear if the disease-free equilibrium is universally stable, which occurs when $R_0<1$. An endemic equilibrium is formed when $R_0>1$, which means that the sickness persists.

1. **Impact of Time Delay:** Time delay significantly influences the stability of the endemic equilibrium. When the delay is small, the equilibrium remains stable. However, beyond a critical delay value, the system undergoes a Hopf bifurcation, leading to periodic oscillations in the disease prevalence.
2. **Critical Conditions for Bifurcation:** Analytical expressions are derived to identify the delay values at which Hopf bifurcation occurs, providing a clear understanding of the transition from stability to oscillatory behavior.
3. **Numerical Validation:** Simulations confirm the theoretical predictions, demonstrating the onset of periodic oscillations and the sensitivity of disease dynamics to time delays.

Suggestions

1. **Delay Minimization:** Public health interventions should aim to minimize delays in diagnosing and treating infections, as longer delays can destabilize the system and result in recurrent outbreaks.
2. **Early Detection and Response:** Strategies that focus on reducing the infectious period, such as faster testing and treatment, can help maintain the stability of the disease-free state.
3. **Policy Design:** Incorporating the effects of delays in epidemiological models can improve the accuracy of disease control policies and help design interventions that prevent periodic disease outbreaks.
4. **Further Research:** Investigate the influence of other factors, such as vaccination or external interventions, in the presence of delays, to develop more comprehensive models for managing infectious diseases.
5. **Community Awareness:** Educating the public about timely medical intervention can be a practical approach to reducing the effects of delays in disease transmission dynamics.

CONCLUSION

Using a conventional incidence rate, we have examined the SIS epidemic model in this specific paper. We found that of all the variables that affect the SIS epidemic model's global stability, the base reproduction number is the most crucial. A disease-free-steady state, which is globally asymptotically stable, will be the only possible outcome if the basic reproduction number is less than one; the disease will inevitably vanish. If there is more than one fundamental reproduction number, then there is a unique endemic stable state. As a result, the disease is likely to spread across the population and become endemic in due time. This study provides a comprehensive analysis of the stability and dynamics of an SIS epidemic model incorporating a time delay to represent the period between infection and recovery. The results demonstrate that the basic reproduction number (R_0) serves as a crucial threshold parameter determining the disease's long-term behavior. When $R_0<1$, the disease-free equilibrium is globally stable, ensuring the eradication of the disease. For $R_0>1$, an endemic equilibrium emerges, and the system's stability depends on the time delay.As the delay increases beyond a critical value, the system experiences a Hopf bifurcation, leading to sustained oscillations in the infected population. This highlights the significant role of delays in influencing disease dynamics and the potential for recurrent outbreaks.The findings emphasize the importance of timely interventions and suggest that reducing delays in diagnosis and treatment can stabilize the system and control disease spread. This work contributes to the understanding of time-delayed epidemic models and offers valuable insights for designing effective public health strategies to mitigate the impact of infectious diseases.

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