

DYNAMIC ANALYSIS OF A FRACTIONAL-ORDER DRUG ADDICTION MODEL WITH GENERALIZED NONLINEAR INCIDENCE RATE*

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Abstract This paper analyzes a fractional-order drug addiction model with a generalized incidence rate of the form $f(P, A)$. The analysis begins by establishing the well-posedness of the model, verifying the existence, uniqueness, non-negativity, and boundedness of solutions. The basic reproduction number R_0 is derived, and two equilibrium points are identified: The drug-free equilibrium and the drug-persistent equilibrium. The local asymptotic stability of each equilibrium is investigated using the Routh-Hurwitz criterion, while global stability is established by constructing appropriate Lyapunov functionals and applying LaSalle's invariance principle. Unlike classical integer-order models that assume memoryless dynamics, the incorporation of the Caputo fractional derivative effectively accounts for the 'memory effects' and historical dependence inherent in the neurological and behavioral progression of substance abuse. The main theoretical findings demonstrate that, under appropriate conditions, drug addiction will eventually die out when $R_0 < 1$, but will persist when $R_0 > 1$. Numerical simulations are conducted to validate the theoretical results, offering insights for intervention strategies and policy-making. In summary, the proposed framework provides a robust tool for understanding addiction dynamics and formulating public health policies to mitigate the impact of substance abuse in communities.

Keywords Caputo fractional derivative, basic reproduction number, local stability, global stability.

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1. Introduction

Substance dependence, commonly known as drug addiction or substance use disorder, is a major public health concern characterized by significant disruptions in brain function and behavior. It manifests as a diminished ability to control the consumption of both legal and illicit drugs

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or medications, ultimately driving compulsive drug-seeking and use, even in the face of severe harmful consequences. This condition involves both physical and psychological dependence, which leads to two core phenomena: Tolerance (requiring more of the substance to achieve the same effect) and withdrawal symptoms when use is discontinued. Addiction commonly involves psychoactive substances such as alcohol, nicotine, opioids, and stimulants, as well as behavioral dependencies like gambling and excessive technology use. Unchecked addiction entrenches a destructive cycle of severe psychological, social, and economic repercussions, creating a detrimental feedback loop of individual hardship and community-wide strain [27,36]. Globally, countries such as the United States, India, and Brazil confront critical challenges posed by rising addiction rates, particularly among the youth who are essential to national productivity [1]. For instance, recent epidemiological reports document a sharp rise in mortality attributable to illicit opioids in North America. In parallel, the misuse of alcohol within adolescent populations in Europe continues to constitute a significant challenge [42]. Understanding the pathophysiological mechanisms of addiction—spanning neurobiological, psychological, and environmental factors—is critical for developing targeted prevention and treatment strategies. Mathematical models have served as instrumental tools in addressing this pervasive public health challenge. They enable the simulation of addiction dynamics and the analysis of its transmission patterns within populations, particularly among younger demographics. By providing a quantitative framework, mathematical modeling aids in analyzing trends, identifying key factors, and predicting intervention outcomes, thereby offering critical insights for the design of evidence-based policies and targeted intervention strategies.

Compartmental modeling is a powerful mathematical approach used to analyze the behavior of complex systems, commonly described by systems of ordinary differential equations. In recent years, it has been extensively employed to simulate a variety of biological processes, including the dynamics of infectious diseases such as COVID-19, HIV, and tuberculosis [2,8,44]. This modeling technique provides critical insights into identifying the most effective and safest strategies for controlling disease outbreaks. In this study, we establish an explicit connection between individuals addicted to drugs and potential drug users. The insights derived from these models are vital for public health efforts aimed at understanding and curbing the spread of addiction. A number of studies have contributed to building this critical foundation by developing models to predict its dynamics [26,35].

