



Research article**Mathematical analysis for a dynamic model of illicit drug consumption****Abdulaziz H. Alharbi^{1,*}, M. S. J. Alzahrani^{2,*}, Fadhel Jday¹ and Aref Alsehaimi³**¹ Department of Mathematics, Jamoum University College, Umm Al-Qura University, Makkah 25375, Saudi Arabia² Mechanical and Industrial Engineering Department, College of Engineering Al-Leith, Umm Al-Qura University, Makkah, Saudi Arabia³ Department of Social Sciences, College of Arts, University of Hail, Saudi Arabia*** Correspondence:** Email: ahhrbe@uqu.edu.sa, msjzahrani@uqu.edu.sa.

Abstract: This paper presents a comprehensive mathematical model for analyzing the dynamics of illicit drug consumption within a population. Building upon and extending classical epidemiological models such as the susceptible, infected, and recovered (SIR) framework, the proposed model categorizes individuals into four compartments: Non-susceptible, susceptible, addicted, and rehabilitated. The model incorporates key social factors such as peer influence, intervention efforts, and the probability of relapse. A nonlinear system of differential equations was developed to describe the transitions between these states. The basic reproduction number R_0 was derived to assess the potential spread of addiction and stability analysis of the equilibrium points was carried out. Numerical simulations explore the impact of various intervention levels, highlighting how increased societal and family involvement can reduce the prevalence of addiction. Furthermore, an optimal control problem was formulated and solved using Pontryagin's maximum principle to determine the most cost-effective intervention strategy over time. The results demonstrate that adaptive, well-balanced policies can significantly reduce both susceptibility and addiction rates while minimizing social and economic costs.

Keywords: Simulation of dynamical systems; infectious disease; mathematical model; population dynamics; optimal control; Pontryagin's maximum principle

Mathematics Subject Classification: 37M05, 37M20, 91D10, 92D25, 49J15

1. Introduction

Dynamic models of illicit drug consumption aid in understanding the interplay between supply, demand, prevention, and harm reduction, informing public health and policy. Early models drew

inspiration from epidemiology, particularly the SIR framework [1], with some demonstrating the model's ability to describe epidemiological dynamics [2] and using the SIR model to predict infectious peak data [3]. SIR models have also been used in recent studies to explore social behaviors such as rumor propagation on social media [4–6]. These models emphasize the similarity between drug diffusion and infectious disease spread [7]. Compartmental models have evolved to include states like “addicted”, “rehabilitated”, and “relapsed” [8–11], .

Later approaches involve stochastic and deterministic dynamic models and multi-agent systems [12, 13], evolutionary game theory, and network analysis. Optimal control models [14] examine the trade-offs in decriminalization and public health investments. The “economon model” [15] has also been introduced to improve understanding the prevalence of the drug addiction.

Despite these advances, challenges remain, including a lack of trustworthy data [16], the influence of psycho-social elements, and nonlinear feedback loops [17]. Future research could benefit from combining machine learning with big data analytics [18].

While epidemiological models like the SIR framework have been adapted to study illicit drug dynamics [1, 13, 17], critical limitations persist. First, most models assume homogeneous mixing and static intervention parameters [13], ignoring spatial heterogeneity and adaptive policymaking—a shortcoming noted in recent reviews of drug policy efficacy [19]. Second, relapse rates are often oversimplified or ignored [8], despite clinical studies showing 40%–60% relapse probabilities among rehabilitated individuals [20]. Third, existing optimal control strategies rarely account for cost-effectiveness in real-world budget constraints [14]. Our work addresses these gaps by: (i) Introducing a secondary transmission term (σ_2) to quantify peer-driven contagion, aligning with social network theories in [21]; (ii) modeling dynamic intervention thresholds ($\delta(t)$) via Pontryagin's principle, responding to calls for adaptive strategies in [22]; and (iii) incorporating empirical relapse data from [8] into compartment transitions. This approach bridges mathematical rigor with policy-relevant insights, offering a tool to evaluate interventions like those debated in [23].

2. Preliminaries

Pontryagin's maximum principle (PMP) [24, 25] is a powerful tool in optimal control theory for finding the best possible control for a dynamical system to achieve a particular objective. The PMP provides necessary conditions for optimality. It is essential to understand the core hypotheses on which the PMP relies to appreciate its applicability.

Pontryagin's maximum principle (PMP) applies under the following assumptions:

H1. State dynamics regularity:

- The vector field $f : \mathbb{R}^n \times \mathcal{U} \times [0, T] \rightarrow \mathbb{R}^n$ is continuous in (x, u, t) .
- f is continuously differentiable in x .

H2. Cost function regularity:

- The running cost $L : \mathbb{R}^n \times \mathcal{U} \times [0, T] \rightarrow \mathbb{R}$ is continuous.
- The terminal cost $\Phi : \mathbb{R}^n \rightarrow \mathbb{R}$ is continuously differentiable.

H3. Admissible controls:

- The control set \mathcal{U} is compact.

- The control $u(t)$ is piecewise continuous (or Lebesgue measurable).

H4. Transversality condition:

- For free terminal state problems, $\lambda(T) = \nabla \Phi(x(T))$.

Theorem 1. (Pontryagin's maximum principle) Under hypotheses H1–H4, if u^* is an optimal control, then there exists an adjoint process $\lambda(t)$ satisfying:

1. The adjoint equation:

$$\dot{\lambda}(t) = -\frac{\partial H}{\partial x}(x^*(t), u^*(t), \lambda(t), t). \quad (2.1)$$

2. The minimization condition:

$$H(x^*(t), u^*(t), \lambda(t), t) = \min_{u \in \mathcal{U}} H(x^*(t), u, \lambda(t), t). \quad (2.2)$$

3. The Hamiltonian is defined as:

$$H(x, u, \lambda, t) = L(x, u, t) + \lambda^\top f(x, u, t). \quad (2.3)$$

Proof. See [24, 25]. □

3. Mathematical model

In this work, we modify the classical SIR models commonly used in mathematical epidemiology (see [7]), where S denotes the susceptible population, I the infectious individuals, and R the recovered group. The population P_t is classified into four groups (P_n , P_s , P_a , P_r) according to the illicit drug abuse model. P_n denotes individuals who are neither current nor potential drug users, and P_s represents the social group most susceptible to illicit drug abuse. The group of people with illicit drug addiction is represented by the compartment P_a . The group of people who have stopped abusing illicit drugs, unconstrainedly or through specialized rehabilitation facilities, is represented by the recovered compartment P_r . The causes of addiction are widespread throughout society; therefore, new individuals are constantly joining the illicit drug-using community. Drug-addicted people P_a can become susceptible P_s at a rate $\sigma_1(P_a + P_s)\frac{P_n}{P_t}$, whereas non-susceptible (P_n) can become susceptible (P_s) by contact rate σ_1 . With the intervention/interference parameter δ , it is feasible to intervene at this point and influence the susceptible person to return to the non-susceptible class (P_n). This parameter represents the efforts of both family and society in rejecting the idea of drug addiction. It also includes the endeavors of competent authorities through awareness courses on the dangers of addiction, as well as the efforts of associations specialized in caring for individuals struggling with drug addiction. Their message emphasizes that addiction is a long-term condition with the potential for relapse, and that ongoing, supportive care such as medication, therapy, and involvement in self-help groups plays a vital role in reducing the risk of future relapses. At a rate $\sigma_2\frac{P_s P_a}{P_t}$, the susceptible P_s by the contact rate σ_2 with drug-addicted P_a may be recruited into the population P_a . The drug abuse rate of ψ may then be stopped by these drug-addicted P_a . Some people who have been removed might return to P_s or they might have recovered and are now in the non-susceptible class P_n . The average

duration of the rehabilitation time is μ^{-1} . Since $(1 - f)$ is the percentage of rehabilitated people, the model is described by the following nonlinear system of equations.

