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STABILITY ANALYSIS AND TURING INSTABILITY OF AN EPIDEMIC REACTION-DIFFUSION MODEL WITH VACCINATION, TWO AWARE CLASSES AND SATURATED TREATMENT

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Abstract. In this paper, we developed a reaction-diffusion epidemic model with two aware classes and saturated treatment considering the impact of vaccination. We divided the total population into six classes, namely susceptible, vaccinated, completely aware, partially aware, infected, and recovered class. We discussed the basic properties of our system. With the help of Dirichlet boundary conditions and positive initial data, we studied the local and global existence in time of solutions. We performed a stability analysis of the equilibrium points, their existence, and found the basic reproduction number $\mathcal{R}_0$ of the system. Then, we established the occurrence conditions of Turing instability for our diffusive model. Finally, we have provided a thorough numerical exploration of our model to illustrate our analytical results by choosing a suitable set of parameters.

1. Introduction

Infectious diseases are one of the main causes of death for millions of people every year. Therefore, mathematical modeling of infectious diseases has recently attracted the attention of many researchers to study the dynamics of transmission of various diseases and their control [3], [16]. Kermac and McKendrick [24] discovered this for the first time in the early 20th century.

Nowadays, vaccination is considered one of the most effective tools in the history of modern medicine for combating infectious diseases [11], [23]. Its primary goal is to reduce the number of susceptible individuals and to induce adaptive immunity against a specific disease. However, vaccination does not necessarily guarantee lifelong immunity, as the acquired immunity may gradually wane over time. Moreover, vaccine efficacy is not always perfect and can vary depending on the type of vaccine, the targeted disease, and the characteristics of the population receiving it. This necessitates the development of mathematical models that consider these aspects.

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Awareness plays a pivotal role in curbing the spread of infections, consequently, there has been an increasing emphasis on public awareness and the impact of information regarding the transmission of infectious diseases to mitigate infection risk [6], [8], [15]. The significance of awareness has driven many researchers to develop mathematical models for controlling disease transmission. Greenhalgh et al. [8] investigated the effects of awareness-raising programs on the dissemination of infectious diseases, demonstrating that such interventions markedly improve disease control. Goel et al. [14] proposed an epidemiological model that distinguishes between two distinct levels of awareness, complete and partial, to reflect actual behavioral dynamics more accurately, and assumed that recovered individuals acquire permanent immunity; however, Kundu et al. [22] demonstrated in a subsequent study that this assumption may not apply to certain diseases such as influenza and COVID-19. They showed that immunity may wane over time, leading recovered individuals to transition back into the susceptible category. Endale et al. [9] underscored the importance of awareness and stressed the necessity for authorities to actively educate the population about their critical role in limiting disease spread.

Treatment plays a crucial role in combating epidemics, especially with advances in medical sciences. Therefore, the treatment rate is often included as a key component in epidemiological models. Initially, researchers employed constant treatment functions to represent the effect of treatment [25], these were later developed into more realistic saturated treatment functions that account for the limitations of medical resources or the efficiency of treatment, as the number of infected individuals increases [21].

In this paper, we developed a reaction-diffusion epidemiological model, centrally incorporating the role of vaccination alongside awareness (both complete and partial) and treatment. We will analyze how the integration of vaccination into disease control strategies, in conjunction with the promotion of health awareness and the provision of effective treatment, significantly contributes to enhancing epidemic control and reducing its complications. We also highlight the critical importance of the diffusion process in our model, as it contributes to capturing the fundamental dynamics of epidemic spread.

This paper is structured as follows: Section 1 introduces the study. Section 2 presents the model formulation. Section 3 discusses some basic properties. Section 4 and section 5 prove the local existence and the global existence, respectively. Section 6 analyzes the stability of the equilibria. Section 7 studies the occurrence conditions for Turing instability. Finally, Section 8 presents numerical simulations.

2. Model formulation

In this section, we formulate the reaction-diffusion epidemic model with vaccination. To derive our system, we divide the total population into five classes: unaware susceptible $S(t)$, vaccinated $V(t)$, completely aware $A_c(t)$, partially aware $A_p(t)$, infected $I(t)$ and recovered class $R(t)$ at time t. The system can be represented as:

$$\begin{cases} \partial_t S - D_1\Delta S = f_1(S,\ V,\ A_c,\ A_p,\ I,\ R), \\ \partial_t V - D_2\Delta V = f_2(S,\ V,\ A_c,\ A_p,\ I,\ R), \\ \partial_t A_c - D_3\Delta A_c = f_3(S,\ V,\ A_c,\ A_p,\ I,\ R), \\ \partial_t A_p - D_4\Delta A_p = f_4(S,\ V,\ A_c,\ A_p,\ I,\ R), \\ \partial_t I - D_5\Delta I = f_5(S,\ V,\ A_c,\ A_p,\ I,\ R), \\ \partial_t R - D_6\Delta R = f_6(S,\ V,\ A_c,\ A_p,\ I,\ R), \end{cases} \quad in\ \mathbb{R}^+ \times \Omega, \tag{2.1}$$

where Ω is an open bounded domain in $\mathbb{R}^N$ with smooth boundary $\partial\Omega$. The notation Δ is the Laplacian operator and the positive constants $D_i,\ i = 1, 2, ..., 6$ are the diffusion coefficients.

