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STABILITY ANALYSIS AND OPTIMAL CONTROL OF A MATHEMATICAL MODEL FOR HEROIN USE DYNAMICS

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Abstract. The widespread abuse of heroin, a potent opioid derived from morphine, continues to pose serious challenges to public health due to its high addiction potential and neurotoxic effects. In response to the growing demand for evidence-based intervention strategies, this study develops a compartmental mathematical model to better understand the transmission dynamics of heroin dependence. The model incorporates a distinct compartment for recovering individuals, explicitly accounting for the risk of relapse. Analytical investigation yields the equilibrium states of the system, and the basic reproduction number \mathcal{R}_0 is derived to characterize the threshold behavior. Stability analyses -both local and global- are conducted for the heroin-free and endemic equilibria using the Routh-Hurwitz criterion and Lyapunov functionals. A sensitivity analysis is performed to identify key parameters that influence the persistence of addiction. To evaluate possible interventions, we introduce optimal control strategies that emphasize awareness programs and nonpharmaceutical approaches. Numerical simulations carried out using MATLAB support the theoretical results and highlight the potential effectiveness of combined strategies in curbing the spread of heroin use. These findings provide a theoretical foundation for informed public health decision-making in the context of opioid addiction.

Keywords: heroin consumption; basic reproduction number; stability analysis; optimal control strategies.

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1. INTRODUCTION

Heroin is a potent pharmaceutical created from morphine. It has been assessed as contributing to huge public health problems due to its extremely addictive nature and the serious impact on the central nervous system. Heroin is found in white or brown and, less commonly, a black sticky substance. It is taken by injection, snorting or smoking and all three methods rapidly deliver the heroin to the brain, making it highly addictive[14]. The increased availability of heroin has contributed to its widespread use across many regions worldwide. Heroin poses serious health risks, including the possibility of respiratory depression, collapsed veins, infections of the heart lining and valves, liver and kidney disease. Additionally, heroin use is frequently associated with risky behaviors such as needle sharing, which significantly increases the risk of contracting infectious diseases like human immunodeficiency virus (HIV) and hepatitis [15].

Statistical data indicate the alarming prevalence of heroin use and its consequences. In the United States, heroin use has risen significantly, with an estimated 103000 new users per year. The opioid crisis has seen a dramatic increase in heroin-related overdose deaths, with approximately 14019 deaths reported annually [16]. In 2021, 60 million people used opioids for non-medical purposes, with 31.5 million using opiates, primarily heroin, according to the United Nations Office on Drugs and Crime (UNODC)[22].

Mathematical modeling is a powerful tool that allows researchers to enhance their understanding of real-world systems. Heroin addiction develops and spreads in communities like an epidemic. Therefore, developing mathematical models of heroin addiction is crucial for providing explanations and interpretations of heroin distribution. Initially, people are susceptible to heroin addiction. They then become active heroin users and pass through control measures or stop using heroin on their own. Although heroin addicts can quit, they frequently run a serious danger of relapsing. For substance use disorders, the typical relapse rate is between 40% to 60%, and for heroin specifically, it can be as high as 90% [11]. An interesting paper by White and Comiskey [5] explores the patterns and dynamics of heroin use and addiction within a population. The study looks at factors that contribute to the spread and persistence of heroin epidemics by classifying the population into three distinct groups: susceptibles, heroin users, and heroin users undergoing treatment. Mulone [13] showed that the steady states of the White

and Comiskey model are stable. The paper [10] discusses the mathematical modeling of drug use dynamics considering four classes: susceptible individuals, occasional drug users, frequent drug users, and individuals undergoing drug use therapy. In [24], the authors address the problem of analyzing the existence and stability of heroin equilibria using the second compound matrix. In [7] a mathematical model that addresses the heroin problem was formulated including a saturation function to represent the treatment dynamics for heroin users. The contribution of [8] lies in the use of a nonlinear incidence function in a very general form and provides a detailed analysis of the global dynamics of the resulting system. Moreover, a growing body of researchers have been devoted to the mathematical modeling of heroin addiction [18, 19, 4].

Heroin has a profound influence on society, and costs billions of dollars each year. It can lead to negative social effects, disrupt families, create panic in the workplace, and destroy an addict's education, often result in criminal activity [2]. Therefore, it is essential to understand the dynamics of heroin addiction and identify the most critical parameters to apply control theory techniques. This helps in determining how these epidemics can be controlled and eradicated from communities [20]. In fact, many researchers have conducted studies on the heroin epidemic using various control interventions, including single or multiple strategies. The authors of [20] construct a mathematical framework for the use of heroin and incorporate two functions as control interventions: prevention education measures and treatment. In [21] a heroin epidemic model with age structure is suggested including the treatment of active heroin users and the education of the susceptible population about heroin addiction as control measures. In [18], the authors included the recovered population in the model and looked at the treatment of heroin addiction with medication as a control variable. The authors of [23] proposed a model for marijuana consumption integrating sensitivity analysis and a comparative evaluation of optimal control strategies.

Motivated by the aforementioned reasons and studies, we propose a mathematical model of the heroin epidemic by extending the White and Comiskey model [5]. Specifically, we add a compartment representing individuals with a history of heroin use who recover either through treatment or by self-decision and who acquire temporary immunity to heroin. The total population is divided into four compartments: susceptible individuals, who have never used heroin

or are at risk of initiating use; heroin users, who actively consume heroin; individuals in therapy, representing those undergoing treatment for heroin addiction; and recovered individuals, consisting of former heroin users who have ceased heroin use either through treatment or self-decision. Recovered individuals are assumed to have temporary immunity to heroin; however, after this period, relapse may occur, and they return to the susceptible class.

This model is rigorously studied from both qualitative and quantitative perspectives. In addition to the stability analysis, we examine how the basic reproduction number responds to variations in key epidemiological parameters. Furthermore, we perform a sensitivity analysis to assess the robustness of the model predictions with respect to changes in critical parameters and to identify the factors that have the greatest impact on the system dynamics. These simulations provide a better understanding of which parameter changes are most influential in population dynamics and help identify the key aspects that must be considered when designing effective policies to address heroin consumption.

More precisely, we use numerical simulations to gain new insights into control efforts aimed at preventing heroin epidemics and to evaluate the efficacy of different intervention measures. We focus on two parameters: the heroin transmission rate and the relapse rate. Based on the principle that prevention is preferable to treatment—which can be costly and may cause side effects—we formulate an optimal control problem to minimize the number of heroin users through awareness campaigns and nonpharmaceutical interventions. Moreover, we conduct a numerical comparison of three strategies: the first combines awareness programs with nonpharmaceutical interventions; the second relies solely on awareness programs; and the third uses only nonpharmaceutical interventions. The results of the model suggest that appropriately implemented strategies can effectively limit the spread of heroin addiction.

The paper is structured as follows. In Section 2, we present the proposed model for heroin propagation and derive the basic reproduction number \mathcal{R}_0 . In Section 3, we examine the existence of equilibrium points and establish the stability of both the heroin-free and the unique endemic equilibrium. We also perform a sensitivity analysis of \mathcal{R}_0 to identify the parameters most influencing heroin transmission. Section 4 formulates the optimal control problem, emphasizing the role of the proposed intervention strategies, and proves the existence of an optimal

solution that minimizes the cost functional. In Section 5, we provide simulation results comparing scenarios with and without control measures, demonstrating the potential of these strategies to reduce the spread of heroin.