$$\frac{dP_n}{dt} = -\sigma_1(P_a + P_s)\frac{P_n}{P_t} + \delta P_s + fpP_r, \quad (3.1)$$

$$\frac{dP_s}{dt} = \sigma_1(P_a + P_s)\frac{P_n}{P_t} - \sigma_2\frac{P_sP_a}{P_t} - \delta P_s, \quad (3.2)$$

$$\frac{dP_a}{dt} = \sigma_2\frac{P_sP_a}{P_t} + (1 - f)pP_r - \psi P_a, \quad (3.3)$$

$$\frac{dP_r}{dt} = \psi P_a - pP_r, \quad (3.4)$$

satisfying

$$P_n + P_s + P_a + P_r = P_t, \quad (3.5)$$

where $P_n(0) \geq 0$, $P_s(0) \geq 0$, $P_a(0) \geq 0$, $P_r(0) \geq 0$, (for more details, see Figure 1).

The total population P_t is assumed to be constant, and we can restrict our attention to four equations. By re-scaling to express the system in terms of population proportions, we define:

$$\beta = \frac{P_s}{P_t}, \quad \alpha = \frac{P_a}{P_t}, \quad r = \frac{P_r}{P_t}, \quad \gamma = \frac{P_n}{P_t}.$$

Therefore, we obtain the following system:

$$\frac{d\gamma}{dt} = -\sigma_1\gamma(\beta + \alpha) + \delta\beta + fpr, \quad (3.6)$$

$$\frac{d\beta}{dt} = \sigma_1\gamma(\beta + \alpha) - \sigma_2\beta\alpha - \delta\beta, \quad (3.7)$$

$$\frac{d\alpha}{dt} = \sigma_2\beta\alpha + (1 - f)pr - \psi\alpha, \quad (3.8)$$

$$\frac{dr}{dt} = \psi\alpha - pr, \quad (3.9)$$

satisfying

$$\gamma + \beta + \alpha + r = 1, \quad (3.10)$$

with $\gamma(0) \geq 0$, $\beta(0) \geq 0$, $\alpha(0) \geq 0$, $r(0) \geq 0$.

Parameter interpretations

- σ_1 : Daily rate of non-users becoming susceptible (day^{-1}).
- σ_2 : Daily peer influence rate leading to addiction (day^{-1}).
- δ : Daily rate of intervention success (day^{-1}).
- $f \in [0, 1]$: Fraction of rehabilitated individuals who relapse.
- ψ : Average rehabilitation duration ($1/\psi$ days).
- p : Rate of permanent recovery after rehabilitation (day^{-1}).

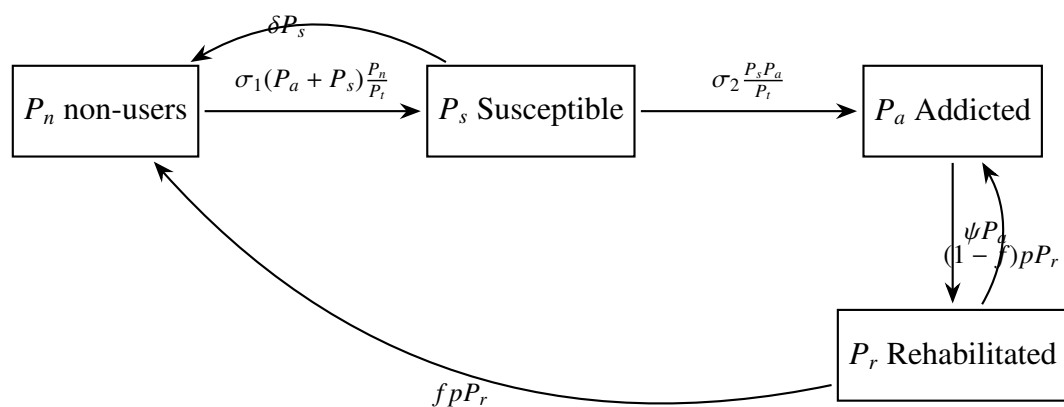


Figure 1. Schematic of the illicit drug consumption model. The four compartments (P_n, P_s, P_a, P_r) represent population states, with arrows showing the transition rates between them.

3.1. Assumptions

1. We make the assumption that drug usage is contagious, meaning that any new user may introduce a new set of others. Drug usage will be regarded as contagious under this approach.
2. Social contact plays a vital role in recruiting individuals who use drugs.
3. All members of the population participate equally in the recruitment process, regardless of whether they take drugs or are at risk of doing so.
4. Since births and deaths are not taken into account in this model, the total population P_t is regarded as constant.

4. Mathematical study

4.1. Estimation of the basic reproduction number R_0

The basic reproduction number, R_0 , serves as a threshold value indicating the average number of new cases caused resulting from one person addicted to illicit drugs. Introducing this concept is crucial for assessing whether addiction can spread within a population and sustain itself or become endemic. In this instance, R_0 represents the average number of secondary drug users influenced by another drug user. There are several methods for calculating R_0 , and in this work, we apply the next-generation operator approach presented in [5]. Using this approach, the infective compartments are denoted by β and α , the non-infective compartments by γ , and the affected individuals are identified by r . We obtain $R_0 = \frac{\sigma_1}{\delta}$, which is at a minimum required for the illicit drug-addiction-free state to be globally asymptotically stable. Conversely, when $R_0 > 1$, there is the potential for the existence of multiple stable endemic states. For $R_0 < 1$, illicit-drug addiction will eventually disappear, and a new addiction cannot be initiated. Given that peer and societal pressure play a major role in this model, the spread of addiction can be seen as a community-driven process, and R_0 can be interpreted as an indicator of how conducive the environment is to sustaining drug use.

4.2. Disease dynamics: Equilibrium points

We distinguish three states of equilibrium points: The equilibrium point where susceptibility is absent, the equilibrium point free from addictive drugs, and the equilibrium point reflecting the coexistence.

Case 1. $\alpha^* = 0$ and $\beta^* = 0$: The steady state free from drug abuse.

From Eq (3.7), $\sigma_1 \gamma^* \alpha^* = 0$, since $\gamma^* \neq 0$, and we deduce that $\alpha^* = 0$. From Eq (3.9), $r^* = 0$, and from Eq (3.10), $\gamma^* = 1$. This is the drug-free equilibrium point.

From Eq (3.7), $\gamma^* = \frac{\delta}{\sigma_1}$, from Eq (3.9), $r^* = 0$, and from Eq (3.10), $\beta^* = \frac{\sigma_1 - \delta}{\sigma_1}$.

Case 2. $\alpha^* = 0$ and $\beta^* \neq 0$: The steady state free from addictive drugs.