We assume the Dirichlet boundary conditions

$$S = S_*,\ V = V_*,\ A_c = A_{c*},\ A_p = A_{p*},\ I = I_*,\ R = R_* \qquad \text{on } \mathbb{R}^+ \times \partial\Omega, \tag{2.2}$$

where S_*, V_*, A_{c*}, A_{p*}, I_*, and R_* are the coordinates of the endemic equilibrium E_*.

The bounded initial data

$$\begin{aligned} &S_0(x) = S(0,x), V_0(x) = V(0,x), A_{c0}(x) = A_c(0,x),\\ &A_{p0}(x) = A_p(0,x), I_0(x) = I(0,x), R_0(x) = R(0,x) \qquad \text{in } \Omega, \end{aligned}$$

where $S_0(x)$, $V_0(x)$, $A_{c0}(x)$, $A_{p0}(x)$, $I_0(x)$, $R_0(x) \in \mathbb{R}_{\geq 0}$. The nonlinear reaction functions f_i, $1 \leq i \leq 6$ are defined as follows

$$f_1(S,\ V,\ A_c,\ A_p,\ I,\ R) = \Lambda - \lambda_1 S - \lambda_2 S - \lambda_3 S - \frac{\beta_1 SI}{1+bI} - \mu S + \omega V,$$

$$f_2(S,\ V,\ A_c,\ A_p,\ I,\ R) = \lambda_1 S - \frac{\beta_2(1-\epsilon)VI}{1+bI} - (\mu+\omega)V,$$

$$f_3(S,\ V,\ A_c,\ A_p,\ I,\ R) = \lambda_2 S - \frac{\beta_3 A_c I}{1+bI} - \mu A_c,$$

$$f_4(S,\ V,\ A_c,\ A_p,\ I,\ R) = \lambda_3 S - \frac{\beta_4 A_p I}{1+bI} - \mu A_p,$$

$$f_5(S,\ V,\ A_c,\ A_p,\ I,\ R) = \frac{\beta_1 SI}{1+bI} + \frac{\beta_2(1-\epsilon)VI}{1+bI} + \frac{\beta_3 A_c I}{1+bI} + \frac{\beta_4 A_p I}{1+bI} - (d+\gamma+\mu)I - \varphi(I),$$

$$f_6(S,\ V,\ A_c,\ A_p,\ I,\ R) = \gamma I + \varphi(I) - \mu R.$$

The nonlinearity φ represents the saturated treatment rate, which is assumed to be a continuously differentiable function on $\mathbb{R}^+$ such that

$$\varphi(I) = \frac{\alpha I}{1+\beta I}, \qquad I \geq 0,\ \alpha,\ \beta > 0, \tag{2.3}$$

and

$$\varphi(0) = 0,\ \frac{\partial\varphi(I)}{\partial I} > 0,\ \lim_{I\to+\infty}\varphi(I) = \frac{\alpha}{\beta} \text{ for all } I > 0. \tag{2.4}$$

The model parameters are defined as:

Λ : the recruitment rate of the susceptible population,
μ : natural death rate of the population,
λ_1 : the rate at which susceptibles are vaccinated,
λ_2 : the fractions of the completely aware susceptible,
λ_3 : the fractions of the partially aware susceptible,
β_1 : the infection rate of unaware susceptible,
β_2 : the infection rate of vaccinated susceptible,
β_3 : the infection rate of completely aware susceptible,
β_4 : the infection rate of partially aware susceptible,
b : inhibition effect,
ϵ : efficacy of the vaccination ($0 \leq \epsilon \leq 1$),
ω : vaccination-induced immunity rate,
d : the natural death rate of the population,
γ : the natural recovery rate of the population,

α : cure rate,
β : impact of treatment delays on the infectious population.

3. Basic properties

Let N denotes the total population at time t, is given by

$$N(t) = S(t) + V(t) + A_c(t) + A_p(t) + I(t) + R(t),$$

biologically, we assume that all model's parameters Λ, d_1, d_2, σ_1, σ_2, σ_3, μ, δ, α, β, γ are positive constants.

System (2.1) has the following properties:

Proposition 3.1. *For the positive initial conditions, the solution of system (2.1) remain positive.*

Proof. The reactions are quasi-positive, i.e.

$$\begin{aligned}
&f_1(0,\ V,\ A_c,\ A_p,\ I,\ R) = (\Lambda + \omega V) \geq 0,\\
&f_2(S,\ 0,\ A_c,\ A_p,\ I,\ R) = \lambda_1 S \geq 0,\\
&f_3(S,\ V,\ 0,\ A_p,\ I,\ R) = \lambda_2 S \geq 0,\\
&f_4(S,\ V,\ A_c,\ 0,\ I,\ R) = \lambda_3 S \geq 0,\\
&f_5(S,\ V,\ A_c,\ A_p,\ 0,\ R) = 0,\\
&f_6(S,\ V,\ A_c,\ A_p,\ I,\ 0) = (\gamma + \tfrac{\alpha I}{1+\beta I})I \geq 0,
\end{aligned}$$

for all S, V, A_c, A_p, I, $R \geq 0$. We deduce via the maximum principle (see Smoller[12]) the preservation of the positivity of the solution. □