Although existing approaches elucidate critical aspects of addiction—such as its population-level transmission and the impact of treatment or intervention programs—there remains an urgent need for more comprehensive frameworks that synthesize socio-behavioral factors, economic variables, and public awareness initiatives. A particularly critical shortcoming is the propensity of many individuals to relapse following recovery or rehabilitation, which adversely affects both their health and social well-being. To address this deficiency, fractional calculus has emerged as a powerful mathematical tool [14]. Recently, fractional and fractal operators have been increasingly adopted in the modeling of complex systems. They provide a powerful framework for describing phenomena like memory, long-range dependence, and fractal behavior, which are often beyond the reach of classical integer-order calculus [31]. In biomathematics, fractional derivatives are highly effective for capturing the hereditary nature of various processes. They have been successfully used to model disease spread, population dynamics, and even environmental pollution systems (such as plastic waste management), where historical states directly shape future evolution [5,15,33]. In contrast to conventional approaches, fractional calculus inherently accounts for the memory and hereditary effects inherent in individuals with a history of addiction, thereby affording a more nuanced understanding of the condition's underlying complexity.

By integrating these factors, fractional calculus augments the predictive accuracy of mathematical models [14]. Similarly, fractal theory has proven valuable across diverse fields, including ecology, social network analysis, and studies of human behavior [12, 37]. Recent advancements have further solidified the efficacy of fractional calculus in epidemiology and behavioral sciences. For instance, contemporary studies (e.g., Izadi et al., Madani et al. [14, 23] and related recent works) have increasingly utilized fractional operators to capture the heavy-tailed decay and relapse phenomena in drug abuse and related complex social contagions, underscoring the urgent necessity of transitioning from integer-order to fractional-order methodologies. By integrating these methodologies, researchers can develop models that faithfully capture the inherent temporal memory and spatial heterogeneity of complex systems in biology, physics, and sociology, ultimately providing deeper insights into dynamics such as persistence, relapse, and propagation patterns.

The incidence rate constitutes a fundamental determinant in developing epidemiological dynamic models. Two primary functional forms of incidence rates are conventionally adopted in existing epidemic modeling frameworks: The bilinear formulation βSI , where β denotes the transmission probability per contact [4, 13], and the standard incidence formulation $\frac{\beta SI}{N}$, with N representing the total population size [20]. The bilinear incidence rate exhibits notable theoretical and practical constraints in precisely characterizing disease transmission dynamics within human populations. To overcome these shortcomings, the epidemiological modeling community has advocated for the adoption of nonlinear incidence functions, which offer a more mechanistically realistic representation of contagion processes. This paradigm shift originated with the foundational work of Capasso and Serio [7], who first formalized the concept of nonlinear incidence rates in their seminal 1973 study of the cholera outbreak in Bari. Their investigation revealed a sublinear growth pattern in disease incidence—instead of increasing proportionally with the number of infectious individuals I , the rate demonstrated a decelerating trend as I rose. This empirical finding motivated the introduction of the saturated incidence rate $\frac{\beta SI}{1+\alpha I}$, which showed strong consistency with collected data. In this formulation, the parameter α accounts for saturation mechanisms, reflecting inhibitory effects such as behavioral adaptations or resource limitations. Since then, numerous researchers [14, 18, 41] have developed and analyzed epidemiological models incorporating various forms of nonlinear incidence rates.

In fractional calculus, the Riemann-Liouville, Caputo, and Grünwald-Letnikov derivatives represent the principal definitions for fractional differentiation. The Caputo fractional derivative is the most frequently used operator among the various fractional-order differential operators employed in model development. This derivative has been widely applied to model the dynamics of infectious disease transmission and in several other studies [11, 17, 43]. Although this methodology has been well-established in fields such as epidemiology and plant virus sciences, its application to modeling drug addiction dynamics remains limited. This reveals a critical research gap, as existing models often neglect memory-driven effects or overlook the fractal characteristics essential for accurately describing drug use dynamics. To address this, we developed a *PEAR* model with a generalized nonlinear incidence rate and the Caputo operator. This framework provides a more realistic representation of the complex temporal behaviors in drug use, offering valuable insights for designing intervention strategies.