From Eq (3.7), $\gamma^* = \frac{\delta}{\sigma_1}$, from Eq (3.9), $r^* = 0$, and from Eq (3.10), $\beta^* = \frac{\sigma_1 - \delta}{\sigma_1}$. This means that $\sigma_1 > \delta$ ($R_0 > 1$). This is the steady state that is free from addictive drugs but allows for the existence of susceptibility.

Case 3. $\beta^* = \frac{f\psi}{\sigma_2}$, where $f\psi < \sigma_2$: Coexistence equilibrium point.

An alternative possible outcome of the model is a scenario where all sub-populations coexist, and illicit drug addiction remains persistently present in the population, representing an endemic state. By substituting the expressions for β and n from Eqs (3.9) and (3.10) into Eq (3.6), we obtain a quadratic equation in α of the form

$$b_1 \alpha^2 + b_2 \alpha + b_3 = 0,$$

which can be solved for α , where the coefficients are given by:

$$b_1 = \sigma_1 \left(1 + \frac{\psi}{\sigma_2} \right) + \frac{2\sigma_1 f\psi}{\sigma_2 \mu} + \frac{\sigma_1 f^2 \psi^2}{\sigma_2^2 \mu^2} > 0, \quad (4.1)$$

$$b_2 = \frac{2\sigma_1 f\psi}{\mu} - \frac{\sigma_1 f\psi^2}{\sigma_2 \mu^2} - \sigma_1 + f\psi, \quad (4.2)$$

$$b_3 = \frac{-\sigma_1 f^2 \psi^2}{\sigma_2 \mu^2} (\sigma_1 - \delta). \quad (4.3)$$

Since $b_1 > 0$, the number of coexistence solutions for α depends on the values of b_2 and b_3 ; there may be zero, one, or two such solutions.

4.3. Stability analysis of equilibrium points

Lemma 2. *The stability of the model's equilibrium points is determined by analyzing the eigenvalues of the Jacobian matrix evaluated at each equilibrium. Specifically:*

- The drug-abuse-free equilibrium is stable if the basic reproduction number ($R_0 := \frac{\sigma_1}{\delta}$), is less than 1.
- The steady-state free from addictive drugs can be stable but allows for the existence of susceptibility only when $\sigma_1 > \delta$ ($R_0 > 1$).
- The alternative coexistence equilibrium point is subject to the conditions of stability: (i) $\sigma_1 > \sigma_2$; (ii) $f\psi > \left(1 - \frac{\sigma_2}{\sigma_1}\right)^2$.

Proof. To analyze the stability of the equilibrium points, we linearize the system by introducing small perturbations around the equilibrium state (γ, β, α) . This is done by substituting:

$$\gamma = \gamma + \epsilon_1, \quad \beta = \beta + \epsilon_2, \quad \alpha = \alpha + \epsilon_3, \quad (4.4)$$

where $\epsilon_1, \epsilon_2, \epsilon_3$ represent small perturbations. We then expand all expressions around the equilibrium points using Taylor's theorem, retaining only the linear terms and discarding higher-order terms in $\epsilon_1, \epsilon_2, \epsilon_3$. The equations in their linearized form can be expressed as follows.

$$\frac{dV}{dt} = JV, \quad (4.5)$$

where

$$J = \begin{pmatrix} -\sigma_1\beta - \sigma_1a & -\sigma_1\gamma + \delta & -\sigma_1\gamma \\ \sigma_2\alpha & \sigma_1\gamma - \sigma_2\alpha - \delta & \sigma_2d \\ f\psi r & \sigma_2\alpha & \sigma_2d - \psi \end{pmatrix},$$

with

$$\begin{aligned} B &= \sigma_1(\beta + \alpha), \\ F &= (1 - f)p, \\ G &= \sigma_1\gamma + fp, \\ V &= \begin{pmatrix} \epsilon_1 \\ \epsilon_2 \\ \epsilon_3 \end{pmatrix}. \end{aligned}$$

The corresponding characteristic equation is derived from,

$$|J - \lambda I| = 0,$$

and takes the following form:

$$\lambda^3 + m_1\lambda^2 + m_2\lambda + m_3 = 0,$$

where

$$\begin{aligned} m_1 &= B + F + \psi + \delta + fp + \sigma_2\alpha - \sigma_1\gamma - \sigma_2\beta, \\ m_2 &= B(G - \delta) + \psi_1\psi_2 + (B + fp)\psi_3 - FG + \psi_4, \\ m_3 &= \psi\psi_5 - \psi\psi_6\psi_7 - (F - \sigma_2\alpha)\psi_8 + F\psi_9, \end{aligned}$$

with

$$\begin{aligned} \psi_1 &= F + \psi - \sigma_2\beta, \\ \psi_2 &= B + \delta + fp + \sigma_2\alpha - \sigma_1\gamma, \\ \psi_3 &= \delta + \sigma_2\alpha - \sigma_1\gamma, \\ \psi_4 &= (F - \sigma_2\alpha)(\sigma_1\gamma - \sigma_2\beta), \end{aligned}$$

$$\begin{aligned}
\psi_5 &= (B(G - \delta) + (B + fp)(\delta + \sigma_2\alpha - \sigma_1\gamma)), \\
\psi_6 &= (FG - (F - \sigma_2a)(\sigma_1\gamma - \sigma_2\beta)), \\
\psi_7 &= (B + \delta + fp + \sigma_2\alpha - \sigma_1\gamma), \\
\psi_8 &= ((\sigma_1\gamma - \sigma_2\beta)(\delta + \sigma_2\alpha - \sigma_1\gamma) + BG), \\
\psi_9 &= (G(B + fp) - (\sigma_1\gamma - \sigma_2\beta)(G - \delta)).
\end{aligned}$$

The Routh-Hurwitz criteria are:

$$m_1 > 0, \quad m_3 > 0, \quad m_1m_2 - m_3 > 0, \quad (4.6)$$

which are used to determine the stability of each case.

Case 1. At the drug-free equilibrium point,

$$(\gamma, \beta, \alpha) = (1, 0, 0),$$

the condition for stability using (4.6) is:

$$\delta > \sigma_1. \quad (4.7)$$

The stability condition for the addiction-free equilibrium point is identical to the condition that the basic reproduction number $R_0 < 1$, preventing the occurrence of an epidemic. This implies that if potential recruits are discouraged from joining addiction more quickly than they are recruited, no addiction will form within the population.

Case 2. $n_u^* = \frac{\delta}{\sigma_1}, \beta^* = \frac{\sigma_1 - \delta}{\sigma_1}, \alpha^* = 0, r^* = 0, \sigma_1 > \delta$.

In this case, it is assumed that the disease has become established, meaning that $R_0 > 1$. The stability conditions derived from Eq (4.6) are as follows:

$$\sigma_1 > \sigma_2 \quad ; \quad f\psi > \left(1 - \frac{\delta}{\sigma_1}\right)\sigma_2. \quad (4.8)$$

Case 3. $\beta^* = \frac{f\psi}{\sigma_2}, f\psi < \sigma_2, n_u^* = 1 - \frac{f\psi}{\sigma_2} - \alpha^*(1 - \frac{\psi}{\mu}), \alpha^*$.