2. THE MATHEMATICAL MODEL AND EQUILIBRIA

The aforementioned works on the heroin epidemic significantly make a contribution to the current body of literature on the topic of heroin. However, most referenced studies mainly focus on the three primary compartments of the population: those who are susceptible, heroin users not undergoing treatment, and heroin users receiving treatment. In our model, we added a compartment for recovered individuals R and introduced a highlighted rate ρ representing the proportion of heroin users who become susceptible again after treatment. The overall population is made up of four compartments: S , which describes the proportion of people who are susceptible to using heroin; U_1 , which is the current population of heroin users; U_2 , which is the total number of heroin users in therapy; and R , which is the proportion of individuals who have overcome heroin addiction and have recovered. We developed a flow diagram (see 2) that depicts the population's inflow and outflow across various compartments. This visual representation allowed us to mathematically formulate the problem through the system:

$$(1) \quad \begin{cases} \frac{dS}{dt} = \Lambda - \beta_1 S U_1 - \alpha S + \rho R, \\ \frac{dU_1}{dt} = \beta_1 S U_1 - (\alpha + \varepsilon + \beta_2) U_1 + p U_2, \\ \frac{dU_2}{dt} = \beta_2 U_1 - (p + \delta + \alpha) U_2, \\ \frac{dR}{dt} = \varepsilon U_1 + \delta U_2 - (\rho + \alpha) R. \end{cases}$$

The model parameters are considered to be positive constants. The biological interpretations of these parameters are provided in Table(1).

The incidence term $\beta_1 S U_1$, known as the bilinear or mass effect infection, indicates that the heroin incidence rate increases with the number of susceptible individuals and addicts.

2.1. Nonnegativity and boundedness. In this section, we demonstrate that our system is well-posed by establishing results related to existence, positivity, and boundedness.

Theorem 1. *System (1) admits a unique solution with a positive initial condition $(S(0), U_1(0), U_2(0), R(0))$ for all time $t > 0$.*

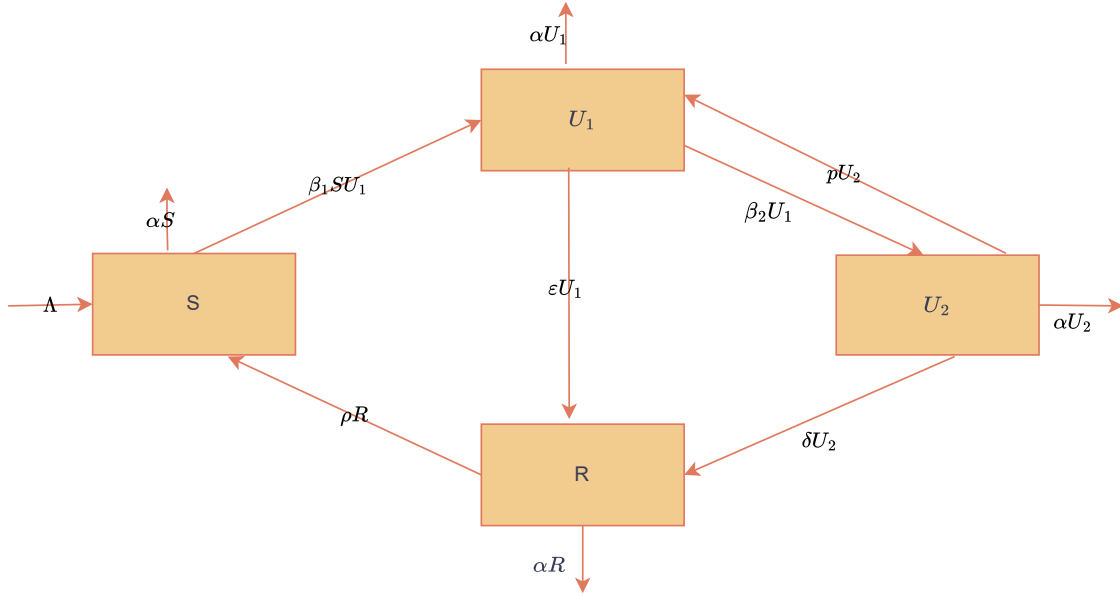


FIGURE 1. Graphical illustration of the heroin transmission model.

TABLE 1. The biological significance of the parameters in model (1)

Parameter	Description
β_1	Heroin transmission rate
β_2	Treatment rate of heroin users
α	Natural mortality rate
p	Relapse rate of people in treatment for heroin
δ	Recovery rate of treated heroin users
ϵ	Rate of personal decision to stop heroin consumption
ρ	Rate of heroin users who become susceptible after treatment
Λ	Recruitment rate of susceptibles

Proof. Suppose $\mathcal{B} = C(\mathbb{R}^+, \mathbb{R}^4)$, is the Banach space of continuous functions with the topology of uniform convergence. It is clear that the right-hand side of system (1) is completely continuous and locally Lipschitzian on \mathcal{B} . According to Cauchy-Lipschitz theorem, there is a unique solution $(S(t), U_1(t), U_2(t), R(t))$ passing through $(S(0), U_1(0), U_2(0), R(0))$ [27]. \square

Theorem 2. *The trajectories $(S(t), U_1(t), U_2(t), R(t))$ of the system (1), with positive initial condition $(S(0), U_1(0), U_2(0), R(0))$, will always be positive for every time t .*

Proof. From the equations of the system (1), we have[26]:

$$\left. \frac{dS}{dt} \right|_{S=0} = \Lambda + \rho R, \quad \left. \frac{dU_1}{dt} \right|_{U_1=0} = pU_2, \quad \left. \frac{dU_2}{dt} \right|_{U_2=0} = \beta_2 U_1, \quad \left. \frac{dR}{dt} \right|_{R=0} = \varepsilon U_1 + \delta U_2$$

we have $\left. \frac{dU_1}{dt} \right|_{U_1=0} = pU_2 \geq 0$, and hence $U_1 \geq 0$ for all $t \geq 0$. Suppose, for the sake of contradiction that U_1 or U_2 become negative, then there exists a $t_1 > 0$ with $t_1 = \inf\{t : U_1(t) = 0, t > 0\}$, such that $\left. \frac{dU_1(t_1)}{dt} \right|_{U_1(t_1)=0} = pU_2 < 0$. But we also have $U_1(t_1) = 0, U_1(t) > 0$ with $t \in [0, t_1)$ and $U_2(t_1) < 0$. Since $U_2(0) \geq 0$, there is a $t_2 > 0$ with $t_2 = \inf\{t : U_2(t) = 0, t \in [0, t_1]\}$. Hence by the definition of $t_2, \dot{U}_2(t_2) \leq 0$. But $\dot{U}_2(t_2) = \beta_2 U_1(t_2) > 0$, Which contradicts our assumption. Therefore, $U_1(t) \geq 0, U_2(t) \geq 0$ for all $t \geq 0$. Consequently, we get $R(t) \geq 0$ and $S(t) \geq 0$ for all $t > 0$. \square

Theorem 3. *The closed set $\Omega = \{(S, U_1, U_2, R) \in \mathbb{R}_+^4 : S + U_1 + U_2 + R \leq \frac{\Lambda}{\alpha}\}$ is positively invariant with respect to system (1).*

Proof. The population function can be examined as follows:

$$P(t) = S(t) + U_1(t) + U_2(t) + R(t)$$

By adding the entire system of equations in model (1)

$$(2) \quad \frac{dP}{dt} = \Lambda - \alpha P$$

The solution to equation (2) is given by

$$P(t) = \frac{\Lambda}{\alpha} + e^{-\alpha t} \left(P(0) - \frac{\Lambda}{\alpha} \right)$$

Since $P(0) = S(0) + U_1(0) + U_2(0) + R(0) \in \Omega$, thus $P(t) \leq \frac{\Lambda}{\alpha}$. \square

Remark 1. *The function P is continuously differentiable on the interval $[0, +\infty[$, and its derivative is given by:*

$$\dot{P}(t) = -\alpha e^{-\alpha t} \left(P(0) - \frac{\Lambda}{\alpha} \right)$$

Therefore, the sign of $\dot{P}(t)$ depends solely on the initial value $P(0)$.

Indeed, for $P(0) > \frac{\Lambda}{\alpha}$, the function $P(t)$ is strictly decreasing and remains between $\frac{\Lambda}{\alpha}$ and $P(0)$.

This ensures the boundedness of P , and subsequently, the boundedness of the state variables S , U_1 , U_2 , and R .

