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Dynamics of an Infectious Disease Model with Transmission Delay from Recovery to Susceptibility

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Abstract:

In this study, an extended SIR model with vaccination and treatment is considered by introducing a delay in the transition from the recovered class back to the susceptible class. This delay reflects the time it takes for individuals to lose immunity after recovery or vaccination. To understand the dynamics of the disease, both local and global stability of the equilibrium points are analyzed. Local stability conditions are derived using the basic reproduction number and characteristic equations, while global stability is established through Lyapunov's method. A numerical example is included to demonstrate how different values of the delay affect the system.

Keywords: characteristic, equations, equilibrium, demonstrate

1. Introduction

Infectious diseases have always been a major concern for human health, especially because of how quickly they can spread and reappear, often when we least expect it. Over the years, researchers and scientists have worked hard to understand how these diseases behave and how we might stop them [10],[11]. One of the most useful tools in this effort has been mathematical modelling, which helps us see the bigger picture by using equations to describe how diseases move through a population [6],[7],[8],[9].

Most models divide the population into groups like those who are susceptible to infection, those who are currently infected, and those who have recovered. These simple models have been very helpful in studying a range of diseases. But real life is often more complicated. One important factor that's sometimes left out is the time it takes for someone who has recovered from an illness, especially someone who hasn't been vaccinated, to become vulnerable again.

In many infections, recovery doesn't mean someone is safe forever. Their immunity might only last a few months or years, and after that, they can get sick again. This delay between recovering and becoming susceptible once more can have a big impact on how a disease spreads, especially in communities where not everyone gets vaccinated or where immunity fades over time.

By including this delay in our models, we get a much clearer understanding of how diseases can re-emerge, even after they seem to be controlled. It also helps us make better decisions about when and how to use tools like vaccination and treatment. For example, if we know there's a time lag before people become susceptible again, we can plan booster vaccinations or public health responses more effectively.

This study looks at what happens when we account for that delay, when recovered individuals slowly lose their immunity and rejoin the group of people who can catch the disease. Our goal is to use this more realistic approach to better understand the ups and downs of disease outbreaks, and to figure out how we can respond in smarter, more timely ways.

The SIR model with treatment and vaccinations, which was introduced in [1], is taken into consideration. In [3], various global stability conditions for the considered model are examined. Further, the influence of time-varying rates of vaccination and treatment is investigated in [2]. The effects of the vaccine delay are highlighted in [4]. In this article, the delay in recovering individuals becoming susceptible again is introduced, and its dynamics on the disease spread are studied.

This document has the following structure. The model is presented, and basic properties are examined in Section 2. Section 3 looks at the model's stability both locally and globally. In Section 4, the numerical examples are discussed. In section 5, the conclusion is provided.

2. Model Description

A three-compartmental, Susceptible-Infected-Recovered (SIR) model is considered. $'u'$ denotes susceptible population, $'v'$ denotes the infected population and $'w'$ denotes the recovered population. The dynamics of the model are described by the following differential equations

$$\begin{aligned} u' &= a - bf(u,v) - du - cV(u) + \alpha w(t-\mu) \\ v' &= b_1 f(u(\mathrm{t}-\tau), v) - rP(v) - d_1 v \\ w' &= rP\big(v(t-\delta)\big) - \alpha w \end{aligned} \tag{1}$$

The susceptible population growth rate is represented by $\boldsymbol{a}$, and the non-linear infection function that illustrates how susceptibles become infected is indicated by $\boldsymbol{f(u,v)}$. $\boldsymbol{b>0}$ indicates that susceptible and infected people interact, and $\boldsymbol{d}$ is the rate at which susceptible people who are inherently immune to the illness and do not become infected are removed from the system. The vaccine function $\boldsymbol{V(u)}$ is the vaccination function which depends on the susceptible population, the successful vaccination rate is $\boldsymbol{c>0}$, and the rate at which susceptible people become infected is $b_1(<b)$. The rate at which an unvaccinated or less immunity person who has recovered may be exposed to infection again and become susceptible again is indicated by the parameter $\boldsymbol{\alpha>0}$.
$\boldsymbol{P(v)}$ is the recovery (by treatment) function of the infected, $\boldsymbol{r>0}$ indicates the rate of recovery, and $\boldsymbol{d_1}$ is the infection-related mortality rate.
Time delay $\boldsymbol{\tau>0}$ indicates the time it takes for susceptible u to become infected when it comes in contact with v. Time delay $\boldsymbol{\delta>0}$ is the time taken by a person to get recovered by the treatment.
We introduce a time delay $\boldsymbol{\mu>0}$. It is the time taken by the recovered individual to become susceptible again due to a lack of immunity or because of no vaccination.