Proposition 3.2. *The solutions of system (2.1) are bounded.*

Proof. We start by adding the equation of system (2.1), we get

$$\begin{aligned}
\partial_t N(t) &= \partial_t(S(t) + V(t) + A_c(t) + A_p(t) + I(t) + R(t)) \\
&= \Lambda - \mu S(t) - \mu V(t) - \mu A_c(t) - \mu A_p(t) - \mu I(t) - \mu R(t) \\
&= \Lambda - \mu(S(t) + V(t) + A_c(t) + A_p(t) + I(t) + R(t)) - dI \\
&\leq \Lambda - \mu(S(t) + V(t) + A_c(t) + A_p(t) + I(t) + R(t)).
\end{aligned} \tag{3.1}$$

This implies that

$$N(t) \leq \frac{\Lambda}{\mu}(1 - e^{-\mu t}) + N(0)e^{-\mu t}.$$

Now for $N(0) \leq \frac{\Lambda}{\mu}$ implies, $N(t) < \frac{\Lambda}{\mu}$, and

$$\lim_{t\to\infty} \sup N(t) \leq \frac{\Lambda}{\mu}. \tag{3.2}$$

Thus, we obtain

$$0 < N(t) \leq \frac{\Lambda}{\mu}.$$

□

Remark. *By Propositions 3.1 and 3.2, the region*

$$\Gamma = \left\{(S,\ V,\ A_c,\ A_p,\ I,\ R) : (S,\ V,\ A_c,\ A_p,\ I,\ R) \geq 0 \ \ \textit{and} \ \ S + A_c + A_p + I + R \leq \frac{\Lambda}{\mu}\right\}$$

is a positively invariant for system (2.1).

4. Local existence of solutions

In this section, we state a local existence result of the solution for system (2.1). Throughout this study, we denote by

$$\begin{aligned} \|u\|_p^p &= \frac{1}{|\Omega|}\int_\Omega |u(x)|^p\,dx,\ 1\le p<+\infty,\\ \|u\|_\infty &= ess\sup_{x\in\Omega}|u(x)|,\\ \|u\|_{C(\overline{\Omega})} &= \max_{x\in\overline{\Omega}}|u(x)|, \end{aligned}$$

the usual norms in spaces $L^p(\Omega)$, $L^\infty(\Omega)$ and $C(\overline{\Omega})$, respectively. Since the functions f_i are continuously differentiable on $\mathbb{R}_+^6$, for all $i=1,...,6$, then for any initial data in $L^\infty(\overline{\Omega})$, it is easy to check directly their Lipschitz continuity on bounded subsets of the domain of a fractional power of the operator

$$O=-\begin{pmatrix} D_1\Delta & 0 & \dots & 0\\ 0 & D_2\Delta & \dots & 0\\ \vdots & \vdots & \ddots & \vdots\\ 0 & 0 & \dots & D_6\Delta \end{pmatrix}$$

Under these assumptions, the following local existence result is well known (see Henry[7] and Friedman[1]).

Proposition 4.1. *The system (2.1)-(2.3) admits a unique, classical local solution* $(S,\ V,\ A_c,\ A_p,\ I,\ R)$ *on* $[0,T_{\max}[\times\Omega$.

If $T_{\max}<\infty$ *then*

$$\lim_{t\nearrow T_{\max}}\left\{\ \|S(t,.)\|_\infty+\|V(t,.)\|_\infty\|A_c(t,.)\|_\infty+\|A_p(t,.)\|_\infty+\|I(t,.)\|_\infty+\|R(t,.)\|_\infty\right\}=\infty,$$

where $T_{\max}$ *denotes the eventual blow-up time.*

5. Global existence of solutions

In this section, our objective is to study the global existence of solutions for reaction-diffusion system (2.1), with the help of Dirichlet boundary condition (2.2) and positive initial data (2.3).To prove this, we have applied the old general method of J. Morgan [13] for m-components systems on the form (2.1) which we summarize in our case (i.e. $m=6$) as follows

Lemma 5.1. *We suppose that the functions* f_j, $1\le j\le 6$ *are of polynomial growth and satisfy*

$$\sum_{j=1}^{6}\alpha_{ij}f_j(S;V;A_c;A_p;I;R)\le E_i,\ i=1,...,6. \tag{5.1}$$

Where $E_i=c_{i1}S+c_{i2}V+c_{i3}A_c+c_{i4}A_p+c_{i5}I+c_{i6}R+k_i$,

$\forall\ S,\ V,\ A_c,\ A_p,\ I,\ R\ge 0$, *and where* α_{ij}, c_{ij}, *and* k_i, $1\le i;j\le 6$ *are positive reals. As a result all solution of system (2.1) are positive and global in time (i.e.* $T_{\max}=+\infty$*) if they satisfies the conditions (2.2) and (2.3).*