2.2. The Basic Reproduction number. The basic reproduction number is a threshold value that indicates how many secondary infections arise from introducing an infected individual into a susceptible population [6]. A threshold typically determines whether the epidemic will continue or eventually end. When it comes to heroin use, \mathcal{R}_0 provides us with the average of the total number of individuals that each heroin consumer first introduced to heroin consumption during their toxicomane career [5].

System (1) has always a heroin-free equilibrium $E_0(\frac{\Lambda}{\alpha}, 0, 0, 0)$. The basic reproduction number will be obtained [3], with

$$\mathcal{F} = \begin{pmatrix} \beta_1 S U_1 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} (\alpha + \varepsilon + \beta_2) U_1 - p U_2 \\ -\beta_2 U_1 + (p + \delta + \alpha) U_2 \end{pmatrix}$$

$$F = \begin{pmatrix} \beta_1 \frac{\Lambda}{\alpha} & 0 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} (\alpha + \varepsilon + \beta_2) & -p \\ -\beta_2 & (p + \delta + \alpha) \end{pmatrix}$$

then

$$(3) \quad \mathcal{R}_0 = \rho(FV^{-1}) = \frac{\frac{\beta_1 \Lambda}{\alpha} (p + \delta + \alpha)}{(\alpha + \varepsilon + \beta_2)(p + \delta + \alpha) - p\beta_2}$$

Theorem 4. *The system (1) has a unique endemic equilibrium $E^* = (S^*, U_1^*, U_2^*, R^*)$ if $\mathcal{R}_0 > 1$.*

Proof. We equate the equation of the system (1) to zero:

$$(4) \quad \beta_2 U_1^* - p U_2^* - \delta U_2^* - \alpha U_2^* = 0$$

Solving the above equation (4) for U_1^* , we get:

$$(5) \quad U_1^* = \frac{p + \delta + \alpha}{\beta_2} U_2^*$$

Substituting the above value in the second and the fourth equation of system (1), and equating it to zero, we get:

$$R^* = \frac{\beta_2 \delta + \varepsilon(p + \delta + \alpha)}{\beta_2(p + \alpha)} U_2^*$$

$$S^* = \frac{\Lambda\beta_2(\rho + \alpha) + \rho\varepsilon(p + \delta + \alpha)U_2^* + \rho\beta_2\delta U_2^*}{\beta_2\alpha(\rho + \alpha) + \beta_1(\rho + \alpha)(p + \delta + \alpha)U_2^*}$$

Now, using the first equation of the system, we obtain:

$$(6) \quad AU_2^* = B$$

with:

$$A = \alpha\beta_1(p + \delta + \alpha)(p + \delta + \alpha + \beta_2)(\rho + \alpha + \varepsilon)$$

$$B = \alpha\beta_2(\rho + \alpha)((\alpha + \varepsilon + \beta_2)(p + \delta + \alpha) - \beta_2p)(\mathcal{R}_0 - 1)$$

It is clear that B is nonnegative if $\mathcal{R}_0 > 1$. Furthermore, S^* , U_1^* and R^* are uniquely determined by U_2^* . It follows that there is a unique heroin-endemic equilibrium if $\mathcal{R}_0 > 1$. \square

3. STABILITY ANALYSIS OF EQUILIBRIA

This section is devoted to the local and global stability analysis of the equilibrium points.

3.1. Local stability. We will evaluate the equilibrium's local stability by evaluating the eigenvalues of the Jacobian matrices of the system(1) and applying the Routh-Hurwitz criterion[25].

Theorem 5. *The heroin-free equilibrium E_0 is asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix of the system(1) is provided as:

$$J = \begin{pmatrix} -\beta_1 U_1 - \alpha & -\beta_1 S & 0 & \rho \\ \beta_1 U_1 & \beta_1 S - (\alpha + \varepsilon + \beta_2) & p & 0 \\ 0 & \beta_2 & -(p + \delta + \alpha) & 0 \\ 0 & \varepsilon & \delta & -(\rho + \alpha) \end{pmatrix}$$

$$J_{(E_0)} = \begin{pmatrix} -\alpha & -\beta_1 \frac{\Lambda}{\alpha} & 0 & \rho \\ 0 & \beta_1 \frac{\Lambda}{\alpha} - (\alpha + \varepsilon + \beta_2) & p & 0 \\ 0 & \beta_2 & -(p + \delta + \alpha) & 0 \\ 0 & \varepsilon & \delta & -(\rho + \alpha) \end{pmatrix}$$

Then, the characteristic equation is of the form

$$(7) \det(J_{(E_0)} - \lambda I) = (\alpha + \lambda)((\rho + \alpha) + \lambda) \left\{ \left(\beta_1 \frac{\Lambda}{\alpha} - (\alpha + \varepsilon + \beta_2) - \lambda \right) (-(p + \delta + \alpha + \lambda)) - p\beta_2 \right\} = 0$$

where λ is an eigenvalue of the Jacobian matrix $J_{(E_0)}$, and I is the 4×4 identity matrix. It is easy to show that the equation (7) admits four real eigenvalues given by:

$$\lambda_1 = -\alpha, \quad \lambda_2 = -(\rho + \alpha),$$

$$\lambda_3 = \frac{C-D}{2} - \frac{1}{2} \sqrt{(C-D)^2 + 4 \cdot \beta_1 \frac{\Lambda}{\alpha} \cdot D \left(1 - \frac{1}{\mathcal{R}_0} \right)},$$

$$\lambda_4 = \frac{C-D}{2} + \frac{1}{2} \sqrt{(C-D)^2 + 4 \cdot \beta_1 \frac{\Lambda}{\alpha} \cdot D \left(1 - \frac{1}{\mathcal{R}_0} \right)},$$

where

$$C = \beta_1 \frac{\Lambda}{\alpha} - (\alpha + \varepsilon + \beta_2), \quad D = p + \delta + \alpha.$$

Since λ_1 , λ_2 , and λ_3 are negative for all biologically meaningful parameter values, the local stability of E_0 depends on the sign of λ_4 . It is easy to show that $\lambda_4 < 0$ if and only if $\mathcal{R}_0 < 1$. Therefore, the heroin-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$, and unstable when $\mathcal{R}_0 > 1$.

Then, all four eigenvalues have negative real parts, which means that E_0 is locally asymptotically stable. \square

Theorem 6. *If $\mathcal{R}_0 > 1$ the endemic equilibrium E^* is asymptotically stable.*

Proof. Indeed, the linearization of model (1) about the endemic equilibrium point gives,

$$J_{E^*} = \begin{pmatrix} -\beta_1 U_1^* - \alpha & -\beta_1 S^* & 0 & \rho \\ \beta_1 U_1^* & \beta_1 S^* - (\alpha + \varepsilon + \beta_2) & p & 0 \\ 0 & \beta_2 & -(p + \delta + \alpha) & 0 \\ 0 & \varepsilon & \delta & -(\rho + \alpha) \end{pmatrix}$$

The characteristic equation of system (1) at E^* is given by the following form:

$$\begin{vmatrix} -\beta_1 U_1^* - \alpha - \lambda & -\beta_1 S^* & 0 & \rho \\ \beta_1 U_1^* & \beta_1 S^* - (\alpha + \varepsilon + \beta_2) - \lambda & p & 0 \\ 0 & \beta_2 & -(p + \delta + \alpha) - \lambda & 0 \\ 0 & \varepsilon & \delta & -(\rho + \alpha) - \lambda \end{vmatrix} = 0$$

equivalent to,

$$\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0$$

replacing S^* by its formula, we find:

$$a_1 = p + \delta + 3\alpha + \frac{p\beta_2}{p + \delta + \alpha} + \beta_1 U_1^* + \rho > 0$$

$$\begin{aligned} a_2 &= \beta_1 U_1^* (p + \delta + 3\alpha + \varepsilon + \beta_2 + \rho) + (2\alpha + \rho) \frac{p\beta_2}{p + \delta + \alpha} + \alpha(\rho + 2\alpha + p + \delta) \\ &\quad + (\rho + \alpha)(p + \delta + \alpha) > 0 \end{aligned}$$

$$\begin{aligned} a_3 &= (\beta_1 U_1^* + \alpha)(p + \delta + \alpha)(\rho + \alpha) + \beta_1 U_1^* (\rho(\alpha + \beta_2) + \alpha(\alpha + \varepsilon + \beta_2)) \\ &\quad + p(\alpha + \varepsilon) + (\delta + \alpha)(\alpha + \varepsilon + \beta_2) > 0 \end{aligned}$$

$$a_4 = \beta_1 U_1^* \rho \alpha (p + \alpha + \delta + \beta_2) + \beta_1 U_1^* \alpha (p(\alpha + \varepsilon) + \alpha(\delta + \alpha)(\alpha + \varepsilon + \beta_2)) > 0$$

