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Dynamics of a Generalized Infectious Disease Model with Transmission, Treatment and Vaccination Delays

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ABSTRACT

This paper presents a generalized SIR model that accounts for real-world factors such as time delays in disease transmission, treatment, vaccine effectiveness, and waning immunity. The model incorporates a delay representing the time required for individuals to become infectious after exposure, delay in the treatment, as well as a separate delay reflecting the period between vaccination and the development of immunity. In addition, a delay is introduced in the return of recovered individuals to the susceptible class, simulating the gradual loss of immunity over time. The stability of the disease-free and endemic equilibria is examined using both local and global analysis. Local stability conditions are derived through the basic reproduction number and characteristic roots, while global stability is explored using a Lyapunov-based approach. Numerical simulations are carried out to demonstrate how varying the delays influences the disease progression and control, offering practical implications for public health strategies.

Keywords: Infectious Disease Model, Time Delays, Equilibria, Stability Analysis.

INTRODUCTION

Infectious diseases have long posed significant challenges to public health, largely due to their ability to spread rapidly and resurface unexpectedly. Over time, considerable research has focused on understanding the patterns of disease transmission and identifying effective strategies to contain outbreaks. Among the tools that have proven invaluable in this effort is mathematical modelling, which allows researchers to represent disease dynamics through equations and simulate how infections progress within a population.

Traditional models often classify individuals into compartments such as susceptible, infected, and recovered, forming the basis of the well-known SIR framework. These compartmental models have helped in analyzing a wide range of infectious diseases [3,5,9,11,14,20]. However, real-world disease spread involves additional complexities that these basic models may not fully capture. One such aspect is the delay in various disease-related processes, which can significantly alter the course of an outbreak [4, 6, 9, 10, 12, 15].

In particular, the delay between infection and the onset of infectiousness, known as the transmission delay, plays a critical role in the spread of many diseases. Similarly, after receiving a vaccine, individuals typically do not develop immunity instantly; this vaccination delay can affect how quickly a population becomes protected. Even the delay in treatment affects the spread of infection. Moreover, immunity, whether from recovery or vaccination, is not always permanent. Individuals may gradually lose their immunity and become susceptible once again. Accounting for this delay in loss of immunity provides a more realistic view of how diseases may re-emerge in populations over time[1-2,13,16, 21].

By integrating these four types of delays-transmission delay, vaccination delay, and immunity waning delay-into our models, we can better understand the timing and scale of outbreaks, as well as assess the effectiveness of public health measures like vaccination campaigns, quarantine periods, and booster shots. These insights are especially important in managing diseases where immunity does not last indefinitely or where vaccine rollout faces logistical challenges.

In this study, we build upon the classical SIR model by incorporating treatment and vaccination strategies, as well as the above-mentioned delays. The model originally proposed in [18] serves as our foundation. Prior studies, such as [7], have explored the impact of time-dependent treatment and vaccination rates, while [8] analysed the global stability of related models. The consequences of delays associated with vaccine efficacy were considered in [17]. In [19] the predictive dynamics of the model is studied. In our work, we extend these ideas by introducing explicit time delays in four key processes: the transmission of infection, the activation of vaccine-induced immunity, treatment delay and the return of recovered individuals to the susceptible class due to immunity loss. We analyze how these delays influence the behavior of the system and the potential for disease persistence or elimination.

The paper is organized as follows: Section 2 introduces the mathematical model and discusses its fundamental properties. In Section 3, we conduct a detailed stability analysis, both local and global, to understand how the system responds to different delay parameters. Section 4 presents numerical simulations that illustrate the influence of delays on the progression and control of the disease. Finally, Section 5 concludes the study with a summary of key findings and potential directions for future research.