Building on the model of Kamal et al. [16], we present and evaluate a new Caputo fractional-order drug addiction model with a generalized nonlinear incidence rate. The principal contributions and novel aspects of our work are summarized as follows:

- In response to the fundamental limitations of existing integer-order models, which unre-

alistically assume memoryless transitions in addiction dynamics, we introduce a novel fractional-order compartmental model using the Caputo derivative. This mathematical choice is biologically justified by the fact that drug addiction is deeply governed by long-term neurological remodeling and behavioral memory.

- We couple this fractional framework with a generalized nonlinear incidence rate $f(P, A)$. Unlike previous models relying on simple bilinear incidence (e.g., Kamal et al. [16]), our generalized approach accommodates the saturation of transmission due to psychological alertness and limited social networks, offering a highly realistic representation of large-scale drug epidemics.

- A comprehensive analytical framework confirms the well-posedness of the model by establishing the existence, uniqueness, boundedness, and positivity of its solutions. Building on this foundation, a detailed stability analysis of the equilibrium points yields valuable insights into the impact of critical parameters on the system's dynamics.

- Sensitivity analysis of key parameters coupled with numerical simulations delivers fresh insights into the role of therapeutic measures and awareness strategies in governing addiction control.

The structure of this paper is organized as follows: Section 2 presents fundamental definitions and key results for fractional derivatives. Section 3 presents the fractional-order model, examining the existence, uniqueness, non-negativity, and boundedness of solutions, deriving R_0 , and analyzing the local/global stability of equilibria. Section 4 provides numerical simulations to validate the theory, along with a sensitivity analysis of R_0 and predictive modeling. Conclusions are drawn in Section 5.

2. Preliminaries

This section revisits essential concepts in fractional calculus, concentrating on the Caputo definition. This definition is adopted due to its strong memory effects, which are indispensable in drug abuse modeling. A particular benefit of the Caputo approach is its preservation of standard initial condition structures, ensuring compatibility with integer-order models. Moreover, its derivative for constant functions is zero, aligning with classical calculus and improving interpretability in phenomenological models.

Definition 2.1 ([31]). Let $g(t) \in L^1([t_0, \tau])$ (L^1 is the set of Lebesgue integrable functions), then the left and right RL integrals of fractional-order α of the function $g(t)$ are, respectively, given as follows

$${}^{RL}I_{t_0}^\alpha g(t) = \frac{1}{\Gamma(\alpha)} \int_{t_0}^t (t-x)^{\alpha-1} g(x) dx,$$

and

$${}^{RL}I_\tau^\alpha g(t) = \frac{1}{\Gamma(\alpha)} \int_t^\tau (x-t)^{\alpha-1} g(x) dx,$$

where $\Gamma(\cdot)$ is the well-known gamma function.

Definition 2.2 ([31]). The left and right RL fractional-order derivatives of the function $g(t)$ are, respectively, defined as follows

$${}^{RL}D_t^\alpha g(t) = \frac{1}{\Gamma(n-\alpha)} \left(\frac{d}{dt} \right)^n \int_{t_0}^t (t-x)^{n-\alpha-1} g(x) dx,$$

and

$${}_t^{RL}D_\tau^\alpha g(t) = \frac{(-1)^n}{\Gamma(n-\alpha)} \left(\frac{d}{dt}\right)^n \int_t^\tau (x-t)^{n-\alpha-1} g(x) dx,$$

where n is any positive integer such that $n-1 < \alpha \leq n$.

Definition 2.3 ([31]). Let $g(t) \in C^n(t_0, \tau)$ (i.e. $g^{(n-1)}(t)$ is absolutely continuous), then the left and right Caputo derivatives of fractional-order α of the function $g(t)$ are, respectively, defined as follows

$${}_t^C D_t^\alpha g(t) = \frac{1}{\Gamma(n-\alpha)} \int_{t_0}^t (t-x)^{n-\alpha-1} g^{(n)}(x) dx,$$

and

$${}_t^C D_\tau^\alpha g(t) = \frac{(-1)^n}{\Gamma(n-\alpha)} \int_t^\tau (x-t)^{n-\alpha-1} g^{(n)}(x) dx,$$

where n is any positive integer such that $n-1 < \alpha \leq n$.

