Mathematics in Applied Sciences and Engineering
Volume **6**, Number **3**, September **2025**, pp.**216-237**
https://ojs.lib.uwo.ca/mase
https://doi.org/10.5206/mase/19428

IMPACT OF VACCINATION AND NONLINEAR INCIDENCE RATE ON THE DYNAMIC OF INFLUENZA STRAINS EPIDEMIC MODEL: OPTIMAL CONTROL APPROACH

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Abstract. In this study, we develop and analyze a mathematical model describing the transmission dynamics of influenza strains, including both drug-resistant and non-resistant variants. Our model generalizes several existing epidemic models by incorporating key biological factors. We first prove the existence, uniqueness, positivity, and boundedness of global solutions to ensure the model is well-posed under natural conditions. Using the next-generation matrix method, we calculate two basic reproduction numbers associated with the model. We identify four biologically relevant equilibrium points: the disease-free equilibrium, the resistant endemic equilibrium, the non-resistant endemic equilibrium, and the coexistence endemic equilibrium. The local stability of these equilibrium is established through standard stability analysis techniques. Furthermore, we propose two optimal control strategies, treatment and media awareness campaigns based on Pontryagin's Maximum Principle to reduce the spread of infection. Numerical simulations performed in Matlab illustrate and support our theoretical results. Finally, the paper concludes with a discussion and suggestions for future research directions.

1. Introduction

Mathematical modeling has become increasingly important in the study of infectious diseases due to its vital role in understanding transmission dynamics and informing public health interventions. The spread of infectious diseases, caused by bacterial or viral agents, continues to pose a significant threat to human health. Notable examples of viral infections include HIV and influenza [4]. Mathematical models provide researchers with powerful tools to analyze the progression of these diseases and to design effective control strategies aimed at minimizing their transmission and impact.

One of the earliest mathematical frameworks for infectious disease modeling is the classical SIR model introduced by Kermack and McKendrick [22], which divides the population into susceptible, infectious, and recovered individuals. While this model has provided a foundation for epidemic theory, subsequent developments have introduced additional compartments and mechanisms to capture more complex biological realities, such as latency, quarantine, vaccination, and treatment effects [20, 23].

In epidemiology, the incidence rate represents the number of new infections occurring per unit of time, and it plays a central role in modeling the dynamics of infectious diseases. A commonly used form is the bilinear (or mass action) incidence rate βSI, where β is the transmission coefficient, and S and I denote the susceptible and infected populations, respectively. This form has been widely applied in classical epidemic models due to its simplicity and foundational significance.

However, various alternative incidence functions have been proposed in the literature to reflect more realistic transmission dynamics based on different modeling assumptions (see Table 1 and references [32, 30, 29]). Each of these forms offers certain advantages and limitations depending on the biological

Received by the editors 22 June 2024; accepted 29 August 2025; published online 6 September 2025.
2020 *Mathematics Subject Classification.* Primary 92D30; Secondary 49J20, 34D20.
Key words and phrases. Epidemic model, influenza disease, vaccination, stability, optimal control.

context. For instance, when the number of infected individuals becomes large, saturation effects may occur limiting the number of new infections due to factors like behavioral change or resource constraints. In such cases, saturated incidence rates, such as those used by Cao et al. [7], provide a more accurate representation.

Moreover, when public awareness and media influence play a role in disease prevention, modified incidence functions incorporating media effects become relevant. One such form is $(\beta_1 - \beta_2 I)SI$, which models the reduction in transmission as the number of infections increases due to heightened public response [30]. Su and Yang [33] also investigated the role of nonlinear incidence rates in modeling hepatitis C virus (HCV) dynamics, further emphasizing the importance of selecting appropriate transmission functions based on disease characteristics.

However, vaccination represents a valuable mechanism to protect individuals against infectious diseases before the human body receives the infection. In general, the vaccine plays the role of the immune system when the organism is not capable of protecting humans against the disease. Several authors have investigated the effect of vaccination on the dynamics of epidemics by presenting a mathematical model which includes the class of vaccinated individuals. For example, Tornatore et al. [34] proposed a generalized SIR model that contains a vaccination class. They determined sufficient conditions for the disease-free equilibrium exponential stability. Foy et al. [17] presented a Covid-19 epidemic model with vaccination. They examined the vaccine distribution strategies in India. [39] models the transmission of SARS-CoV-2 in various environments, emphasizing that the persistence of the virus depends on the basic reproduction number (R_0). It also suggests control measures through numerical simulations and theoretical analysis.ve In [37], the authors proposed and analyzed a periodic stochastic SIR model with pulse vaccination.

Several mathematicians have developed epidemic models which generalize the famous models of Kermack and Mckendrick by proposing new classes of individuals as the class of individuals in quarantine, the category of exposed individuals, the susceptible confined individuals class, or by the integration of parameters which are associated with biological situations. Hethcote et al. [19] proposed and analyzed an epidemic model with a class of individuals in quarantine and different types of incidence functions. Li and Muldowney [23] investigated the global stability of a four-compartment model that included the exposed class of individuals. The majority of existing model considers the spread of a single type of epidemic, but there are other situations where there are two types of infections. For example, Baba et al. [3] proposed and analyzed a model that sketch the dissemination of influenza in a group of person with two categories of infective population, namely, the infective individuals with resistant and non-resistant strains. They provided a detailed stability analysis of the system.

A considerable body of research has been devoted to the mathematical modeling of influenza transmission, reflecting its recurring seasonal burden and pandemic potential. According to the World Health Organization, seasonal influenza epidemics cause an estimated 3–5 million cases of severe illness and 290,000–650,000 respiratory deaths worldwide each year [38]. Early models of influenza incorporated the SEIR framework to capture latency and recovery dynamics [2]. Subsequent refinements introduced age structure, heterogeneity in contact patterns, and vaccination strategies to better represent realistic population dynamics [15, 26]. With the emergence of antiviral therapies, models were developed to assess the effectiveness of treatment and prophylaxis in mitigating influenza spread [24]. More recent works have addressed the coexistence of drug-sensitive and drug-resistant strains, highlighting the evolutionary pressure exerted by widespread antiviral use and its consequences for epidemic control [1, 18]. Other contributions have incorporated waning immunity, seasonal forcing, and cross-immunity between

strains, providing insights into influenza's long-term dynamics and periodic recurrence [11, 35]. Collectively, these models illustrate the necessity of accounting for resistance, vaccination, and immunity to adequately capture influenza epidemiology.

The model proposed by Baba et al. [3] is presented by the following ordinary differential equations system

$$\begin{cases} \dfrac{dS(t)}{dt} = b - lS(t) - \alpha S(t)I_N(t) - \dfrac{\beta S(t)I_R(t)}{1+k_1 I_R(t)}, \\ \dfrac{dI_N(t)}{dt} = \alpha S(t)I_N(t) - (l+\mu)I_N(t), \\ \dfrac{dI_R(t)}{dt} = \dfrac{\beta S(t)I_R(t)}{1+k_1 I_R(t)} - (l+\gamma)I_R(t), \\ \dfrac{dR(t)}{dt} = \mu I_N(t) + \gamma I_R(t) - lR(t). \end{cases} \tag{1.1}$$

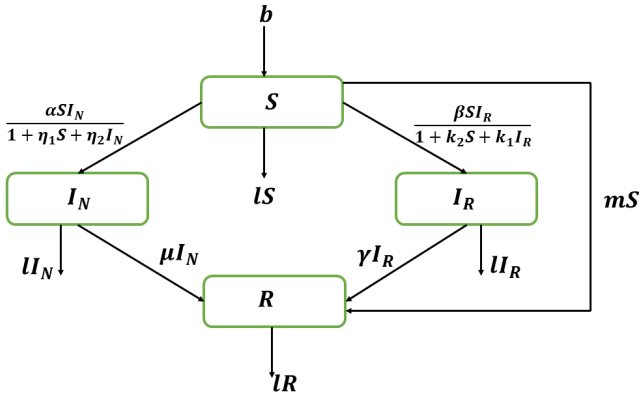


FIGURE 1. Schematic of influenza strains (1.2)

Where $S(t)$ is the group of the susceptible individuals in population, $I_N(t)$ is the group of infected non-resistant individuals, $I_R(t)$ is the group of infected resistant individuals. The parameters of the system are supposed all positive, where b is the recruitment rate of the population, l represents the natural death rate of the population, α and β are the infection rate by non-resistant strain and resistance strain, respectively. μ and γ are the recovery rates of the infective non-resistant I_N and infective resistance I_R individuals, respectively.

