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Hybrid Reaction–Diffusion Epidemic Models: Dynamics and Emergence of Oscillations

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ABSTRACT

In this paper, we construct a hybrid epidemic mathematical model based on a reaction–diffusion system of the SIR (susceptible-infected-recovered) type. This model integrates the impact of random factors on the transmission rate of infectious diseases, represented by a probabilistic process acting at discrete time steps. The hybrid model couples a continuous reaction–diffusion system, which describes the spatiotemporal dynamics of the infectious disease, with a discrete probabilistic process that models potential change in the transmission rate. We establish properties of both biological and mathematical interest in the hybrid model, including the existence of global solutions, stability analysis of equilibrium points, and the emergence of oscillatory behaviors. Additionally, we extend the hybrid model by including vaccination. The dynamics and emergence of oscillations in the hybrid model are investigated under various scenarios, which are illustrated through numerical simulations.

1 | Introduction

According to the centers for disease control and prevention (CDC), an epidemic is defined as a sudden increase in the number of cases of a disease above what is normally expected in a population of a specific area. An outbreak shares the same definition as an epidemic but is often used to describe a more geographically limited event. On the other hand, a pandemic refers to an epidemic that has spread across multiple countries or continents, usually affecting a large number of people [1]. Epidemics can refer to diseases or other health-related behaviors, such as smoking or excessive drinking, when their rates significantly exceed the expected occurrence in a community or region [2]. The primary aim of epidemiologists is to first understand the causes of a disease, then predict its progression, and ultimately develop strategies to control it, including comparisons of various potential approaches [3].

Mathematical models of infectious disease transmission have become increasingly important in guiding public health policy [4]. Since Bernoulli developed the first mathematical model of infectious disease transmission in 1760 to assess the effects of variolation [5], numerous additional models have been proposed. These models play a vital role in interpreting data, formulating hypotheses, designing experiments for testing, diagnosing based on observed signs and symptoms, and supporting decision-making processes [4].

In compartmental models, a population is divided into compartments, with each compartment representing individuals based on their current state in the epidemic. When a system of ordinary differential equations is used to analyze the disease's progression over time, these models are referred to as *deterministic*. The foundations of compartmental epidemiological models were established by public health physicians such as Sir R. A. Ross, W. H. Hamer, A. G. McKendrick, and W. O. Kermack between 1900 and 1935 [3]. The basic compartmental models used to describe the transmission of communicable diseases are presented in a series of three papers by Kermack and McKendrick: the first in 1927 [6], followed by subsequent works in 1932 [7] and 1933 [8]. The Kermack–McKendrick theory laid the foundation for the SIR (susceptible-infectious-recovered) models and their variants. The compartmental representations are as follows: susceptible individuals (S) are those who have never been infected and are susceptible to the disease; infected individuals (I) are those currently infected and capable of transmitting the disease; and recovered individuals (R) are those who have recovered and developed immunity. The SIR models have been extended to study a wide range of infectious diseases (see, e.g., [9–14]).

A general SIR model with vital dynamics (i.e., births and deaths), studied by authors such as [15–17], is described by the following system of equations:

$$\begin{cases} \frac{dS}{dt} = \mu N - \mu S - \beta SI, \quad S(0) = S_0 \geq 0\,, \\ \frac{dI}{dt} = \beta SI - \nu I - \mu I, \quad I(0) = I_0 \geq 0\,, \\ \frac{dR}{dt} = \nu I - \mu R, \quad R(0) = R_0 \geq 0\,, \end{cases} \tag{1}$$

with $S(t) + I(t) + R(t) = N$, at time t, and N represents the total population. The inflow of newborns into susceptible class occurs at a rate μN, and the deaths in the three compartments are μS, μI, and μR, respectively. The deaths balance the births, keeping the total population N constant [15].

In model (1), the standard incidence term $\beta IS/N$ is used, where β, often referred to as the *transmission coefficient*, represents the average number of sufficient contacts for transmission per person per unit of time. The expression $\beta IS/N$ represents the number of new cases per unit of time; see, for example, [15] and references cited therein.

In compartmental models, variations in the transmission rate (β) play a crucial role in determining the spread of the disease. Temporal fluctuations in the transmission rate can significantly affect disease propagation; for methods to infer time-varying transmission rates, see, for example, [18, 19]. A high transmission rate leads to rapid disease spread, increasing the likelihood of outbreaks and epidemics throughout the population. Conversely, a low transmission rate results in slower disease spread, which may limit the disease to isolated outbreaks or containment within specific regions.

Many researchers have studied SIR models incorporating random perturbations in the transmission rate to account for the significant impact of environmental fluctuations. For further information on such models, see, for example, [20–25] and references cited therein.

Deterministic SIR-type compartmental models have been extended to reaction–diffusion equations to estimate the asymptotic rate of spatial spread; see, for example, [11, 26] and references therein. Reaction–diffusion compartmental models account for the spatial structure of disease propagation and the mobility of individuals. Additionally, the inclusion of diffusion terms allows for the examination of how spatial heterogeneity can influence disease dynamics [26]. Reaction–diffusion SIR-type models have been both analytically and numerically studied; see, for instance, [27–33]. Various studies have also investigate the role of vaccination in controlling the spread of infectious diseases [30, 34–36].

Hybrid models in epidemiology provide a robust framework for epidemic modeling by combining deterministic and probabilistic approaches, offering comprehensive insights into disease dynamics and control strategies. By addressing complexity, uncertainty, and heterogeneity, these models enhance our understanding of infectious disease transmission and support evidence-based decision-making in public health interventions [37–40]. Recent studies have explored epidemic hybrid models, including abstract hybrid models with applications to the COVID-19 pandemic; see, for example, [41, 42].

In this paper, our aim is to enhance the integration of transmission rate fluctuations within a continuous spatiotemporal model, described by a reaction–diffusion system. A similar conceptual approach was proposed in [43], where a continuous reaction–diffusion system representing the spatiotemporal dynamics of a forest ecosystem was coupled with a discrete probabilistic process to model the occurrence of extreme events. Here, we propose a novel SIR-type hybrid model that combines deterministic and probabilistic components. The deterministic part of the hybrid model is given by a continuous spatiotemporal SIR reaction–diffusion system, previously studied in [28], while the probabilistic component is represented by a discrete probabilistic process that accounts for fluctuations in the transmission rate. In this probabilistic process, the timing of transmission rate changes is determined by discrete random variables. Furthermore, the frequency and intensity of these changes are incorporated into the model as free parameters, allowing for the exploration of different scenarios. In this way, our hybrid model thus operates by coupling two spatial scales: a regional scale for the transmission dynamics of the SIR model and a local scale for the variations in the transmission rate. It also integrates two modeling formalisms: a continuous-deterministic formalism for population dynamics and a discrete–probabilistic formalism for transmission rate changes. Furthermore, we extend the reaction–diffusion SIR model by incorporating a vaccination parameter. To the best of our knowledge, this multiscale, multiformalism SIR-type parametric model has not been previously considered.

This paper is structured as follows. Section 2 presents a detailed formulation of our hybrid model and establishes its well-posedness at a theoretical level. In Section 3, we provide sufficient conditions on the intensity of transmission rate changes to ensure the stability of the disease-free equilibrium, both with and without the inclusion of a vaccination coverage parameter. Section 4 features several numerical experiments designed to illustrate the theoretical stability results.

2 | Continuous-Deterministic and Discrete–Probabilistic SIR Hybrid Model

In this section, we describe the construction of a hybrid model that captures the complex evolution of an epidemic. Initially, the mechanisms of birth, mortality, infection transmission, and spatial diffusion are modeled using a reaction–diffusion system of the SIR type. We then incorporate the effects of various factors that influence the transmission rate of infectious diseases–such as ecological changes, air quality, climate, human behavior, demographics, and virus mutations, which can occur randomly (see, e.g., [44–46])–by coupling the reaction–diffusion system with a probabilistic process operating at discrete time intervals.

2.1 | Spatiotemporal SIR Model

Let $\Omega \subset \mathbb{R}^m$ denote a bounded domain with a smooth boundary $\partial\Omega$ and $m \in \{1, 2\}$. We assume that a population of individuals distributed within Ω is affected by an epidemic. This population is divided into three subgroups: susceptible individuals S, infected individuals I, and recovered individuals R.

Following the works of [26, 30, 36, 47] and [48], we describe the epidemic's progression using the following reaction–diffusion system:

$$\begin{cases} \frac{\partial S(x,t)}{\partial t} = d_1 \Delta S(x,t) + \mu N(t) - \mu S(x,t) - \beta S(x,t) I(x,t), & x \in \Omega, \quad t > 0, \\ \frac{\partial I(x,t)}{\partial t} = d_2 \Delta I(x,t) - (\mu + \nu) I(x,t) + \beta S(x,t) I(x,t), & x \in \Omega, \quad t > 0, \\ \frac{\partial R(x,t)}{\partial t} = d_3 \Delta R(x,t) - \nu I(x,t) + \mu R(x,t), & x \in \Omega, \quad t > 0. \end{cases} \tag{2}$$

Here, x and t represent the spatial and temporal variables, respectively. For each $x \in \Omega$ and $t \geq 0$, the functions $S(x,t)$, $I(x,t)$, and $R(x,t)$ denote the number of susceptible, infected, and recovered individuals at position x and time t, respectively.

The parameters d_1, d_2, d_3, μ, β, and ν are positive coefficients. Specifically, d_1, d_2, and d_3 represent the diffusion rates of susceptible, infected, and recovered individuals, respectively. We assume that the birth and death rates are equal and that newborns are susceptible to the disease, having no inherent immunity. Therefore, both the natural birth and death rates are denoted by the parameter μ. The recovery rate from infected to recovered individuals is represented by the parameter ν, and the coefficient β models the transmission rate from susceptible to infected individuals. Finally, the total population

size at time t, denoted by $N(t)$, is given by the following expression:

$$N(t) = \int_{\Omega} [S(x,t) + I(x,t) + R(x,t)]dx \ .$$

The reaction–diffusion system (2) is supplemented by the following initial conditions at $t = 0$

$$S(x,0) = S_0(x) \geq 0, \quad I(x,0) = I_0(x) \geq 0, \quad R(x,0) = R_0(x) \geq 0, \quad x \in \Omega, \tag{3}$$

and by the homogeneous Neumann boundary condition:

$$\frac{\partial S(\xi,t)}{\partial \upsilon} = \frac{\partial I(\xi,t)}{\partial \upsilon} = \frac{\partial R(\xi,t)}{\partial \upsilon} = 0, \qquad \xi \in \partial\Omega, \quad t > 0, \tag{4}$$

where υ denotes the outward unit normal vector on the boundary $\partial\Omega$ of Ω. The Neumann boundary condition (4) implies that no infection occurs across the boundary.