Then we have,

$$\begin{aligned} a_1 a_2 - a_3 &= (\beta_1 U_1^*)^2 (\rho + 3\alpha + \varepsilon + p + \delta + \beta_2) + \left(\frac{p\beta_2}{p + \delta + \alpha}\right)^2 (\rho + 2\alpha) \\ &\quad + \beta_1 U_1^* \{(\rho + 2\alpha + p + \delta) + p(2\alpha + \rho + \beta_2 + p + \delta) \\ &\quad + \alpha(2\rho + 5\alpha + 2p + 2\delta + \varepsilon + \beta_2) + (\delta + \alpha)(\rho + 2\alpha + p + \delta) \\ &\quad + \rho(\rho + 2\alpha + \varepsilon + p + \delta)\} + \alpha(\alpha + p + \delta)(p + \delta + 3\alpha + \rho) \\ &\quad + (\rho + \alpha)\{p\beta_2 + \rho\alpha + 2\alpha^2 + (p + \delta + \alpha)(p + \delta + 3\alpha + \rho)\} > 0 \end{aligned}$$

$$\begin{aligned} a_2 a_3 - a_1 a_4 &= (\beta_1 U_1^*)^2 (\rho + 2\alpha + \varepsilon + \beta_2 + p + \delta) \{p(\alpha + \varepsilon) + \rho\beta_2 + \alpha(\alpha + \varepsilon + \beta_2) \\ &\quad + \rho\beta_2 + (\delta + \alpha)(\alpha + \varepsilon + \beta_2)\} + (\beta_1 U_1^*)^2 \alpha \rho (\beta_2 + \rho + 2\alpha + \varepsilon) \\ &\quad + (\beta_1 U_1^* + \alpha)(\rho + \alpha) \{\beta_1 U_1^* (p + \delta + \alpha)(\rho + 3\alpha + \varepsilon + \beta_2 + p + \delta) \\ &\quad + p\beta_2(\rho + 2\alpha) + \alpha(p + \delta + \alpha)(\rho + 2\alpha + p + \delta)\} \end{aligned}$$

$$\begin{aligned}
& + \beta_1 U_1^* \alpha^2 \{ p\beta_2 + \rho + \alpha + \rho(p + \delta + \alpha)(\rho + 2\alpha + p + \delta) \\
& + (p + \delta + \alpha)(\rho + 2\alpha + p + \delta) \} + \frac{p\beta_2}{p + \delta + \alpha} \beta_1 U_1^* \{ \rho(\rho + \alpha)(\alpha + \beta_2) \\
& + \alpha(\rho + 2\alpha)(\alpha + \varepsilon + \beta_2) + \alpha p(\alpha + \varepsilon) + p\rho\varepsilon + \rho\delta(\varepsilon + \beta_2) + \rho\alpha(\alpha + \varepsilon + \beta_2) \\
& + \alpha(\delta + \alpha)(\alpha + \varepsilon + \beta_2) \} + \beta_1 U_1^* (\rho + \alpha)(p + \delta + \alpha) \{ \alpha(\alpha + \varepsilon + \beta_2) \\
& + p(\alpha + \varepsilon) + (\delta + \alpha)(\alpha + \varepsilon + \beta_2) \} \\
& + \beta_1 U_1^* (p + \delta + \alpha)(\rho + \alpha) \{ (p + \delta + \alpha)(\rho + \alpha) + \rho\alpha \} \\
& + \beta_1 U_1^* \rho\beta_2 \{ \alpha(p + \delta) + \rho(p + \delta + \alpha) \} \\
& + \alpha(\rho + \alpha)(p + \delta + \alpha) \{ (\rho + \alpha)(p + \delta + \alpha) + \alpha(\rho + 2\alpha + p + \delta) \} > 0.
\end{aligned}$$

By the Routh-Hurwitz criterion [25], it follows that the endemic equilibrium E^* is locally asymptotically stable. \square

3.2. Global stability. In this part, we examine the global stability of the heroin-free and endemic stable states by building an appropriate Lyapunov function. First, we focus on the heroin-free equilibrium's global stability.

Theorem 7. *If $\mathcal{R}_0 \leq 1$, the heroin-free equilibrium E_0 of system (1) is globally asymptotically stable.*

Proof. Consider the following Lyapunov function:

$$V = (p + \delta + \alpha)U_1 + pU_2$$

then,

$$\begin{aligned}
\dot{V} & = ((p + \delta + \alpha)\beta_1 S - (\alpha + \varepsilon + \beta_2) + p\beta_2)U_1 \\
& \leq (\beta_1 \frac{\Lambda}{\alpha} (p + \delta + \alpha) - (\alpha + \varepsilon + \beta_2)((p + \delta + \alpha) + p\beta_2))U_1 \\
& \leq (\alpha + \varepsilon + \beta_2)((p + \delta + \alpha) + p\beta_2)(\mathcal{R}_0 - 1)U_1 \\
& \leq 0 \quad \text{if } \mathcal{R}_0 \leq 1
\end{aligned}$$

The largest compact invariant set in $\{(S, U_1, U_2, R), / \frac{dV}{dt} = 0\}$ is the singleton E_0 . Applying

LaSalle's invariance principle [1], we conclude that the heroin-free equilibrium E_0 of system (1) is globally asymptotically stable in Ω if $\mathcal{R}_0 \leq 1$. \square

Theorem 8. *If $\mathcal{R}_0 > 1$ and $\rho = 0$, the endemic equilibrium E^* is globally stable.*

Proof. Examine the Lyapunov function given by:

$$V = (S - S^* - S^* \ln \frac{S}{S^*}) + (U_1 - U_1^* - U_1^* \ln \frac{U_1}{U_1^*}) + \frac{pU_2^*}{\beta_2 U_1^*} (U_2 - U_2^* - U_2^* \ln \frac{U_2}{U_2^*})$$

Therefore, the derivative form of the function V according to time is given as:

$$\begin{aligned} \dot{V} &= (1 - \frac{S^*}{S}) \frac{dS}{dt} + (1 - \frac{U_1^*}{U_1}) \frac{dU_1}{dt} + \frac{pU_2^*}{\beta_2 U_1^*} (1 - \frac{U_2^*}{U_2}) \frac{dU_2}{dt} \\ \dot{V} &= (1 - \frac{S^*}{S}) [\Lambda - \beta_1 S U_1 - \alpha S + \rho R] + (1 - \frac{U_1^*}{U_1}) [\beta_1 S U_1 + p U_2 - (\alpha + \varepsilon + \beta_2) U_1] \\ &\quad + \frac{pU_2^*}{\beta_2 U_1^*} (1 - \frac{U_2^*}{U_2}) [\beta_2 U_1 - (p + \delta + \alpha) U_2] \\ &= (1 - \frac{S^*}{S}) [\beta_1 S^* U_1^* + \alpha S^* - \rho R^* - \beta_1 S U_1 - \alpha S + \rho R] \\ &\quad + (1 - \frac{U_1^*}{U_1}) [\beta_1 S U_1 + p U_2 - (\frac{\beta_1 S^* U_1^* + p U_2^*}{U_1^*}) U_1] + \frac{pU_2^*}{\beta_2 U_1^*} (1 - \frac{U_2^*}{U_2}) [\beta_2 U_1 - \frac{\beta_2 U_1^*}{U_2^*} U_2] \end{aligned}$$