On the other hand, to fight against an epidemic, the vaccine has long been the most powerful means (see [37]). Therefore, we assume that susceptible individuals are vaccinated with a success rate m (with $0 \leq m \leq 1$). While $m = 1$ represents an idealized case of perfect vaccine efficacy, this assumption allows us to explore the full range of vaccination outcomes, from no protection to complete immunity, providing insight into the potential impact of vaccination on disease dynamics. We also model the transmission of the epidemic by the incidence of Beddington-DeAngelis [5]. We note that the incidence of Beddington-DeAngelis generalizes several incidences existing in the literature (see, [13, 14, 12]). Then, the following

epidemic model is considered with vaccination and Beddington-DeAngelis incidence rate

$$\begin{cases} \dfrac{dS(t)}{dt} = b - lS(t) - mS(t) - \dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - \dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}, \\ \dfrac{dI_N(t)}{dt} = \dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - (l+\mu)I_N(t), \\ \dfrac{dI_R(t)}{dt} = \dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} - (l+\gamma)I_R(t), \\ \dfrac{dR(t)}{dt} = \mu I_N(t) + mS(t) + \gamma I_R(t) - lR(t). \end{cases} \tag{1.2}$$

Noting that the three first equations of (1.2) do not depend on the fourth equation, the fourth equation can be removed without loss of generality. Therefore, we can only study the following system:

$$\begin{cases} \dfrac{dS(t)}{dt} = b - lS(t) - mS(t) - \dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - \dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}, \\ \dfrac{dI_N(t)}{dt} = \dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - (l+\mu)I_N(t), \\ \dfrac{dI_R(t)}{dt} = \dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} - (l+\gamma)I_R(t), \end{cases} \tag{1.3}$$

with initial conditions

$$S(0) \geq 0,\ I_N(0) \geq 0,\ I_R(0) \geq 0. \tag{1.4}$$

This paper aims to analyze the impact of vaccination and nonlinear incidence on the transmission dynamics of influenza involving two strains: drug-resistant and non-resistant. We derive two basic reproduction numbers, which are expressed in terms of model parameters, and use them to establish sufficient conditions for the local stability of four biologically meaningful equilibrium points. Furthermore, we formulate and analyze an optimal control problem to reduce both the number of infections and the associated treatment costs, incorporating vaccination and media awareness strategies. Unlike classical models based solely on bilinear or standard incidence, our model adopts a Beddington-DeAngelis incidence rate, which captures saturation and behavioral effects, thus providing a more realistic representation of disease spread. In particular, our framework generalizes several existing models (e.g., [32, 30]) by including nonlinear transmission dynamics and control strategies, thereby offering a broader and more flexible platform for studying the interplay between vaccination, drug resistance, and public health interventions in influenza epidemics.

The remainder of this paper is organized as follows. In Section 2, we analyze the equilibrium points of the model (1.3) and establish the existence, positivity, and boundedness of its solutions to ensure that the system is both mathematically and biologically well-posed. Section 3 is devoted to the formulation and characterization of the optimal control problem. In Section 4, we provide numerical simulations to support and illustrate our theoretical findings. Finally, the paper concludes with a summary of the main results and suggestions for future research directions.

TABLE 1. Some incidence rates

Incidence rates	**Expressions**	**References**
Standard incidence rate	$\frac{\beta SI}{N}$	[10]
Saturated incidence rate	$\frac{\beta SI}{1+\eta I}$	[8]
Beddington-DeAngelis	$\frac{\beta SI}{1+\eta_1 S+\eta_2 I}$	[5]
Crowley-Martin	$\frac{\beta SI}{1+\eta_1 S+\eta_2 I+\eta_1\eta_2 SI}$	[9]
Media coverage incidence	$\beta_1-\beta_2\frac{I}{I+M}$	[25]

2. Basic mathematical analysis

2.1. **Existence of the solution.** The proposed model below describes the dynamics of a biological population; therefore, the solutions of the system must remain non-negative and bounded. To this end, we define the following set:

$$\omega=\left\{(S,I_R,I_N)\in\mathbb{R}^3_+:\quad 0\leq S+I_R+I_N\leq\frac{b}{l}\right\}.$$

We can also verify the existence and uniqueness of the solutions of the system. To proceed, we rewrite system (1.3) as:

$$\psi(t)=AX(t)+B(X(t)),$$

where

$$X(t)=\begin{bmatrix}S(t)\\I_N(t)\\I_R(t)\\R(t)\end{bmatrix},\quad \psi(t)=\begin{bmatrix}\frac{dS(t)}{dt}\\\frac{dI_N(t)}{dt}\\\frac{dI_R(t)}{dt}\\\frac{dR(t)}{dt}\end{bmatrix},$$

$$A=\begin{bmatrix}-(l+m)&0&0&0\\0&-(l+\mu)&0&0\\0&0&-(l+\gamma)&0\\m&\mu&\gamma&-l\end{bmatrix},$$

and

$$B(X(t))=\begin{bmatrix}b-\frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)}-\frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}\\\frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)}\\\frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}\\0\end{bmatrix}.$$

There exist a Lipschtiz constant L such that

$$\|B(X_1(t))-B(X_2(t))\|\leq L\|X_1(t)-X_2(t)\|,$$

then

$$\|\psi(X_1)-\psi(X_2)\|\leq W\|X_1(t)-X_2(t)\|,$$

where $W = max(L, \|A\|) < \infty$. This shows that the function ψ is uniformly Lipschitz continuous, and with the restrictions on $S(t) \geq 0$, $I_R(t) \geq 0$, $I_N(t) \geq 0$ and $R(t) \geq 0$, we prove the existence of a solution for the system (1.3) [6].

2.2. **Positivity.** Since the population can not be non negative at any stage, the following theorem shows that every solution of (1.3), with non negative initials values (1.4) will remain in $\mathbb{R}^4_+$.

Theorem 2.1. *For any initial conditions (1.4), the solutions $S(t)$, $I_R(t)$, $I_N(t)$, $R(t)$ of system (1.3) are positive for all $t \geq 0$.*

Proof. Taking the first equation of system (1.3)

$$\begin{aligned}\frac{dS(t)}{dt} &= b - lS(t) - mS(t) - \frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - \frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} \\ &\geq -lS(t) - mS(t) - \frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - \frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)},\end{aligned}$$

then

$$S'(t) + G(t)S(t) \geq 0, \tag{2.1}$$

where

$$G(t) = l + m + \frac{\alpha I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} + \frac{\beta I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}.$$

Multiplying the two sides of (2.1) by $\exp\left(\int_0^t G(s)ds\right)$, we get

$$\exp\left(\int_0^t G(s)ds\right) S'(t) + G(t)\exp\left(\int_0^t G(s)ds\right) S(t) \geq 0,$$

then

$$\frac{d}{dt}\left[\exp\left(\int_0^t G(s)ds\right) S(t)\right] \geq 0.$$

Integrating the last inequality between 0 and t we obtain:

$$\int_0^t \frac{d}{dt}\left[\exp\left(\int_0^t G(s)ds\right) S(s)\right] ds \geq 0,$$

then

$$S(t) \geq S(0)\left[\exp\left(-\int_0^t G(s)ds\right)\right],$$

this implies $S(t) \geq 0$. In a similar way, we can prove that $I_R(t) \geq 0$, $I_N(t) \geq 0$ and $R(t) \geq 0$. □

2.3. **Boundlessness of the solution.** The following theorem presents the boundlessness of solution.

Theorem 2.2. *The set ω is positively invariant with respect to system (1.3), with the initial conditions (1.4).*

Proof. Let's assume that $N = S + I_N + I_R$, then

$$\begin{aligned}\frac{dN(t)}{dt} &= b - lS(t) - mS(t) - \frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - \frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} \\ &+ \frac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - (l+\mu)I_N(t) + \frac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} \\ &- (l+\gamma)I_R(t),\end{aligned}$$

this gives

$$\begin{aligned}\frac{dN(t)}{dt} &= b - lN(t) - mS(t) - \mu I_N(t) - \gamma I_R(t)\\ &\leq b - lN(t).\end{aligned}$$

According to Birkhoff and Rota's [6] differential inequality, we get

$$N(t) \leq \frac{b}{l} + N(0)\exp\left(-lt\right),$$

with $N(0)$ the initial condition. Letting $t \to \infty$, we obtain

$$\lim_{t\to\infty} N(t) \leq \frac{b}{l}.$$

□

Remark 2.1. According to the Theorem 2.1 and 2.2, we confirm that the problem is mathematically and biologically well defined.