Lemma 2.1 ([30]). Let $f(x) \in C[a, b]$ and ${}_a^C D_x^\alpha f(x) \in C(a, b]$ for $0 < \alpha \leq 1$, then

$$f(x) = f(a) + \frac{1}{\Gamma(\alpha+1)} ({}_a^C D_x^\alpha f)(\xi) (x-a)^\alpha$$

with $a \leq \xi \leq x$, $\forall x \in (a, b]$.

Using Lemma 2.1, we state the following corollary.

Corollary 2.1 ([30]). Suppose that $g(x) \in C[0, b]$ and ${}_0^C D_x^\alpha g(x) \in C(0, b]$ for $0 < \alpha \leq 1$. If ${}_0^C D_x^\alpha g(x) \geq 0$, $\forall x \in (0, b)$, then the function g is non-decreasing and if ${}_0^C D_x^\alpha g(x) \leq 0$, $\forall x \in (0, b)$, then the function g is non-increasing for all $x \in (0, b)$.

Lemma 2.2 ([21]). Let $\alpha \in (0, 1)$ and consider a continuous function $x : [t_0, \infty) \rightarrow \mathbb{R}$ satisfying the following condition

$${}_0^C D_t^\alpha x(t) + \mu x(t) \leq \nu, t \geq t_0, \mu, \nu \in \mathbb{R}, \mu \neq 0.$$

Then, we have the inequality

$$x(t) \leq \left(x(t_0) - \frac{\nu}{\mu}\right) E_\alpha(-\mu(t-t_0)^\alpha) + \frac{\nu}{\mu},$$

for all $t \geq t_0$, where E_α is the Mittag-Leffler function of one parameter defined by

$$E_\alpha(t) = \sum_{k=0}^{\infty} \frac{t^k}{\Gamma(\alpha k + 1)}.$$

Definition 2.4 ([32]). Let $F(s)$ be the Laplace transform of $f(t)$. Then,

$$\mathcal{L}\{{}_0^C D_t^\alpha f(t), s\} = s^\alpha F(s) - \sum_{i=0}^{n-1} s^{\alpha-i-1} f^{(i)}(0), \alpha \in (n-1, n]; n \in \mathbb{N}.$$

Definition 2.5 ([9]). The Mittag-Leffler function $E_{l,m}(x)$ is given by

$$E_{l,m}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(ln+m)}, x \in \mathbb{R}, l > 0, m > 0,$$

and satisfies the property

$$E_{l,m}(x) = xE_{l,l+m}(x) + \frac{1}{\Gamma(m)}.$$

Furthermore, the Laplace transform of $t^{m-1}E_{l,m}(\pm\lambda t^l)$ is given as follows:

$$\mathcal{L}\left[t^{m-1}E_{l,m}(\pm\lambda t^l)\right] = \frac{s^{l-m}}{s^l \mp \lambda}.$$

Lemma 2.3 ([20]). *Let $0 < \alpha < 1$ and $g \in C[0, T]$ be a positive-valued function. Then, for all $t \in [0, T]$, one has ${}_0^c D_t^\alpha(g(t) - g^* - g^* \ln \frac{g(t)}{g^*}) \leq \left(1 - \frac{g^*}{g(t)}\right) {}_0^c D_t^\alpha g(t)$, for all $g^* \in \mathbb{R}_+$.*

Lemma 2.4 ([29]). *Let $x \in \mathbb{R}_+$ be a continuous and differentiable function. Then, for any $t \geq 0, \alpha \in (0, 1]$, and $\bar{x} > 0$, we have*

$${}_0^c D_t^\alpha \left(x(t) - \bar{x} - \int_{\bar{x}}^{x(t)} \frac{h(\bar{x})}{h(s)} ds \right) \leq \left(1 - \frac{h(\bar{x})}{h(x(t))} \right) {}_0^c D_t^\alpha x(t),$$

where $h : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ is a differentiable and strictly increasing function.