The following are the assumptions for the infection function, the recovery function and the vaccination function [1].

(i). $f(u,v)\geq 0, \forall u \; and \; \forall v$

(ii). $f(u,0) \geq 0, \forall u$

(iii). $f(0,v) \geq 0, \ \forall v$

(iv). $f(0,0) = 0,$

(v). $P(v) \geq 0, \forall v$

(vi). $P(0) = 0,$

(vii). $V(u) \geq 0, \forall u$

(viii) $V(0) = 0.$

(ii). $f(0,0) = 0.$

By following the procedure as in [4], we can prove the result on Positivity and boundedness of the solutions of the model (1) and state the result as

Theorem 2.1: All solutions of the system (1) with nonnegative initial conditions are nonnegative and bounded [4].

In the next section, we will examine the local and global stability of the model (1) at equilibria

3. Equilibria and Stability

3.1 Equilibrium

In studying how diseases spread and persist within a population through mathematical models like Model (1), we typically identify two possible long-term scenarios, known as equilibrium points. These describe the steady states a population may reach over time in response to the infection dynamics. For such models, the two main types of equilibrium are the disease-free equilibrium and the endemic equilibrium.

The disease-free equilibrium represents a state in which the infection has been eliminated from the population. In this case, there are no infected or recovered individuals. It is expressed as $(u^*, 0,0)$, where $u^* > 0$ denotes a healthy population of susceptible individuals with no presence of disease.

In contrast, the endemic equilibrium reflects a scenario in which the disease continues to exist within the population over the long term. Here, all population groups—susceptible, infected, and recovered or immune—maintain positive values. This equilibrium is denoted by (u^*, v^*, w^*), where each component is greater than zero. It implies that while some

individuals remain uninfected, a consistent number of people are carrying or recovering from the disease.

Understanding the stability of the model at equilibrium points is crucial to determining whether an infection will disappear or persist as a permanent feature of the population's health environment. They are also essential in assessing public health initiatives and establishing acceptable levels of disease control and elimination.

3.2 Basic Reproduction Number

The number of secondary infections caused by an infectious individual moving through a vulnerable community is determined by the Basic Reproduction Number (BRN), a threshold in epidemiology. It is represented by R_0.
For model (1) using the next-generation matrix method, we get the BRN as

$R_0 = \frac{b_1 f_v(u^*,0)}{rP'(0)+d_1}$.

(One can refer to [4] for the derivation of R_0)

3.3 Local Stability

Studying the local stability of the equilibrium points in the model is crucial to comprehending how a disease develops or disappears over time [14], [17]. By examining local stability around equilibrium points, we may observe how the system responds to minor changes. A point is considered stable if the system regains equilibrium following a minor disruption; if not, it may lead to further spread or decline of the infection.

First, we state the conditions for local stability at the disease-free equilibrium.

Theorem 3.1: System (1) will be unstable at disease-free equilibrium for $R_0 > 1$ and will be locally stable if
(i) $R_0 < 1$, (ii)$\alpha e^{-\lambda\mu} < bf_u(u^*,0) + d + cV'(u^*)$, (iii)$re^{-\lambda\delta}\frac{\partial P}{\partial v_\delta}(0) < \alpha$

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

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

$A_1 = -bf_u(u^*,0) - d - cV'(u^*) + \alpha e^{-\lambda\mu}$
$Q_1 = -bf_v(u^*,0)$
$B_1 = b_1 f_v(u^*,0) - r\,P'(0) - d_1 + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*,0)$
$C_1 = -\alpha + re^{-\lambda\delta}\frac{\partial P}{\partial v_\delta}(0)$.
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 $C_1 < 0$.
$A_1 < 0 \implies \alpha e^{-\lambda\mu} < bf_u(u^*, 0) + d + cV'(u^*)$,
$B_1 < 0 \implies R_0 < 1$,
$C_1 < 0 \implies re^{-\lambda\delta} \frac{\partial P}{\partial v_\delta}(0) < \alpha.$
Thus proved.