2.4. **Equilibrium points.** In this section, we compute the equilibrium points of system (1.3) in order to analyze their stability. To do this, we solve the system defined by

$$\begin{aligned}&b - lS(t) - mS(t) - \frac{\alpha S(t) I_N(t)}{1+\eta_1 S(t) + \eta_2 I_N(t)} - \frac{\beta S(t) I_R(t)}{1 + k_2 S(t) + k_1 I_R(t)} = 0,\\ &\left(\frac{\alpha S(t)}{1+\eta_1 S(t) + \eta_2 I_N(t)} - (l+\mu)\right) I_N(t) = 0,\\ &\left(\frac{\beta S(t)}{1 + k_2 S(t) + k_1 I_R(t)} - (l+\gamma)\right) I_R(t) = 0.\end{aligned}$$

The equilibrium points are given as follows:

(1) Disease-free equilibrium state. The free equilibrium state stands for the absence of infection. Thus, all the infected groups will be empty, and the whole population will incorporate only susceptible. Hence, the free equilibrium of the model is giving by

$$E_0\left(\frac{b}{l+m}, 0, 0\right).$$

(2) Non-Resistant equilibrium state. The non-resistant equilibrium state stands for the absence of infection in the resistant group. Thus, we have

$$E_{NR}\left(S^*, I_N^*, 0\right),$$

such that

$$S^* = \frac{l + \mu + \eta_2(l+\mu) I_N^*}{\alpha - (l+\mu)\eta_1},$$

and

$$I_N^* = \frac{b\alpha - b(l+\mu)\eta_1 - (l+m)(l+\mu)}{\eta_2(l+m)(l+\mu) + (l+\mu)[\alpha - (l+\mu)\eta_1]},$$

which only exists when

$$\alpha - (l+\mu)\eta_1 > 0,$$

and

$$b\alpha - b(l+\mu)\eta_1 - (l+m)(l+\mu) > 0.$$

This implies

$$\alpha - (l+\mu)\eta_1 > \frac{(l+m)(l+\mu)}{b}. \tag{2.2}$$

(3) Resistant equilibrium. The resistant equilibrium state stands for the absence of infection in the non-resistant group. Thus, we have

$$E_R\left(S^*, 0, I_R^*\right),$$

such that

$$S^* = \frac{l+\gamma+k_1(l+\gamma)I_R^*}{\beta-(l+\gamma)k_2},$$

and

$$I_R^* = \frac{b\beta - b(l+\gamma)k_2 - (l+m)(l+\gamma)}{k_1(l+m)(l+\gamma)+(l+\gamma)[\beta-(l+\gamma)k_2]},$$

which only exists when

$$\beta - (l+\gamma)k_2 > 0,$$

and

$$b\beta - b(l+\gamma)k_2 - (l+m)(l+\gamma) > 0.$$

This implies

$$\beta - (l+\gamma)k_2 > \frac{(l+m)(l+\gamma)}{b}. \tag{2.3}$$

(4) Endemic equilibrium state. The endemic equilibrium state, is when the infection is present in both resistant and non-resistant compartments. Thus, we have

$$E_e\left(S^*, I_N^*, I_R^*\right),$$

such that

$$I_N^* = \frac{(\alpha-\eta_1(l+\mu))S^* - (l+\mu)}{(l+\mu)\eta_2},$$

$$I_R^* = \frac{(\beta-k_2(l+\gamma))S^* - (l+\gamma)}{(l+\gamma)k_2},$$

and

$$S^* = \frac{\eta_2 k_1 b + \eta_2(l+\gamma) + k_1(l+\mu)}{k_1\eta_2(l+m) + k_1[\alpha-(l+\mu)\eta_1] + \eta_2[\beta-(l+\gamma)k_2]},$$

which exists when

$$(\beta-k_2(l+\gamma))S^* - (l+\gamma) > 0, \tag{2.4}$$

and

$$(\alpha-\eta_1(l+\mu))S^* - (l+\mu) > 0. \tag{2.5}$$

2.5. **Basic reproduction number.** Using the next-generation matrix method developed by Van den Driessche and Watmoung [36], we can express the basic reproduction number of the system (1.3). Therefore, the infected compartments are I_N and I_R. The outflow term $\mathfrak{V}$ and the nonlinear terms with new infection $\mathfrak{F}$ are as follows:

$$\mathfrak{F} = \begin{bmatrix} \dfrac{\alpha S I_N}{1+\eta_1 S+\eta_2 I_N} \\ \dfrac{\beta S I_R}{1+k_2 S+k_1 I_R} \end{bmatrix},$$

and

$$\mathfrak{V} = \begin{bmatrix} (l+\mu) I_N \\ (l+\gamma) I_R \end{bmatrix}.$$

The Jacobian matrices $\mathcal{F}$ and $\mathcal{V}$, associated with $\mathfrak{F}$ and $\mathfrak{V}$, respectively, are given as follows:

$$\mathcal{F} = \begin{bmatrix} \frac{\alpha S(1+\eta_1 S+\eta_2 I_N)-\eta_2 \alpha S I_N}{(1+\eta_1 S+\eta_2 I_N)^2} & 0 \\ 0 & \frac{\beta S(1+k_2 S+k_1 I_R)-k_1 \beta S I_R}{(1+k_2 S+k_1 I_R)^2} \end{bmatrix},$$

and

$$\mathcal{V} = \begin{bmatrix} (l+\mu) & 0 \\ 0 & (l+\gamma) \end{bmatrix},$$

Then, the matrix $\mathcal{F} \times \mathcal{V}^{-1}$ is given as follows:

$$\mathcal{F} \times \mathcal{V}^{-1} = \begin{bmatrix} \frac{\alpha S(1+\eta_1 S+\eta_2 I_N)-\eta_2 \alpha S I_N}{(l+\mu)(1+\eta_1 S+\eta_2 I_N)^2} & 0 \\ 0 & \frac{\beta S(1+k_2 S+k_1 I_R)-k_1 \beta S I_R}{(l+\gamma)(1+k_2 S+k_1 I_R)^2} \end{bmatrix}.$$

The linearized form of the matrix $\mathcal{F} \times \mathcal{V}^{-1}$ at the equilibrium E_0 is expressed as follows:

$$(\mathcal{F} \times \mathcal{V}^{-1})(E_0) = \begin{bmatrix} \frac{\alpha b}{(l+m+\eta_1 b)(l+\mu)} & 0 \\ 0 & \frac{\beta b}{(l+m+k_2 b)(l+\gamma)} \end{bmatrix}.$$

The dominant eigenvalue of $\mathcal{F} \times \mathcal{V}^{-1}(E_0)$ represents the basic reproduction numbers, and it's given by $\mathcal{R}_0 = \rho(\mathcal{F} \times \mathcal{V}^{-1})$ (with ρ the spectrum of $\mathcal{F} \times \mathcal{V}^{-1}(E_0)$). Then,

$$\mathcal{R}_{01} = \frac{\alpha b}{(l+m+\eta_1 b)(l+\mu)},$$

or

$$\mathcal{R}_{02} = \frac{\beta b}{(l+m+k_2 b)(l+\gamma)}.$$

Remark 2.2. In deterministic epidemic models, the basic reproduction number plays a crucial role. It represents the expected number of secondary cases produced by a single infectious individual introduced into a population composed entirely of susceptible individuals, over the course of the infectious period. This threshold quantity determines whether the infection will die out when it is less than one, or lead to an epidemic outbreak when it exceeds one.

2.6. **Stability.** In this section, we prove the theorem for the stability of the equilibrium points of system (1.3).

First, we derive the Jacobian matrix of system (1.3) in terms of the variables S, I_N, I_R:

$$J = \begin{bmatrix} -(l+m)-j_{11}-j_{12} & -j_{22} & -j_{33} \\ j_{11} & j_{22}-(l+\mu) & 0 \\ j_{12} & 0 & j_{33}-(l+\gamma) \end{bmatrix},$$

where

$$\begin{cases} j_{11} = \dfrac{\alpha I_N(1+\eta_2 I_N)}{\left(1+\eta_1 S+\eta_2 I_N\right)^2}, \\ j_{12} = \dfrac{\beta I_R(1+k_1 I_R)}{\left(1+k_2 S+k_1 I_R\right)^2}, \\ j_{22} = \dfrac{\alpha S(1+\eta_1 S)}{\left(1+\eta_1 S+\eta_2 I_N\right)^2}, \\ j_{33} = \dfrac{\beta S(1+k_2 S)}{\left(1+k_2 S+k_1 I_R\right)^2}. \end{cases}$$

Theorem 2.3. *The disease-free equilibrium point E_0 is locally stable when* $\max\{\mathcal{R}_{01}, \mathcal{R}_{02}\} < 1$, *unstable when* $\min\{\mathcal{R}_{01}, \mathcal{R}_{02}\} > 1$.

Proof. For E_0, the Jacobian matrix becomes as follows:

$$J_{E_0} = \begin{bmatrix} -(l+m) & -\dfrac{\alpha b}{l+m+\eta_1 b} & -\dfrac{\beta b}{l+m+k_2 b} \\ 0 & \dfrac{\alpha b}{l+m+\eta_1 b} - (l+\mu) & 0 \\ 0 & 0 & \dfrac{\beta b}{l+m+k_2 b} - (l+\gamma) \end{bmatrix},$$

Then, the eigenvalues of J_{E_0} are

$$\begin{aligned} \lambda_1 &= -(l+m) < 0, \\ \lambda_2 &= \frac{\alpha b}{l+m+\eta_1 b} - (l+\mu) = (l+\mu)[\frac{\alpha b}{(l+m+\eta_1 b)(l+\mu)} - 1] \\ &= (l+\mu)[\mathcal{R}_{01} - 1] < 0, \\ \lambda_3 &= \frac{\beta b}{l+m+k_2 b} - (l+\gamma) = (l+\gamma)[\frac{\beta b}{(l+m+k_2 b)(l+\gamma)} - 1] \\ &= (l+\gamma)[\mathcal{R}_{02} - 1] < 0. \end{aligned}$$

According to stability theory [28], the equilibrium point E_0 is locally stable if $\lambda_2 < 0$ and $\lambda_3 < 0$. This condition is satisfied when $\mathcal{R}_{01} < 1$ and $\mathcal{R}_{02} < 1$.