Model Description

We study a compartmental model consisting of three groups: susceptible $'u'$, infected $'v'$, and recovered $'w'$ individuals. The model incorporates time delays and is governed by the following set of differential equations:

$$u' = a - bf(u,v) - du - cV(u(t-\eta)) + \alpha w(t-\mu)$$
$$v' = b_1 f(u(t-\tau),v) - rP(v) - d_1 v$$
$$w' = rP(v(t-\delta)) - \alpha w \quad (1)$$

Here:

- a: Rate of entry into the susceptible class.
- $f(u,v)$: Infection function based on contact between susceptible and infected individuals.
- b: Infection contact rate.
- d: Natural removal rate of susceptibles (e.g., through immunity or external causes).
- $V(u)$: Vaccination function dependent on the susceptible population.
- c: Rate of successful vaccination.
- $b_1 < b$: Reduced infection rate due to partial protection or immunity.
- $P(v)$: Recovery function through treatment.
- r: Treatment recovery rate.
- d_1: Disease-induced death rate.
- α: Rate of immunity loss leading to re-susceptibility.

Time Delays

- $\tau > 0$: Delay representing the incubation period (time from exposure to infectiousness).
- $\delta > 0$: Delay in treatment.
- $\mu > 0$: Delay for recovered individuals to become susceptible again due to waning immunity.
- $\eta > 0$: Delay in Vaccination

Model Assumptions [1]

- $f(u,v) \geq 0, P(v) \geq 0, V(u) \geq 0 \ \ \forall u \ and \ \forall v$
- $f(u,0) \geq 0, \forall u$
- $f(0,v) \geq 0, \forall v$
- $f(0,0) = 0, P(0) = 0, V(0) = 0.$

These conditions ensure that infection, recovery, and vaccination behave in a biologically realistic manner.

Using the approach outlined in [17], we establish that the solutions of model (1) remain both positive and bounded, and present the result as follows.

Theorem 2.1

If the initial conditions are nonnegative, then all solutions to system (1) remain nonnegative and bounded for all $t \geq 0$ [17].

In the next section, we analyze the local and global stability of the equilibrium points of the system, taking into account the effects of the time delays.

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STABILITY ASPECTS

Existence of equilibria

When using mathematical models like Model (1) to study how diseases behave in a population, one of the key steps is identifying the long-term behavior of the system. This involves finding what are known as equilibrium points**,** which describe the possible steady states that the population may settle into over time. Two important types of equilibrium often arise in such models: the disease-free equilibriumand theendemic equilibrium**.**

The disease-free equilibrium refers to a situation where the infection has been completely cleared from the population. In this case, there are no infected or recovered individuals—only susceptible ones remain. Mathematically, it is written as $(u^*, 0,0)$, where $u^* > 0$ represents the size of the healthy, uninfected population.

On the other hand, the endemic equilibrium describes a condition in which the disease continues to exist in the population over time. Here, all three groups—susceptible, infected, and recovered—are present in the system with positive values. This state is written as (u^*, v^*, w^*), with each term greater than zero. It suggests a persistent level of infection, even if it doesn't grow or shrink.

Understanding whether these points are stable helps us predict whether the disease will eventually disappear or continue circulating in the population. This kind of analysis also plays a key role in evaluating how effective public health strategies—like vaccination and treatment—might be in controlling or eliminating the disease.

The basic reproduction number, written as R_0, is an important concept used to estimate how a disease might spread. It tells us how many people, on average, one infected person can pass the disease to in a population where everyone is still vulnerable. If R_0 is greater than 1, the disease is likely to spread; if it is less than 1, it will likely die out.

In the case of Model (1), we use what's called thenext-generation matrix methodto calculate R_0. This method helps us understand how new infections are generated and spread through the population, based on the model's structure. We get $R_0 = \frac{b_1 f_v(u^*,0)}{rP'(0)+d_1}$ (refer to [17] for detailed derivation of R_0)

Local Stability

Looking at how stable the equilibrium points are in the model helps us understand whether a disease will fade away or continue to spread over time. Local stability, in particular, tells us how the system behaves when there's a small change in the number of infections or recoveries. If the system returns to its original state after a slight disturbance, the equilibrium is said to be stable. But if the system moves further away from that point, it means the disease might spread more or eventually die out, depending on the situation.

We'll begin by outlining the conditions that determine whether the disease-free equilibrium remains stable when small changes occur in the system.