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

Theorem 3.2: System (1) is locally stable at the endemic equilibrium point (u^*, v^*, w^*), if
(i) $\alpha e^{-\lambda\mu} < bf_u(u^*, v^*) + d + cV'(u^*)$,
(ii) $b_1 f_v(u^*, v^*) + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*) < r\,P'(v^*) + d_1$,
(iii) $re^{-\lambda\delta} \frac{\partial P}{\partial v_\delta}(v^*) < \alpha.$

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

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

$A_2 = -bf_u(u^*, v^*) - d - cV'(u^*) + \alpha e^{-\lambda\mu}$
$Q_2 = -bf_v(u^*, v^*)$
$B_2 = b_1 f_v(u^*, v^*) - r\,P'(v^*) - d_1 + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*)$
$C_2 = -\alpha + re^{-\lambda\delta} \frac{\partial P}{\partial v_\delta}(v^*)$.
Clearly A_2, B_2 and $C_2$are the eigenvalues.
$A_2 < 0 \implies \alpha e^{-\lambda\mu} < bf_u(u^*, v^*) + d + cV'(u^*)$,
$B_2 < 0 \implies b_1 f_v(u^*, v^*) + b_1 e^{-\lambda\tau} f_{u_\tau}(u^*, v^*) < r\,P'(v^*) + d_1$,
$C_2 < 0 \implies re^{-\lambda\delta} \frac{\partial P}{\partial v_\delta}(v^*) < \alpha.$
For the above 3 conditions, the eigenvalues of the system will be negative, which implies the system is locally stable

Next, we examine the conditions for global stability of the model.

3.4. Global stability at endemic equilibria

In models of type (1) that describe the spread of infectious diseases, where the disease may remain present in the population over time, it's important to understand how the system behaves in the long run. This is where the idea of global stability becomes useful [5].

If an equilibrium point is said to be globally stable, it means that no matter how the disease starts—whether the number of infected people is high or low—the system will eventually move toward and settle at that same point. When this point represents an endemic

equilibrium, it tells us that the disease will continue to exist in the population at a steady level over time. Even if there are temporary changes, like sudden increases or decreases in cases, the system will return to this balance in the long run. This kind of analysis helps researchers understand whether the disease will die out or continue spreading.

We use the approach of Lyapunov stability to examine the conditions for the model (1) to be globally stable.

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

$$\begin{aligned}(u-u^*)' &= -b(f(u,v)-f(u^*,v^*)) - d(u-u^*) - c(V(u)-V(u^*)) + \\ &\quad \alpha(w(t-\mu)-w^*) \\ (v-v^*)' &= b_1(f(u(\mathrm{t}-\tau),v)-f(u^*,v^*)) - r(P(v)-P(v^*)) - d_1(v-v^*) \\ (w-w^*)' &= r(P\big(v(t-\delta)\big)-P(v^*)) - \alpha(w-w^*)\end{aligned} \quad (2)$$

To prove the result of global stability, we assume the following Lipschitz conditions on the functions f, V and P.

$$K_1|u-u^*| + K_2|v-v^*| \le |f(u,v)-f(u^*,v^*)| \le K_3|u-u^*| + K_4|v-v^*|$$

$$M_1|u-u^*| \le |V(u)-V(u^*)| \le M_2|u-u^*|$$

$$N_1|v-v^*| \le |P(v)-P(v^*)| \le N_2|v-v^*| \quad (3)$$

We state

Theorem 3.3. The system (1) is globally stable at the equilibrium point if the functions of system (1) satisfy Lipschitz conditions (3) and the parameters of the system satisfy $bK_1 + d + cM_1 > b_1K_3$ and $bK_2 + d_1 + rN_1 > b_1K_4 + rN_2$.