3. Fractional-order model formulation and analysis

In this section, a drug addiction model with a generalized nonlinear incidence function is formulated. Furthermore, the positivity and boundedness of the solutions are rigorously verified, and the stability properties of the model are systematically analyzed.

3.1. Model formulation

Very recently, Kamal et al. [16] introduced a drug addiction model (3.1) that subdivides the population affected by drug abuse into distinct groups: Potential users $P(t)$, experimental users $E(t)$, addicted individuals $A(t)$, and recovered individuals $R(t)$. The population transition dynamics are governed by the following assumptions:

(A-1) Potential users (P) maintain a drug-free lifestyle, having never used or been exposed to addictive substances.

(A-2) The influence of active drug users is restricted to susceptible individuals and has no effect on those with established addiction.

(A-3) Addicted individuals (A) are now active participants in drug-related activities, having progressed from initial exposure to full engagement in addictive behaviors.

(A-4) Recovered individuals (R) have attained sustainable recovery through a range of empirically supported interventions, including public health initiatives, psychological and ethical counseling, and engagement with media and faith-based practices.

$$\begin{cases} \frac{dP}{dt} = \Lambda - \sigma PA - \mu P, \\ \frac{dE}{dt} = \sigma PA - \alpha E - \tau_D E - \mu E, \\ \frac{dA}{dt} = \alpha E - \tau_A A - \mu A, \\ \frac{dR}{dt} = \tau_D E + \tau_A A - \mu R, \end{cases} \quad (3.1)$$

where the detailed description of the parameters can be found in [16].

A key limitation of the integer-order model (3.1) is its inability to account for memory effects and hereditary properties, which are fundamental to the dynamics of biological processes. To overcome this limitation without sacrificing numerical tractability or general applicability, we introduce the Caputo fractional operator in place of the standard time derivative. By virtue of their nonlocal characteristics, fractional derivatives enable the accurate capture of memory-driven behavior resulting from long-term social interactions and neural remodeling. This approach provides a more physiologically realistic framework with an enhanced ability to model complex system dynamics. Meanwhile, since model (3.1) employs a bilinear incidence rate that suffers from unbounded growth, and considering various biologically feasible incidence rates, we introduce a class of generalized incidence functions. The new drug addiction model takes the form

$$\begin{cases} {}^c_0D_t^\alpha P = \Lambda - \sigma f(P, A) - \mu P, \\ {}^c_0D_t^\alpha E = \sigma f(P, A) - \gamma E - \tau E - \mu E, \\ {}^c_0D_t^\alpha A = \gamma E - \xi A - \mu A, \\ {}^c_0D_t^\alpha R = \tau E + \xi A - \mu R, \end{cases} \quad (3.2)$$

with initial conditions

$$P(0) = P^0 > 0, E(0) = E^0 > 0, A(0) = A^0 > 0, R(0) = R^0 > 0,$$

where ${}^c_0D_t^\alpha$ is the Caputo fractional-order derivative and $0 < \alpha \leq 1$. The descriptions of the parameters in the fractional-order PEAR drug addiction model (3.2) are shown in Table 1 [16].

Table 1. Parameter meanings for model (3.2).

Parameter	Description
Λ	Recruitment rate of new potential users.
σ	Rate at which potential users experiment with drugs.
γ	Rate at which experimental users become addicted.
ξ	Rate at which addicted individuals recover.
τ	Rate at which experimental users recover without addiction.
μ	Natural death rate of individuals.