□

Theorem 2.4. *The following statements hold:*

(i) *If $\mathcal{R}_{01} > 1$ and $\mathcal{R}_{02} < 1$, then the equilibrium point E_{NR} is locally stable.*
(ii) *If $\mathcal{R}_{02} > 1$ and $\mathcal{R}_{01} < 1$, then the equilibrium point E_R is locally stable.*

Proof. (i) For E_{NR}, the Jacobian matrix becomes as follows:

$$J_{E_{NR}} = \begin{bmatrix} H_{11} & H_{12} & H_{13} \\ H_{21} & H_{22} & H_{23} \\ H_{31} & H_{32} & H_{33} \end{bmatrix},$$

where

$$
\begin{aligned}
H_{11} &= -(l+m) - \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
H_{12} &= -\frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
H_{13} &= -\frac{\beta S^*}{1+k_2 S^*},\\
H_{21} &= \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
H_{22} &= -(l+\mu) + \frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
H_{23} &= 0,\\
H_{31} &= 0,\\
H_{32} &= 0,\\
H_{33} &= -(l+\gamma) + \frac{\beta S^*}{1+k_2 S^*}.
\end{aligned}
$$

The first eigenvalue of the matrix $J_{E_{NR}}$ is given by

$$
\lambda_1 = H_{33} < \frac{(\frac{l+m}{b} S^* - 1)(l+m+\gamma)}{1+k_2 S^*}.
$$

Using the inequality $b-(l+m)S^* = (l+\mu)I_N^* > 0$, we deduce that $\lambda_1 < 0$. The other two eigenvalues λ_2 and λ_3 of matrix $J_{E_{NR}}$ are the roots of the following equation:

$$
\lambda^2 - (H_{11}+H_{22})\lambda + H_{11}H_{22} - H_{21}H_{12} = 0.
$$

We must prove that the real parts of λ_2 and λ_3 are strictly negative, for this it is sufficient to show that $-(H_{11}+H_{22}) > 0$ and $H_{11}H_{22} - H_{21}H_{12} > 0$. Thus, we have

$$
\begin{aligned}
&-(H_{11}+H_{22})\\
&= l+m + \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} + l + \mu - \frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\\
&= l+m + \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} + \frac{\alpha S^*}{1+\eta_1 S^* + \eta_2 I_N^*}\frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\\
&= l+m + \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} + \frac{\alpha S^* \eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} > 0.
\end{aligned}
$$

And,

$$
\begin{aligned}
&H_{11}H_{22} - H_{21}H_{12}\\
&= \left(-(l+m) - \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\right) \times \left(-(l+\mu) + \frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\right)\\
&+ \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} \times \frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\\
&= (l+m)(l+\mu) + \frac{(l+\mu)\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} - \frac{(l+m)\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\\
&= \frac{(l+m)\alpha S^*}{1+\eta_1 S^* + \eta_2 I_N^*} + \frac{(l+\mu)\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} - \frac{(l+m)\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2}\\
&= \frac{(l+\mu)\alpha I_N^*(1+\eta_2 I_N^*) + (l+m)\alpha S^* \eta_2 I_N^*}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} > 0.
\end{aligned}
$$

According to the Routh–Hurwitz criteria [28], the eigenvalues λ_2 and λ_3 have negative real parts. Therefore, the equilibrium point E_{NR} is locally stable.

(ii) Similarly, we can prove that the equilibrium E_R of system (1.3) is locally stable if $\mathcal{R}_{01} < 1$ and $\mathcal{R}_{02} > 1$. □

Theorem 2.5.
Assume that the conditions (2.4) and (2.5) holds, if $\mathcal{R}_{01} > 1$ and $\mathcal{R}_{02} > 1$, then E_e is locally stable.

Proof. For E_e, the Jacobian matrix becomes as follows:

$$
J_{E_e} = \begin{bmatrix} K_{11} & K_{12} & K_{13} \\ K_{21} & K_{22} & K_{23} \\ K_{31} & K_{32} & K_{33} \end{bmatrix},
$$

where

$$
\begin{aligned}
K_{11} &= -(l+m) - \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} - \frac{\beta I_R^*(1+k_1 I_R^*)}{(1+k_2 S^* + k_1 I_R^*)^2}\\
&= -(l+m) - K_{21} - K_{31},\\
K_{12} &= -\frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} = -(l+\mu) - K_{22},\\
K_{13} &= -\frac{\beta S^*(1+k_2 S^*)}{(1+k_2 S^* + k_1 I_R^*)^2} = -(l+\gamma) - K_{33},\\
K_{21} &= \frac{\alpha I_N^*(1+\eta_2 I_N^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
K_{22} &= -(l+\mu) + \frac{\alpha S^*(1+\eta_1 S^*)}{(1+\eta_1 S^* + \eta_2 I_N^*)^2} = -\frac{\alpha S^* \eta_2 I_N^*}{(1+\eta_1 S^* + \eta_2 I_N^*)^2},\\
K_{23} &= 0,\\
K_{31} &= \frac{\beta I_R^*(1+k_1 I_R^*)}{(1+k_2 S^* + k_1 I_R^*)^2},\\
K_{32} &= 0,\\
K_{33} &= -(l+\gamma) + \frac{\beta S^*(1+k_2 S^*)}{(1+k_2 S^* + k_1 I_R^*)^2} = -\frac{\beta k_1 S^* I_R^*}{(1+k_2 S^* + k_1 I_R^*)^2}.
\end{aligned}
$$

The eigenvalues λ_1, λ_2 and λ_3 of the matrix J_{E_e} are the roots of the following equation:

$$\lambda^3 + q_1\lambda^2 + q_2\lambda + q_3 = 0,$$

where

$$\begin{aligned}
q_1 &= -(K_{33} + K_{11} + K_{22}),\\
q_2 &= K_{33}K_{11} + K_{22}K_{33} + K_{11}K_{22} - K_{21}K_{12} - K_{31}K_{13},\\
q_3 &= K_{33}K_{21}K_{12} + K_{31}K_{13}K_{22} - K_{33}K_{11}K_{22}.
\end{aligned}$$

According to Routh-Hurwitz criteria [28], the equilibrium E_{NR} is locally stable if $q_i > 0$, $\forall i = 1, 2, 3$ and $q_1 \times q_2 > q_3$.

$$\begin{aligned}
q_1 &= -(K_{33} + K_{11} + K_{22})\\
&= -K_{33} + K_{21} + K_{31} - K_{22} + l + m\\
&= \frac{\beta k_1 S^* I_R^*}{(1 + k_2 S^* + k_1 I_R^*)^2} + \frac{\alpha I_N^*(1 + \eta_2 I_N^*)}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} + \frac{\beta I_R^*(1 + k_1 I_R^*)}{(1 + k_2 S^* + k_1 I_R^*)^2}\\
&+ \frac{\alpha S^* \eta_2 I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} + l + m,\\
&= \frac{\alpha I_N^*(1 + \eta_2 S^* + \eta_2 I_N^*)}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} + \frac{\beta I_R^*(1 + k_1 S^* + k_1 I_R^*)}{(1 + k_2 S^* + k_1 I_R^*)^2} + l + m\\
&= q_{11} + q_{12} + l + m > 0,
\end{aligned}$$

$$\begin{aligned}
q_2 &= K_{33}K_{11} + K_{22}K_{33} + K_{11}K_{22} - K_{21}K_{12} - K_{31}K_{13},\\
&= -(l + m)K_{33} - K_{33}K_{21} + K_{33}K_{22} - (l + m)K_{22} - K_{31}K_{22}\\
&+ (l + \mu)K_{21} + (l + \gamma)K_{31}\\
&= \frac{\beta k_1 S^* I_R^* I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2 (1 + k_2 S^* + k_1 I_R^*)^2}\\
&\times (k_1(1 + \eta_2 I_N^*) + \eta_2 k_1 S^* + \eta_2(1 + k_1 I_R^*))\\
&+ \frac{\alpha I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} ((l + \mu)(1 + \eta_2 I_N^*) + (l + m)\eta_2 S^*)\\
&+ \frac{\beta I_R^*}{(1 + k_2 S^* + k_1 I_R^*)^2} ((l + m)k_1 S^* + (l + \gamma)(1 + k_1 I_R^*))\\
&= q_{21} + q_{22} + q_{23} > 0,
\end{aligned}$$

$$\begin{aligned}
q_3 &= K_{33}K_{21}K_{12} + K_{31}K_{13}K_{22} - K_{33}K_{11}K_{22}\\
&= -(l + \mu)K_{33}K_{21} - (l + \gamma)K_{31}K_{22} + (l + m)K_{33}K_{21}\\
&= (l + \mu)\frac{\beta k_1 S^* I_R^*}{(1 + k_2 S^* + k_1 I_R^*)^2} \frac{\alpha I_N^*(1 + \eta_2 I_N^*)}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2}\\
&+ (l + \gamma)\frac{\beta I_R^*(1 + k_1 I_R^*)}{(1 + k_2 S^* + k_1 I_R^*)^2} \frac{\alpha S^* \eta_2 I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2}\\
&+ (l + m)\frac{\beta k_1 S^* I_R^*}{(1 + k_2 S^* + k_1 I_R^*)^2} \frac{\alpha S^* \eta_2 I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} > 0.
\end{aligned}$$

Then $q_i > 0$, $\forall i = 1, 2, 3$.