It is seen that the system (2.1) satisfies all the conditions as stated in lemma 5.1 except the condition (5.1), which can be verified, using the positivity of the solutions for all times, as follows

$$\begin{cases} f_1(S;V;A_c;A_p;I;R) \leq (-\lambda_1-\lambda_2-\lambda_3-\mu)S+\omega V+\Lambda; \\ (f_1+f_2)(S;V;A_c;A_p;I;R) \leq (-\lambda_2-\lambda_3-\mu)S-\mu V+\Lambda; \\ (f_1+f_2+f_3)(S;V;A_c;A_p;I;R) \leq (-d_3-\mu)S-\mu V-\mu A_c+\Lambda; \\ (f_1+f_2+f_3+f_4)(S;V;A_c;A_p;I;R) \leq -\mu S-\mu V-\mu A_c-\mu A_p+\Lambda; \\ (f_1+f_2+f_3+f_4+f_5)(S;V;A_c;A_p;I;R) \leq -\mu S-\mu V-\mu A_c-\mu A_p-(d+\gamma+\mu)I+\Lambda; \\ (f_1+f_2+f_3+f_4+f_5+f_6)(S;V;A_c;A_p;I;R) \leq -\mu S-\mu V-\mu A_c-\mu A_p-(d+\mu)I-\mu R+\Lambda, \end{cases}$$

the following proposition establishes that solutions of system (2.1)-2.3 exists globally in time.

Proposition 5.2. *The system (2.1) has a non-negative, global solution in time if it satisfies the conditions (2.2) and (2.3).*

6. Stability analysis

In this section, we aim to show the existence of equilibrium solutions for system (2.1) and we provide the value of the basic reproductive number. Then, we study the stability properties of the equilibrium solutions of our system (2.1).

6.1. **Existence of model's equilibrium solutions.** In order to find equilibriums for system (2.1), we solve the following algebraic system

$$\begin{cases} \Lambda-\lambda_1 S-\lambda_2 S-\lambda_3 S-\frac{\beta_1 SI}{1+bI}-\mu S+\omega V=0, \\ \lambda_1 S-\frac{\beta_2(1-\epsilon)VI}{1+bI}-(\mu+\omega)V=0, \\ \lambda_2 S-\frac{\beta_3 A_c I}{1+bI}-\mu A_c=0, \\ \lambda_3 S-\frac{\beta_4 A_p I}{1+bI}-\mu A_p=0, \\ \frac{\beta_1 SI}{1+bI}+\frac{\beta_2(1-\epsilon)VI}{1+bI}+\frac{\beta_3 A_c I}{1+bI}+\frac{\beta_4 A_p I}{1+bI}-(d+\gamma+\mu)I-\varphi(I)=0, \\ \gamma I+\varphi.(I)-\mu R=0. \end{cases} \tag{6.1}$$

We obtain the following equilibrium solution

6.1.1. *Disease-free equilibrium (E_0).* At disease-free equilibrium

$$I=0, \text{ and } R=0,$$

hence

$$\begin{aligned} E_0 &= (S_0,V_0,A_{c0},A_{p0},I_0,R_0) \\ &= (\frac{\Lambda}{\mu+\lambda_1+\lambda_2+\lambda_3-\omega\frac{\lambda_1}{\mu+\omega}}, \frac{\lambda_1}{\mu+\omega}S_0, \frac{\lambda_2}{\mu}S_0, \frac{\lambda_3}{\mu}S_0, 0, 0). \end{aligned} \tag{6.2}$$

6.1.2. *Endemic equilibrium (E_*).* At endemic equilibrium

$$S\neq 0,\ V\neq 0,\ A_c\neq 0,\ A_p\neq 0,\ I\neq 0,\ R\neq 0,$$

hence

$$E_*=(S_*,V_*A_{c*},A_{p*},I_*,R_*), \tag{6.3}$$

where

$$\begin{array}{l} S_* = (\Lambda + \omega V_*)/(\lambda_1 + \lambda_2 + \lambda_3 + \mu + \frac{\beta_1 I_*}{1+bI_*}), \\ V_* = (\lambda_1 S_*)/(\mu + \omega + \frac{\beta_2(1-\epsilon)I_*}{1+bI_*}), \\ A_{c*} = (\lambda_2 S_*)/(\mu + \frac{\beta_3 I_*}{1+bI_*}), \\ A_{p*} = (\lambda_3 S_*)/(\mu + \frac{\beta_4 I_*}{1+bI_*}), \\ R_* = \frac{1}{\mu}(\varphi(I_*) + \gamma I_*), \end{array} \tag{6.4}$$

where I_* is the positive solution of the following equation

$$AI_*^2 + BI_* + C = 0, \tag{6.5}$$

with

$$\begin{array}{lcl} A & = & b\beta(d+\gamma+\mu), \\ B & = & \beta(d+\gamma+\mu) + b(d+\gamma+\mu) + \alpha b - \beta(\beta_1 S_* + \beta_2 V_*(1-\epsilon) + \beta_3 A_{c*} + \beta_4 A_{p*}), \\ C & = & (d+\gamma+\mu) + \alpha - (\beta_1 S_* + \beta_2 V_*(1-\epsilon) + \beta_3 A_{c*} + \beta_4 A_{p*}). \end{array}$$