The condition for stability using (4.6) is

$$\theta_1(\theta_2 - \psi(1 - f)(Q\sigma_1 - \delta)) > \theta_3,$$

where

$$\begin{aligned}
\theta_1 &= \psi(1 - f) + \mu + \delta + \sigma_2\alpha - \sigma_1Q, \\
\theta_2 &= \sigma_2\alpha\psi - \sigma_1Q\sigma_2\alpha + \mu(\delta + \sigma_2\alpha - \sigma_1Q), \\
\theta_3 &= (\psi\sigma_1(\frac{f\psi}{\sigma_2} + \alpha) + \mu f\psi - \sigma_1\mu Q)\sigma_2\alpha, \\
Q &= 1 - \frac{2f\psi}{\sigma_2} - \alpha(2 - \frac{\psi}{\mu}).
\end{aligned}$$

□

5. Optimal control on the intervention/interference rate

Preventive awareness involves conducting extensive awareness campaigns designed to educate the public, particularly young people, about the dangers of drugs and the abuse of prescription medications, and the destructive effects they cause on the human body and mind, and the resulting harms to the addict's personality, which can extend to their family and society. These awareness campaigns take several forms of activities, including:

Giving lectures and holding seminars in universities and schools. Printing books, brochures, bulletins, publications, and posters, and distributing them to young people. Holding cultural competitions in coordination with universities, on health and social harms, with valuable financial rewards distributed to the winners. Producing radio and television programs that address the health and social harms of drug abuse, misuse of medical drugs, and electronic publishing. Participating in local, regional, and international meetings and seminars to gain experience and benefit from the experiences of countries. Urging individuals involved in drug abuse to go to one of the Hope Complexes to receive the necessary treatment for addiction, taking into account complete confidentiality in treatment, in addition to receiving treatment free of charge.

The primary aim of the optimal strategy is to lower the susceptible and drug-addicted individuals $(P_s(t), P_a(t))$ within the population by using an optimal intervention/interference rate. Hence, we assume that the intervention/interference rate $\delta(t)$ changes throughout the time period $[0, T]$, where T is a fixed positive constant. Hence, the objective is to determine the optimal control function $\delta = \delta(t)$ within the set of admissible controls.

$$\mathbf{P}_{ad} = \{\delta(t) : 0 \leq \delta_{\min} \leq \delta(t) \leq \delta_{\max}, 0 \leq t \leq T, \delta(t) \text{ is piecewise continuous}\}.$$

By minimizing the following functional:

$$J(\delta) = \frac{\kappa_s}{2} \int_0^T P_s^2(t) dt + \frac{\kappa_a}{2} \int_0^T P_a^2(t) dt + \frac{\kappa_\delta}{2} \int_0^T \delta^2(t) dt,$$

for suitable choices of the constants $\kappa_s > 0$, $\kappa_a > 0$, and $\kappa_\delta > 0$, our objective is to reduce the number of susceptible and drug-addicted individuals while minimizing the control cost. According to the theory presented in [26], it can be readily shown that the optimal control and the corresponding state exist. Letting $\varphi = (P_n, P_s, P_a, P_r)^t$, the system of Eqs (3.1)–(3.4) can be reformulated as follows:

$$\dot{\varphi} = B\varphi + H_1(\varphi) = H_2(\varphi) \quad (5.1)$$

$$\text{with } B = \begin{pmatrix} 0 & \delta & 0 & fp \\ 0 & -\delta & 0 & 0 \\ 0 & 0 & -\psi & (1-f)p \\ 0 & 0 & \psi & -p \end{pmatrix} \text{ and } H_1(\varphi) = \begin{pmatrix} -\sigma_1 P_n (P_s + P_a) \\ \sigma_1 P_n (P_s + P_a) - \sigma_2 \frac{P_s P_a}{P_t} \\ \sigma_2 \frac{P_s P_a}{P_t} \\ 0 \end{pmatrix}.$$

Theorem. The continuous function H_2 satisfies a uniform Lipschitz condition.

Proof. It is clear that the continuous function H_1 is uniformly Lipschitz, given that it meets the required conditions.

$$\begin{aligned}
\|H_1(\varphi_1) - H_1(\varphi_2)\|_1 &\leq \sigma_1 \left| P'_n(P'_s + P'_a) - P_n(P_s + P_a) \right| + \sigma_1 \left| P_n(P_s + P_a) - P'_n(P'_s + P'_a) \right| \\
&+ \frac{\sigma_2}{P_t} \left| P'_s P'_a - P_s P_a \right| + \frac{\sigma_2}{P_t} \left| P_s P_a - P'_s P'_a \right| \\
&= 2\sigma_1 \left| P'_n(P'_s + P'_a) - P'_n(P_s + P_a) + P'_n(P_s + P_a) - P_n(P_s + P_a) \right| \\
&+ 2\frac{\sigma_2}{P_t} \left| P'_s P'_a - P_s P'_a + P_s P'_a - P_s P_a \right| \\
&\leq 2\sigma_1 P_t \left| (P'_s + P'_a) - (P_s + P_a) \right| + 2\sigma_1 P_t \left| P'_n - P_n \right| + 2\sigma_2 \left| P'_s - P_s \right| + 2\sigma_2 \left| P'_a - P_a \right| \\
&\leq L_1 \|\varphi_1 - \varphi_2\|_1,
\end{aligned}$$

where $L_1 = \max(4\sigma_1 P_t, 4\sigma_2)$. The matrix B satisfies

$$\|B\varphi_1 - B\varphi_2\|_1 \leq L_2 \|\varphi_1 - \varphi_2\|_1,$$

where $L_2 = \max(\delta, \psi, p)$, thus,

$$\|H_2(\varphi_1) - H_2(\varphi_2)\|_1 \leq \delta \|\varphi_1 - \varphi_2\|_1$$

with $\delta = \max(L_1, L_2)$. Therefore, the continuous function H_2 is uniformly Lipschitz. \square

Therefore, the system described by (5.1) possesses a unique solution. Applying Pontryagin's maximum principle [22, 26, 27], the control problem can be analyzed through the Hamiltonian function as outlined below:

$$\begin{aligned}
H &= \frac{\kappa_s}{2} P_s^2 + \frac{\kappa_a}{2} P_a^2 + \frac{\kappa_\delta}{2} \delta^2 + \lambda_1 \frac{dP_n}{dt} + \lambda_2 \frac{dP_s}{dt} + \lambda_3 \frac{dP_a}{dt} + \lambda_4 \frac{dP_r}{dt} \\
&= \frac{\tilde{\kappa}_s}{2} P_s^2 + \frac{\tilde{\kappa}_a}{2} P_a^2 + \frac{\tilde{\kappa}_\delta}{2} \delta^2 + \lambda_1 (-\sigma_1 P_n (P_s + P_a) + \delta P_s + f p P_r) \\
&+ \lambda_2 (\sigma_1 P_n (P_s + P_a) - \sigma_2 \frac{P_s P_a}{P_t} - \delta P_s) + \lambda_3 (\sigma_2 \frac{P_s P_a}{P_t} + (1 - f) p P_r - \psi P_a) + \lambda_4 (\psi P_a - p P_r).
\end{aligned} \tag{5.2}$$

The adjoint variables $\lambda_1, \lambda_2, \lambda_3$, and λ_4 are solutions of the following adjoint system:

$$\begin{cases} \frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial P_n} = \lambda_1 \sigma_1 (P_s + P_a) + \lambda_2 \sigma_1 (P_s + P_a), \\ \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial P_s} = -\kappa_s P_s + \lambda_1 (\sigma_1 P_n - \delta) + \lambda_2 (-\sigma_1 P_n + \sigma_2 \frac{P_a}{P_t} + \delta) - \lambda_3 \sigma_2 \frac{P_a}{P_t}, \\ \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial P_a} = -\kappa_a P_a + \lambda_1 \sigma_1 P_n + \lambda_2 (-\sigma_1 P_n + \sigma_2 \frac{P_s}{P_t}) + \lambda_3 (-\sigma_2 \frac{P_s}{P_t} + \psi) - \lambda_4 \psi, \\ \frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial P_r} = -f p \lambda_1 - (1 - f) p \lambda_3 + p \lambda_4, \end{cases} \tag{5.3}$$

satisfying $\lambda_1(T) = 0$, $\lambda_2(T) = 0$, $\lambda_3(T) = 0$, and $\lambda_4(T) = 0$.