Remark 1. We briefly show that $N(t)$ is constant over time. Let the total number of individuals in Ω at $t = 0$ de defined as follows:

$$N_0 = \int_{\Omega} [S(x,0) + I(x,0) + R(x,0)]dx \ .$$

By summing the equations in system (2) and integrating over Ω, we obtain the following:

$$\frac{\partial}{\partial t} \int_{\Omega} [S(x,t) + I(x,t) + R(x,t)]dx = \int_{\Omega} d_1 \Delta S(x,t) + d_2 \Delta I(x,t) + d_3 \Delta R(x,t)dx \ .$$

Using Green's formula and applying the Neumann boundary condition (4), we conclude that

$$\frac{\partial}{\partial t} \int_{\Omega} [S(x,t) + I(x,t) + R(x,t)]dx = 0 \ .$$

This implies that $N(t)$ is constant, that is,

$$N(t) = \int_{\Omega} [S(x,t) + I(x,t) + R(x,t)]dx = N_0 \quad \text{for all } t \geq 0 \ .$$

Since the first two equations of system (2) are independent of R, system (2) can be rewritten as follows:

$$\begin{cases} \frac{\partial S(x,t)}{\partial t} = d_1 \Delta S(x,t) + \mu N - \mu S(x,t) - \beta S(x,t) I(x,t), & x \in \Omega, \quad t > 0, \\ \frac{\partial I(x,t)}{\partial t} = d_2 \Delta I(x,t) - (\mu + \nu) I(x,t) + \beta S(x,t) I(x,t), & x \in \Omega, \quad t > 0, \end{cases} \tag{5}$$

with initial conditions

$$S(x,0) = S_0(x), \quad I(x,0) = I_0(x), \quad x \in \Omega, \tag{6}$$

and the homogeneous Neumann boundary condition

$$\frac{\partial S(\xi,t)}{\partial \upsilon} = \frac{\partial I(\xi,t)}{\partial \upsilon} = 0 \qquad \xi \in \partial\Omega, \quad t > 0. \tag{7}$$

After this reduction, R can be determined using the following relation:

$$R(x,t) = N - S(x,t) - I(x,t) \ .$$

To prove the existence of global solutions of the system, we first define the following subspace of the Sobolev space $W^{2,2}(\Omega)$

$$H^2_N(\Omega) = \left\{ u \in W^{2,2}(\Omega), \quad \frac{\partial u}{\partial \upsilon} = 0 \text{ on } \partial\Omega \right\} \ .$$

We then introduce the subregion Θ defined by

$$\Theta = \left\{ (S, I)^\top \in \left(L^2(\Omega)\right)^2 \;\middle|\; 0 < S + I \leq \max\left\{ \|S_0(x) + I_0(x)\|_{L^\infty(\Omega)}, N \right\} \right\}. \tag{8}$$

The following theorem establishes the existence of global solutions to the initial boundary value problem defined by Equations (5–7).

Theorem 1. *For each initial condition $(S_0, I_0)^\top \in \Theta$, the initial boundary value problem given by Equations (5 – 7) has a unique global solution $(S(x,t), I(x,t))$ in the function space*

$$S,\ I \in \mathscr{C}\left([0,+\infty), L^2(\Omega)\right) \cap \mathscr{C}\left((0,+\infty), H_N^2(\Omega)\right) \cap \mathscr{C}^1\left((0,+\infty), L^2(\Omega)\right). \tag{9}$$

Furthermore, the global solution $(S(x,t), I(x,t))$ is positive and bounded.

Proof. First, we prove that the initial boundary value problem given by Equations (5–7) admits a unique local solution. To achieve this, we reformulate it as the following abstract Cauchy problem:

$$\begin{cases} \frac{dU}{dt} + AU = F(U), \quad t > 0\,, \\ U(0) = U_0\,, \end{cases}$$

in the Hilbert space $Y = L^2(\Omega) \times L^2(\Omega)$, where $U = (S, I)$ and $A = diag(-d_1\Delta + 1, -d_2\Delta + 1)$.

It is known that A is a positive definite, self-adjoint, and sectorial operator with an angle strictly less than $\frac{\pi}{2}$, and its domain is $D(A) = H_N^2(\Omega) \times H_N^2(\Omega)$. Moreover, A admits fractional powers A^θ, for $0 \leq \theta \leq 1$, where

- if $0 \leq \theta < \frac{3}{4}$, the domain is $D(A^\theta) = H^{2\theta}(\Omega) \times H^{2\theta}(\Omega)$;
- if $\frac{3}{4} < \theta \leq 1$, the domain is $D(A^\theta) = H_N^{2\theta}(\Omega) \times H_N^{2\theta}(\Omega)$.

Now, let $\theta \in (\frac{3}{4}, 1)$. The nonlinear operator F is defined by

$$F(U) = \begin{pmatrix} \mu N + (1-\mu)S - \beta SI \\ \beta SI - (1+\mu+\nu)I \end{pmatrix}, \quad U = (S, I) \in D\left(A^\theta\right).$$

Since $\frac{3}{4} < \theta \leq 1$, we have the following continuous embedding

$$D\left(A^\theta\right) = H_N^{2\theta}(\Omega) \times H_N^{2\theta}(\Omega) \subset L^\infty(\Omega) \times L^\infty(\Omega). \tag{10}$$

Elementary computations show that $F(U)$ satisfies the following estimation:

$$\|F(U) - F(V)\|_Y \leq C\left(\|U\|_{\mathcal{L}} + \|V\|_{\mathcal{L}} + 1\right)\|U - V\|_Y,$$

where $\mathcal{L} = (L^\infty(\Omega))^2$ and $C = \max((\mu+1), 2\beta) > 0$.

Using the embedding (10), we deduce that there exists $\tilde{C}(\Omega, C) > 0$ such that

$$\|F(U) - F(V)\|_Y \leq \tilde{C}\left(\left\|A^\theta U\right\|_Y + \left\|A^\theta V\right\|_Y + 1\right)\|U - V\|_Y, \quad U, V \in D\left(A^\theta\right).$$

Therefore, by applying the existence and uniqueness theorem for the abstract Cauchy problem (see [49], Theorem 4.4), the initial boundary value problem given by Equations (5–7) admits a unique local solution satisfying

$$S, I \in \mathscr{C}\left([0, T_m], L^2(\Omega)\right) \cap \mathscr{C}\left((0, T_m], H_N^2(\Omega)\right) \cap \mathscr{C}^1\left((0, T_m], L^2(\Omega)\right), \qquad T_m > 0,$$

where the final time $T_m > 0$ depends on the initial condition U_0.

Next, let $U_0 = (S_0, I_0)$ be a positive initial condition. By applying Lemma 2.2 from [26], we conclude that the solution $U(t) = (S(t), I(t))$ is positive in $(0, T_m)$.

To demonstrate that the solution $U(t) = (S(t), I(t))$ is bounded, we use Theorem 2.1 from [26]. Specifically, for all $x \in \Omega$ and $t > 0$, the following inequality holds:

$$0 < S(x,t) + I(x,t) \leq \max\{||S_0(x) + I_0(x)||_{L^\infty(\Omega)}, N\} .$$

Applying Corollary 4.3 from [49], it follows that $T_m = +\infty$. Hence, the initial boundary value problem (5–7) possesses a unique global solution $U = (S, I)$ in the following function space:

$$\mathscr{C}\left([0,+\infty), L^2(\Omega)\right) \cap \mathscr{C}\left((0,+\infty), H_N^2(\Omega)\right) \cap \mathscr{C}^1\left((0,+\infty), L^2(\Omega)\right).$$

The proof is complete. □

We now turn to the study of the homogeneous steady states of system (5), which are the solutions of the following system:

$$\begin{cases} \mu N - \mu S - \beta S I = 0, \\ -(\mu + \nu) I + \beta S I = 0 . \end{cases} \tag{11}$$

To analyze these steady states, we first introduce the basic reproduction number R_0, defined by the following:

$$R_0 = \frac{N\beta}{\mu + \nu}. \tag{12}$$

The basic reproduction number R_0 is a key threshold quantity in epidemiology, as it helps determine whether an infection can invade and persist in a new host population. The following theorem is proved in [26].

Theorem 2. *The following assertions hold.*

For all $\mu > 0$, $\nu > 0$ and $\beta > 0$, system (5) admits a homogeneous steady state, denoted by $E^0 = (S_f, 0)$, where $S_f = N$.

If $R_0 > 1$, system (5) admits a second homogeneous steady state, denoted by $E^* = (S^*, I^*)$, where $S^* = \frac{\mu+\nu}{\beta}$ and $I^* = \frac{N\mu}{\mu+\nu} - \frac{\mu}{\beta}$.

The steady state E^0, known as the disease-free equilibrium, represents the absence of infected individuals in the population. The steady state E^* is referred to as the *endemic equilibrium* and corresponds to the persistence of the infection within the population.

We now briefly recall the global stability results of the homogeneous steady states of the SIR model (5).

By virtue of Theorem 2, the region Θ defined by (8) is invariant under the flow induced by system (5). The stability of the steady states E^0 and E^* within this invariant region is established by the following theorem, which was proved in [26].