Hence if $\rho = 0$, we have:

$$\dot{V} = \beta_1 S^* U_1^* (2 - \frac{S}{S^*} - \frac{S^*}{S}) + \alpha S^* (2 - \frac{S}{S^*} - \frac{S^*}{S}) + p U_2^* (2 - \frac{U_2}{U_1} - \frac{U_1}{U_2}) \leq 0$$

On the other hand, the set $\{(S, U_1, U_2, R), / \frac{dV}{dt} = 0\}$ is the singleton (S^*, U_1^*, U_2^*, R^*) . Then, by LaSalle's invariance principle[1], E^* is globally asymptotically stable when $\mathcal{R}_0 > 1$. \square

Remark 2. *It is possible for condition $\rho = 0$ might not always hold, meaning the global stability of the heroin-endemic equilibrium E^* could be unproven. However, Figure (3) indicates that the heroin-endemic equilibrium of the system (1) remains globally asymptotically stable even in this context.*

3.3. Sensitivity analysis of \mathcal{R}_0 . Our objective now is to identify the parameters that influence the spread of heroin use. In the context of our model, we perform a sensitivity analysis to investigate how the basic reproduction number \mathcal{R}_0 responds to variations in key model parameters. The value of \mathcal{R}_0 in system(1) is influenced by seven parameters, the per capita contact

rate β_1 , the treatment rate of heroin users β_2 , the relapse rate p , the recovery rate δ of treated individuals, the personal decision rate ε to stop heroin use, the natural mortality rate α , and the recruitment rate Λ . Among these, α and Λ are considered uncontrollable.

To quantify the impact of the remaining parameters on \mathcal{R}_0 , we compute the normalized forward sensitivity indices, following the methodology introduced by Arriola and Hyman [12] and Chitnis et al. [17].

$$\begin{aligned}\chi_{\beta_1}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \beta_1} \frac{\beta_1}{\mathcal{R}_0} = 1. \\ \chi_p^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0} = \frac{\alpha \beta_2 p (\delta + \alpha)}{(p + \delta + \alpha)(\alpha(\alpha + \varepsilon)(p + \delta + \alpha) + \alpha \beta_2 p (\delta + \alpha))}. \\ \chi_{\beta_2}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \beta_2} \frac{\beta_2}{\mathcal{R}_0} = \frac{-\alpha \beta_2 p (\delta + \alpha)}{\alpha(\alpha + \varepsilon)(p + \delta + \alpha) + \alpha \beta_2 p (\delta + \alpha)}. \\ \chi_{\varepsilon}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \varepsilon} \frac{\varepsilon}{\mathcal{R}_0} = \frac{-\varepsilon(p + \delta + \alpha)}{(\alpha + \varepsilon + \beta_2)(p + \delta + \alpha) - p \beta_2}. \\ \chi_{\delta}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \delta} \frac{\delta}{\mathcal{R}_0} = \frac{-p \delta \beta_2}{((\alpha + \varepsilon + \beta_2)(p + \delta + \alpha) - p \beta_2)(p + \delta + \alpha)}.\end{aligned}$$

These sensitivity indices provide a means to quantify the relative impact of parameter variations on the basic reproduction number \mathcal{R}_0 . Among all influencing parameters, particular attention is directed toward β_1 and p , which are selected as control variables in our model. An increase in β_1 leads to a proportional rise in \mathcal{R}_0 , while p also contributes positively to its growth, though to a lesser extent.

In contrast, β_2 , ε , and δ exhibit an inverse relationship with \mathcal{R}_0 : increasing any of these parameters results in a corresponding decrease in the reproduction number.

These results reveal that even slight perturbations in β_1 or p can lead to significant shifts in \mathcal{R}_0 , underlining the necessity of precise parameter tuning and targeted interventions in the design of effective mitigation strategies.

4. THE OPTIMAL CONTROL PROBLEM

Optimal control theory [9] is widely used as a feasible and efficient option for decision makers to develop and simulate control strategies. According to the results of \mathcal{R}_0 's sensitivity analysis, and in order to control the spread of the heroin epidemic, it is imperative to focus more on reducing β_1 , as it is the most influential parameter with a strong positive impact on

the model output. Additionally, particular attention should be given to the parameter p , since long-term relapse from individuals in treatment back to active heroin use contributes to the persistence of heroin users within the community. Therefore, in our model (1) we formulate a set of control variables $(c_1(t), c_2(t))$ to minimize the total number of heroin users and the associated cost through awareness campaigns and the implementation of nonpharmaceutical interventions, where:

- c_1 represents the effect of educational campaigns in social media, television, newspapers... This encourages people to participate in preventive programs, and its objective is to reduce the infection contact rate and improve the optimal control of heroin spread. This control variable will be incorporated into the incidence term as $(1 - c_1(t))$, since an increase in control reduces the level of interaction.
- c_2 represents the implementation of nonpharmaceutical interventions, such as social reintegration, that help patients rebuild or establish positive relationships and develop a structured routine. The goal is to reduce the rate of relapse p of people who receive heroin use therapy.

According to the previous discussion, we get the acceptable set of control variables as follows:

$$U_{ad} = \{(c_1, c_2) : \text{mesurable } 0 \leq c_1(t) \leq 1, \quad 0 \leq c_2(t) \leq p_{max} \leq 1 \quad 0 \leq t \leq T_f\}$$

The control is completely ineffective when $c_j(t) = 0 \quad j = 1, 2$ and completely effective when $c_1(t) = 1$ and $c_2(t) = p_{max}$, where p_{max} indicates the maximum proportion of individuals in therapy who relapse. T_f represents the final time.

Firstly, we define the total cost functional to be minimized by implementing the proposed control strategies. The objective is to reduce the number of active heroin users while accounting for the costs associated with applying awareness campaigns and nonpharmaceutical interventions.

$$J(c_1(t), c_2(t)) = \int_0^{T_f} (A_1 U_1(t) + A_2 c_1^2(t) + A_3 c_2^2(t)) dt,$$

where A_1 , A_2 , and A_3 are positive weighting parameters that reflect the relative importance of the respective terms in the cost functional. A quadratic cost on the control variables is considered, which is a common choice in optimal control theory due to its convexity and ability to

capture the nonlinear nature of the effort or resources required to implement awareness campaigns and nonpharmaceutical interventions.

We then examine the sufficient conditions for the existence of an optimal control pair $(c_1^*(t), c_2^*(t))$ that minimizes J subject to the system dynamics:

$$(8) \quad \begin{aligned} \frac{dS}{dt} &= \Lambda - (1 - c_1(t))\beta_1 S U_1 - \alpha S + \rho R, \\ \frac{dU_1}{dt} &= (1 - c_1(t))\beta_1 S U_1 - (\alpha + \varepsilon + \beta_2)U_1 + (p - c_2(t))U_2, \\ \frac{dU_2}{dt} &= \beta_2 U_1 - (p + \delta + \alpha)U_2, \\ \frac{dR}{dt} &= (\delta + c_2(t))U_2 + \varepsilon U_1 - (\rho + \alpha)R. \end{aligned}$$

with initial conditions:

$$(S(0), U_1(0), U_2(0), R(0)) \in \mathbb{R}_+^4.$$

Theorem 9. *There exists an optimal control (c_1^*, c_2^*) such that*

$$J((c_1^*, c_2^*)) = \min_{U_{ad}} J((c_1, c_2))$$

Proof. The existence of an optimal control pair can be readily established by verifying that:

- (1) The state variables and the control are nonnegative and belong to a nonempty set.
- (2) U_{ad} is closed and convex.
- (3) Model system (8) is linear in control variables c_1 and c_2 with coefficients dependent on state variables.
- (4) $L(S, U_1, U_2, R, (c_1, c_2)) = A_1 U_1(t) + A_2 c_1^2(t) + A_3 c_2^2(t) \geq A_2 c_1^2(t) + A_3 c_2^2(t)$

We find that our control set U_{ad} and objective function J satisfy all the assumptions that guarantee the existence of an optimal system [28]. So there exists a control (c_1, c_2) such that $J((c_1^*, c_2^*)) = \min(J(c_1, c_2))$. \square

In the following, we characterize the optimal control of the system with the aid of Pontryagin's Maximum Principle [9].