The derivatives of the Hamiltonian are given by

$$\frac{\partial H}{\partial \delta} = \kappa_\delta \delta + (\lambda_1 - \lambda_2) P_s.$$

Therefore, $\frac{\partial H}{\partial \delta} = 0$ has a unique solution given by

$$\delta^*(t) = \frac{(\lambda_2 - \lambda_1)P_s}{\kappa_\delta},$$

provided that $\kappa_\delta \neq 0$ and $0 < \delta_{\min} \leq \frac{(\lambda_2 - \lambda_1)P_s}{\kappa_\delta} \leq \delta_{\max}$. In summary, the control can be characterized as follows:

$$\left\{ \begin{array}{l} \text{if } \frac{\partial H}{\partial \delta} < 0 \text{ at } t, \text{ then } \delta(t) = \delta_{\max}, \\ \text{if } \frac{\partial H}{\partial \delta} > 0 \text{ at } t, \text{ then } \delta(t) = \delta_{\min}, \\ \text{if } \frac{\partial H}{\partial \delta} = 0 \text{ at } t, \text{ then } \delta(t) = \delta^*(t) = \frac{(\lambda_2 - \lambda_1)P_s}{\kappa_\delta}. \end{array} \right.$$

5.1. Numerical examples

We perform numerical results on the system (3.6)–(3.10). The ode45 solver in MATLAB was utilized to solve the initial value problem numerically. The theoretical results derived will be validated by adjusting certain parameter values that significantly influence the basic reproduction number R_0 and, consequently, the stability dynamics. We have the following subdivision of $[0, T]$:

$$[0, T] = \bigcup_{i=0}^{N-1} [t_i, t_{i+1}], \quad \text{where } t_i = i \, dt, \quad dt = \frac{T}{N}.$$

Let P_n^i , P_s^i , P_a^i , and P_r^i denote the approximations of $P_n(t)$, $P_s(t)$, $P_a(t)$, and $P_r(t)$, respectively, at time t_i . An improved Gauss-Seidel-like implicit finite-difference scheme is used to approximate the state variables over time.

$$\left\{ \begin{array}{l} \frac{P_n^{i+1} - P_n^i}{dt} = -\sigma_1 \frac{P_n^{i+1}}{P_t} (P_s^i + P_a^i) + \delta P_s^i + f p P_r^i, \\ \frac{P_s^{i+1} - P_s^i}{dt} = \sigma_1 \frac{P_n^{i+1}}{P_t} (P_s^{i+1} + P_a^i) - \sigma_2 \frac{P_s^{i+1} P_a^i}{P_t} - \delta P_s^{i+1}, \\ \frac{P_a^{i+1} - P_a^i}{dt} = \sigma_2 \frac{P_s^{i+1} P_a^{i+1}}{P_t} + (1 - f) p P_r^i - \psi P_a^{i+1}, \\ \frac{P_r^{i+1} - P_r^i}{dt} = \psi P_a^{i+1} - p P_r^{i+1}. \end{array} \right.$$

In the following examples, we consider an initial state value $(P_n, P_s, P_a, P_r) = (40, 30, 20, 10)$ with a final time $T = 300$, and we use the parameter values $\sigma_1 = 0.5$, $\sigma_2 = 0.8$, $f = 0.44$, $\psi = 1$, and $p = 0.1$.

Case of low intervention ($\delta = 0.05$). In this example, we consider $\delta = 0.05$, which implies that the basic reproduction number is $R_0 > 1$.

Figure 2 illustrates the evolution of the system under a very low intervention rate. With $\delta = 0.05$, the model simulates a scenario in which societal and familial efforts to reintegrate susceptible individuals into the non-susceptible class are minimal. As a result, the susceptible population remains elevated, and the number of addicted individuals tends to increase or stabilize at a high level over time. This outcome highlights the significant influence of the intervention parameter δ on the long-term dynamics of drug addiction and underlines the importance of strong preventive and rehabilitative measures to control its spread.

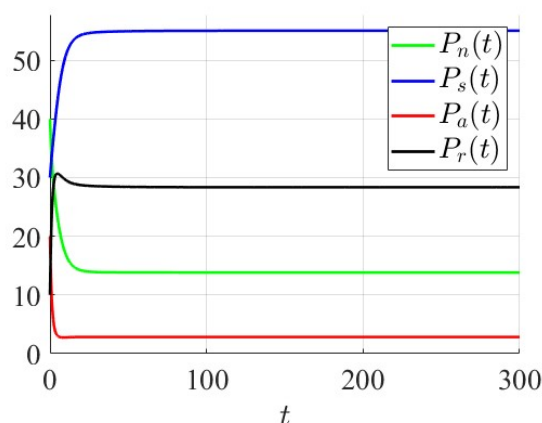


Figure 2. Influence of the intervention parameter $\delta = 0.05$.

Case of a moderate intervention rate ($\delta = 0.5$). In this case (see Figure 3), we consider $\delta = 0.5$, which implies that the basic reproduction number is $R_0 = 1$. The chosen parameter scheme reflects a dynamic where societal and familial interventions play a significant, yet not dominant, role in counteracting the spread of drug addiction. With a moderate intervention rate ($\delta = 0.5$) and a relatively high susceptibility rate ($\sigma_1 = 0.5$), individuals are at considerable risk of becoming susceptible due to social influence. The high transition rate from susceptibility to addiction ($\sigma_2 = 0.8$) indicates that contact with addicted individuals poses a substantial threat of progression into addiction. Although there is a rehabilitation pathway in place, the recovery rate ($\psi = 0.2$) and the fraction of successful reintegration into the non-susceptible class ($f = 0.44$) suggest that more than half of those rehabilitated are likely to relapse, highlighting partial but insufficient effectiveness of the treatment system.

This scheme overall suggests a scenario where, despite ongoing efforts to reduce susceptibility and promote recovery, the system may still lean toward a persistent or increasing prevalence of addiction unless further enhancements are made to intervention strategies and rehabilitation success rates.