Theorem 3. *If $R_0 < 1$, then the disease-free equilibrium E^0 is globally asymptotically stable in Θ. Conversely, if $R_0 > 1$, then the endemic equilibrium E^* is globally asymptotically stable in Θ .*

2.2 | Modeling Random Variations in Disease Transmission Using Discrete Probabilistic Process

Our aim is to incorporate the possibility of random changes in the transmission rate of the disease. Various factors can influence and alter the transmission rate, such as virus mutations, human behaviors, public health measures, or medical interventions (see, e.g., [50–52]). These random modifications lead to new dynamics in the spread of the epidemic that cannot be adequately captured by a purely continuous system. To model these random changes in the transmission rate, we couple the reaction–diffusion system (5) with a probabilistic process acting at discrete time steps. This probabilistic process is able to interrupt the trajectory of the reaction–diffusion system (5) and to make it restart in a new parameter regime. Following [43], we introduce three parameters τ, p, and $\mathcal{J}$:

- $\tau > 0$ represents the time delay between two potential changes in the transmission rate β. This establishes a time discretization given by

$$\mathcal{T} = \{0, \tau, 2\tau, \ \dots \ , k\tau, \ \dots \ \}; \tag{13}$$

- $p \in [0, 1]$ denotes the probability of occurrence of a change in the transmission rate at a time step $t \in \mathcal{T}$. For simplicity, we assume that p remains constant over time;
- $\mathcal{J}$ is a random variable in the interval $[J_{\min}, J_{\max}]$, where $-1 \leq J_{\min} < J_{\max})$. This variable determines the intensity of the changes in the transmission rate.

More specifically, let $\Xi(t)$ denote the probabilistic event corresponding to the occurrence of a change in the transmission rate at time $t \in \mathcal{T}$. At each time step $k\tau \in \mathcal{T}$, with $k \geq 0$, we consider a Bernoulli variable X_k with parameter p, which determines the probability of the event $\Xi(t)$:

$$\mathbb{P}(\Xi(k\tau)) = \mathbb{P}(X_k = 1) = p, \quad k > 0. \tag{14}$$

We assume that the Bernoulli variables $(X_k)_{k\geq 0}$ are pairwise independent. The sequence of Bernoulli variables $(X_k)_{k\geq 0}$ then determines a probabilistic sequence of transmission rates $(\beta_k)_{k\geq 0}$. This sequence is initialized with $\beta_0 = \beta$ and iterated as follows: If a change occurs at time $t^* = k^*\tau$, then the trajectory of the reaction–diffusion system (5) and (6) is interrupted and restarted according to the dynamics of the reaction–diffusion system (5), with a new transmission parameter β_{k^*+1}, determined by

$$\beta_{k^*+1} = (1 + \mathcal{J})\beta_{k^*}, \tag{15}$$

where $\mathcal{J}$ is randomly chosen from $[J_{\min}, J_{\max}]$, according to a probability law that can be arbitrarily chosen.

Finally, the new initial condition is defined by

$$S_{k^*\tau}(x) = S(x, k^*\tau) \ , \qquad I_{k^*\tau}(x) = I(x, k^*\tau) \ , \qquad x \in \Omega. \tag{16}$$

Altogether, Equations (5–7) and (13–16) define a hybrid model that couples both a deterministic–continuous system and a discrete–probabilistic process. For simplicity, we refer to this hybrid model as $(\mathcal{H})$.

2.3 | Global Solution of the SIR Hybrid Model

Our goal now is to prove that the hybrid model $(\mathcal{H})$, as defined in Sections 2.1 and 2.2, admits relevant solutions. The following theorem addresses this.

Theorem 4. *For any initial condition $U_0 = (S_0, I_0)^\top \in \Theta$, any realization of a sequence of pairwise independent Bernoulli variables $(X_k)_{k\geq 0}$, and any realization of the random intensity variable $\mathcal{J}$ within the interval $[J_{\min}, J_{\max}]$, the hybrid model $(\mathcal{H})$ defined by Equations (5 – 7) and (13 – 16) admits a unique global solution. This solution is denoted $\mathbb{U}(t) = (S(t), I(t))^\top$, defined on $[0, +\infty)$ with values in Θ.*

Furthermore, for each pair of positive integers (k, k') such that $k < k'$ with $X_k = X_{k'} = 1$ and $X_j = 0$ for $k < j < k'$, the restriction $\mathbb{U}_{|[k\tau, k'\tau)}$ of $\mathbb{U}(t)$ to the time interval $[k\tau, k'\tau)$ satisfies

$$S_{|_{[k\tau, k'\tau)}}, \ I_{|[k\tau, k'\tau)} \in \mathscr{C}\left([k\tau, k'\tau), L^2(\Omega)\right) \cap \mathscr{C}\left((k\tau, k'\tau), H^2_N(\Omega)\right) \cap \mathscr{C}^1\left((k\tau, k'\tau), L^2(\Omega)\right). \tag{17}$$

Proof. We construct the global solution $\mathbb{U}(t)$ of the hybrid model $(\mathcal{H})$ by induction on k.

First, consider the initial condition $U_0 = (S_0, I_0)^\top \in \Theta$. By Theorem 1, the initial boundary value system (5–7) admits a unique global solution $U(t, U_0) = (S(t), I(t))^\top$, such that

$$S, \ I \in \mathscr{C}\left([0, +\infty), L^2(\Omega)\right) \cap \mathscr{C}\left((0, +\infty), H^2_N(\Omega)\right) \cap \mathscr{C}^1\left((0, +\infty), L^2(\Omega)\right).$$

Now, let $\beta_0 = \beta$ and let k_1 denote the first positive integer such that the Bernoulli variable X_{k_1} satisfies $X_{k_1} = 1$. We construct the solution $\mathbb{U}(t)$ of the hybrid model on the time interval $[0, k_1\tau]$ by setting

$$\begin{cases} \mathbb{U}(t) = U(t, U_0), & t \in [0, k_1\tau), \\ \mathbb{U}(k_1\tau) = (S_1, I_1)^\top \ , \end{cases}$$

where S_1 and I_1 are given by

$$S_1(x) = S(x, k_1\tau) \ , \quad I_1(x) = I(x, k_1\tau) \ , \quad x \in \Omega \ .$$

Since Θ is invariant under the flow induced by the reaction–diffusion system (5), we have $\mathbb{U}(t) \in \Theta$ for all $t \in [0, k_1\tau]$. Next, we define the new transmission rate as

$$\beta_1 = (1 + \mathcal{J})\beta_0 \ ,$$

where $\mathcal{J}$ is a randomly chosen from the interval $[J_{\min}, J_{\max}]$. For the time interval $[k_1\tau, +\infty)$, we consider the initial boundary value problem (5–7) with the new transmission rate β_1, the new initial condition $U_1 = (S_1, I_1)^T$, and the new initial time $k_1\tau$. We denote the resulting global solution by $\tilde{U}(t, U_1) = (\tilde{S}(t), \tilde{I}(t))$, which is defined on $[k_1\tau, +\infty)$ and whose existence is again guaranteed by Theorem 1.

Next, we consider the first integer $k_2 > k_1$ such that the Bernoulli variable X_{k_2} satisfies $X_{k_2} = 1$. Then the solution $\mathbb{U}(t)$ of the hybrid model is extended to $[0, k_2\tau)$ by setting

$$\begin{cases} \mathbb{U}(t) = \tilde{U}(t, U_1) \ , & t \in [k_1\tau, k_2\tau) \ , \\ \mathbb{U}(k_2\tau) = (S_2, I_2)^\top \ , \end{cases}$$

where $S_2(x) = \tilde{S}(x, k_2\tau)$ and $I_2(x) = \tilde{I}(x, k_2\tau)$ for $x \in \Omega$. Again, we have $\mathbb{U}(t) \in \Theta$ for all $t \in [k_1\tau, k_2\tau]$. We then define the new transmission rate β_2 as $\beta_2 = (1 + \mathcal{J})\beta_1$.

The initial boundary value problem (5–7) is restarted with the new transmission rate β_2, the new initial condition $U_2 = (S_2, I_2)^T$, and the new initial time $t_0 = k_2\tau$.

Finally, by repeating this process, we construct the global solution $\mathbb{U}(t)$ of the hybrid model $(\mathcal{H})$ on $[0, +\infty)$ by induction. This solution satisfies $\mathbb{U}(t) \in \Theta$ for all $t \geq 0$. The proof is complete. □

Theorem 4 shows that the solutions of the hybrid model $(\mathcal{H})$ are probabilistic sequences of interrupted trajectories of the reaction–diffusion system (5). Therefore, the hybrid model $(\mathcal{H})$ can be interpreted as an infinite-dimensional piecewise-deterministic Markov process, as defined, for example, in [38].

3 | Stability Analysis of the Hybrid Model

In this section, we examine the hybrid SIR reaction–diffusion model in order to investigate the impact of random perturbations of the transmission rate β on the asymptotic stability of the disease-free equilibrium, E^0, during the probabilistic process.

3.1 | Set of Solutions and Stability in Probability

First, define the set of solutions for the hybrid model $(\mathcal{H})$ as follows:

$$\mathcal{O} = \left\{ \mathbb{U}(t, U_0) \in \mathscr{C}([0, +\infty), \Theta) \ \mid \ \mathbb{U}(t, U_0) \quad \text{solution of} \quad (\mathcal{H}) \right\}, \tag{18}$$

where $\mathbb{U}(t, U_0)$ denotes a solution of the hybrid model $(\mathcal{H})$ with the initial condition $U_0 \in \Theta$.

Let $(\mathcal{O}, \mathbb{F}, \mathbb{P})$ be a probability space, where $\mathbb{F}$ is the σ-algebra determined by the sequence of Bernoulli variables $(X_k)_{k\geq 0}$ and the probabilistic sequence $(\beta_k)_{k\geq 0}$ of the transmission parameters. Additionally, $\mathbb{P}$ denotes the associated probability measure.

We define the stability in probability of the equilibrium E^0 for our hybrid model $(\mathcal{H})$, as presented in [53].

Definition 1. The disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ is said to be stable in probability if for any realization of a sequence of pairwise independent Bernoulli variables $(X_k)_{k\geq 0}$, any realization of the random intensity variable $\mathcal{J}$ within the interval $[J_{\min}, J_{\max}]$, and any $r > 0$, the following property holds:

$$\lim_{||U_0 - E^0||_Y \to 0} \mathbb{P}\left\{ \sup_{t\geq 0} \left(||\mathbb{U}(t, U_0) - E^0||_Y \right) > r \right\} = 0 \ .$$

Otherwise, for the equilibrium E^0, it is said to be unstable in probability.