Next, let's show that $q_1 \times q_2 - q_3 > 0$

$$\begin{aligned}
q_1 \times q_2 - q_3 &= (q_{11} + q_{12} + (l+m))(q_{21} + q_{22} + q_{23}) - q_3 \\
&= q_{11}q_{21} + q_{12}q_{21} + (l+m)q_2 + q_{22}q_{11} + q_{23}q_{12} + q_{23}q_{11} \\
&+ q_{12}q_{22} - q_3q_{23}q_{11} + q_{12}q_{22} - q_3 \\
&= \frac{\beta I_R^*}{(1+k_2S^*+k_1I_R^*)^2}\frac{\alpha I_N^*(1+\eta_2S^*+\eta_2I_N^*)}{(1+\eta_1S^*+\eta_2I_N^*)^2} \\
&\times ((l+m)k_1S^* + (l+\gamma)(1+k_1I_R^*)) \\
&+ \frac{\alpha I_N^*}{(1+\eta_1S^*+\eta_2I_N^*)^2}\frac{\beta I_R^*(1+k_1S^*+k_1I_R^*)}{(1+k_2S^*+k_1I_R^*)^2} \\
&\times ((l+\mu)(1+\eta_2I_N^*) + (l+m)\eta_2S^*) \\
&- (l+\mu)\frac{\beta k_1S^*I_R^*}{(1+k_2S^*+k_1I_R^*)^2}\frac{\alpha I_N^*(1+\eta_2I_N^*)}{(1+\eta_1S^*+\eta_2I_N^*)^2} \\
&- (l+\gamma)\frac{\beta I_R^*(1+k_1I_R^*)}{(1+k_2S^*+k_1I_R^*)^2}\frac{\alpha S^*\eta_2I_N^*}{(1+\eta_1S^*+\eta_2I_N^*)^2} \\
&- (l+m)\frac{\beta k_1S^*I_R^*}{(1+k_2S^*+k_1I_R^*)^2}\frac{\alpha S^*\eta_2I_N^*}{(1+\eta_1S^*+\eta_2I_N^*)^2} \\
&= \frac{\beta I_R^*}{(1+k_2S^*+k_1I_R^*)^2}\frac{\alpha I_N^*}{(1+\eta_1S^*+\eta_2I_N^*)^2}((l+m)k_1\eta_2S^* \\
&+ (1+\eta_2I_N^*)((l+m)k_1S^* + (l+\gamma)(1+k_1I_R^*)) \\
&+ (1+k_1I_R^*)((l+m)\eta_2S^* + (l+\mu)(1+\eta_2I_N^*))) > 0.
\end{aligned}$$

According to the Routh–Hurwitz criteria [28], the eigenvalues λ_1, λ_2 and λ_3 have negative real parts. Therefore, the equilibrium E_e is locally stable. □

3. Optimal control problem

Health problems and epidemics have always been of a great issue for governments in all countries. Several governments have proposed health intervention strategies to manage and eradicate the spread of infectious diseases. The problem is that the cost of transforming these strategies into the field is very high. Therefore, it's a must to find an efficient method that reduces the number of infections and the costs associated to it.

In this section, we investigate two optimal control strategies:

- **Treatment Control Strategy:** The first control mechanism considered is the *treatment control*, denoted by the time-dependent variable $w_1(t)$. This control represents the implementation of therapeutic interventions aimed at reducing the infectious period of individuals carrying the influenza virus, particularly in both the drug-resistant and non-resistant classes. By increasing the rate at which infected individuals receive treatment, the infectious population is expected to decline, thereby reducing overall disease transmission. Mathematically, this control enters the model through the recovery terms, effectively enhancing the transition rate from the infectious compartments to the recovered class. The goal of incorporating $w_1(t)$ is to optimize the allocation of medical resources while minimizing the total number of infections and treatment costs over a finite time horizon.
- **Media Awareness Control Strategy:** The second control strategy focuses on *media awareness campaigns*, modeled by the time-dependent control variable $w_2(t)$. This control targets

the susceptible population and aims to reduce the effective contact rate by promoting behavioral changes such as improved hygiene, social distancing, and increased vaccine uptake. Media awareness can significantly alter disease dynamics by lowering the probability of exposure and subsequent infection. In the model, $w_2(t)$ modifies the incidence function to reflect reduced transmission due to informed behavioral responses. The objective of this strategy is to suppress the spread of both influenza strains by raising public awareness, while also considering the associated costs of running such campaigns.

After considering the proposed controls $w_1(t)$ and $w_2(t)$, we get the following controlled system

$$\begin{cases} \dfrac{dS(t)}{dt} = b - lS(t) - mS(t) - (1-w_1(t))\dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} \\ \qquad\qquad - (1-w_1(t))\dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)}, \\ \dfrac{dI_N(t)}{dt} = (1-w_1(t))\dfrac{\alpha S(t)I_N(t)}{1+\eta_1 S(t)+\eta_2 I_N(t)} - (l+\mu+w_2(t))I_N(t), \\ \dfrac{dI_R(t)}{dt} = (1-w_1(t))\dfrac{\beta S(t)I_R(t)}{1+k_2 S(t)+k_1 I_R(t)} - (l+\gamma+w_2(t))I_R(t), \\ \dfrac{dR(t)}{dt} = (\mu+w_2(t))I_N(t) + mS(t) + (\gamma+w_2(t))I_R(t) - lR(t). \end{cases} \tag{3.1}$$

The aim is to minimize the following objective functional

$$J(w_1,w_2) = \int_0^T \left(I_N(t) + I_R(t) + \frac{C_1 w_1^2(t)}{2} + \frac{C_2 w_2^2(t)}{2} \right) dt, \tag{3.2}$$

where the parameters $C_1 \geq 0$ and $C_2 \geq 0$ balance the size of the terms and represent the weight factor's characterization based on the costs and benefits of the treatment. The purpose is to minimize the objective function presented in the equation (3.2) by decreasing the number of the infected. To put it differently, we are looking for an optimal control $w^* = (w_1^*, w_2^*)$ such that

$$J(w_1^*, w_2^*) = min\{J(w_1,w_2) : (w_1,w_2) \in W\}, \tag{3.3}$$

where W is the control's set defined by:

$$W = \{w = (w_1,w_2) : w_i \ mesurable, 0 \leq w_i(t) \leq 1, \ for \ i = 1,2 \ and \ t \in [0,t_f]\}.$$

3.1. **Optimal Control Existence.** According to Fliming and Rishel [16], we can obtain the existence of the optimal control w^*. Thus, the five following steps must be checked :

1. W is a nonempty set.
2. W is convex and closed.
3. The system solution is bounded by a linearity in the state and control variables.
4. The objective function integrand is convex.
5. There are $C_1 > 0$, $C_2 > 0$ and $\Psi > 1$ such that the integrand $L(S, I_N, I_R, R)$ of the objective functional satisfies

$$L(S, I_N, I_R, R) \geq C_1 + C_2(\|w_1\|^2 + \|w_2\|^2)^{\Psi/2}$$

The following theorem shows the existence of the optimal control

Theorem 3.1. *Given the controlled system (3.1). There exists an optimal control* $(w_1^*, w_2^*) \in W$ *such that*

$$J(w_1^*, w_2^*) = min\{J(w_1,w_2) : (w_1,w_2) \in W\}, \tag{3.4}$$

Proof. The existence of the optimal control can be obtained using a result by Fleming and Rishel [16], checking the above steps. According to Lukes [27]. An existence result was used to give the existence of solution of system (3.1) with bounded coefficients, which gives condition 1. By definition the control set W is convex and closed. Using the boundedness of the solution and it's linearity in w, the right side of (3.1) verify the third condition. The integrand in the objective functional (3.2) is convex on W. In addition, we can easily see that there exist a constant $\Psi > 1$ and positive numbers C_1 and $C_2 > 0$ satisfying

$$L(S, I_N, I_R, R) \geq C_1 + C_2(\|w_1\|^2 + \|w_2\|^2)^{\Psi/2}.$$

This complete the proof. □

3.2. **Optimal Control Characterization.** This subsection provides the necessary conditions for the optimal control problem using the Pontryagin's Maximum Principle [31]. In order to characterize the optimal control $w^* = (w_1^*, w_2^*)$, the Hamiltonian H is defined from the formulation of objective functional (3.2) as follows:

$$H = I_N(t) + I_R(t) + \frac{C_1}{2}w_1^2(t) + \frac{C_2}{2}w_2^2(t) + \sum_{i=1}^{4}\lambda_i g_i, \tag{3.5}$$

where g_i is the right hand side of the differential equation of each state variable of the system (3.1). Using Pontryagin's maximum principle [31], we can determine the optimal control $w^* = (w_1^*, w_2^*)$ for the system (3.1) and its associated trajectory $X^* = (S^*, I_N^*, I_R^*, R^*)^T$. Then the following theorem is stated:

Theorem 3.2. *Given the optimal control $w^* = (w_1^*, w_2^*)$ and the corresponding solution $X^* = \begin{pmatrix} S^* & I_N^* & I_R^* & R^* \end{pmatrix}^T$ of the system (3.1), there exists adjoint functions λ_1, λ_2, λ_3 and λ_4 satisfying the following equations:*

$$\begin{aligned}
\lambda_1' &= \lambda_1 l + (\lambda_1 - \lambda_4)m \\
&\quad + (\lambda_1 - \lambda_2)(1 - w_1)\frac{\alpha I_N^*(1 + \eta_1 S^* + \eta_2 I_N^*) - \alpha S^* I_N^* \eta_1}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} \\
&\quad + (\lambda_1 - \lambda_3)(1 - w_1)\frac{\beta I_R^*(1 + k_2 S^* + k_1 I_R^*) - \beta S^* I_R^* k_2}{(1 + k_2 S^* + k_1 I_R^*)^2}, \\
\lambda_2' &= -1 + \lambda_2 l + (\lambda_2 - \lambda_4)(\mu + w_2) \\
&\quad + (\lambda_1 - \lambda_2)(1 - w_1)\frac{\alpha S^*(1 + \eta_1 S^* + \eta_2 I_N^*) - \alpha S^* I_N^* \eta_2}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2}, \\
\lambda_3' &= -1 + \lambda_3 l + (\lambda_3 - \lambda_4)(\gamma + w_2) \\
&\quad + (\lambda_1 - \lambda_3)(1 - w_1)\frac{\beta S^*(1 + k_2 S^* + k_1 I_R^*) - \beta S^* I_R^* k_1}{(1 + k_2 S^* + k_1 I_R^*)^2}, \\
\lambda_4' &= \lambda_4 l,
\end{aligned}$$

with the transversal conditions at time t_f:

$$\lambda_i(t_f) = 0, \quad \forall i = 1, ..., 4.$$

Moreover, for $t \in [0, t_f]$, the optimal controls w_1^* and w_2^* are given by

$$w_1^* = min\left(1, max(0, \frac{(\lambda_2 - \lambda_1)}{C_1}\frac{\alpha S^* I_N^*}{(1 + \eta_1 S^* + \eta_2 I_N^*)} + C)\right), \tag{3.6}$$

with

$$C = \frac{(\lambda_3 - \lambda_1)}{C_1} \frac{\beta S^* I_R^*}{(1 + k_2 S^* + k_1 I_R^*)},$$

and

$$w_2^* = min\left(1, max(0, \frac{(\lambda_2 - \lambda_4) I_N^*}{C_2} + \frac{(\lambda_3 - \lambda_4) I_R^*}{C_2})\right). \tag{3.7}$$

Proof. Let the Hamiltonian H defined by (3.5). The adjoint equations and transversality conditions can be obtained by using Pontryagins Maximum Principle [31], such that

$$\begin{aligned}
\lambda_1' = -\frac{\partial H}{\partial S} &= \lambda_1 l + (\lambda_1 - \lambda_4) m \\
&\quad + (\lambda_1 - \lambda_2)(1 - w_1) \frac{\alpha I_N^* (1 + \eta_1 S^* + \eta_2 I_N^*) - \alpha S^* I_N^* \eta_1}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2} \\
&\quad + (\lambda_1 - \lambda_3)(1 - w_1) \frac{\beta I_R^* (1 + k_2 S^* + k_1 I_R^*) - \beta S^* I_R^* k_2}{(1 + k_2 S^* + k_1 I_R^*)^2}, \\
\lambda_2' = -\frac{\partial H}{\partial I_N} &= -1 + \lambda_2 l + (\lambda_2 - \lambda_4)(\mu + w_2) \\
&\quad + (\lambda_1 - \lambda_2)(1 - w_1) \frac{\alpha S^* (1 + \eta_1 S^* + \eta_2 I_N^*) - \alpha S^* I_N^* \eta_2}{(1 + \eta_1 S^* + \eta_2 I_N^*)^2}, \\
\lambda_3' = -\frac{\partial H}{\partial I_R} &= -1 + \lambda_3 l + (\lambda_3 - \lambda_4)(\gamma + w_2) \\
&\quad + (\lambda_1 - \lambda_3)(1 - w_1) \frac{\beta S^* (1 + k_2 S^* + k_1 I_R^*) - \beta S^* I_R^* k_1}{(1 + k_2 S^* + k_1 I_R^*)^2}, \\
\lambda_4' = -\frac{\partial H}{\partial R} &= \lambda_4 l,
\end{aligned}$$

with the transversal conditions at t_f given by $\lambda_1(t_f) = 0$, $\lambda_2(t_f) = 0$, $\lambda_3(t_f) = 0$ and $\lambda_4(t_f) = 0$. The optimal controls w_1^* and w_2^* can be obtained from the following optimal conditions:

$$\frac{\partial H}{\partial w_1} = 0 \quad and \quad \frac{\partial H}{\partial w_2} = 0,$$

this gives

$$\begin{aligned}
&\frac{\partial H}{\partial w_1} = C_1 w_1 + (\lambda_1 - \lambda_2) \frac{\alpha S I_N}{1 + \eta_1 S + \eta_2 I_N} + (\lambda_1 - \lambda_3) \frac{\beta S I_R}{1 + k_2 S + k_1 I_R} = 0 \quad \text{and} \\
&\frac{\partial H}{\partial w_2} = C_2 w_2 + (\lambda_4 - \lambda_2) I_N + (\lambda_4 - \lambda_3) I_R = 0.
\end{aligned}$$

Finally, we can easily obtain w_1^* and w_2^* in the form (3.6) and (3.7) respectively. □

4. Numerical Simulation

In this section, we present a series of numerical simulations conducted using Matlab to validate and illustrate the theoretical findings obtained in the previous sections. These simulations serve to confirm the analytical results concerning the stability of equilibrium points, the behavior of the model under varying parameter values, and the effectiveness of the proposed control strategies. In Table 2, we present the parameter values for the numerical simulation.

TABLE 2. Parameter values in the simulation.

Parameter	Figure 2-(a)	Figure 2-(b)	Figure 2-(c)	Figure 2-(d)
α	0.2	0.2	0.6	0.6
β	0.2	0.6	0.2	0.6
b	0.8	0.8	0.8	0.8
l	0.2	0.2	0.2	0.2
μ	0.9	0.9	0.9	0.9
γ	0.75	0.75	0.75	0.75
m	0.2	0.2	0.2	0.2
k_1	0.015	0.015	0.015	0.015
k_2	0.1	0.1	0.1	0.1
η_1	0.001	0.001	0.001	0.001
η_2	0.01	0.01	0.01	0.01
$\mathcal{R}_{01}$	$0.3629 < 1$	$0.3629 < 1$	$1.0887 > 1$	$1.0887 > 1$
$\mathcal{R}_{02}$	$0.3509 < 1$	$1.0526 > 1$	$0.3509 < 1$	$1.0526 > 1$