6.2. **Basic reproductive number.** We define the basic reproductive number $\mathcal{R}_0$ of our diffusive model (2.1) by using the next generation matrix method [17], which is given by

$$\mathcal{R}_0 = \frac{\Lambda(\beta_1 + \beta_2(1-\epsilon)\frac{\lambda_1}{\mu+\omega} + \beta_3\frac{\lambda_2}{\mu} + \beta_4\frac{\lambda_3}{\mu})}{(d+\gamma+\mu)(\mu+\lambda_1+\lambda_2+\lambda_3-\omega\frac{\lambda_1}{\mu+\omega})}. \tag{6.6}$$

Proof. To calculate $(\mathcal{R}_0)$, we involve only the infected compartment I from our model. Thus, let $\mathcal{F} = \begin{pmatrix} \frac{\beta_1 SI}{1+bI} + \frac{\beta_2(1-\epsilon)VI}{1+\epsilon I} + \frac{\beta_3 A_c I}{1+bI} + \frac{\beta_4 A_p I}{1+bI} \\ 0 \end{pmatrix}$, $\mathcal{V} = \begin{pmatrix} (d+\gamma+\mu)I + \varphi(I) \\ 0 \end{pmatrix}$. The Jacobian matrix of $\mathcal{F}$ and $\mathcal{V}$ at E_0 with respect to the infected compartment I, are

$$F = \begin{pmatrix} \beta_1 S_0 + \beta_2(1-\epsilon)V_0 + \beta_3 A_{c_0} + \beta_4 A_{p_0} \\ 0 \end{pmatrix}, \; V = \begin{pmatrix} d+\gamma+\mu \\ 0 \end{pmatrix}.$$

The basic reproductive number $\mathcal{R}_0$ is defined as the spectral radius of G, denoted by $\rho(G)$, given as

$$\mathcal{R}_0 = \frac{\beta_1 S_0 + \beta_2(1-\epsilon)V_0 + \beta_3 A_{c_0} + \beta_4 A_{p_0}}{d+\gamma+\mu}. \tag{6.7}$$

Let us substitute V_0, A_{c_0} and A_{p_0} in (6.7), then we have,

$$\mathcal{R}_0 = \frac{\beta_1 S_0 + \beta_2(1-\epsilon)\frac{\lambda_1}{\mu+\omega}S_0 + \beta_3\frac{\lambda_2}{\mu}S_0 + \beta_4\frac{\lambda_3}{\mu}S_0}{d+\gamma+\mu}.$$

Thus, with a direct computation, we obtain (6.6). □

6.3. **Stability of the equilibrium solutions.** In this section, we move to study the local stability of the equilibria of system (2.1) as described in the following theorem.

Theorem 6.1. *The disease-free equilibrium E_0 of system (2.1) is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. To prove the local stability of the disease-free equilibrium, we must examine if all the eigenvalues of the linearizing operator have negative real parts, then the solution is locally asymptotically stable. □

We define the linearizing operator

$$L(E_0) = \begin{pmatrix} D_1\Delta - H_1 & \omega & 0 & 0 & -\beta_1 S_0 & 0 \\ \lambda_1 & D_2\Delta - H_2 & 0 & 0 & -\beta_2(1-\epsilon)\frac{\lambda_1}{\mu+\omega} S_0 & 0 \\ \lambda_2 & 0 & D_3\Delta - H_3 & 0 & -\beta_3\frac{\lambda_2}{\mu} S_0 & 0 \\ \lambda_3 & 0 & 0 & D_4\Delta - H_4 & -\beta_4\frac{\lambda_3}{\mu} S_0 & 0 \\ 0 & 0 & 0 & 0 & D_5\Delta + H_5 & 0 \\ 0 & 0 & 0 & 0 & \gamma + \alpha & D_6\Delta - H_6 \end{pmatrix},$$

where

$$H_1 = \lambda_1 + \lambda_2 + \lambda_3 + \mu,\ H_2 = \mu + \omega,\ H_3 = \mu,\ H_4 = \mu$$

$$H_5 = S_0(\beta_1 + \beta_2(1-\epsilon)\frac{\lambda_1}{\mu+\omega} + \beta_3\frac{\lambda_2}{\mu} + \beta_4\frac{\lambda_3}{\mu}) - (d + \gamma + \mu) - \alpha,\ H_6 = \mu.$$

To determine the local stability of E_0, we need to examine the eigenvalues of the Jacobian. The corresponding Jacobian matrix is

$$J_i(E_0) = \begin{pmatrix} -D_1\lambda_i - H_1 & \omega & 0 & 0 & -\beta_1 S_0 & 0 \\ \lambda_1 & -D_2\lambda_i - H_2 & 0 & 0 & -\beta_2(1-\epsilon)\frac{\lambda_1}{\mu+\omega} S_0 & 0 \\ \lambda_2 & 0 & -D_3\lambda_i - H_3 & 0 & -\beta_3\frac{\lambda_2}{\mu} S_0 & 0 \\ \lambda_3 & 0 & 0 & -D_4\lambda_i - H_4 & -\beta_4\frac{\lambda_3}{\mu} S_0 & 0 \\ 0 & 0 & 0 & 0 & -D_5\lambda_i + H_5 & 0 \\ 0 & 0 & 0 & 0 & \gamma + \alpha & -D_6\Delta - H_6 \end{pmatrix},$$

we obtain for all $i \geq 0$

$$\begin{cases} r_{i1} = -D_1\lambda_i - H_1, \\ r_{i2} = -D_2\lambda_i - H_2, \\ r_{i3} = -D_3\lambda_i - H_3, \\ r_{i4} = -D_4\lambda_i - H_4, \\ r_{i5} = -D_5\lambda_i + H_5, \\ r_{i6} = -D_6\Delta - H_5. \end{cases}$$

It can be seen that r_{i1}, r_{i2}, r_{i3}, r_{i4}, r_{i5} and r_{i6} have negative real parts, which implies that E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$.