Definition 2. The disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ is said to be locally asymptotically stable in probability if, in addition to stability in probability, for any realization of the sequence of pairwise independent Bernoulli variables $(X_k)_{k\geq 0}$ any realization of the random intensity variable $\mathcal{J} \in [J_{\min}, J_{\max}]$, the following property holds:

$$\lim_{||U_0 - E^0||_Y \to 0} \mathbb{P}\left\{ \lim_{t\to +\infty} \left(||\mathbb{U}(t, U_0) - E^0||_Y \right) = 0 \right\} = 1 \ .$$

Definition 3. The disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ is said to be globally asymptotically stable in probability, if it is stable in probability, and for any initial condition $U_0 \in \Theta$, any realization of a sequence of pairwise independent Bernoulli variables $(X_k)_{k\geq 0}$, and any realization of the random intensity variable $\mathcal{J} \in [J_{\min}, J_{\max}]$, the following property holds:

$$\mathbb{P}\left\{ \lim_{t\to +\infty} \left(||\mathbb{U}(t, U_0) - E^0||_Y \right) = 0 \right\} = 1 \ .$$

3.2 | Stability Sufficient Conditions

We now investigate the stability characteristics of the hybrid model $(\mathcal{H})$ with respect to the probabilistic parameters.

First, we define the following two sets with an empty intersection:

$$\Lambda_0 = \left\{ \beta > 0 | \beta < \frac{\mu + \nu}{N} \right\} \quad \text{and} \quad \Lambda_+ = \left\{ \beta > 0 | \beta > \frac{\mu + \nu}{N} \right\} .$$

As a consequence of Theorem 1, when β belongs to Λ_0, the basic reproduction number satisfies $R_0 < 1$. This condition implies the global asymptotic stability of the disease-free equilibrium, E^0.

Conversely, when β belongs to Λ_+, the global asymptotic stability of the endemic equilibrium point, E^*, is guaranteed.

Let us now consider the following assumptions.

- (H_1) The disease-free equilibrium, E^0, of the reaction–diffusion systems (5) and (6) is globally asymptotically stable (i.e., $\beta \in \Lambda_0$).
- (H_2) There exist $\alpha, \varrho > 0$ sufficiently small such that

$$V_\alpha(E^0) \cap V_\varrho(E^*) = \emptyset \ ,$$

  where $V_\alpha(E^0)$ and $V_\varrho(E^*)$ are neighborhoods of E^0 and E^*, respectively.

In the following proposition, we prove a sufficient condition on the intensity $\mathcal{J}$ that ensures the persistence of solutions of the hybrid model $(\mathcal{H})$ in a neighborhood of the disease-free equilibrium, E^0, over the entire time interval $\mathcal{T}$.

Proposition 1. *Let the assumption (H_1) hold, and let δ be the maximum value of the sequence $(\beta_k)_{k\in\mathbb{N}} \in \Lambda_0$. If the following inequality is satisfied,*

$$J_{\max} \leq \frac{\mu + \nu}{N\delta} - 1, \tag{19}$$

then the disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ is globally asymptotically stable in probability, for all values of $p \in (0,1)$ and for all $\mathcal{J} \in [J_{\min}, J_{\max}]$.

Proof. Let $(X_k)_{k\geq 0}$ be a sequence of pairwise independent Bernoulli variables indicating changes in the transmission coefficient $\Xi(k\tau)$, at the time instances $t = k\tau$.

Assume that there exists at least one positive integer k_1 such that $X_{k_1} = 1$. If no such k_1 exists, the result follows directly from assumption (H_1).

At time $t_1 = k_1\tau$, the transmission coefficient becomes $\beta_{k_1} = (1+\mathcal{J})\beta$, where $\mathcal{J}$ is randomly selected from the interval $[J_{\min}, J_{\max}]$. Due to condition (19) on $J_{\max}$, the coefficient β_{k_1} remains in Λ_0. Therefore, the basic reproduction number is less than one, which implies that the trajectories of the hybrid system will converge to the disease-free equilibrium, E^0, after a certain time T within the time interval $[k_1\tau, +\infty)$. In other words, the following convergence is valid

$$\lim_{t\to+\infty} ||\mathbb{U}(t, U_0) - E^0||_{_Y} = 0, \quad \text{with probability 1 .}$$

More generally, consider the positive integers $k_1, \ldots, k_n$ such that the Bernoulli variable $(X_{k_i})_i$ satisfies $X_{k_i} = 1$, for all $i \in \{1, \ldots, n\}$. By induction, we construct a probabilistic sequence of transmission rates $(\beta_{k_i})_i$ belonging to Λ_0.

Thus, using similar reasoning as for the the first change, we conclude that the disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ is globally asymptotically stable with probability 1. □

If we consider the hybrid model $(\mathcal{H})$ with a sequence of decreasing transmission coefficients, we obtain the following result.

Corollary 1. *Let the assumption (H_1) hold, and assume that $J_{\max} \leq 0$. Then for all $p \in (0,1)$ and all $\mathcal{J} \in [J_{\min}, J_{\max}]$, the solution of the hybrid model $(\mathcal{H})$ converges to the disease-free equilibrium E^0.*

Proof. Let $(X_k)_{k>0}$ be a sequence of pairwise independent Bernoulli variables and suppose that there exists a positive integer k_1 such that $X_{k_1} = 1$.

Using formula (15) and assuming that all $\mathcal{J}$ are negative, the new transmission coefficient satisfies $\beta_{k_1} \leq \beta$.

Under the assumption (H_1), it follows that $\beta_{k_1} \in \Lambda_0$. Or equivalently,

$$||\mathbb{U}(t, U_0) - E^0||_{_Y} \to 0 \quad \text{as} \quad t \to +\infty \quad \text{with probability 1 fo any} \quad U_0 \in \Theta.$$

More generally, if there exist positive integers $k_1, \ldots, k_n$ such that $X_{k_i} = 1$, for all $i \in \{1, \ldots, n\}$, then we obtain a sequence of transmission coefficients $(\beta_{k_i})_i$ which decreases over time and remains within Λ_0. Therefore, the global stability in probability of the trajectories of the hybrid model $(\mathcal{H})$, with n perturbations of β, holds.

By induction, it follows that for any sequence of pairwise independent Bernoulli variables $(X_k)_{k>0}$ and any negative $\mathcal{J}$, the solution of the hybrid model $\mathbb{U}(t, U_0)$ converges to E^0 with probability 1. □

The results above can be interpreted as follows: If the intensity $\mathcal{J}$ is negative or possibly positive but within a small range (as long as it satisfies the condition on $J_{\max}$), then the trajectories of the hybrid model will remain in the vicinity of the disease-free equilibrium E_0. This implies that the stability of E^0 is preserved with probability 1, even in the presence of transmission rate changes with negative intensity or slightly positive intensity. In epidemiological terms, this means that when the transmission rate decreases or slightly increases (due to external factors), the disease will not spread widely. As a result, the population will stay closer to the disease-free state, indicating that the disease remains under control and does not lead to widespread outbreaks.

In the following proposition, we investigate the hybrid model $\mathbb{U}(t, U_0)$ with an increasing of the transmission coefficient.

Proposition 2. *Consider the hybrid model $\mathbb{U}(t, U_0)$ under the assumptions (H_1) and (H_2), with a sufficiently large τ and*

$$J_{\min} \geq \frac{\mu+\nu}{N\epsilon} - 1 > 0, \tag{20}$$

where ϵ represents the smallest value of the sequence $(\beta_k)_{k\in\mathbb{N}}$ within the set Λ_0. Then, for all $p \in (0,1)$ and for all $\mathcal{J} \in [J_{\min}, J_{\max}]$, the disease-free equilibrium, E^0, of the hybrid model $(\mathcal{H})$ becomes unstable in probability.

Proof. Consider a sequence $\left(X_l\right)_{l>0}$ of pairwise independent Bernoulli variables, and assume that here exists $k_n > 0$, such that the Bernoulli variable X_{k_n} satisfies $X_{k_n} = 1$. Under the condition given in Equation (20), for any $\mathcal{J}$ randomly chosen in the interval $[J_{\min}, J_{\max}]$, we can easily show that the corresponding transmission coefficient β_{k_n} belongs to Λ_+. Moreover, since τ is assumed to be sufficiently large, the solution of the hybrid model $\mathbb{U}(t, U_0)$ will leave the neighborhood $V_\alpha(E^0)$ after a time $t > k_n\tau$ with probability 1.

For a given time $t_{k_m} > t_{k_n}$, where the Bernoulli variable X_{k_m} satisfies $X_{k_m} = 1$, we can show that β_{k_m} belongs to Λ_+, due to the fact that the intensity $\mathcal{J}$ is randomly chosen from a nonnegative interval. Furthermore, assuming that τ is sufficiently large, the solution of the hybrid model $\mathbb{U}(t, U_0)$ will eventually converge to the endemic equilibrium E^*. According to the assumption (H_2), this convergence implies the instability in probability of the disease-free equilibrium E^0.

By repeating the preceding reasoning over the sequence of times $(t_{k_n})_n$, where the Bernoulli variables $(X_{k_n})_n$ satisfy $X_{k_n} = 1$, we conclude by induction that the disease-free equilibrium E^0 becomes unstable in probability. □

Proposition 2 shows that when the intensity $\mathcal{J}$ is sufficiently large, the trajectory of the hybrid model leaves the neighborhood of the disease-free equilibrium, E^0, and converges toward the endemic equilibrium, E^*. Consequently, in the modified SIR model with frequent probabilistic changes in transmission rate and higher intensity, the system is unable to return to the disease-free equilibrium, E^0, after a certain time. This result suggests that larger fluctuations in the transmission rate lead to a persistent spread of the disease, making eradication of the infection unattainable.

3.3 | Emergence of Oscillations

In this section, we present the conditions on the intensity $\mathcal{J}$ and the time delay τ under which the hybrid SIR model $\mathbb{U}(t, U_0)$ exhibits oscillatory behavior or not.

Initially, we examine the case where the solution of the hybrid model $\mathbb{U}(t, U_0)$ exhibits oscillations between the neighborhood of the disease-free equilibrium E^0 and the neighborhood of the endemic equilibrium E^*.