Theorem 3.1: System (1) will be locally stable at disease free equilibrium point $(u^*, 0,0)$ if

(i) $R_0 < 1$, (ii)$\alpha e^{-\lambda\mu} < bf_u(u^*, 0) + d + ce^{-\lambda\eta} V_{u_\eta}(u^*)$, (iii)$re^{-\lambda\delta} P_{v_\delta}(0) < \alpha$.

Proof: The characteristic equation for the system (1) at $(u^*, 0,0)$ is given by

$$\begin{bmatrix} \lambda - A_1 & Q_1 & 0 \\ 0 & \lambda - B_1 & 0 \\ 0 & 0 & \lambda - C_1 \end{bmatrix} = 0$$

$$A_1 = -bf_u(u^*, 0) - d - ce^{-\lambda\eta} V_{u_\eta}(u^*) + \alpha e^{-\lambda\mu}$$
$$Q_1 = -bf_v(u^*, 0)$$

$$B_1 = b_1 f_v(u^*, 0) - r\,P'(0) - d_1 + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, 0)$$
$$C_1 = -\alpha + re^{-\lambda\delta} P_{v_\delta}(0)\,.$$

Clearly A_1, B_1 and C_1 are the eigenvalues.

A system of the form (1) is unstable if any of its eigenvalues have a positive real part, and it is stable if all the eigenvalues are negative.

If $B_1 > 0$, then clearly $R_0 > 1$.Thus, if $R_0 > 1$ then the system is unstable.

For the system to be stable $A_1 < 0$, $B_1 < 0$, and $C_1 < 0$.

$$A_1 < 0 \implies \alpha e^{-\lambda\mu} < bf_u(u^*, 0) + d + ce^{-\lambda\eta} V_{u_\eta}(u^*),$$
$$B_1 < 0 \implies R_0 < 1,$$
$$C_1 < 0 \implies re^{-\lambda\delta} P_{v_\delta}(0) < \alpha.$$

Thus proved.

Next, we state the conditions for local stability at the endemic equilibrium.

Theorem 3.2: For the conditions

(i) $\alpha e^{-\lambda\mu} < bf_u(u^*, v^*) + d + ce^{-\lambda\eta} V_{u_\eta}(u^*)$,

(ii) $b_1 f_v(u^*, v^*) + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*) < r\, P'(v^*) + d_1$,

(iii) $re^{-\lambda\delta} P_{v_\delta}(v^*) < \alpha$,

system (1) is locally stable at the endemic equilibrium point (u^*, v^*, w^*)

Proof: The characteristic equation for the system (1) at (u^*, v^*, w^*) is given by

$$\begin{bmatrix} \lambda - A_2 & Q_1 & 0 \\ 0 & \lambda - B_2 & 0 \\ 0 & 0 & \lambda - C_2 \end{bmatrix} = 0$$

$$A_2 = -bf_u(u^*, v^*) - d - ce^{-\lambda\eta} V_{u_\eta}(u^*) + \alpha e^{-\lambda\mu}$$
$$Q_2 = -bf_v(u^*, v^*)$$
$$B_2 = b_1 f_v(u^*, v^*) - r\, P'(v^*) - d_1 + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*)$$
$$C_2 = -\alpha + re^{-\lambda\delta} P_{v_\delta}(v^*).$$

Clearly A_2, B_2 and C_2 are the eigenvalues.

$$A_2 < 0 \implies \alpha e^{-\lambda\mu} < bf_u(u^*, v^*) + d + ce^{-\lambda\eta} V_{u_\eta}(u^*),$$
$$B_2 < 0 \implies b_1 f_v(u^*, v^*) + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*) < r\, P'(v^*) + d_1,$$
$$C_2 < 0 \implies re^{-\lambda\delta} P_{v_\delta}(v^*) < \alpha.$$

Under the above three conditions, all the eigenvalues of the system are negative, indicating that the equilibrium point is locally stable.

We now turn our attention to analyzing the conditions required for global stability of the model.

Global Stability At Endemic Equilibria

When we're trying to understand how a disease behaves over time using models like (1), it's not just the immediate spread that matters; we also want to know what happens in the long run. That's where the concept of global stability becomes really important.