3.2. Positivity and boundedness

This subsection establishes the nonnegativity and boundedness of solutions to system (3.2) for any positive initial condition. We assume the generalized incidence rate $f(P, A)$ is nonnegative and continuously differentiable on the interior of $\mathbb{R}_+^2$. Based on the foundational assumptions in [29], we introduce the following hypotheses (H_1) to (H_5) , which are essential for the subsequent analysis.

$$(H_1) : f(0, A) = 0, f(P, 0) = 0, \text{ for all } P, A > 0,$$

$$(H_2) : \frac{\partial f(P, 0)}{\partial P} = 0, \text{ for all } P > 0,$$

$$(H_3) : 0 < \frac{\partial f(P, A)}{\partial P} \leq \theta, \text{ for all } P, A > 0, \theta \text{ is a positive constant,}$$

$$(H_4) : 0 < \frac{\partial f(P, A)}{\partial A} \leq \frac{f(P, A)}{A}, \text{ for all } P, A > 0,$$

$(H_5) : \frac{\partial^2 f(P, A)}{\partial A^2} \leq 0$, for all $P, A \geq 0$.

The function f includes a number of specific incidence rates. For instance:

1. $f(x, y) = \beta x^p, p \geq 1$ [34].
2. $f(x, y) = (\beta - \frac{\beta_1 y}{m+y})x, \beta > \beta_1, m > 0$ [39, 40].
3. $f(x, y) = \frac{\beta x}{x+\alpha y}, \alpha > 0$ [22].
4. $f(x, y) = \frac{\beta x}{1+\alpha y^p}, \alpha > 0, 0 < p \leq 1$ [19].
5. $f(x, y) = \frac{\beta x}{1+\alpha_1 x+\alpha_2 y+\alpha_3 xy}, \alpha_1, \alpha_2, \alpha_3 \geq 0$ [3, 28, 29].

Since the recovered population $R(t)$ is absent from the other equations of system (3.2), we proceed with the following reduced system without loss of generality.

$$\begin{cases} {}^c_0 D_t^\alpha P = \Lambda - \sigma f(P, A) - \mu P, \\ {}^c_0 D_t^\alpha E = \sigma f(P, A) - \gamma E - \tau E - \mu E, \\ {}^c_0 D_t^\alpha A = \gamma E - \xi A - \mu A. \end{cases} \quad (3.3)$$

Theorem 3.1. *For any nonnegative initial condition $X(0) = (P(0), E(0), A(0))^T$, there is always a unique solution for model (3.3) on $[0, +\infty)$.*

Proof. Let $X(t) = (P(t), E(t), A(t))^T$. The model (3.3) can be written as follows

$${}_0^c D_t^\alpha X(t) = F(X(t)), \forall t \geq 0,$$

where

$$F(X(t)) = \begin{pmatrix} \Lambda - \sigma f(P, A) - \mu P \\ \sigma f(P, A) - \gamma E - \tau E - \mu E \\ \gamma E - \xi A - \mu A \end{pmatrix}.$$

Utilizing hypotheses (H_1) and (H_3) and the Lagrange mean value theorem, we can get that $f(P, A) \leq \theta P$.

Let

$$Q_1 = \begin{pmatrix} -\mu & 0 & 0 \\ 0 & -(\gamma + \tau + \mu) & 0 \\ 0 & \gamma & -(\xi + \mu) \end{pmatrix},$$

$$Q_2 = \begin{pmatrix} -\sigma\theta & 0 & 0 \\ \sigma\theta & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad Q_0 = \begin{pmatrix} \Lambda \\ 0 \\ 0 \end{pmatrix}.$$

Consequently, one can obtain $\|F(X(t))\| \leq \|Q_0\| + (\|Q_1\| + \|Q_2\|)\|X\|$.

It therefore follows from Lemma 4 in [6] that a unique solution to model (3.3) exists globally on $[0, +\infty)$. $\square$

Consider the set

$$\Omega = \{(P, E, A) \in \mathbb{R}_+^3 : P \geq 0, E \geq 0, A \geq 0\}.$$

We now prove that every solution of the model (3.3) is non-negative and lies in Ω .