For the stability of the positive equilibrium, E_* will be analyzed in the next section.

7. Turing instability (DDI)

In this section, we shall study the Turing instability for our diffusive model.

Turing instability, or diffusion-driven instability (DDI), is first introduced by Turing [5]. This concept is defined as follows.

Definition 7.1. *The reaction-diffusion system*

$$\partial_t u = D\Delta u + f(u),$$

exhibits Turing instability (DDI), if the system is without diffusion

$$\partial_t u = f(u),$$

has a locally stable equilibrium state, then the equilibrium is locally unstable in the presence of diffusion [4].

The function f (we assume it is regular) describes the reaction dynamics, D is a diagonal matrix of diffusion coefficients, and the boundary conditions are supposed of Neumann and homogenous.

According to the steps of Qian and Murray [10], we shall try to study the sufficient conditions for DDI.

Proposition 7.2. *The sufficient condition for DDI is that either*

(i) the largest diagonal element of $\mathbf{J}_{E_*}$ is greater than zero ($\mathbf{j}_{rr} > 0$) with corresponding diffusion coefficient very small ($D_{rr} \ll 1$); or

(ii) the smallest diagonal cofactor of $\mathbf{J}_{E_*}$ is less than zero ($cof(\mathbf{J}_{E_*})_{ss} < 0$) with corresponding diffusion very large ($D_{ss} \gg 1$).

For the small diffusions, we apply Proposition (7.2) to system (2.1) with Neumann homogenous boundary conditions.

The Jacobian matrix of system (2.1) at the endemic equilibrium $E_*(S_*,\ A_{c*},\ A_{p*},\ I_*,\ R_*)$ is given by

$$\mathbf{J}_{E_*} = \begin{pmatrix} -(\lambda_1+\lambda_2+\lambda_3+\mu) - \frac{\beta_1 I_*}{1+bI_*} & \omega & 0 & 0 & -\frac{\beta_1 S_*}{(1+bI_*)^2} & 0 \\ \lambda_1 & -(\mu+\omega) - \frac{\beta_2(1-\epsilon)I_*}{1+bI_*} & 0 & 0 & -\frac{\beta_2(1-\epsilon)V_*}{(1+bI_*)^2} & 0 \\ \lambda_2 & 0 & -(\frac{\beta_3 I_*}{1+bI_*}+\mu) & 0 & -\frac{\beta_3 A_{c*}}{(1+bI_*)^2} & 0 \\ \lambda_3 & 0 & 0 & -(\frac{\beta_4 I_*}{1+bI_*}+\mu) & -\frac{\beta_4 A_{p*}}{(1+bI_*)^2} & 0 \\ \frac{\beta_1 I_*}{1+bI_*} & \frac{\beta_2(1-\epsilon)I_*}{1+bI_*} & \frac{\beta_3 I_*}{1+bI_*} & \frac{\beta_4 I_*}{1+bI_*} & \mathbf{j}_{55} & 0 \\ 0 & 0 & 0 & 0 & \gamma + \frac{\alpha}{(1+\beta I_*)^2} & -\mu \end{pmatrix},$$

where

$$\mathbf{j}_{55} = \frac{\beta_1 S_* + \beta_2(1-\epsilon)V_* + \beta_3 A_{c*} + \beta_4 A_{p*}}{(1+bI_*)^2} - (d+\gamma+\mu) - \frac{\alpha}{(1+\beta I_*)^2},$$

is the largest diagonal element of $\mathbf{J}_{E_*}$, which is positive under the following condition

$$\beta_1 S_* + \beta_2(1-\epsilon)V_* + \beta_3 A_{c*} + \beta_4 A_{p*} > (1+bI_*)^2 \left[(d+\gamma+\mu) + \frac{\alpha}{(1+\beta I_*)^2}\right]. \quad (7.1)$$

Proposition 7.2 gives us the following theorem.