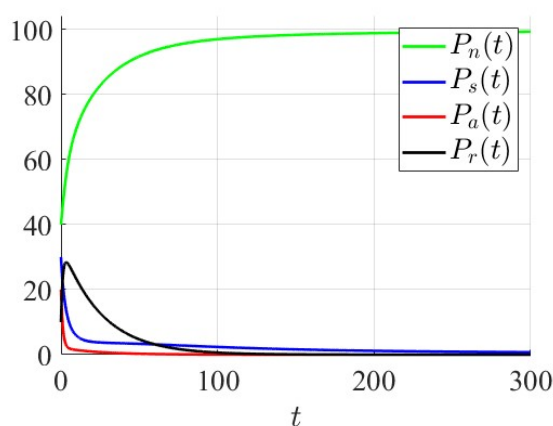


Figure 3. Influence of the intervention parameter $\delta = 0.5$.

Case of high intervention ($\delta = 0.8$). In this scenario (see Figure 4), the model exhibits a rapid decline in the number of susceptible ($P_s(t)$), addicted ($P_a(t)$), and rehabilitated ($P_r(t)$) individuals, while the non-susceptible population ($P_n(t)$) increases sharply and stabilizes near the total population size. This behavior reflects the strong impact of a high intervention rate, where susceptible individuals are quickly redirected back into the non-susceptible class before they progress to addiction. As a result, the spread of addiction is effectively suppressed early in the simulation, and the system evolves toward a drug-free equilibrium. The figure clearly demonstrates that with sustained and effective intervention, it is possible to eliminate addiction from the population over time.

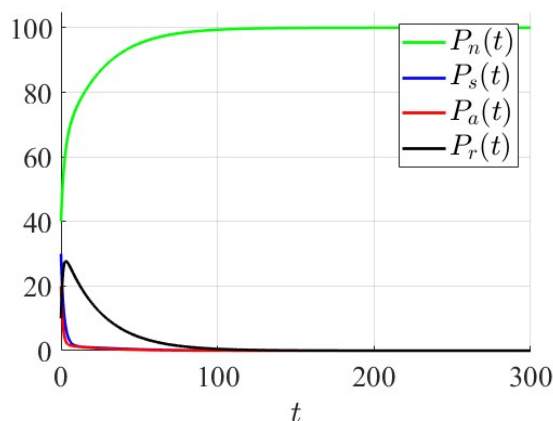


Figure 4. Influence of the intervention parameter $\delta = 0.8$.

5.2. Numerical examples for the control problem

The numerical scheme employed for the control problem is presented below. Consider the subdivision of the interval $[0, T]$ as follows: $[0, T] = \bigcup_{i=0}^{N-1} [t_i, t_{i+1}]$, $t_i = idt$, $dt = \frac{T}{N}$. Let P_n^i , P_s^i , P_a^i , P_r^i ,

$\lambda_1^i, \lambda_2^i, \lambda_3^i, \lambda_4^i$, and δ^i approach $P_n(t), P_s(t), P_a(t), P_r(t), \lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t)$, and the control $\delta(t)$ at the time t_i . An enhanced version of the Gauss-Seidel-like implicit finite-difference scheme will be used to approximate the state variables, while a first-order backward-difference scheme will be employed to approximate the adjoint variables.

$$\left\{ \begin{array}{l} \frac{P_n^{i+1} - P_n^i}{dt} = -\sigma_1 \frac{P_n^{i+1}}{P_t} (P_s^i + P_a^i) + \delta^i P_s^i + f p P_r^i, \\ \frac{P_s^{i+1} - P_s^i}{dt} = \sigma_1 \frac{P_n^{i+1}}{P_t} (P_s^{i+1} + P_a^i) - \sigma_2 \frac{P_s^{i+1} P_a^i}{P_t} - \delta^i P_s^{i+1}, \\ \frac{P_a^{i+1} - P_a^i}{dt} = \sigma_2 \frac{P_s^{i+1} P_a^{i+1}}{P_t} + (1-f) p P_r^i - \psi P_a^{i+1}, \\ \frac{P_r^{i+1} - P_r^i}{dt} = \psi P_a^{i+1} - p P_r^{i+1}, \\ \frac{\lambda_1^{N-i} - \lambda_1^{N-i-1}}{dt} = \lambda_1^{N-i-1} \sigma_1 (P_s^{i+1} + P_a^{i+1}) + \lambda_2^{N-i} \sigma_1 (P_s^{i+1} + P_a^{i+1}), \\ \frac{\lambda_2^{N-i} - \lambda_2^{N-i-1}}{dt} = -\kappa_s P_s^{i+1} + \lambda_1^{N-i-1} (\sigma_1 P_n^{i+1} - \delta^i) + \lambda_2^{N-i-1} (-\sigma_1 P_n^{i+1} + \sigma_2 \frac{P_a^{i+1}}{P_t} + \delta^i) - \lambda_3^{N-i} \sigma_2 \frac{P_a^{i+1}}{P_t}, \\ \frac{\lambda_3^{N-i} - \lambda_3^{N-i-1}}{dt} = -\kappa_a P_a^{i+1} + \lambda_1^{N-i-1} \sigma_1 P_n^{i+1} + \lambda_2^{N-i-1} (-\sigma_1 P_n^{i+1} + \sigma_2 \frac{P_s^{i+1}}{P_t}) + \lambda_3^{N-i-1} (-\sigma_2 \frac{P_s^{i+1}}{P_t} + \psi) - \lambda_4^{N-i} \psi, \\ \frac{\lambda_4^{N-i} - \lambda_4^{N-i-1}}{dt} = -f p \lambda_1^{N-i-1} - (1-f) p \lambda_3^{N-i-1} + p \lambda_4^{N-i-1}. \end{array} \right.$$

Therefore, δ^{i+1} will be computed as follows: $\delta^{i+1} = \frac{(\lambda_2^{N-i-1} - \lambda_1^{N-i-1}) P_s^{i+1}}{\kappa_\delta}$ provided that $\kappa_\delta \neq 0$ and

$0 < \delta_{\min} \leq \delta^{i+1} \leq \delta_{\max}$. Consequently, the following algorithm will be utilized.

$P_n^0 \leftarrow P_n(0), P_s^0 \leftarrow P_s(0), P_a^0 \leftarrow P_a(0), P_r^0 \leftarrow P_r(0), \lambda_1^N \leftarrow 0, \lambda_2^N \leftarrow 0, \lambda_3^N \leftarrow 0, \lambda_4^N \leftarrow 0, \delta^0 \leftarrow \delta(0)$,
for $i = 0$ to $N - 1$ **do**