Theorem 10. *Let (c_1^*, c_2^*) be the optimal control and $\tilde{S}, \tilde{U}_1, \tilde{U}_2, \tilde{R}$ the corresponding optimal states of the control system (8). Then there exists an adjoint variable $\theta = (\theta_1, \theta_2, \theta_3, \theta_4)$ that*

satisfies:

$$(9) \quad \left\{ \begin{array}{l} \frac{d\theta_1}{dt} = (\theta_1 - \theta_2)(1 - c_1)\beta_1 U_1 + \theta_1 \alpha, \\ \frac{d\theta_2}{dt} = -A_1 + (\theta_1 - \theta_2)(1 - c_1)\beta_1 S + \theta_2(\alpha + \varepsilon + \beta_2) - \theta_3\beta_2 - \theta_4\varepsilon, \\ \frac{d\theta_3}{dt} = -\theta_2(p - c_2) + \theta_3(p + \delta + \alpha) - \theta_4(\delta + c_2), \\ \frac{d\theta_4}{dt} = -\theta_1\rho + \theta_4(\rho + \alpha), \\ \theta_i(T_f) = 0 \quad \text{for } i = 1, 2, 3, 4. \end{array} \right.$$

The optimal control pair (c_1^*, c_2^*) is characterized by:

$$c_1^*(t) = \frac{\beta_1 \tilde{S} \tilde{U}_1}{2A_2} (\theta_2 - \theta_1).$$

$$c_2^*(t) = \frac{\tilde{U}_2}{2A_3} (\theta_2 - \theta_4).$$

From the characteristics of the control set, we get:

$$c_1^*(t) = \min\left\{\max\left\{0, \frac{\beta_1 \tilde{S} \tilde{U}_1}{2A_2} (\theta_2 - \theta_1), 1\right\}\right\}.$$

$$c_2^*(t) = \min\left\{\max\left\{0, \frac{\tilde{U}_2}{2A_3} (\theta_2 - \theta_4)\right\}, p_{max}\right\}.$$

Proof. We define the Hamiltonian as:

$$H(S, U_1, U_2, R, (c_1, c_2), \theta) = L(S, U_1, U_2, R, (c_1, c_2)) + \theta_1 \dot{S} + \theta_2 \dot{U}_1 + \theta_3 \dot{U}_2 + \theta_4 \dot{R}$$

where L is the Lagrangian function. Let (c_1^*, c_2^*) be the optimal control and $\tilde{S}, \tilde{U}_1, \tilde{U}_2, \tilde{R}$ the corresponding optimal state variables.

From Pontryagin's maximum principle, there exist functions $\theta_1, \theta_2, \theta_3, \theta_4$ that satisfy the adjoint equations:

$$\frac{d\theta_1}{dt} = -\frac{\partial H}{\partial S} \quad \frac{d\theta_2}{dt} = -\frac{\partial H}{\partial U_1} \quad \frac{d\theta_3}{dt} = -\frac{\partial H}{\partial U_2} \quad \frac{d\theta_4}{dt} = -\frac{\partial H}{\partial R}$$

with transversality conditions $\theta_i(T_f) = 0 \quad i = 1, 2, 3, 4$. Therefore, we obtain the adjoint system

(9). Finally, by applying optimality condition $\frac{\partial H}{\partial c_j}|_{c_j=c_j^*} = 0 \quad \text{for } j \in \{1, 2\}$, we get:

$$c_1^*(t) = \frac{\beta_1 \tilde{S} \tilde{U}_1}{2A_2} (\theta_2 - \theta_1).$$

$$c_2^*(t) = \frac{\tilde{U}_2}{2A_3} (\theta_2 - \theta_4).$$

□

5. NUMERICAL SIMULATIONS AND INTERPRETATIONS

In this section devoted to numerical simulations, we aim to validate the theoretical results regarding the stability of equilibrium points for both cases where the basic reproduction number \mathcal{R}_0 is less than or greater than 1. Since control measures are only meaningful when heroin transmission persists, we will focus particularly on the case where $\mathcal{R}_0 > 1$. We will illustrate the sensitivity of this threshold to various parameters and numerically implement an optimal control strategy for this scenario.

5.1. Stability. First, we consider the case when, $\mathcal{R}_0 < 1$ by using the values given in the table (2). The dynamics of the model (1) is presented in (2). This figure shows that the susceptible population S persists and invariably converges to $\frac{\Lambda}{\alpha}$ for different initial conditions. While heroin users U_1 , heroin users in treatment U_2 and recovered R decline to zero. Under this condition, we examine numerically the stability of the heroin-free equilibrium E_0 .

Parameter	Value	References
Λ	1.5	[7]
α	0.01	[7]
β_1	0.001	[7]
δ	0.1	[7]
β_2	0.9	[7]
p	0.467	[7]
ε	0.015	[7]
ρ	0.01	Assumed

TABLE 2. Model parameters at heroin-free equilibrium

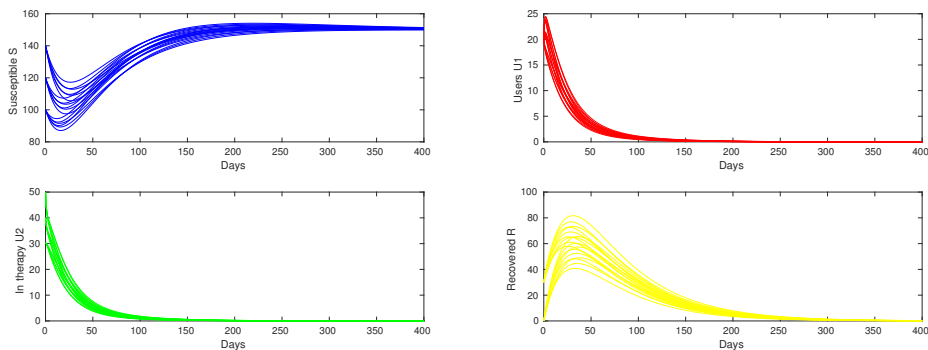


FIGURE 2. State variables of system (2) with parameters as presented in Table (2).

Next, we focus on the case when $\mathcal{R}_0 > 1$, keeping the same parameter values as those given in table (2) except for $\beta_1 = 0.002$. In this case, $\mathcal{R}_0 = 1.5261$. The dynamics of the model are presented in (3). This figure shows that the susceptible population S , heroin users U_1 , heroin users in treatment U_2 , and the temporarily recovered class R persist. We observed that the epidemic initially spreads rapidly, causing a decline in the number of susceptible individuals S and stabilizes at a lower value, while the other compartments increase before stabilizing. In summary, the heroin epidemic spreads rapidly at first before reaching equilibrium. This case will be referred to as the uncontrolled scenario.

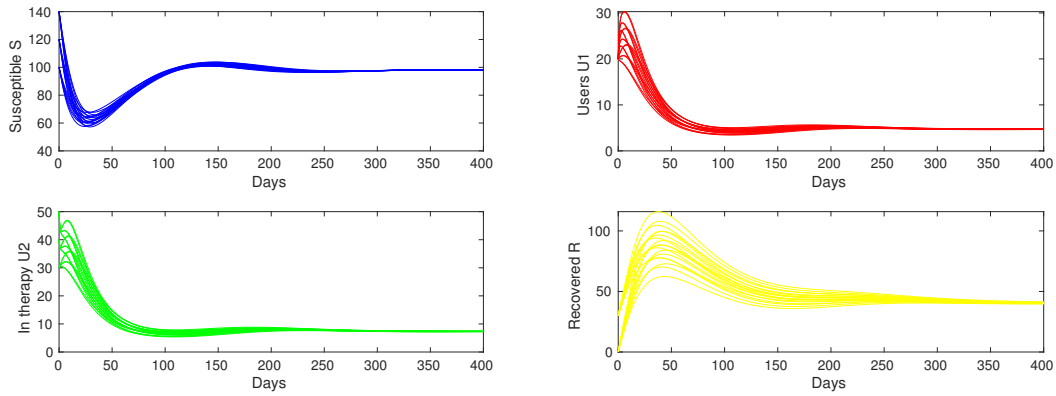


FIGURE 3. State variables of system (2) when $\mathcal{R}_0 > 1$, with different initial conditions.