Simply put, if a system is globally stable, it means that no matter how things start-whether there are just a handful of infections or a sudden outbreak-it will eventually settle into a stable pattern. If this stable point is what we call an

endemic equilibrium, it means the disease won't go away entirely, but it will stay at a constant level within the population. Even if the number of cases rises or falls for a while, over time, the system will naturally move back to that steady state.

Studying global stability helps us figure out whether a disease is likely to fade out on its own or become a lasting issue in the community. In our case, we use the Lyapunov stability method to look at these long-term patterns and see if the model always returns to equilibrium, no matter where it starts.

To establish global stability, we begin by assuming that the functions f, V and P satisfy certain Lipschitz conditions.

$$K_1|u-u^*| + K_2|v-v^*| \le |f(u,v)-f(u^*,v^*)| \le K_3|u-u^*| + K_4|v-v^*|$$
$$M_1|u-u^*| \le |V(u)-V(u^*)| \le M_2|u-u^*|$$
$$N_1|v-v^*| \le |P(v)-P(v^*)| \le N_2|v-v^*| \quad (2)$$

The following inequality we will be used in the next result

For any real numbers $a$and b, $ab \le \frac{1}{4\theta}a^2 + \theta b^2$ (3)

As the equilibrium point (u^*, v^*, w^*) is the solution of the system (1). We can rewrite the system as

$$(u-u^*)' = -b(f(u,v)-f(u^*,v^*)) - d(u-u^*) - c(V(u(t-\eta)) - V(u^*)) + \alpha(w(t-\mu)-w^*)$$
$$(v-v^*)' = b_1(f(u(\mathrm{t}-\tau),v)-f(u^*,v^*)) - r(P(v)-P(v^*)) - d_1(v-v^*)$$
$$(w-w^*)' = r(P(v(t-\delta)) - P(v^*)) - \alpha(w-w^*) \quad (4)$$

We state

Theorem 3.3. The system (1) is globally stable at the equilibrium point if the functions of system (1) satisfy Lipschitz conditions (2) and the parameters of the system satisfy

(i) $bK_1 + d + cM_1 + \frac{bK_2}{4\theta_1} - \frac{\alpha}{4\theta_2} - \frac{b_1K_3}{4\theta_1} > 0,$

(ii) $bK_2\theta_1 - b_1K_4 + rN_1 + d_1 - b_1K_3\theta_1 - \frac{rN_2}{4\theta_3} > 0,$

(iii) $\alpha(1-\theta_2) - rN_2\theta_3 > 0.$

Proof: Let the Lyapunov function be

$$L = |u-u^*|^2 + |v-v^*|^2 + |w-w^*|^2 + \alpha|u-u^*| \int_{t-\mu}^{t} |w(s)-w^*|^2\,ds + b_1K_3|v-v^*| \int_{t-\tau}^{t} |u(s)-u^*|^2\,ds + rN_2|w-w^*| \int_{t-\delta}^{t} |v(s)-v^*|^2 - cM_1|u-u^*| \int_{t-\eta}^{t} |u(s)-u^*|^2.$$

Then the dini derivative along the solutions of (1) using (4) is

$$\begin{aligned} D^+ \le\ & |u-u^*|[-b|f(u,v)-f(u^*,v^*)| - d|u-u^*| - c|V(u(t-\eta))-V(u^*)| \\ & + \ \alpha|w(t-\mu)-w^*|] \\ & + |v-v^*|[b_1|f(u(\mathrm{t}-\tau),v)-f(u^*,v^*)| - r|P(v)-P(v^*)| \\ & - d_1|v-v^*|] + |w-w^*|\left[r\left|P\left(v(t-\delta)\right)-P(v^*)\right| - \alpha|w-w^*|\right] \\ & + \alpha|w-w^*| - \alpha|w(t-\mu)-w^*| + b_1K_3|u-u^*| - b_1K_3|u(\mathrm{t}-\tau)-u^*| \\ & + rN_2|v-v^*| - rN_2|v(t-\delta)-v^*| - cM_1|u-u^*|^2 \\ & + cM_1|u-u^*||u(\mathrm{t}-\eta)-u^*| \end{aligned}$$