Theorem 5. *Let $J_{\max}$ be a sufficiently large positive parameter to ensure that there exists a probabilistic change in transmission rate β_k that belongs to Λ_+, assuming that τ is sufficiently large and that the assumptions (H_1) and (H_2) hold. Under these conditions, for all $p \in (0,1)$ and for all $\mathcal{J} \in [J_{\min}, J_{\max}]$, the solution of the hybrid model $\mathbb{U}(t, U_0)$ exhibits oscillations between a neighborhood $V_\alpha(E^0)$ of the disease-free equilibrium, E^0, and a neighborhood $V_\varrho(E^*)$ of the endemic equilibrium, E^*.*

Proof. Let $(X_k)_{k\geq 0}$ be a sequence of pairwise independent Bernoulli variables determining changes in the transmission coefficient $\Xi(k\tau)$ at times $t_k = k\tau$ with a randomly varying intensity $\mathcal{J}$. Consider the following sequence of realizations $(X_k)_{k\geq 0}$ satisfying for all $j \geq 0$:

$$\begin{cases} X_k = 1, \text{ and } \mathcal{J} \geq \frac{\mu+\nu}{N\epsilon} - 1 & k_1 \times j \leq k < k_1 \times j + k_2, \\ X_k = 1, \text{ and } \mathcal{J} \in [J_{\min}, \frac{\mu+\nu}{N\delta} - 1] & k_1 \times j + k_2 \leq k < k_1 \times j + k_3, \\ X_k = 0 & k_1 \times j + k_3 \leq k < k_1 \times (j+1), \end{cases} \tag{21}$$

where k_1, k_2, and k_3 are positive integers such that $k_1 > k_3 > k_2$ and ϵ and δ represent, respectively, the smallest and largest values among all potential occurrences of probabilistic changes β_k within the set Λ_0.

With this sequence $(X_k)_{k\geq 0}$, the solution $\mathbb{U}(t, U_0)$ of the hybrid model $(\mathcal{H})$ undergoes k_3 successive changes in the transmission rate β. Specifically, there are k_2 changes where the new transmission parameter is in Λ_+ and $k_3 - k_2$ changes where the new transmission rate is in Λ_0. These changes alternate with a time interval of length $(k_1 - k_3)\tau$ without any changes.

Assuming that τ is sufficiently large and if k_2 is sufficiently large, then the solution $\mathbb{U}(t, U_0)$ is attracted to a neighborhood $V_\varrho(E^*)$ of the endemic equilibrium in the time interval $[k_1 \times j\tau, (k_1 \times j + k_2)\tau]$ for all $j \geq 0$.

If k_3 is sufficiently large, $\mathbb{U}(t, U_0)$ is attracted to a neighborhood $V_\alpha(E^0)$ of the disease-free equilibrium in the time interval $[(k_1 \times j + k_2)\tau, (k_1 \times j + k_3)\tau]$ for $j \geq 0$. Since E^0 is globally asymptotically stable when k_1 is sufficiently large, the solution $\mathbb{U}(t, U_0)$ reaches a neighborhood $V_\alpha(E^0)$ in the time interval $[(k_1 \times j + k_3)\tau, k_1 \times (j+1)\tau]$ for all $j \geq 0$.

Consequently, the solution $\mathbb{U}(t, U_0)$ of the hybrid model $(\mathcal{H})$ oscillates between $V_\alpha(E^0)$ and $V_\varrho(E^*)$, completing the proof. □

In epidemic terms, the emergence of oscillations in the hybrid SIR model reflects wave-like epidemic patterns, where the number of infections fluctuates periodically over time. Understanding these oscillations is crucial for predicting the timing and intensity of epidemic waves, which can help inform public health strategies aimed at mitigating the impact of infectious diseases.

For the stability of the hybrid model $(\mathcal{H})$ in situations where the solution temporarily converges to a neighborhood of E^* over a short time interval, consider the following hypothesis:

(H_3) Let $(t_{k_n})_{k_n>0}$ be a sequence of times in $\mathcal{T}$ such that $t_{k_n} < t_{k_n+1}$. Assume that

$$\text{if} \quad \beta_{k_n} \in \Lambda_+ \quad \text{then} \quad \beta_{k_n+1} \in \Lambda_0 \ .$$

Let $\tau^* := \max_{k_n \geq 1} |t_{k_n} - t_{k_n+1}|$ denote the maximum time interval between two successive changes in transmission rates in the sequence $(t_{k_n})_{k_n>0}$.

We can establish the following theorem.

Theorem 6. *Assume that conditions (H_1) – (H_3) hold and that τ^* is sufficiently small. Then, for all $p \in (0,1)$ and for all $\mathcal{J} \in [J_{\min}, J_{\max}]$ where $J_{\max}$ is a sufficiently large positive parameter, the solution of the hybrid model $(\mathcal{H})$ converges to the disease-free equilibrium E^0.*

Proof. Let $(t_{k_n})_{k_n>0}$ be a sequence of times that satisfies (H_3). Then, the solution of the hybrid model, $\mathbb{U}(t, U_0)$, can be attracted to $V_\varrho(E^*)$ during the time interval $[t_{k_n}, t_{k_n+1}]$ for all $k_n > 0$. This follows from the same argument used in the proof of Theorem 5. Under the assumption that τ^* is very small, the hybrid model does not have enough time to align with the endemic equilibrium E^*. Hence, the solution $\mathbb{U}(t, U_0)$ remains close to E^0 throughout the entire time interval $\mathcal{T}$. □

Theorem 6 can be interpreted as follows: When there are alternating high and low intensity changes in β within a sufficiently short time frame, the asymptotic stability of E^0 is preserved with probability 1. Essentially, this means that timely and effective interventions can lead to the elimination of the disease, even when population behaviors and intervention intensities fluctuate. This highlights the importance of rapid response and adaptable policies in managing and eradicating epidemics.

To investigate the stability properties of the disease-free equilibrium, E^0, within the hybrid model $(\mathcal{H})$, when the transmission rate β_0 belongs to Λ_+, the following proposition is established.

Proposition 3. *If $\beta_0 \in \Lambda_+$ and τ is sufficiently large, then for all $p \in (0,1)$ and for all $\mathcal{J} \in [J_{\min}, J_{\max}]$ satisfying*

$$J_{\max} \leq \frac{\mu + \nu}{N\lambda} - 1 \ ,$$

where λ is the maximum value of all possible occurrences of β within the set Λ_+, the trajectories of the hybrid model leave the neighborhood of E^* and are attracted to a neighborhood of the disease-free equilibrium, E^0, after a time T.

Proof. Consider a sequence $\left(X_k\right)_{k>0}$ of pairwise independent Bernoulli variables and assume that there exists a positive integer $k > 0$ such that $X_k = 1$. Given the condition

$$J_{\max} \leq \frac{\mu + \nu}{N\lambda} - 1 \ ,$$

for any $\mathcal{J}$ randomly chosen from $[J_{\min}, J_{\max}]$, we can show that β_k becomes an element of Λ_0, provided τ is sufficiently large. Thus, the solution of the hybrid model $\mathbb{U}(t, U_0)$ leaves the neighborhood $V_\alpha(E^*)$ and is attracted to a neighborhood of E^0 with probability 1, after a time $t > k\tau$.

More generally, by applying the same reasoning to all Bernoulli variables $X_k = 1$, we deduce that the solution of the hybrid model ($\mathcal{H}$) will certainly be attracted to a neighborhood of E^0. □

In other words, when the intensity of transmission rate changes remains within a certain range, the dynamics of the disease in the hybrid model stabilize, even in scenarios where the value of the transmission coefficient β_0 suggests a higher risk of instability.

3.4 | Hybrid SIR Model With Vaccination

In this scenario, let $\beta_0 \in \Lambda_+$ and consider the deterministic SIR model (2) with vaccination described by the following system:

$$\begin{cases} \frac{\partial S(x,t)}{\partial t} = d_1 \Delta S(x,t) + \mu N - \mu S(x,t) - \beta S(x,t) I(x,t) - uS(x,t), & x \in \Omega, \quad t > 0, \\ \frac{\partial I(x,t)}{\partial t} = d_2 \Delta I(x,t) - (\mu + \nu) I(x,t) + \beta S(x,t) I(x,t), & x \in \Omega, \quad t > 0, \\ \frac{\partial R(x,t)}{\partial t} = d_3 \Delta R(x,t) - \nu I(x,t) + \mu R(x,t) + uS(x,t), & x \in \Omega, \quad t > 0, \end{cases} \tag{22}$$

where u represents the vaccination coverage of susceptible individual. We assume that the vaccination parameter satisfies $0 \leq u \leq u_{max} \leq 1$. The vaccination strategy influences the transmission dynamics, potentially reducing the infection rate and affecting the spread of the epidemic. System (22) can be simplified to the first two equations concerning S and I.

Our goal is to establish a condition on the vaccination parameter that ensures disease eradication by a final time T (i.e., $I(\cdot, T) = 0$).

We now present the particular stability properties of equilibrium related to the first two equations of system (22). Some properties of the SIR model with vaccination coverage are discussed in [34]. for a system of ordinary differential equations. By applying similar arguments, we can establish the following lemma.

Lemma 1. ([34]). *We define the induced reproduction number as follows:* $R(u) = \frac{\beta\mu N}{(\mu+\nu)(\mu+u)}$. *The following properties hold:*

1. *The equilibrium of system* (22) *corresponds to the disease-free equilibrium* $E_u^0 = \left(\frac{\mu N}{\mu+u}, 0\right)$. *Additionally, if* $R(u) > 1$, *the system admits an endemic equilibrium* $E_u^* = (S^*, I^*)$, *where* $S^* = \frac{\mu N}{(\mu+u)R(u)}$ *and* $I^* = \frac{\mu+u}{\beta}(R(u) - 1)$.
2. $R(u)$ *is a decreasing function of* u, *indicating that vaccination reduces the vaccine-induced reproduction number*.
3. *In the absence of vaccination, we have* $R(u = 0) = R_0 = \frac{\beta N}{\mu+\nu}$.
4. *We have* $R(u) \leq R_0$. *Consequently, if* $R_0 < 1$, *then* $R(u) < 1$. *Thus,* E_u^0 *is asymptotically stable if* $R(u) < 1$.
5. *If* $R_0 > 1$, *we have the following inequality:* $\frac{\mu}{\mu+u} R_0 \leq R(u) \leq R_0$. *Since* $\frac{\mu}{\mu+u} \leq 1$, *if* $u > \mu\left(\frac{\beta N}{\mu+\nu} - 1\right) := u_c$, *then* $R(u) < 1$.

Now, we can present the condition on the vaccination parameter for the hybrid model with vaccination, as defined by Equations ((22), (6), (7), (13), (14), (15), and (16)), where $R_0 > 1$, to ensure the asymptotic stability of E_u^0.