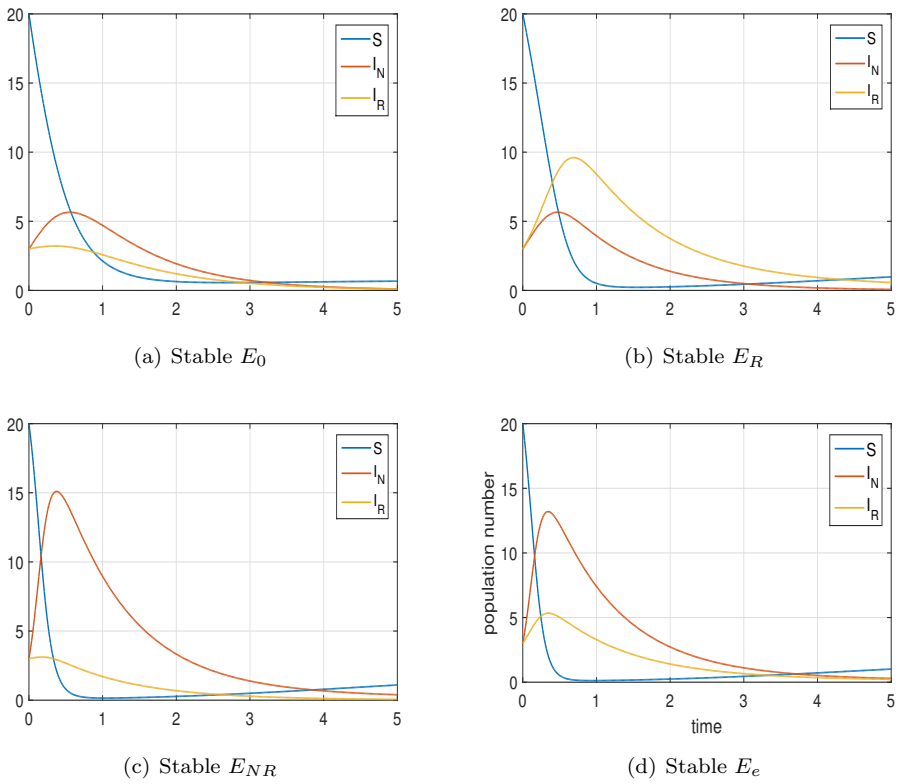
Where the initial values of each compartment are $S(0) = 20$, $I_N(0) = 3$, $I_{NR}(0) = 3$ and $R(0) = 0$. Since $\mathcal{R}_{01} = 0.3629 < 1$ and $\mathcal{R}_{02} = 0.3509 < 1$, the conditions of Theorem 2.3 are satisfied, then the free equilibrium E_0 is locally stable, Figure 2-(a) shows the results. Based on the same parameters, we take a large value of infection rate for resistant strain ($\beta = 0,6$), and by a simple computation we get $\mathcal{R}_{01} = 0.3629 < 1$ and $\mathcal{R}_{02} = 1.0526 > 1$. According to Theorem 2.4, the resistant equilibrium E_R is locally stable. Figure 2-(b) illustrate the obtained result. By increasing the rate of infection for the non-resistant strain ($\alpha = 0,6$), we get $\mathcal{R}_{01} = 1.0887 > 1$ and $\mathcal{R}_{02} = 0.3509 < 1$. Then, we see that the non-resistant equilibrium E_{NR} is locally stable (see Figure 2-(c)). Using the same parameters in Table 2, and changing the values of α and β by $0,6$ and $0,6$ respectively. We can see that the endemic equilibrium E_e is locally stable. Figure 2-(d) is a illustration of this result.

Figure 3 illustrates the effect of increasing the vaccination rate ($m = 0$, $m = 0.5$, and $m = 0.9$) on the dynamics of the different population compartments. As the vaccination rate increases, there is a notable reduction in the number of infected individuals, both resistant and non-resistant, along with a corresponding rise in the number of recovered individuals. Biologically, this demonstrates the critical role of vaccination in decreasing the susceptible population, thereby reducing the transmission potential of the disease. High vaccination coverage leads to herd immunity, effectively curbing the spread of infection and promoting disease extinction.

Figure 4 shows the impact of implementing two control strategies: treatment w_1 and media awareness campaigns w_2. When both controls are applied, the number of infected individuals in both I_R and I_{NR} compartments decreases significantly, while the number of recovered individuals increases. From a biological standpoint, treatment contributes directly to reducing the infectious period, while media awareness enhances behavioral changes such as isolation and improved hygiene, which lower transmission rates. The combined effect of these controls not only accelerates recovery but also prevents new infections, reinforcing their effectiveness in controlling and potentially eliminating the disease.

5. Conclusion

This paper proposes a mathematical model with Beddington-DeAngelis incidence rate and vaccination that describes the propagation of influenza strains in a human population. We prove the existence, uniqueness, boundlessness, and positivity of global positive solutions for the system to ensure that



(a) Stable E_0 (b) Stable E_R

(c) Stable E_{NR} (d) Stable E_e

FIGURE 2. Demonstration of the stability of the four equilibria within the respective parameter ranges. (a) Stable E_0 when parameters are taken as in column 2 in Table 2; (b) Stable E_R when parameters are taken as in column 3 in Table 2; (c) Stable E_{NR} when parameters are taken as in column 4 in Table 2; (d) Stable E_e when parameters are taken as in column 5 in Table 2.

our model is consistent with natural conditions. We establish the stability of equilibrium points and characterize stability as a function of two basic reproduction numbers, i.e.:

1. If $\max\{\mathcal{R}_{01}, \mathcal{R}_{02}\} < 1$ the disease-free equilibrium point E_0 is locally stable, and unstable when $\min\{\mathcal{R}_{01}, \mathcal{R}_{02}\} > 1$.
2. If $\mathcal{R}_{01} > 1$ and $\mathcal{R}_{02} < 1$, then the equilibrium point E_{NR} is locally stable.
3. If $\mathcal{R}_{02} > 1$ and $\mathcal{R}_{01} < 1$, then the equilibrium point E_R is locally stable.
4. If $\mathcal{R}_{01} > 1$ and $\mathcal{R}_{02} > 1$, then E_e is locally stable.

We apply an optimal control approach to reduce the number of infected individuals with influenza strains by targeting treatment and media awareness programs. The effectiveness of these strategies is illustrated through numerical simulations.

For future investigations, it is important to further refine and expand the model to better capture the complex dynamics of influenza transmission. This includes exploring the memory effect on the system dynamics through fractional derivatives, incorporating additional factors such as mutation, vaccination heterogeneity, age-structure, and stochastic effects. These extensions will improve the model's realism and its applicability for guiding public health interventions. Moreover, further studies on global stability,

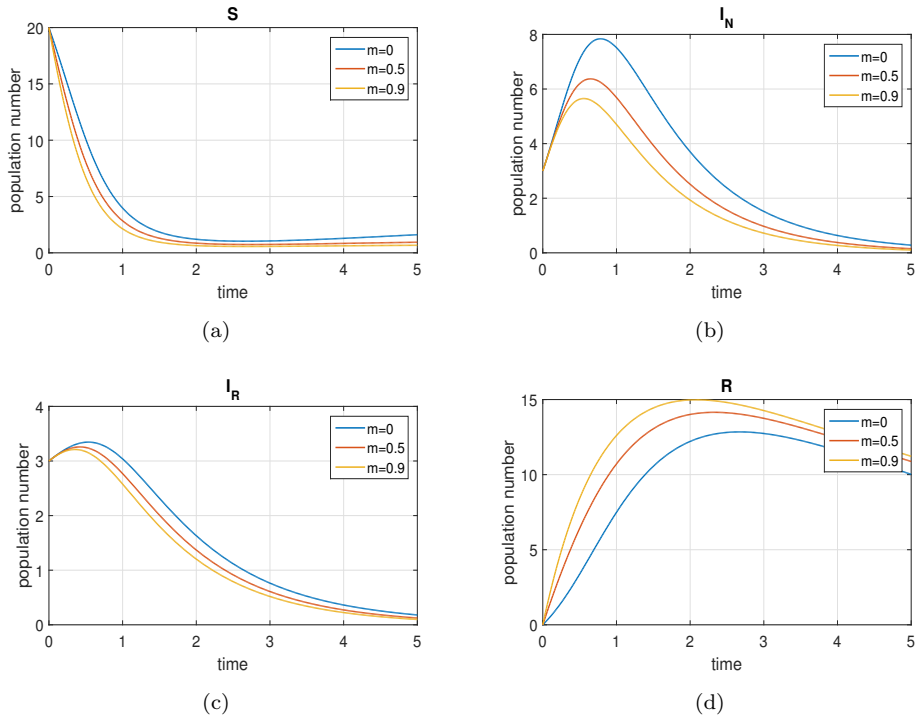
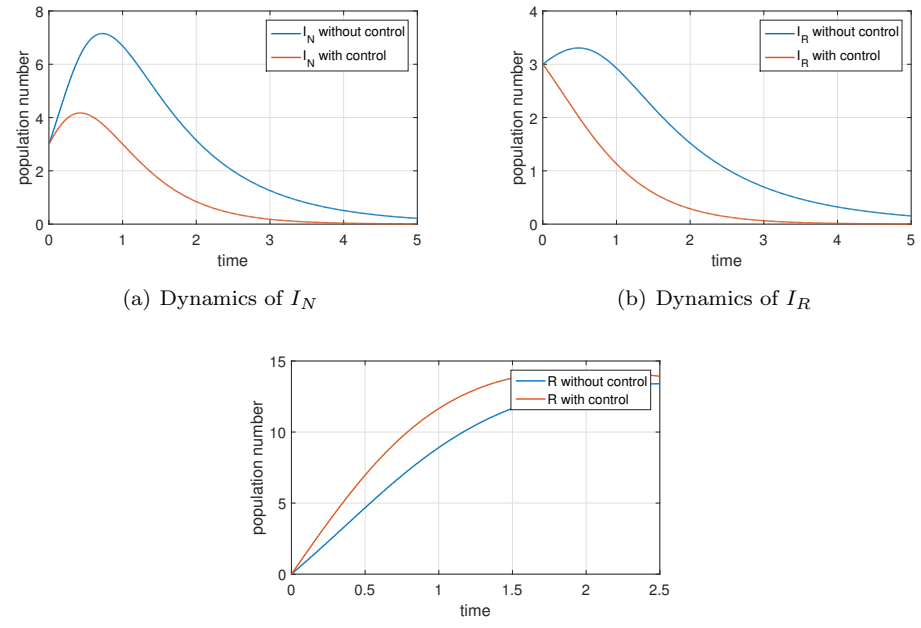


FIGURE 3. The dynamics (S, I_N, I_R, R) for various value of vaccination m.