Applying conditions (2) and simplifying, we get

$$\begin{aligned} D^+ \le\ & -b(K_1|u-u^*|^2 + K_2|u-u^*||v-v^*|) - d|u-u^*|^2 - cM_1|u-u^*||u(t-\eta)-u^*| \\ & + \alpha|w(t-\mu)-w^*||u-u^*| + b_1(K_3|u(\mathrm{t}-\tau)-u^*||v-v^*| + K_4|v-v^*|^2) \\ & - rN_1|v-v^*|^2 - d_1|v-v^*|^2 + rN_2|w-w^*||v(t-\delta)-v^*| - \alpha|w-w^*|^2 \\ & + \alpha|w-w^*||u-u^*| - \alpha|u-u^*||w(t-\mu)-w^*| + b_1K_3|v-v^*||u-u^*| \\ & - b_1K_3|v-v^*||u(\mathrm{t}-\tau)-u^*| + rN_2|w-w^*||v-v^*| \\ & - rN_2|w-w^*||v(t-\delta)-v^*| - cM_1|u-u^*|^2 + cM_1|u-u^*||u(\mathrm{t}-\eta)-u^*| \\ D^+ \le\ & -bK_1|u-u^*|^2 - bK_2|u-u^*||v-v^*| - d|u-u^*|^2 + b_1K_4|v-v^*|^2 - rN_1|v-v^*|^2 \\ & - d_1|v-v^*|^2 - \alpha|w-w^*|^2 + \alpha|w-w^*||u-u^*| + b_1K_3|v-v^*||u-u^*| \\ & + rN_2|w-w^*||v-v^*| - cM_1|u-u^*|^2 \end{aligned}$$

From (3), we assume there exist $\theta_1, \theta_2, \theta_3 > 0$, such that

$$|u-u^*||v-v^*| \le \frac{1}{4\theta_1}|u-u^*|^2 + \theta_1|v-v^*|^2,$$

$$|u-u^*||w-w^*| \le \frac{1}{4\theta_2}|u-u^*|^2 + \theta_2|w-w^*|^2$$

$$|v-v^*||w-w^*| \le \frac{1}{4\theta_3}|v-v^*|^2 + \theta_3|w-w^*|^2$$

Using these inequalities, we have

$$\begin{aligned} D^+ \le\ & -bK_1|u-u^*|^2 - bK_2\left(\frac{1}{4\theta_1}|u-u^*|^2 + \theta_1|v-v^*|^2\right) - d|u-u^*|^2 + b_1K_4|v-v^*|^2 \\ & - rN_1|v-v^*|^2 - d_1|v-v^*|^2 - \alpha|w-w^*|^2 + \alpha\left(\frac{1}{4\theta_2}|u-u^*|^2 + \theta_2|w-w^*|^2\right) \\ & + b_1K_3\left(\frac{1}{4\theta_1}|u-u^*|^2 + \theta_1|v-v^*|^2\right) + rN_2\left(\frac{1}{4\theta_3}|v-v^*|^2 + \theta_3|w-w^*|^2\right) \\ & - cM_1|u-u^*|^2 \end{aligned}$$

Simplifying we get

$$D^+ \le -\left(bK_1 + d + cM_1 + \frac{bK_2}{4\theta_1} - \frac{\alpha}{4\theta_2} - \frac{b_1K_3}{4\theta_1}\right)|u-u^*|^2 - \Big(bK_2\theta_1 - b_1K_4 + rN_1 + d_1 - b1K3\theta1 - rN24\theta3v - v*2 - \alpha1 - \theta2 - rN2\theta3w - w*2.$$

By our assumption on parameter, we get $D^+ \le 0$.

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Hence by LyapunovTheory $u \longrightarrow u^*, v \longrightarrow v^*$and $w \longrightarrow w^*$.

Therefore, the system is (1) is globally stable at equilibria(u^*, v^*, w^*).