Theorem 3.2. *If the initial conditions of model (3.3) are in the region Ω , then all the possible solutions of the model lie within Ω .*

Proof. Using Lemma 2.1 in combination with hypothesis (H_1) , we demonstrate the positivity of the solutions. Since

$$\begin{aligned} {}^c_0D_t^\alpha P|_{P=0} &= \Lambda > 0, \forall E, A \geq 0, \\ {}^c_0D_t^\alpha E|_{E=0} &= \sigma f(P, A) \geq 0, \forall P, A \geq 0, \\ {}^c_0D_t^\alpha A|_{A=0} &= \gamma E \geq 0, \forall P, E \geq 0. \end{aligned} \quad (3.4)$$

If $(P(0), E(0), A(0)) \in \Omega$, due to (3.4) and Corollary 2.1, the vector field on the boundary hyperplanes $P = 0$, $E = 0$, and $A = 0$ is strictly pointing into the interior of Ω or is tangent to the boundary. Thus, the region Ω is a positively invariant set, which guarantees that the solution of the fractional-order model (3.3) remains nonnegative for all $t \geq 0$.

Next, we prove the boundedness of the proposed model.

Summing all equations of system (3.3), we see that the total population size $N(t) = P(t) + E(t) + A(t)$ satisfies the inequality:

$${}^c_0D_t^\alpha N(t) = \Lambda - \mu P - (\tau + \mu) E - (\xi + \mu) A \leq \Lambda - \mu N(t).$$

By Lemma 2.2, it follows that

$$N(t) \leq \left(N(0) - \frac{\Lambda}{\mu} \right) E_\alpha(-\mu t^\alpha) + \frac{\Lambda}{\mu}$$

where $E_\alpha(t) = \sum_{k=0}^{\infty} \frac{t^k}{\Gamma(\alpha k + 1)}$ is the Mittag-Leffler function with parameter α [9]. Therefore,

$$\limsup_{t \rightarrow +\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Hence, the solutions of model (3.3) are non-negative and ultimately bounded. $\square$

3.3. Stability of equilibrium points

In this section, we are dedicated to deriving all possible equilibrium points and computing the basic reproduction number using the next-generation matrix method [10], which will be subsequently illustrated through numerical simulations under various scenarios. Furthermore, we establish the local and global stability theories for the equilibrium points of the fractional-order model (3.3).

3.3.1. Drug-free equilibrium

In the absence of drug use, the entire population belongs to the potential compartment P , with no individuals in the experimental (E) or addicted (A) compartments, i.e., $E = 0$ and $A = 0$. Under this condition, system (3.3) possesses a unique drug-free equilibrium given by $T_0(P_0, E_0, A_0) = T_0\left(\frac{\Lambda}{\mu}, 0, 0\right)$. To determine the existence and uniqueness of a drug-persistent equilibrium $T_*(P_*, E_*, A_*)$, we first derive the basic reproduction number R_0 for model (3.3) using the next-generation matrix technique [10].

Theorem 3.3. *The basic reproduction number of model (3.3) takes the following form: $R_0 = \frac{\gamma \sigma \frac{\partial f(P_0, 0)}{\partial A}}{(\xi + \mu)(\gamma + \tau + \mu)}$.*

Proof. Letting $U(t) = (E(t), A(t))^T$, the model (3.3) can be written as follows:

$${}_0^c D_t^\alpha U = \mathcal{F}(X) - \mathcal{V}(X),$$

where

$$\mathcal{F}(X) = \begin{pmatrix} \sigma f(P, A) \\ 0 \end{pmatrix}, \quad \mathcal{V}(X) = \begin{pmatrix} (\gamma + \tau + \mu) E \\ -\gamma E + (\xi + \mu) A \end{pmatrix}.$$

Let F and V be Jacobian matrices of $\mathcal{F}(X)$ and $\mathcal{V}(X)$ at T_0 , respectively, then

$$F = \begin{pmatrix} 0 & \sigma \frac{\partial f(P_0, 0)}{\partial A} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \gamma + \tau + \mu & 0 \\ -\gamma & \xi + \mu \end{pmatrix}.$$

The basic reproduction number of model (3.3) is defined as the spectral radius of the next-generation matrix $F \cdot V^{-1}$, so

$$R_0 = \frac{\gamma \sigma \frac{\partial f(P_0, 0)}{\partial A}}{(\xi + \mu)(\gamma + \tau + \mu)}.$$

□

3.3.2. Drug-persistent equilibrium

In this subsection, we prove the existence and uniqueness of the drug-persistent equilibrium.