5.2. Sensitivity.

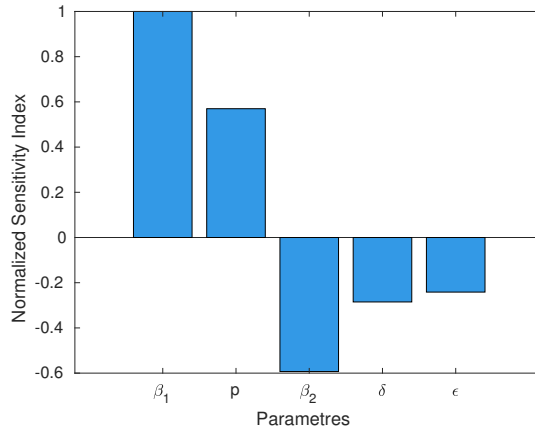


FIGURE 4. the tornado plot of \mathcal{R}_0 .

(8) presents a plot quantifying the sensitivity of the basic reproduction number \mathcal{R}_0 to different parameters in its expression, using the same values as in the previous subsection corresponding to the case where $\mathcal{R}_0 > 1$. Each bar represents the impact of varying one parameter at a time on the resulting value of \mathcal{R}_0 . As confirmed by the theoretical analysis in Section (3.3), p and β_1 , representing the heroin transmission rate and the relapse rate, respectively, have the most significant positive influence, highlighting their key role in sustaining heroin transmission.

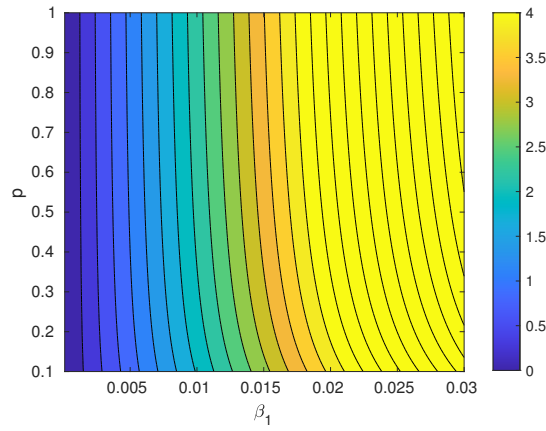


FIGURE 5. The contour plot of \mathcal{R}_0 as a function of β_1 and p .

The (5) shows a contour plot illustrating the variation of \mathcal{R}_0 as a function of the two most influential parameters, β_1 and p , and how its value changes across their defined ranges. Specifically, the figure explores the behavior of \mathcal{R}_0 when p varies from 0.1 to 1 and β_1 ranges from 0 to 0.03. The contour lines and color shading provide a clear and detailed visualization of the magnitude of \mathcal{R}_0 and its sensitivity to variations in these key parameters.

5.3. Optimal control. The next objective is to compare the dynamic behavior of the population without control and under three distinct control strategies: implementing only educational campaigns, only nonpharmaceutical interventions, and a combination of both. Furthermore, the effectiveness of each strategy is assessed by comparing their respective impacts. To this end, the optimality system (9) was solved numerically, and the results are presented graphically using the same parameter values as in the previous simulations, corresponding to the case where $\mathcal{R}_0 = 1.5261 > 1$.

The weight values are $A_1 = 1$, $A_2 = 1$ and $A_3 = 1000$ assuming that nonpharmaceutical interventions are significantly more expensive per individual than awareness programs due to the high rate of relapse, their repetitive nature, and the need for qualified personnel, the value of A_3 is considered to be higher than that of A_2 . The simulations are performed with the initial conditions, $S(0) = 140, U_1(0) = 50, U_2(0) = 30$ and $R(0) = 5$. (6) is considered as a case where no control is implemented. The susceptible population S shows a sudden decline around 50, followed by an increase after day 30, eventually stabilizing with oscillations around 100 individuals. In contrast, the recovered population R , initially increases toward 110 individuals, then declines to reach approximately 50 individuals by day 160.

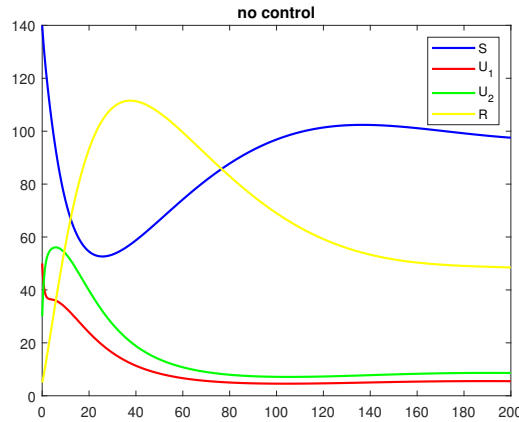


FIGURE 6. The behavior of the variables with no control.

We proceed with a comparative analysis of the numerical results obtained from three possible strategies to control the spread of heroin.

5.3.1. Strategy 1: Implementation of single control $c_1(t)$ only. With this strategy, we consider only awareness campaigns control $c_1(t)$ to minimize the objective function $J(c_1, c_2)$ while the nonpharmaceutical interventions control $c_2(t)$ is set to zero. In (7) the number of susceptible individuals S increased significantly, reaching 160 around day 100. In contrast, the number of heroin users U_1 exhibits a sharp decline and approaches zero starting from day 50. However, the population R increases rapidly at first, reaching 50 on day 40, then decreases and remains at a lower level as time passes, stabilizing at nearly zero on day 200 compared with the case of

no control. We conclude that awareness programs play a crucial role in reducing the population of heroin users. (8) shows the profile of optimal control c_1 . Initially, the intensity of the control intervention is relatively high, indicating a significant mobilization of resources aimed at achieving rapid impact. However, this intensity gradually decreases over time. This trend reflects the fact that effectively raising awareness within the target population requires a certain duration. Indeed, awareness of the harmful effects of regular heroin use does not occur instantly. It requires continuous efforts and repeated exposure to prevention messages.

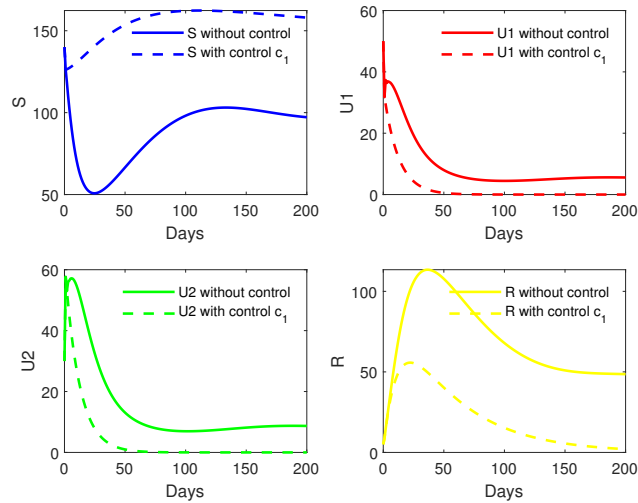


FIGURE 7. The behavior of the variables without control and with control $c_1(t)$

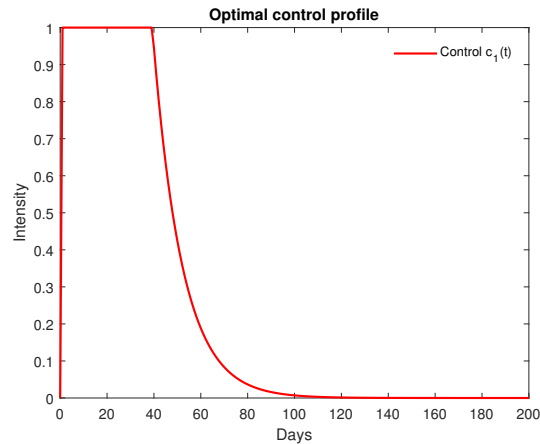


FIGURE 8. profile of control $c_1(t)$

5.3.2. Strategy 2: Implementation of single control $c_2(t)$ only. In this case, we consider only the nonpharmaceutical interventions control c_2 to minimize the objective function $J(c_1, c_2)$. In this case, (9), we observe that the susceptible population S initially decreased significantly, reaching around 76 on day 30, before increasing to 140 on day 170. However, the population U_1 decreases rapidly and stabilizes near zero starting around day 100. The number of recovered individuals R increases gradually, reaching a peak of 108 around day 28, after which it stabilizes at approximately 20 beyond day 200. The intensity profile of control c_2 is given in (10). It shows that the intensity of the control c_2 gradually decreases over time and eventually drops to zero.