Theorem 7. *Assume that* $R(u = 0) > 1$, *and let* τ *be sufficiently large. For all* $p \in (0, 1)$ *and for all* $\mathcal{J} \in [J_{\min}, J_{\max}]$, *if*

$$u > \mu\left(\frac{\rho N}{\mu + \nu} - 1\right), \tag{23}$$

where ρ *is the smallest value of all possible changes in* β *within the set* Λ_+, *then the trajectories of the hybrid model with vaccination converge to the disease-free equilibrium,* E_u^0.

Proof. Let $(X_k)_{k\geq 0}$ be a sequence of pairwise independent Bernoulli variables representing changes in the transmission coefficient for the system (22), occurring at time instances $t = k\tau$. Suppose k_1 is a positive integer such that $X_{k_1} = 1$. If $u > \mu(\frac{\beta_0 N}{\mu+\nu} - 1)$, then the solution of the hybrid model ($\mathcal{H}$) converges to the disease-free equilibrium E_u^0 after time $t = t_{k_1}$, given that $R(u) < 1$.

More generally, by applying the same reasoning to all subinterval $[t_{k_i}, t_{k_{i+1}}]$ where $X_{k_i} = 1$ for all $i \in \{i = 1, \cdots, n\}$ and using condition (23), we can ensure that $R(u) < 1$ for $k_n\tau \in \mathcal{T}$. The proof is complete. □

This theorem suggests that if the vaccination parameter is chosen appropriately within the hybrid model framework, the epidemic can be eradicated.

4 | Numerical Simulations

In this section, we present numerical simulations for the hybrid model ($\mathcal{H}$) to illustrate how probabilistic behaviors can influence the dynamics of the epidemic. The simulations were conducted using MATLAB R2018a on a desktop computer equipped with an Intel Core i7-1255U processor and 16 GB of RAM.

4.1 | Two-Dimensional SIR Model

In this example, we consider system (5) in the two-dimensional spatial domain $\Omega = [0, 10] \times [0, 10]$, with the initial condition $U_0(x, y) := (S_0(x, y), I_0(x, y))$ defined as follows:

$$\begin{aligned} S_0(x, y) &= 100(e^{-(x-5)^2} + e^{-(x-10)^2} + e^{-(x-6)^2})(e^{-(y-5)^2} + e^{-(y-10)^2} + e^{-(y-2)^2}), \\ I_0(x, y) &= 10(e^{-(x-5)^2} + e^{-(x-7)^2} + e^{-(x-10)^2})(e^{-(y-5)^2} + e^{-(y-7)^2} + e^{-(y-8)^2}). \end{aligned} \tag{24}$$

Note that the initial conditions (24) are chosen to better reflect real-world disease scenarios, such as the spread of the disease across different regions (Figure 1).

For the reaction–diffusion model, we use the following parameter values:

$$\nu = 25, \quad \mu = 1.4, \quad \beta = 0.13, \quad N = 200, \quad d_1 = 6, \quad d_2 = 1.2\ .$$

Under these conditions, hypothesis (H_1) is satisfied (i.e., $\beta \in \Lambda_0$), and we have $R_0 = 0.985$. Therefore, the disease-free equilibrium is asymptotically stable.

The disease-free state of the two-dimensional system is illustrated graphically in Figure 2.

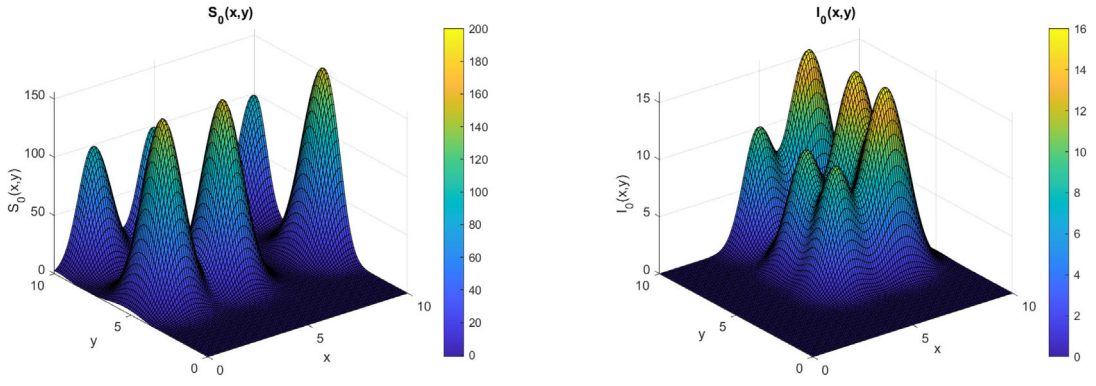

FIGURE 1 | Initial condition (24) for system (5) in the two-dimensional spatial domain Ω. (a) 3D plot showing the density of susceptible individuals $S(x, y)$ at $t = 0$. (b) 3D plot showing the density of infected individuals $I(x, y)$ at $t = 0$. [Colour figure can be viewed at wileyonlinelibrary.com]

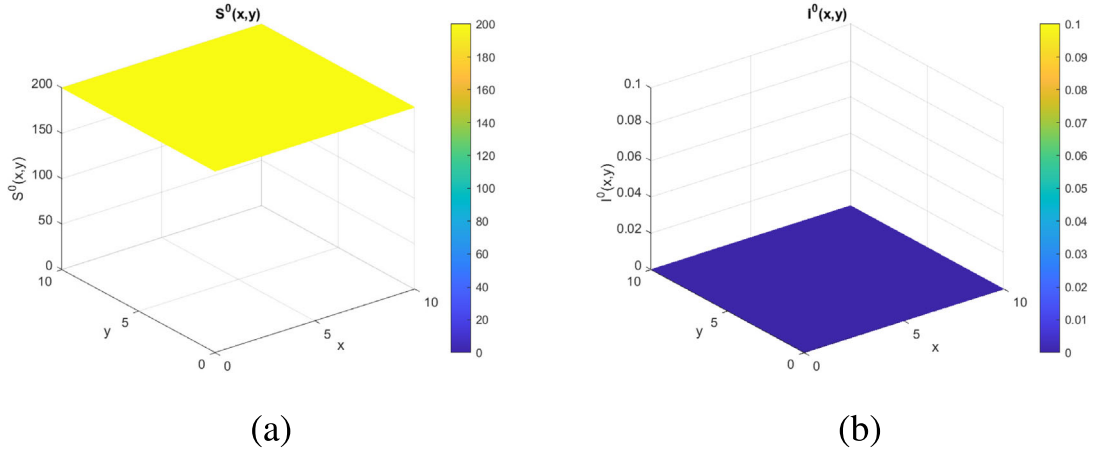

FIGURE 2 | The disease-free state, $E^0 = (S^0(x, y), I^0(x, y))$, associate with system 5 and positive initial conditions (24). (a) 3D plot showing the density of susceptible individuals $S^0(x, y)$. (b) 3D plot showing the density of infected individuals $I^0(x, y)$. [Colour figure can be viewed at wileyonlinelibrary.com]

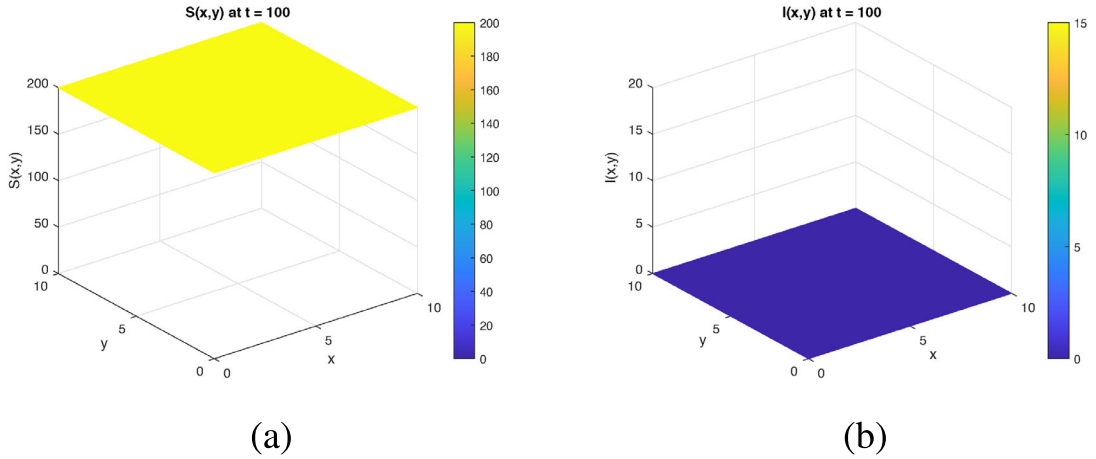

FIGURE 3 | Dynamics of the hybrid system ($\mathcal{H}$) for the parameters in Case 1 (Section 4.1.1): $\tau = 2,\ p = 0.3,\ J \in [-1, 0.1]$. (a) 3D plot showing the density of susceptible individuals $S(x, y)$ at $t = 100$. (b) 3D plot showing the density of infected individuals $I(x, y)$ at $t = 100$. The results indicate that the global solution of the hybrid system ($\mathcal{H}$) converges to the disease-free equilibrium. [Colour figure can be viewed at wileyonlinelibrary.com]

4.1.1 | Case 1: Decrease or Small Increase in Transmission Rate

Consider $\tau = 2,\ p = 0.3$, and $\mathcal{J} \in [-1, 0.1]$ as the parameters of the probabilistic process. Under these conditions, Proposition 1 is satisfied. The hybrid model $\mathbb{U}(t, U_0) = (S, I)$ converges to the disease-free equilibrium $E^0 = (200, 0)$ at $t = 100$, as illustrated in Figure 3.

4.1.2 | Case 2: High Intensity of Change

In this scenario, we consider $\tau = 1,\ p = 0.3$, and $\mathcal{J} \in [1, 3]$, which aligns with the context of Proposition 2. Figure 4 shows that the solution of the hybrid model converges to the endemic equilibrium, E^*, at various time points, reflecting the changes in the parameter β. For example, at $t = 4$, we have $\beta = 0.4811$, resulting in $E^* = (54.87, 7.69)$. At $t = 100,\ \beta = 0.3312$, leading to $E^* = (80.01, 6.35)$. Consequently, the system fails to recover the disease-free equilibrium E^0 throughout the observed time interval.