(a) Dynamics of I_N

(b) Dynamics of I_R

(c) Dynammics of R

FIGURE 4. The dynamics ($I_N(t)$,$I_R(t)$, $R(t)$) without and with the controls w_1 and w_2.

cost-effectiveness of control measures, and incorporating parameter estimation from epidemiological data will enhance the model's predictive accuracy and practical relevance.

References

[1] M.E. Alexander and S.M. Moghadas, *Bifurcation analysis of an SIR model with vaccination-dependent non-linear incidence*, Mathematical Biosciences, **189**(2)(2004), 75–96.

[2] V. Andreasen, J. Lin and S.A. Levin, *The dynamics of cocirculating influenza strains conferring partial cross-immunity*, Journal of Mathematical Biology **35**(7)(1997), 825–842.

[3] I.A. Baba, H. Ahmad, M.D. Alsulami, K.M. Abualnaja and M. Altanji, *A mathematical model to study resistance and non-resistance strains of influenza*, Results in Physics **26** (2021), 104-390.

[4] C.A. Beauchemin and A. Handel, *A review of mathematical models of influenza A infections within a host or cell culture: lessons learned and challenges ahead*, BMC Public Health **11**(2011), 1-15.

[5] J.R. Beddington, *Mutual interference between parasites or predators and its effect on searching efficiency*, The Journal of Animal Ecology, **44**(1)(1975), 331-340.

[6] G. Birkhoff and G.C. Rota, *Ordinary Differential Equations*, Ginn, Boston, 1982.

[7] Q. Cao, Y. Liu and W. Yang, *Global dynamics of a diffusive SIR epidemic model with saturated incidence rate and discontinuous treatments*, International journal of dynamics and control. **10**(6)(2022), 1770-1777.

[8] V. Capasso and G. Serio, *A generalization of the Kermack-McKendrick deterministic epidemic model*, Mathematical Biosciences **42**(1-2)(1978), 43-61.

[9] P.H. Crowley and E.K. Martin, *Functional responses and interference within and between year classes of a dragonfly population*, J.N. Am. Benthol. Soc. **8**(3)(1989), 211-221.

[10] M.C.M. De Jong, O. Diekmann and H. Heesterbeek, *How does transmission of infection depend on population size?* in Epidemic Models: Their Structure and Relation to Data (edited by D. Mollison), Cambridge University Press, 1995.

[11] J. Dushoff, J.B. Plotkin, S.A. Levin and D.J.D. Earn, *Dynamical resonance can account for seasonality of influenza epidemics*, Proceedings of the National Academy of Sciences USA, National Academy of Sciences **101**(48)(2004), 16915–16916.

[12] A. El Koufi, O. Mourad and El Fatini, *Proposition of a novel SIRS epidemic model with double epidemics and coexisting epidemics*, International Journal of Biomathematics. **17**(2024): 2450093.

[13] A. El Koufi, *The power of delay on a stochastic epidemic model in a switching environment*, Complexity **1**(2022): 5121636.

[14] A. El Koufi, J. Adnani, A. Bennar and N. Yousfi, *Dynamics of a stochastic SIR epidemic model driven by Lévy jumps with saturated incidence rate and saturated treatment function*, Stochastic Analysis and Applications **40**(6)(2022), 1048-1066.

[15] N.M. Ferguson, A.P. Galvani and R.M. Bush, *Ecological and immunological determinants of influenza evolution*, Nature, Springer Nature **422**(6930)(2003), 428–433.

[16] W. Fleming and R. Rishel, *Deterministic and Stochastic Optimal Control*, Springer Verlag, New York, 1975.

[17] B.H. Foy, B. Wahl, K. Mehta, A. Shet, G.I. Menon and C. Britto, *Comparing COVID-19 vaccine allocation strategies in India: A mathematical modelling study*, Int J Infect Dis **103**(2021), 431-438.

[18] A. Handel, I.M. Longini and R. Antia, *Antiviral resistance and the control of pandemic influenza: the roles of stochasticity, evolution and model details*, Journal of Theoretical Biology **256**(1)(2009), 117–125.

[19] H. Hethcote, M. Zhien and L. Shengbing, *Effects of quarantine in six endemic models for infectious diseases*, Mathematical biosciences **180**(1-2)(2002), 141-160.

[20] H.W. Hethcote, *The mathematics of infectious diseases*, SIAM Review **42**(4)(2000), 599–653.

[21] V. Hutson, Y. Lou and K. Mischaikow, *Converegnce in competition models with small diffusion coefficients*, J. Diff. Eqns. **211**(2005), 135–161.

[22] W.O. Kermack, and A.G. McKendrick, *Contributions to the mathematical theory of epidemics–I*, Bulletin of mathematical biology. **53**(1-2)(1991), 33-55.

[23] M.Y. Li and P.K. Muldowney, *Global stability for the SEIR model in epidemiology*, Mathematical Biosciences **125**(2)(1995), 155–164.

[24] M. Lipsitch, T. Cohen, B. Cooper, J.M. Robins, S. Ma, L. James, G.G. Gopalakrishna, S.K. Chew, C.C. Tan, M.H. Samore, D. Fisman and M. Murray, *Transmission dynamics and control of severe acute respiratory syndrome*, Science **300**(5627)(2003), 1966–1970.

[25] Y. Liu and J.A. Cui, *The impact of media coverage on the dynamics of infectious disease*, International Journal of Biomathematics **1**(01)(2008), 65-74.

[26] I.M. Longini, M.E. Halloran, A. Nizam and Y. Yang, *Containing pandemic influenza with antiviral agents*, American Journal of Epidemiology, Oxford University Press, **159**(7)(2004), 623–633.
[27] D.L. Lukes, *Differential Equations: Classical to Controlled*, Mathematics in Science and Engineering, Academic Press, New York, Vol. 162, Academic Press, New York, USA, 1982.
[28] M. Martcheva, *An introduction to mathematical epidemiology*, Springer New York **Vol. 61**, 2015, pp. 9-31.
[29] L. Meng and W. Zhu, *Analysis of SEIR epidemic patch model with nonlinear incidence rate, vaccination and quarantine strategies*, Mathematics and Computers in Simulation **200**(2022), 489-503.
[30] J. Pang and J.A. Cui, *An SIRS epidemiological model with nonlinear incidence rate incorporating media coverage*, In 2009 Second International Conference on Information and Computing Science IEEE, **3**(2009), 116-119.
[31] L.S. Pontryagin, *Mathematical theory of optimal processes*, CRC Press, 1987.
[32] R. Prem Kumar, S. Basu, D. Ghosh, P.K. Santra and G.S. Mahapatra, *Dynamical analysis of novel COVID-19 epidemic model with non-monotonic incidence function*, Journal of Public Affairs **22**(2022): e2754.
[33] R. Su and W. Yang, *Global stability of a diffusive HCV infections epidemic model with nonlinear incidence*, Journal of Applied Mathematics and Computing **68**(4)(2022), 2685-2697.
[34] E. Tornatore, P. Vetro and S.M. Buccellato, *SIVR epidemic model with stochastic perturbation*, Neural Computing and Applications **24**(2014), 309-315.
[35] J. Truscott and N.M. Ferguson, *Evaluating the adequacy of gravity models as a description of human mobility for epidemic modelling*, PLoS Computational Biology, Public Library of Science **8**(10) (2012): e1002699.
[36] P. Van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Mathematical biosciences **180**(1-2)(2002), 29-48.
[37] F. Wang, X. Wang, S. Zhang and C. Ding, *On pulse vaccine strategy in a periodic stochastic SIR epidemic model*, Chaos, Solitons & Fractals **66**(2014), 127-135.
[38] World Health Organization, *Influenza (seasonal) fact sheet*, WHO, Geneva, Switzerland, 2025. https://www.who.int/news-room/fact-sheets/detail/influenza-(seasonal), accessed August 31, 2025.
[39] J. Xu, Y. Lei, T. Abdullah and G. Huang, *Dynamical analysis of a COVID-19 model with human-to-human and environment-to-human transmissions and distributed delays,* Mathematics in Applied Sciences and Engineering **5**(2020), 36-56.

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