Theorem 7.3. *For a sufficiently small diffusion D_5 , (7.1) provides a sufficient condition for diffusion-driven instability for the reaction-diffusion system (2.1).*

For the large diffusion, simple calculation reveals that if the cofactors of the first diagonal elements of $\mathbf{J}_{E_*}$ are less than zero under the condition that follows

$$\begin{pmatrix} -(\mu+\omega) - \frac{\beta_2(1-\epsilon)I_*}{1+bI_*} & 0 & 0 & -\frac{\beta_2(1-\epsilon)V_*}{(1+bI_*)^2} & 0 \\ 0 & -(\frac{\beta_3 I_*}{1+bI_*}+\mu) & 0 & -\frac{\beta_3 A_{c*}}{(1+bI_*)^2} & 0 \\ 0 & 0 & -(\frac{\beta_4 I_*}{1+bI_*}+\mu) & -\frac{\beta_4 A_{p*}}{(1+bI_*)^2} & 0 \\ \frac{\beta_2(1-\epsilon)I_*}{1+bI_*} & \frac{\beta_3 I_*}{1+bI_*} & \frac{\beta_4 I_*}{1+bI_*} & \mathbf{j}_{55} & 0 \\ 0 & 0 & 0 & \gamma + \frac{\alpha}{(1+\beta I_*)^2} & -\mu \end{pmatrix} < 0,$$

i.e.,

$$\mu\left(\frac{\beta_3 I_*}{1+bI}+\mu\right)\left(\frac{\beta_4 I_*}{1+bI}+\mu\right)\left[\left((\mu+\omega)+\frac{\beta_2(1-\epsilon)I_*}{1+bI}\right)\times \mathbf{j}_{55}+\frac{\beta_2^2(1-\epsilon)^2 V_* I_*}{(1+bI_*)^3}\right]<0. \tag{7.2}$$

Applying proposition 7.2 , we have the following theorem

Theorem 7.4. *A sufficient condition for diffusion-driven instability for the reaction-diffusion system (2.1) is given by (7.2) for sufficiently large diffusion D_1.*

Remark. *By Theorem 7.3 and 7.4 the positive equilibrium E^* of system (2.1)) is locally unstable. This implies that the reaction-diffusion system (2.1) shows Turing instability.*

8. NUMERICAL SIMULATION

In this section, numerical experiments are performed and presented in two examples to understand the dynamical aspect of the proposed model. We have used MATLAB R2020a to perform numerical simulations of the model system (2.1). Table 1 states the parameter values selected for the examples.

Parameter values	Example 1	Example 2
Λ	5	1
λ_1	0.02	0.5
λ_2	0.02	0.2
λ_3	0.1	0.01
β_1	0.03	0.02
β_2	0.005	0.1
β_3	0.001	0.005
β_4	0.002	0.009
ϵ	0.7	0.8
ω	0.01	0.05
μ	0.03	0.01
b	0.1	0.05
d	0.01	0.008
γ	0.05	0.005
α	0.05	0.03
β	0.1	0.09

TABLE 1. Parameter values for Examples 1 and 2.

8.1. **First Example.** In this first example, we use the parameter values from the first column of Table 1.

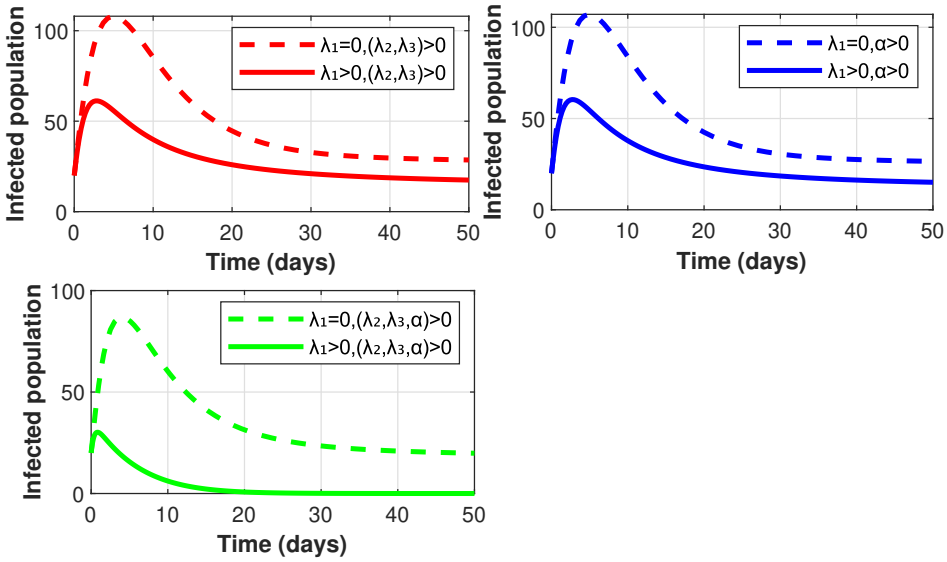

FIGURE 1. Impact of vaccination on infected individuals in the presence of two aware classes and saturated treatment.

Figure 1 demonstrates the impact of incorporating vaccination into awareness and treatment strategies for infected individuals. In the case relying solely on awareness (completely and partially aware classes) and treatment, the number of infected individuals rises sharply to a high peak before gradually declining, indicating that these interventions alone are insufficient to effectively curb the spread of the disease. In contrast, when vaccination is introduced alongside awareness and treatment, a significant reduction in both the peak and duration of infections is observed. This demonstrates that vaccination substantially enhances the effectiveness of epidemic control by reducing the transmission rate, thereby emphasizing the importance of integrating vaccination programs into public health policies in conjunction with awareness and treatment efforts.