We present in Table 1 several values of β and their corresponding R_0 in the hybrid model ($\mathcal{H}$).

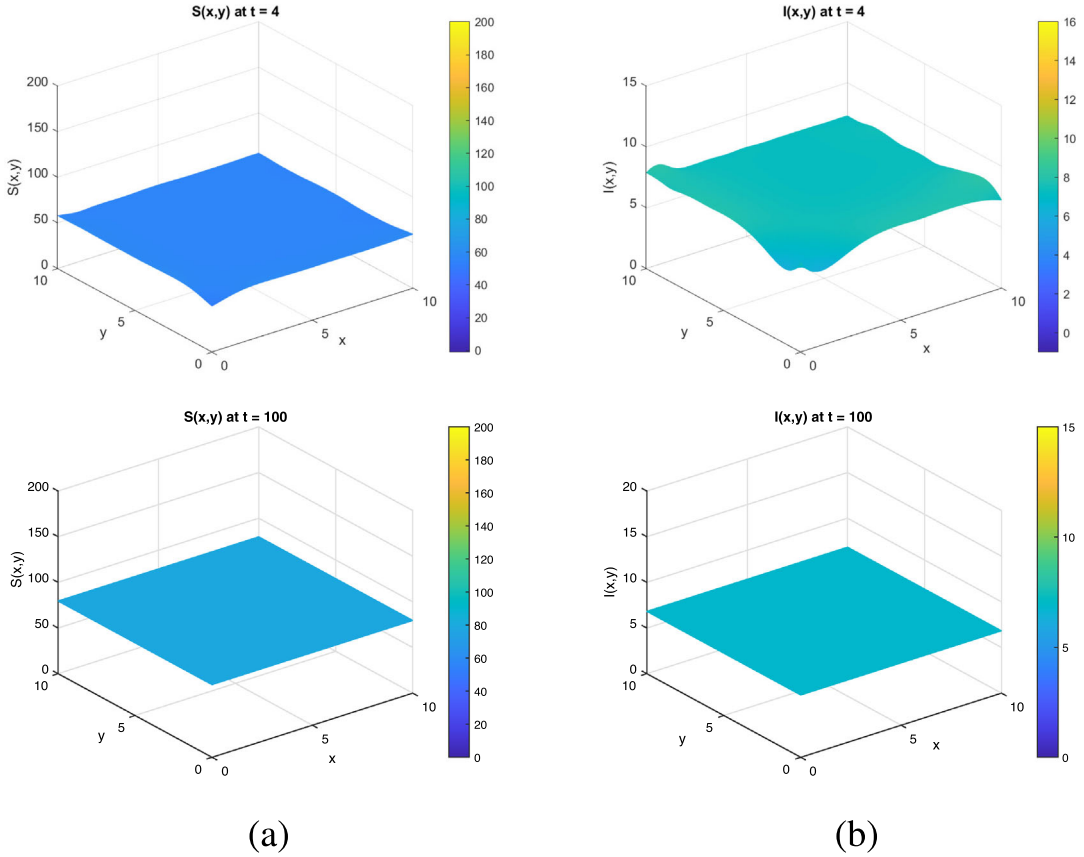

FIGURE 4 | Dynamics of the hybrid system ($\mathcal{H}$) for the parameters corresponding to Case 2 (Section 4.1.2): $\tau = 1,\ p = 0.3,\ \mathcal{J} \in [1, 3]$. (a) 3D plots showing the density of susceptible individuals at $t = 4$ and $t = 100$. (b) 3D plots showing the density of infected individuals at $t = 4$ and $t = 100$. We observe that the global solution of the hybrid system ($\mathcal{H}$) converges to the endemic equilibrium. [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 1 | Values of β and R_0 at various times during the evolution of the hybrid model ($\mathcal{H}$) when the intensity of change in the transmission coefficient β is contained within the interval $[1, 3]$.

Time t	**5**	**20**	**40**	**60**	**100**
Transmission rate β	0.4797	0.3220	0.3397	0.3036	0.3312
Reproduction number R_0	3.6203	2.4301	2.5637	2.2913	2.4996

4.1.3 | Case 3: Emergence of Oscillations

In this case, let $\tau = 1,\ p = 0.3$, and $\mathcal{J} \in [-0.8, 1.8]$. According to Theorem 5, the solution of the hybrid model ($\mathcal{H}$) exhibits oscillations between a neighborhood of E^0 and a neighborhood of E^*. The graphs in Figure 5 illustrate that from $t = 1$ to $t = 10$, there are at least two oscillations between these neighborhoods.

Table 2 presents several values of β and the corresponding reproduction number R_0 for the hybrid model ($\mathcal{H}$) with the specified parameters. We observe that the multiple epidemic waves are related to variations in R_0 within our constructed hybrid SIR model.

Figure 6 presents plots of susceptible and infected individuals, $S(10, 5, :)$ and $I(5, 10, :)$ versus time for different time delays $\tau = 0.1, 1, 2, 5, 10$. These plots illustrate that the time delay between changes in the transmission coefficient within the probabilistic process can acts as a bifurcation parameter, significantly influencing the stability behavior of the equilibrium.

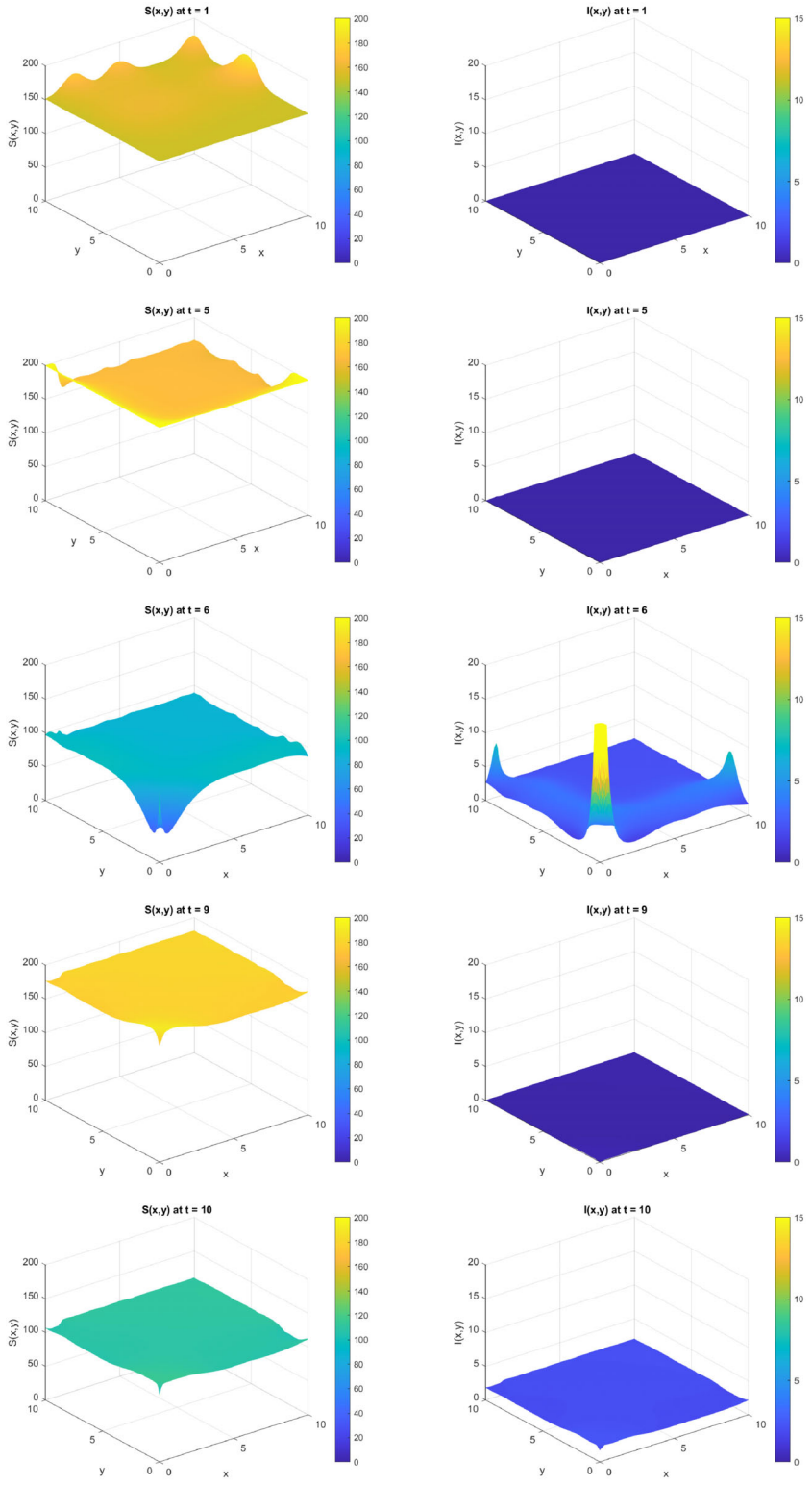

FIGURE 5 | Dynamics of the hybrid system ($\mathcal{H}$) for the parameters corresponding to Case 3: $\tau = 1,\ p = 0.3,\ \mathcal{J} \in [-0.8, 1.8]$. (a) 3D plots showing the density of susceptible individuals at various times: $t = 1, 5, 6, 9, 10$. (b) 3D plots showing the density of infected individuals at the same times. The global solution of the hybrid model ($\mathcal{H}$) exhibits oscillations between a neighborhood of the disease-free equilibrium and a neighborhood of the endemic equilibrium. [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 2 | Several values of β and R_0 in the Case 3 of the hybrid model ($\mathcal{H}$), where $\mathcal{J} \in [-0.8, 1.8]$.