Remark 3.1: The results discussed earlier clearly indicate that the system is capable of maintaining both local and global stability, even when time delays are introduced. While delays are often associated with instability in many dynamic systems, that is not always the case here. The key factor lies in how the model is formulated-specifically, in the careful selection of parameter values and the structure of the nonlinear components.

When the parameters are chosen thoughtfully, and the nonlinear terms are designed to meet certain mathematical conditions or constraints, the system can continue to behave in a stable manner over time. This means that delays, by themselves, are not inherently destabilizing. Instead, it is the interplay between the delays, parameters, and nonlinear dynamics that determines the system's overall behavior. A well-constructed model can effectively absorb or manage the impact of delays without losing stability.

This insight is important, as it reassures us that delayed effects-common in real-world systems such as biological processes, communication networks, or control systems-do not automatically compromise the system's reliability. With proper mathematical modeling, stability can still be ensured. In the following section, we will illustrate this concept through a some of numerical examples. These examples will demonstrate how the behavior of the system changes as the delay parameters are varied, providing a clearer picture of the role delays play and how stability is affected in practice.

Numerical Examples

In this section, we explore several numerical examples based on system (1), where the parameter values, the functional forms of the nonlinear terms. To study the impact of delay on the system's behavior, we vary the delay parameter. The system of delay differential equations is solved using MATLAB's built-in dde23 solver, which is specifically designed for such problems. The resulting solutions are then plotted to visualize how the dynamics of the model evolve as μ\muμ changes. These simulations help provide deeper insight into the role of delay in shaping the system's overall behavior.

Consider the system

$$u' = 11 - 4f(u,v) - 0.5u - 2V(u(t-\eta)) + 2w(t-\mu)$$
$$v' = 3f(u(\mathrm{t}-\tau), v) - 1.5P(v) - 3.5v$$
$$w' = 1.5P\big(v(t-\delta)\big) - 2w \qquad (5)$$

Letting the functional values to be$f(u,v) = uv, V(u) = u, P(v) = v$, for $\theta_1 = \theta_2 = \theta_3 = 0.5$ the system (5) satisfies the constrains of Theorem 3.3. Therefore the system is stable globally at equilibrium point (1.6. 2, 1). The behaviour of the solution of the system (5) for various values of delays can be seen in Figures 1-4.