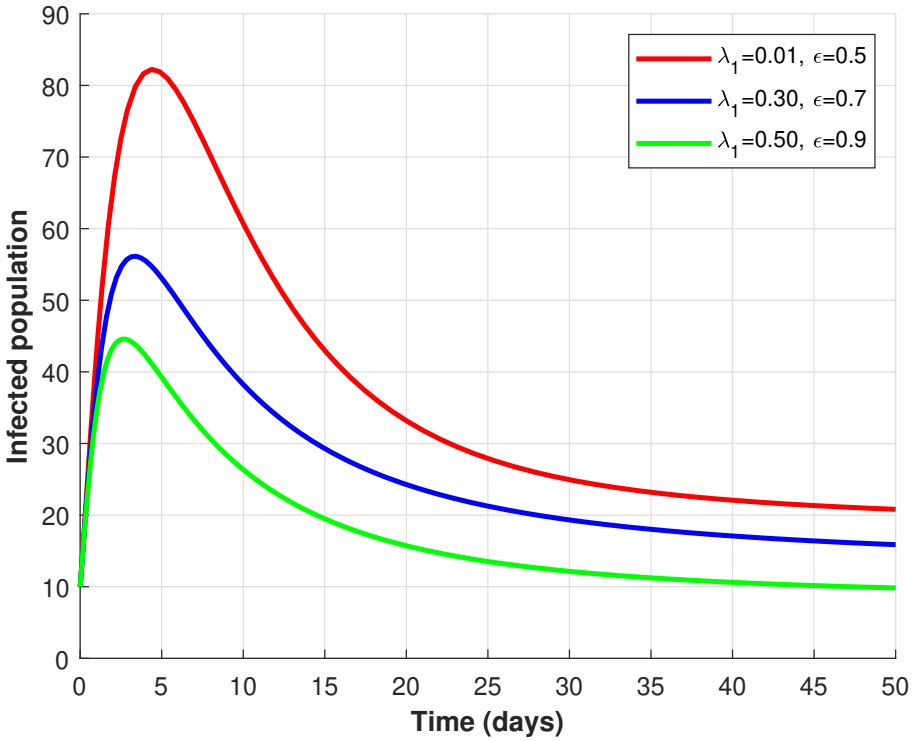

FIGURE 2. Impact of vaccination rate and vaccine efficacy on the infected population

Figure 2 shows three curves, each representing a different scenario based on vaccination rate and vaccine efficacy. The number of infected individuals decreases as both the vaccination rate and vaccine efficacy increase. This demonstrates the importance of vaccination in limiting the spread of the disease, especially when a highly effective vaccine is used in conjunction with a high vaccination rate.

8.2. **Second Example.** This example aims to explore the occurrence of Turing instability for large diffusion. Taking $D_1 = 10$, $D_2 = 1.5$, $D_3 = 1.4$, $D_4 = 0.2$, $D_5 = 0.01$, $D_6 = 0.5$. We use the parameter values from the second column of Table 1, the endemic equilibrium of the model system (2.1) at these parameter values is $E_*(1.103, 1.701, 2.901, 0.086, 38.841, 24.295)$. Thus, the inequality of Theorem 7.4 is satisfied.

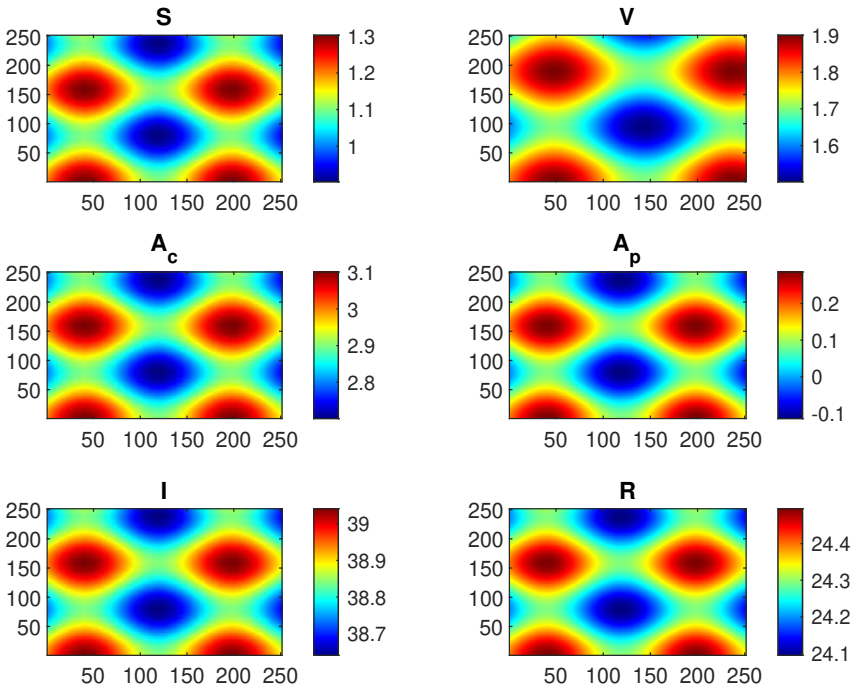

FIGURE 3. Turing Patterns of model (2.1) at parameter values from the second column of Table 1, for compartments S, V, A_c, A_p, I, and R

Figure 3 displays the evolution of Turing Patterns of all population compartments of model (2.1) for relatively larger values of diffusion $D_1 = 1.5$, which is discussed in Theorem 7.4

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