Time t	**1**	**4**	**5**	**6**	**10**
Transmission rate β	0.0670	0.3699	0.0484	0.3563	0.0209
Reproduction number R_0	0.5075	2.8022	0.3666	2.6992	0.1583

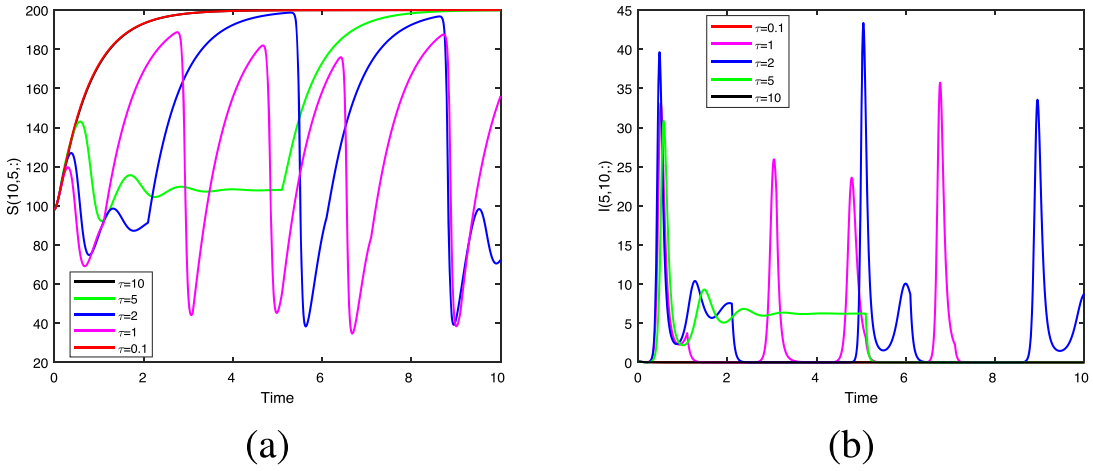

FIGURE 6 | Global solution of the hybrid system ($\mathcal{H}$) for various time delays in the transmission coefficient, $\tau = 0.1, 1, 2, 5, 10$, using the parameters from Case 4. (a) Plots of susceptible individuals at $x = 10$, $y = 5$ versus time. (b) Plots of infected individuals at $(5, 10, :)$ versus time. The results show that τ acts as a bifurcation parameter that alters the stability behavior of the equilibrium. [Colour figure can be viewed at wileyonlinelibrary.com]

Remark 2. The parameter τ serves as a bifurcation parameter in the epidemic hybrid model. Specifically, if τ is small enough, the stability of the disease-free equilibrium is preserved. However, when τ is sufficiently large, oscillations emerge (see Figure 6). Thus, analogous to the transmission coefficient, τ randomly influences the dynamics of disease spread.

4.1.4 | Case 4: Switching Between High and Low Intensity of Change

This scenario corresponds to Theorem 6. Let $p = 0.3$ and $\mathcal{J}$ be an element of $[-0.8, 2]$. Assuming that hypothesis (H_3) holds, we observe that $\mathbb{U}(t, U_0)$ converges to the state $E^0 = (S^0(x, y), I^0(x, y))$, as presented in Figure 2, by time $t = 100$.

4.1.5 | Case 5: $\beta_0 \in \Lambda_+$ and Negative Intensity of Change or Vaccination Strategy

Firstly, we consider the scenario for the two-dimensional system with the following parameter values:

$$\nu = 25, \quad \mu = 1.4, \quad \beta = 0.5, \quad N = 200, \quad d_1 = 6, \quad d_2 = 1.2\ .$$

In this case, the reproduction number R_0 is calculated as $R_0 = 3.7879 > 1$.

Consider $\tau = 1$, $p = 0.3$, and $\mathcal{J} \in [-0.8, 0]$ as the parameters of the probabilistic process. Figure 7 shows that the solution $\mathbb{U}(t, U_0)$ is close to the disease-free equilibrium E^0 at time $t = 100$, even though $R_0 > 1$.

For the vaccine SIR model, consider the first two equations of system (22) with $u = 0.78$ and the intensity $\mathcal{J}$ in $[-0.8, 2]$, while keeping all other parameter values as in the beginning of Case 5. We observe in Figure 8 that the trajectories of the hybrid model ($\mathcal{H}$) converge to the disease-free equilibrium $E^0_{0.78} = (128.44, 0)$ at time $t = 6$.

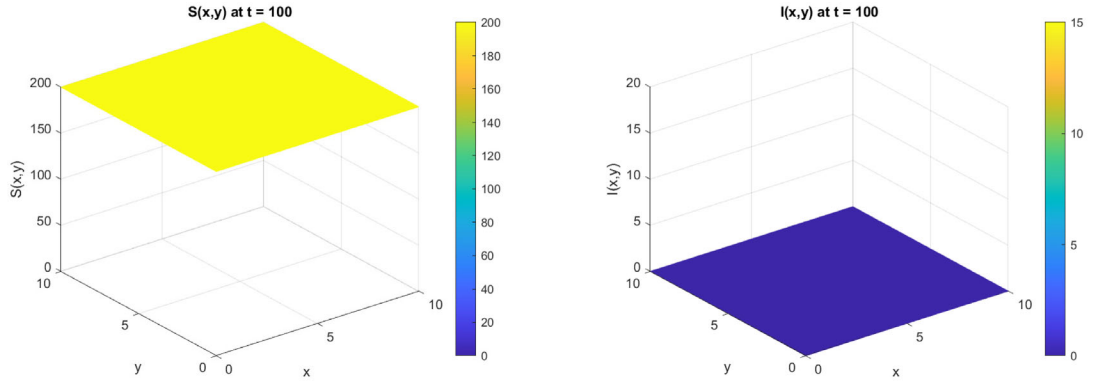

FIGURE 7 | Dynamics of the hybrid system ($\mathcal{H}$) for the parameters corresponding to Case 5: $\beta_0 = 0.5,\ \tau = 1,\ \mathcal{J} \in [-0.8, 0]$. (a) 3D plot of the density of susceptible individuals at time $t = 100$. (b) 3D plot of the density of infected individuals at time $t = 100$. We observe that the solution of the hybrid model is close to the disease-free equilibrium E^0 at time $t = 100$, even though $R_0 > 1$. [Colour figure can be viewed at wileyonlinelibrary.com]

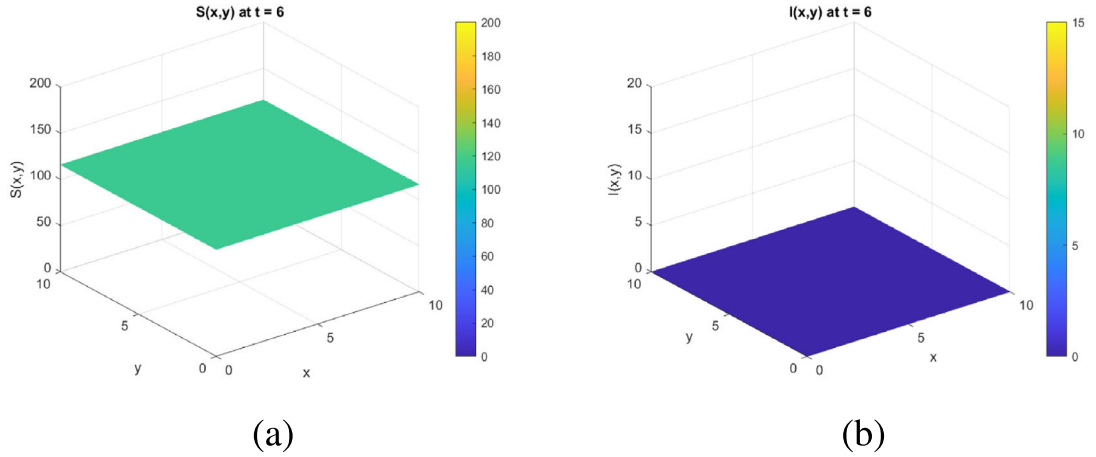

FIGURE 8 | Solution of the hybrid system ($\mathcal{H}$) with vaccination for the parameters corresponding to Case 5: $\beta_0 = 0.5,\ \tau = 1,\ \mathcal{J} \in [-0.8, 2]$ and vaccine parameter $u = 0.78$. (a) 3D plot of the density of susceptible individuals at time $t = 6$. (b) 3D plot of the density of infected individuals at time $t = 6$. We observe that the global solution of the hybrid system with vaccination parameter converges to the disease-free equilibrium at time $t = 6$. [Colour figure can be viewed at wileyonlinelibrary.com]

5 | Conclusion

In this paper, we studied the dynamics and stability of a hybrid reaction–diffusion SIR-type model under the influence of external factors that alter the transmission coefficient. We constructed the hybrid model by coupling a deterministic, continuous process that describes the evolution of an infectious disease with discrete, probabilistic processes that account for potential changes in the transmission coefficient. The hybrid model incorporates a random parameter to simulate varying transmission rates, allowing us to explore scenarios involving both increases and decreases in the rate. We established the well-posedness of our parametric model and verified stability conditions theoretically, supported by numerical simulations.

The main results highlighted in our work include the following:

- A decrease or a small increase in the transmission coefficient preserves the stability of the disease-free equilibrium of the SIR hybrid model.
- When the transmission coefficient changes with higher intensity, the SIR model can attract the endemic state.

- Oscillatory behaviors between the disease-free state and the endemic state can occur, depending in the intensity $\mathcal{J}$ and the time delay between two successive changes in the transmission coefficient.
- The speed of the disease spread can be controlled if there exists a switch between high and low intensity of the transmission rates in the hybrid model with a small time delay.
- When the transmission coefficient is large, and if the intensity or the vaccination parameter satisfies certain theoretical conditions, the SIR model can attract the disease-free state.

In the near future, we aim to explore two key research directions. The first involves developing an optimal control strategy for the reaction–diffusion hybrid epidemic model. This will include devising numerical methods to construct optimal control policies within an abstract framework and investigate how these methods can be applied to real-world scenarios where hybrid models and external factors interact. The second direction focuses on extending the model to a more general framework where the transmission coefficient is a function of both space and time. This extension aims to provide a more realistic representation of epidemic dynamics in reaction–diffusion models.

Author Contributions

Asmae Tajani: conceptualization, investigation, writing – original draft, writing – review and editing, methodology, validation, formal analysis, visualization. **Cristiana J. Silva:** conceptualization, investigation, funding acquisition, writing – original draft, methodology, validation, visualization, writing – review and editing, formal analysis, project administration, supervision. **Guillaume Cantin:** conceptualization, investigation, funding acquisition, writing – original draft, methodology, validation, visualization, writing – review and editing, formal analysis, project administration, supervision.

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Conflicts of Interest

The authors declare no conflicts of interest.

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