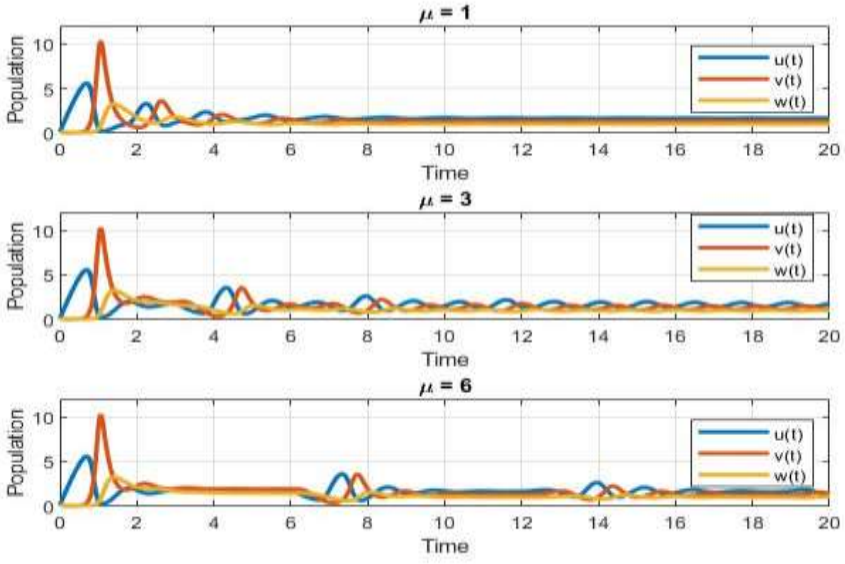

Figure 1: Solution profile of (5) for different values of μ

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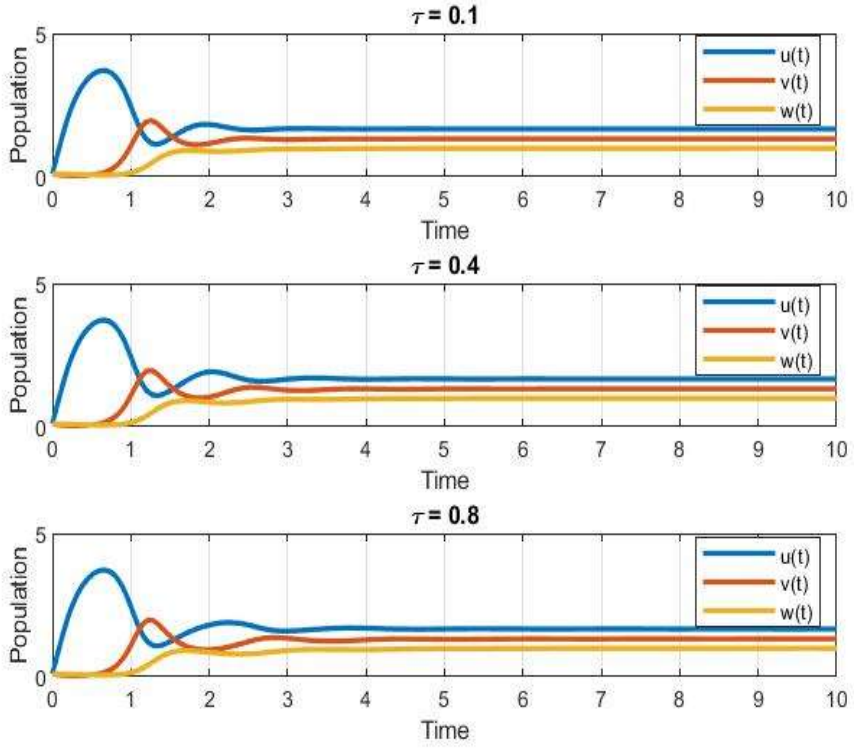