Proof: Let the Lyapunov function be

$$L = |u-u^*| + |v-v^*| + |w-w^*| + \alpha\int_{t-\mu}^{t}|w(s)-w^*|\,ds + b_1K_3\int_{t-\tau}^{t}|u(s)-u^*|\,ds + rN_1\int_{t-\delta}^{t}|v(s)-v^*|.$$

Then the dini derivative along the solutions of (1) equations (2) are

$$\begin{aligned}D^+L \le\ & -b|f(u,v)-f(u^*,v^*)| - d|u-u^*| - c|V(u)-V(u^*)| + \alpha|w(t-\mu)- \\ & w^*| + b_1|f(u(\mathrm{t}-\tau),v)-f(u^*,v^*)| - r|P(v)-P(v^*)| - d_1|v-v^*| + \\ & r\big|P\big(v(t-\delta)\big)-P(v^*)\big| - \alpha|w-w^*| + \alpha|w-w^*| - \alpha|w(t-\mu)-w^*| + \\ & b_1K_3|u-u^*| - b_1K_3|u(\mathrm{t}-\tau)-u^*| + rN_1|v-v^*| - rN_1|v(t-\delta)-v^*|\end{aligned}$$

Using conditions (3), we get

$$D^+ \leq -b(K_1|u-u^*| + K_2|v-\ v^*|) - d|u-u^*| - cM_1|u-u^*| + \alpha|w(t-\mu) - w^*| + b_1(K_3|u(\mathrm{t}-\tau) - u^*| + K_4|v-\ v^*|) - rN_1|v-\ v^*| - d_1|v-v^*| + rN_2|v(t-\delta) -\ v^*| - \alpha|w-w^*| + \alpha|w-w^*| - \alpha|w(t-\mu) - w^*| + b_1K_3|u-u^*| - b_1K_3|u(\mathrm{t}-\tau) - u^*| + rN_2|v-v^*| - rN_2|v(t-\delta) - v^*|$$

$$D^+ \leq (-bK_1 - d - cM_1 + b_1K_3)|u-u^*| + (-bK_2 + b_1K_4 - d_1 - rN_1 + rN_2)|v-\ v^*|$$

$$D^+ \leq -(bK_1 + d + cM_1 - b_1K_3)|u-u^*| - (bK_2 - b_1K_4 + d_1 + rN_1 - rN_2)|v-\ v^*|$$

By our assumptions on the parameter $D^+ \leq 0$.

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

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

Remark 3.1: It is clear from the previous results that the system can still achieve both local and global stability even in the presence of delays. This outcome depends on how the parameters are chosen and how the nonlinear terms are structured. By appropriately choosing parameter values and ensuring that the functional forms of the nonlinear terms satisfy specific constraints, the stability of the system can still be preserved. This shows that delays do not necessarily destabilize the system if the model is constructed carefully.

In the next section numerical examples are demonstrated to show the behavior of the model when the delay vary.

4. Numerical Examples

In this section, we present several examples of system (1) by fixing the parameters, functional forms, and the delays τ and δ. The delay μ is varied to examine how it affects the behaviour of the model. The delay differential equations are solved using MATLAB's dde23 solver, and the corresponding solutions are plotted to illustrate the dynamics.

Consider the system

$$\begin{aligned} u' &= 10 - 3f(u,v) - u - 2V(u) + 3w(t-\mu) \\ v' &= 3f(u(\mathrm{t}-\tau), v) - 1.5P(v) - 3.5v \\ w' &= 1.5P\big(v(t-\delta)\big) - 3w \end{aligned} \qquad (4)$$

(i) Letting the functional values to be $f(u,v) = uv$, $V(u) = u$, $P(v) = v$ and fixing the delays τ =0.1 and δ =0.1 the system (4) 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 (4) for various values delay μ can be seen in Figure 1.