$$\begin{aligned}
P_n^{i+1} &\leftarrow \frac{P_t P_n^i + dt(\delta^i P_t P_s^i + f p P_t P_r^i)}{P_t + dt\sigma_1(P_s^i + P_a^i)}, \\
P_s^{i+1} &\leftarrow \frac{P_t P_s^i + dt\sigma_1 P_n^{i+1} P_a^i}{P_t + dt(-\sigma_1 P_n^{i+1} + \sigma_2 P_a^i + \delta^i P_t)}, \\
P_a^{i+1} &\leftarrow \frac{P_a^i + dt(1-f)p P_r^i}{1 + dt\left(-\sigma_2 \frac{P_s^{i+1}}{P_t} + \psi\right)}, \\
P_r^{i+1} &\leftarrow \frac{P_r^i + dt\psi P_a^{i+1}}{1 + p dt}, \\
\lambda_1^{N-i-1} &\leftarrow \frac{\lambda_1^{N-i} + dt\lambda_2^{N-i}\sigma_1(P_s^{i+1} + P_a^{i+1})}{1 + dt\sigma_1(P_s^{i+1} + P_a^{i+1})}, \\
\lambda_2^{N-i-1} &\leftarrow \frac{\lambda_2^{N-i} + dt\left(\kappa_s P_s^{i+1} - \lambda_1^{N-i-1}(\sigma_1 P_n^{i+1} - \delta^i) + \lambda_3^{N-i}\sigma_2 \frac{P_a^{i+1}}{P_t}\right)}{1 + dt\left(-\sigma_1 P_n^{i+1} + \sigma_2 \frac{P_a^{i+1}}{P_t} + \delta^i\right)}, \\
\lambda_3^{N-i-1} &\leftarrow \frac{\lambda_3^{N-i} + dt\left(\kappa_a P_a^{i+1} - \lambda_1^{N-i-1}\sigma_1 P_n^{i+1} - \lambda_2^{N-i-1}(-\sigma_1 P_n^{i+1} + \sigma_2 \frac{P_s^{i+1}}{P_t}) + \lambda_4^{N-i}\psi\right)}{1 + dt\left(-\sigma_2 \frac{P_s^{i+1}}{P_t} + \psi\right)}, \\
\lambda_4^{N-i-1} &\leftarrow \frac{\lambda_4^{N-i} + p dt(f\lambda_1^{N-i-1} + (1-f)\lambda_3^{N-i-1})}{1 + p dt}, \\
\delta^{i+1} &\leftarrow \max\left(\min\left(\frac{(\lambda_2^{N-i-1} - \lambda_1^{N-i-1})P_s^{i+1}}{\kappa_\delta}, \delta_{\max}\right), \delta_{\min}\right).
\end{aligned}$$

end

Algorithm 1: Optimal intervention/interference strategy procedure.

In the examples that follow, we consider the control δ to be a function of time, denoted by $\delta(t)$, subject to the bounds $\delta_{\min} = 0.01$ and $\delta_{\max} = 5$, with an initial value $\delta(0) = 0.05$. We take an initial state value into account $(12, 16, 0.01, 0.01)$ and a final time $T = 10$. As shown in Figures 5–7, the optimal solution exhibits a high degree of smoothness. The control values tend to decrease as the κ_δ values increase, whereas they rise with increasing values of κ_s and κ_a . It is worth noting that the final numbers of susceptible and drug-addicted individuals remain unchanged despite variations in κ_s , κ_a , and κ_δ .

The optimal strategy can decrease the number of susceptible and drug-addicted individuals and

optimize the control values (costs) reflecting the intervention/interference rate.

Optimal control strategy with $R_0 > 1$. This example illustrates the impact of an optimal intervention strategy in the case where the basic reproduction number satisfies $R_0 > 1$. The control variable $\delta(t)$, constrained within an admissible range, is determined in such a way that it minimizes a cost functional balancing the social cost of addiction and the intervention effort. The resulting trajectories, shown in Figure 5, demonstrate a substantial reduction in both the susceptible and addicted populations over time. The non-susceptible class $P_n(t)$ increases steadily, indicating successful reintegration and prevention. These results underscore the effectiveness of dynamically adjusting the intervention rate to respond to the evolving state of the system, particularly when the potential for addiction spread is high.

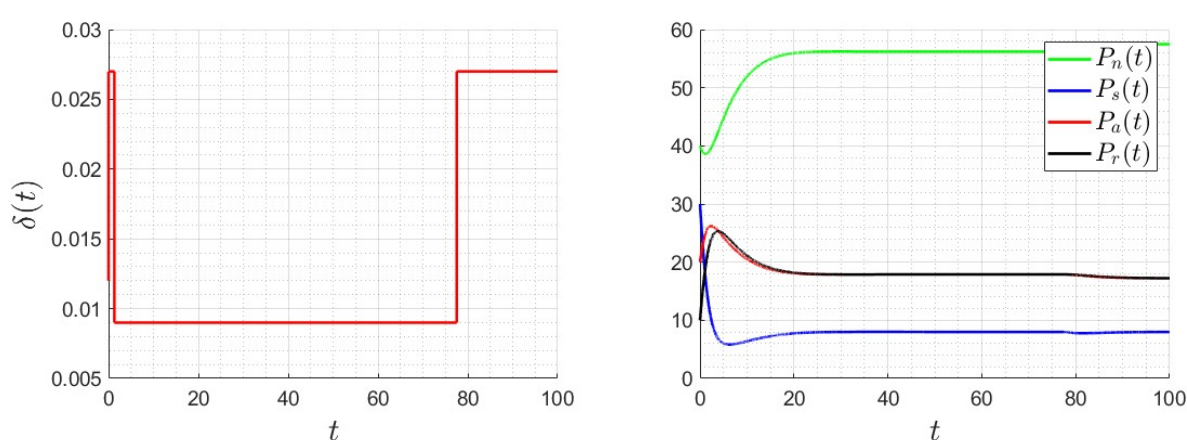


Figure 5. Impact of the optimal technique for $\kappa_s = 1$, $\kappa_a = 1$, $\kappa_\delta = 1$.

Optimal control strategy with $R_0 < 1$. In this example, the optimal control strategy is implemented under the condition that the basic reproduction number satisfies $R_0 < 1$. The dynamics shown in the figure indicate that the addicted population $P_a(t)$ experiences a short initial increase but then rapidly declines, converging toward a very low level. The susceptible population $P_s(t)$ is quickly reduced and remains minimal throughout the simulation. Concurrently, the non-susceptible population $P_n(t)$ increases consistently, eventually dominating the total population, which reflects the effectiveness of early and sustained interventions. The rehabilitated class $P_r(t)$ stabilizes at a moderate level, suggesting ongoing but controlled recovery processes. Overall, this scenario confirms that when $R_0 < 1$, the addiction can be contained efficiently, and optimal control strategies reinforce the decline while minimizing intervention costs.

Symmetric intervention trade-offs. In this scenario, we choose equal weight values for the cost functional, namely $\kappa_s = 10$, $\kappa_a = 10$, and $\kappa_\delta = 10$, reflecting a balanced concern between reducing the number of susceptible and addicted individuals, and the cost of applying intervention efforts. The numerical results demonstrate that the addicted population $P_a(t)$ steadily decreases over time, indicating effective containment of addiction. Meanwhile, the susceptible population $P_s(t)$ initially decreases and then stabilizes at a moderate level, reflecting a controlled yet non-negligible risk of

addiction. The non-susceptible population $P_n(t)$ shows consistent growth, which signifies the success of intervention in promoting prevention and recovery. The rehabilitated population $P_r(t)$ gradually declines as fewer individuals require long-term treatment. Overall, the use of equal weights in the cost functional yields a well-balanced outcome, achieving addiction control without excessive intervention intensity.