Figure 2 Solution profile of (5) for different values of τ

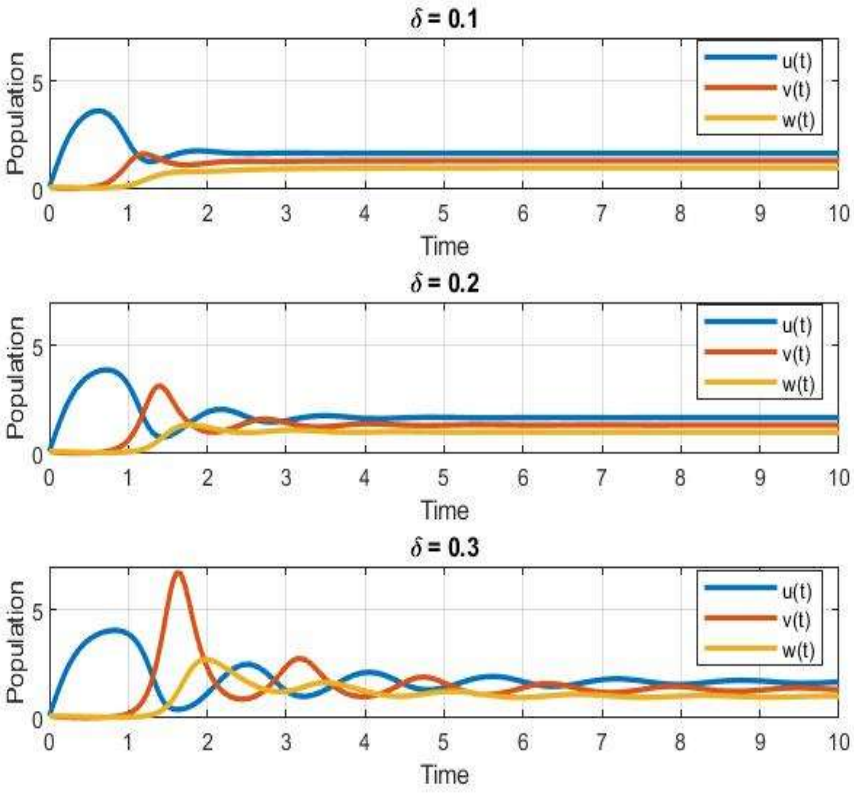

Figure 3 Solution profile of (5) for different values of δ

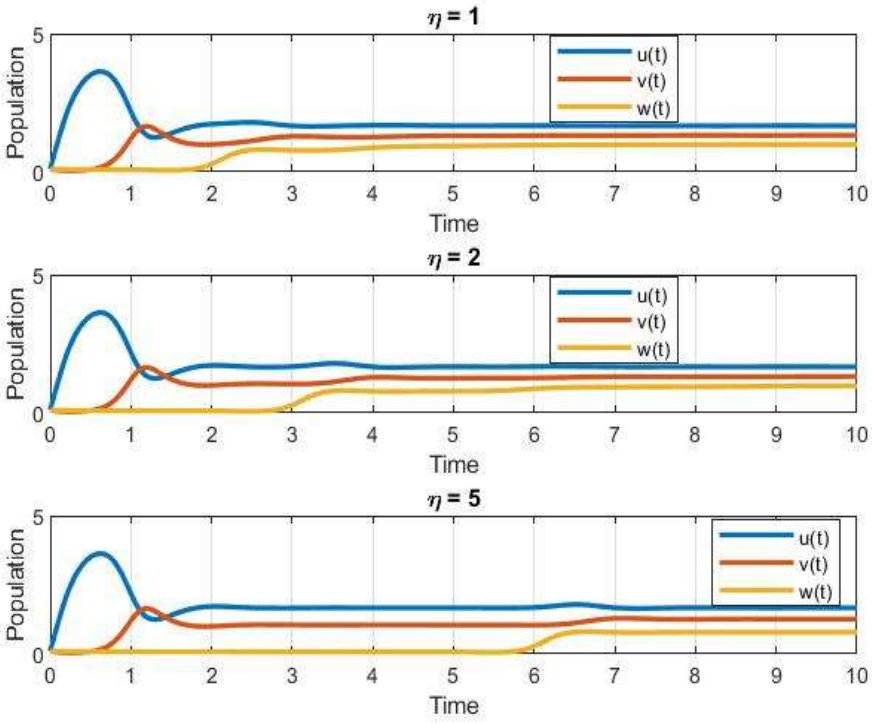

Figure 4 Solution profile of (5) for different values of η

Remark 4.1
Looking at Figures 1 to 4, we notice a clear trend-when the delays increases, the system takes longer to settle into a steady state. In simple terms, a larger delay slows down how quickly the system stabilizes. However, the system still behaves in a predictable and controlled way.This steady behavior, even in the presence of longer delays, is due to the way the model has been carefully designed. The parameters and equations were chosen to meet specific conditions that help maintain balance. So, even though the system reacts more slowly, it doesn't spiral out of control or become unstable.Instead, the system gradually moves toward stability, without showing any major swings or irregular behavior.

This tells us that the model is robust—it can handle a certain amount of delay without losing its overall balance or performance.

CONCLUSION

This study explores the long-term dynamics of an SIR model that incorporates real-world complexities through various delays—specifically in infection transmission, vaccination, treatment, and the loss of immunity over time. These delays reflect more accurate disease progression patterns, where individuals do not respond to infection or interventions instantly, and immunity may wane after recovery.By introducing these time lags into the model, we gain a clearer understanding of how diseases behave when real-world processes like delayed vaccination effects, postponed treatment responses, and gradual return to susceptibility are taken into account. The analysis reveals that, although such delays can slow down the system's movement toward equilibrium, they do not inherently lead to instability.Our findings show that, with appropriate parameter selection and well-structured nonlinear terms, the system can maintain both local and global stability even in the presence of multiple delays. Numerical simulations confirm that while the path to a steady state may become longer with increasing delays, the system still moves toward stability without dramatic oscillations or divergence.These results emphasize the importance of including biologically and socially relevant delays in epidemic models. Accounting for treatment delays, time lags in vaccination, and the gradual loss of immunity leads to a more realistic and dependable framework for understanding disease dynamics and planning effective intervention strategies.

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