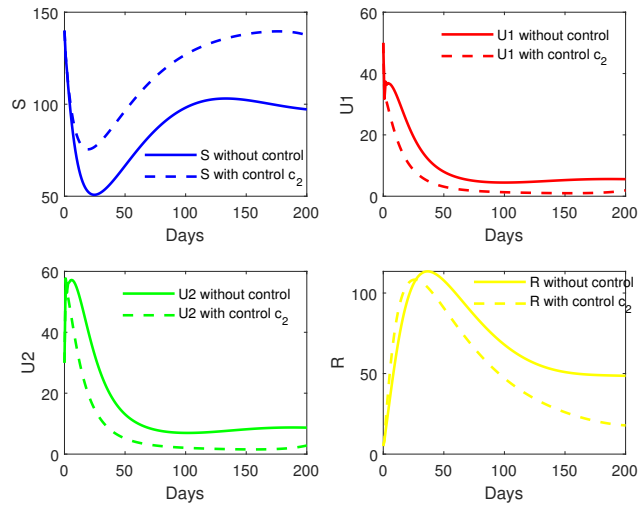


FIGURE 9. The behavior of the variables without control and with control $c_2(t)$.

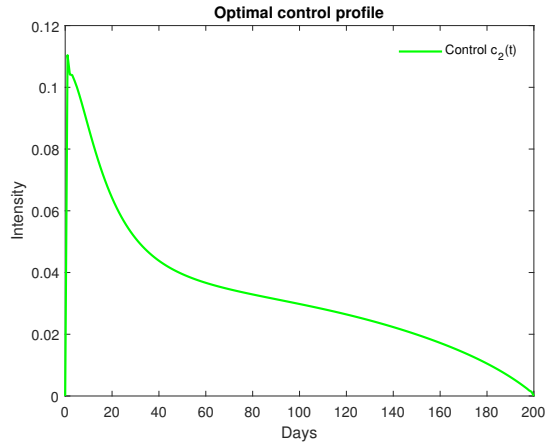


FIGURE 10. profile of control $c_2(t)$.

5.3.3. Strategy 3: Combination of control $c_1(t)$ and $c_2(t)$. In this strategy, we combine awareness campaigns and nonpharmaceutical interventions (c_1, c_2) . The simulations are extended using the same parameter values as in the previous two cases. The dynamics of population densities are illustrated in (11). We observed that the susceptible population S gradually increases to reach a value of 160 around day 70. However, the number of heroin users U_1 fell dramatically and eventually stabilized around zero around day 20. The population R increases to a plateau of approximately 60 individuals on day 30, before declining and stabilizing near zero on day 180. The corresponding optimal control profiles are given in (12), The control c_1 is activated immediately and reaches nearly 1. It then decreases rapidly, indicating that the model recommends a strong initial intervention to reduce the rate of new heroin users. As for c_2 , it starts to increase later and reaches a plateau around the maximum allowed value. After that, it decreases in time and becomes zero.

Among all the strategies, we can therefore conclude very clearly that the optimal strategy becomes more effective and faster to increase the size of susceptible and recovered populations while reducing the number of heroin users in the population when awareness campaigns and nonpharmaceutical interventions are combined.

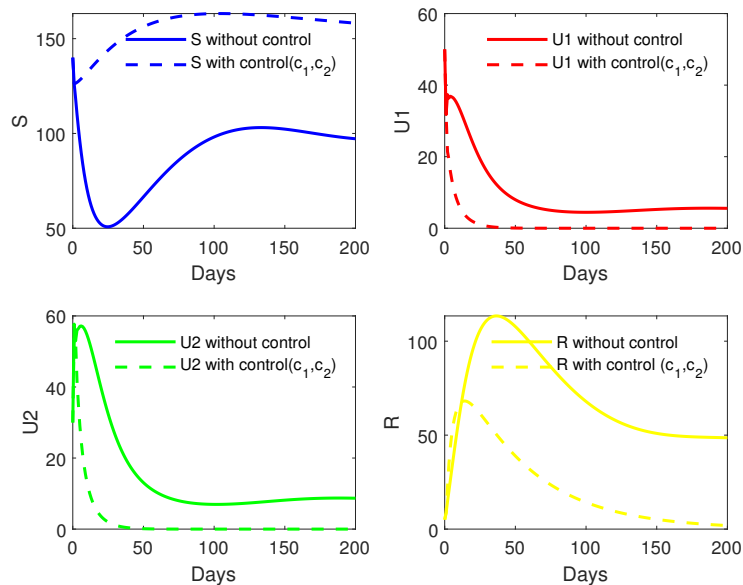


FIGURE 11. The behavior of the variables without control and with control (c_1, c_2)

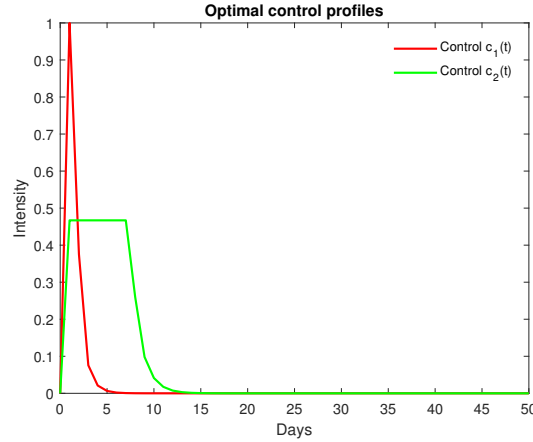


FIGURE 12. profile of control (c_1, c_2) .

6. CONCLUSION

Heroin, a traditional opioid derived from natural sources, continues to play a significant role in global drug addiction. Its transmission is often driven by widespread misconceptions about its addictive nature. Many people start heroin use in the mistaken belief that occasional or limited consumption will not result in dependence. This misjudgment leads to a lack of caution, allowing addiction to develop gradually and often without immediate awareness. Addressing this psychological misconception is crucial for the development of effective prevention strategies and public health interventions. Therefore, the aim of this study is to explore the impact of awareness and nonpharmaceutical interventions on the development of the heroin epidemic within a mathematical framework. We constructed a mathematical model to examine how a heroin epidemic behaves with treatment and relapse. Based on the expression of the basic reproduction number \mathcal{R}_0 , we derived sufficient conditions for the extinction and persistence of heroin use. The findings from the sensitivity analysis of \mathcal{R}_0 guide us towards the strongest and most influential parameters of the proposed model. We formulated control measures coupled with awareness campaigns and nonpharmaceutical interventions. Pontryagin’s Maximum Principle is used to describe the characteristics of these optimal control strategies to minimize the heroin users, as well as the cost burden. The numerical results support the findings of the theoretical analysis. Moreover, the simulations demonstrate that \mathcal{R}_0 plays a vital role in the

spread of heroin use. In addition, we numerically compared the three control strategies, considering both single interventions and their combination. Our results show that of all the strategies, the most effective is the combination of awareness programs and nonpharmaceutical interventions.

Our model is only valid for heroin epidemic assumed to spread through direct contact. However, it can be easily extended to more complex settings by incorporating additional interventions, accounting for the heterogeneity of contact patterns, and introducing stochastic effects. These extensions represent promising directions for future research.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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