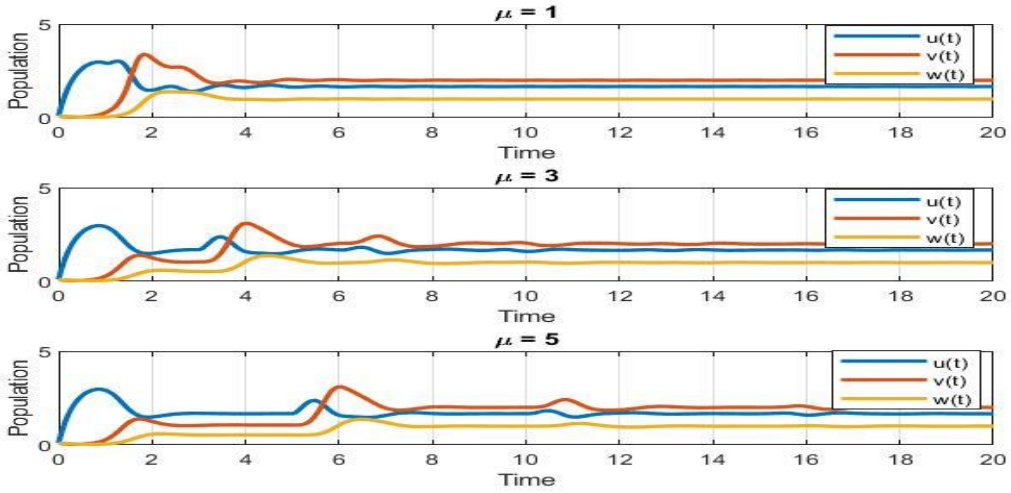

Figure 1: Solution profile of system (1) for various values of the delay μ, where the functional values are $f(u,v) = uv$, $V(u) = u$, $P(v) = v$ and fixed delays τ =0.1 and δ =0.1.

(i) For the functional values $f(u,v) = uv$, $V(u) = \frac{u}{u+1}$, $P(v) = \tanh(v)$ and fixing the delays τ =0.13 and δ =0.13 the system (4) satisfies Global stability conditions of Theorem 3.3. Thus the solutions will reach equilibrium point (1.4, 2.9, 0.5). The behaviour of the solution of the system (4) for different values of the delay μ can be seen in Figure 2.

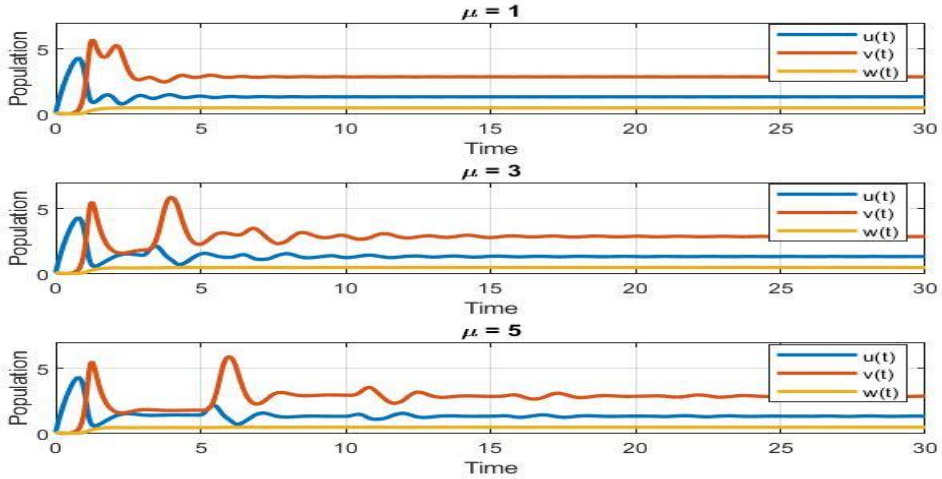

Figure 2: Solution profile of system (1) for various values of the delay μ, where the functional values are $f(u,v) = uv$, $V(u) = \frac{u}{u+1}$, $P(v) = tah(v)$ and fixed delays τ =0.13 and δ =0.13.

Remark 4.1: From Figures 1 and 2, we can see that when the delay μ\muμ becomes larger, the system takes longer to settle down to a steady state. This means the presence of delay slows down how quickly the system reaches stability. However, since the equations and parameters used in the model are chosen carefully to meet specific conditions, the system still behaves in a controlled manner. Even with increasing delay, the system doesn't drift too far or become unstable. It may take more time, but it eventually moves toward the stable point without showing large fluctuations or unexpected changes. This shows that the model is stable and can handle delays up to a certain extent without losing its overall balance.

5. Discussion

This study highlights the effect of immunity loss over time on the long-term behaviour of an SIR model that includes both vaccination and treatment. By introducing a delay in the return of recovered individuals to the susceptible group, the model reflects a more realistic scenario where immunity is not permanent. The analysis shows that, despite this delay, the system can remain stable under the right conditions. Both local and global stability are preserved when the parameters are chosen carefully. Numerical results support this by showing that while the delay may slow down the path to equilibrium, it does not disturb the system's overall stability. These results underline the importance of accounting for delays when modeling diseases where immunity may fade or vaccination is not fully effective. With appropriate assumptions, such models can still offer dependable insights into how a disease might behave over time.

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