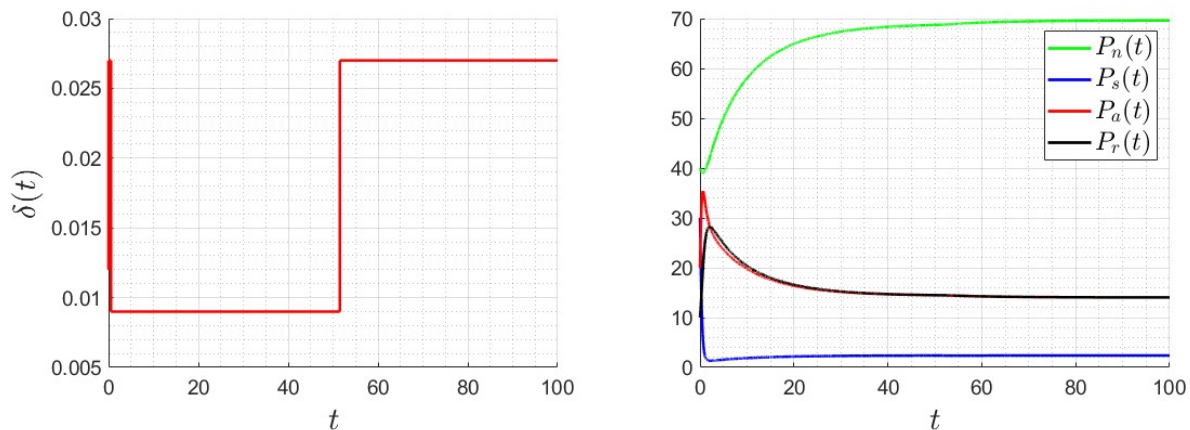


Figure 6. Impact of the optimal technique for $\kappa_s = 1, \kappa_a = 1, \kappa_\delta = 1$.

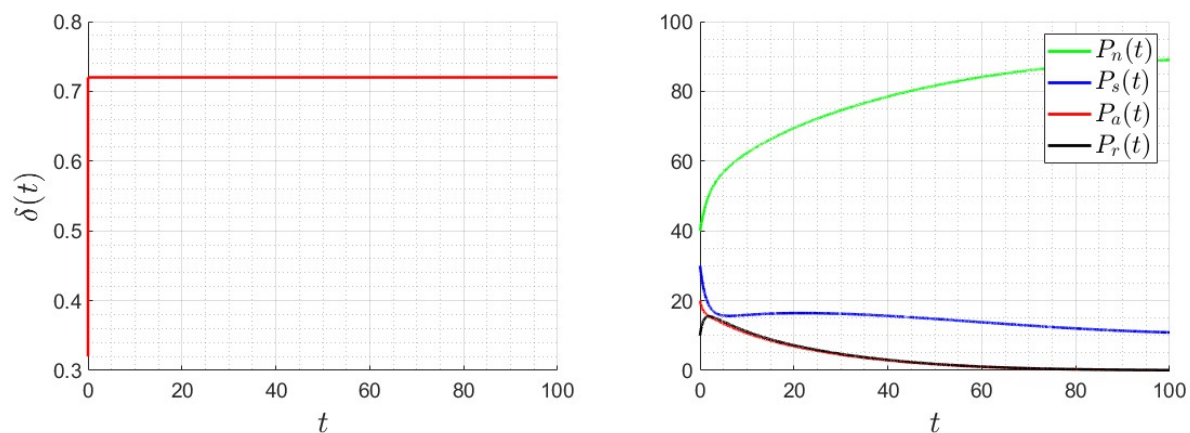


Figure 7. Impact of the optimal technique for $\kappa_s = 10, \kappa_a = 10, \kappa_\delta = 10$.

Control strategy prioritizing susceptible individuals. In this case (see Figure 8), the cost functional is configured with $\kappa_s = 10, \kappa_a = 1$, and $\kappa_\delta = 0.1$, emphasizing a strong penalization of the susceptible population, while allowing more flexibility in both the addicted population and the intervention cost. The simulation results indicate that the susceptible population $P_s(t)$ is rapidly and significantly reduced, reflecting the model's prioritization of minimizing this category. Meanwhile, the addicted population $P_a(t)$ also decreases but at a slower rate, suggesting that addiction control is less aggressive compared to susceptibility reduction. The non-susceptible population $P_n(t)$ increases steadily, benefiting from the reduced transitions from P_n to P_s . The rehabilitated group $P_r(t)$ stabilizes

at a moderate level. This scheme illustrates how tuning the weights in the cost functional can guide the control strategy towards protecting individuals at risk, even if it allows a relatively higher level of addiction or intervention expenditure.

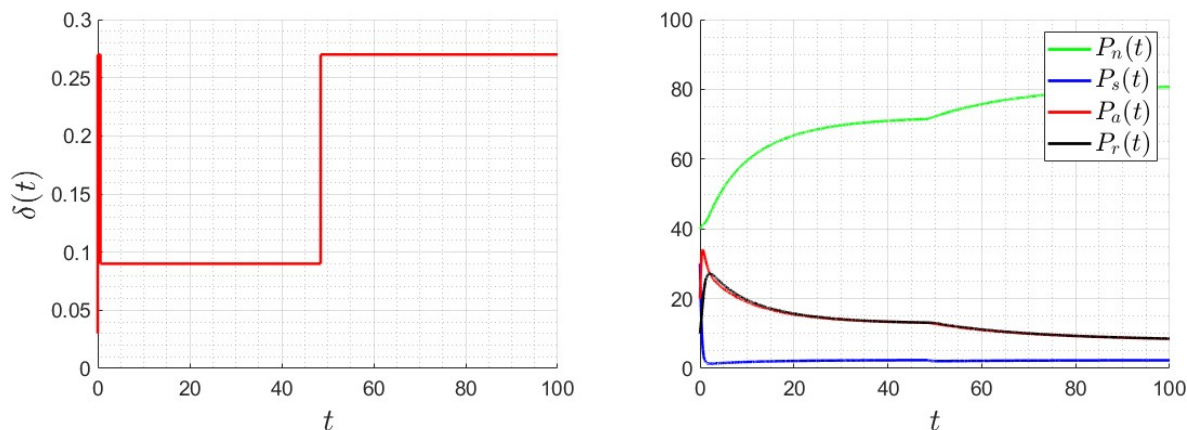


Figure 8. Impact of the optimal technique for $\kappa_s = 10$, $\kappa_a = 1$, $\kappa_\delta = 0.1$.

6. Conclusions

While this study advances mathematical modeling of illicit drug dynamics by addressing key literature gaps, such as peer-driven transmission (σ_2), adaptive interventions ($\delta(t)$), and empirical relapse rates [1–6], it remains a theoretical exploration. Our results, derived from compartmental and optimal control frameworks, provide mechanistic insights into addiction thresholds (e.g., R_0) and control strategies, and their theoretical nature necessitates validation with real-world data (e.g., regional rehabilitation records and policy constraints). Future collaborations with public health agencies could test these frameworks in targeted settings. Ultimately, this work advances addiction modeling as a *dynamic, relapsing process* and offers a template for translating mathematical rigor into policy-ready tools.

Author contributions

Abdulaziz H. Alharbi: Methodology, formal analysis, investigation, software, writing—original draft preparation, writing—review and editing, visualization, project administration, funding acquisition, supervision; M. S. J. Alzahrani: Methodology, formal analysis, investigation, writing—original draft preparation, writing—review and editing, visualization; Fadhel Jday: Methodology, formal analysis, investigation, software, writing—original draft preparation, writing—review and editing, visualization; Aref Alsehami: Writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare no conflict of interest.

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