Theorem 3.4. *If $R_0 > 1$, the model (3.3) has a unique drug-persistent equilibrium $T_*(P_*, E_*, A_*)$.*

Proof. The drug-persistent equilibrium of the model (3.3) is determined by the following system of algebraic equations:

$$\begin{cases} \Lambda - \sigma f(P, A) - \mu P = 0, \\ \sigma f(P, A) - \gamma E - \tau E - \mu E = 0, \\ \gamma E - \xi A - \mu A = 0. \end{cases} \quad (3.5)$$

Solving the above equations yields $P = \frac{\Lambda - (\gamma + \tau + \mu)E}{\mu}$, $A = \frac{\gamma E}{\xi + \mu}$, where $E \in \left[0, \frac{\Lambda}{\gamma + \tau + \mu}\right]$.

Based on the second equation of the model (3.5), we define

$$\Phi(E) = \sigma f\left(\frac{\Lambda - (\gamma + \tau + \mu)E}{\mu}, \frac{\gamma E}{\xi + \mu}\right) - (\gamma + \tau + \mu)E.$$

Using hypothesis (H_1) , we deduce $\Phi(0) = 0$ and $\Phi\left(\frac{\Lambda}{\gamma + \tau + \mu}\right) = -\Lambda < 0$.

Given the continuous differentiability of $\Phi(E)$ with respect to E in $\left[0, \frac{\Lambda}{\gamma + \tau + \mu}\right]$, we can further deduce that:

$$\Phi'(E) = -\frac{\sigma(\gamma + \tau + \mu)}{\mu} \frac{\partial f\left(\frac{\Lambda - (\gamma + \tau + \mu)E}{\mu}, \frac{\gamma E}{\xi + \mu}\right)}{\partial P} + \frac{\sigma \gamma}{\xi + \mu} \frac{\partial f\left(\frac{\Lambda - (\gamma + \tau + \mu)E}{\mu}, \frac{\gamma E}{\xi + \mu}\right)}{\partial A} - (\gamma + \tau + \mu).$$

Provided that $\Phi'(0) > 0$, there exists at least one positive root E_* of $\Phi(E) = 0$ in the interval $\left(0, \frac{\Lambda}{\gamma + \tau + \mu}\right]$.

From hypothesis (H_2) , we observe that

$$\Phi'(0) = -\frac{\sigma(\gamma + \tau + \mu)}{\mu} \frac{\partial f(P_0, 0)}{\partial P} + \frac{\sigma\gamma}{\xi + \mu} \frac{\partial f(P_0, 0)}{\partial A} - (\gamma + \tau + \mu) = (\gamma + \tau + \mu)(R_0 - 1).$$

Therefore, $R_0 > 1$ implies $\Phi'(0) > 0$. Consequently, model (3.3) admits at least one drug-persistent equilibrium $T_*(P_*, E_*, A_*)$, where

$$P_* = \frac{\Lambda - (\gamma + \tau + \mu)E_*}{\mu}, A_* = \frac{\gamma E_*}{\xi + \mu}.$$

We proceed with the proof of uniqueness by substituting the expressions for $T_*(P_*, E_*, A_*)$ into the equations of model (3.5). Thus we have

$$\gamma + \tau + \mu = \frac{\sigma f(P_*, A_*)}{E_*} = \frac{\sigma\gamma f(P_*, A_*)}{(\xi + \mu)A_*}. \quad (3.6)$$

Combining (3.6) with hypothesis (H_3) and (H_4) , we obtain

$$\begin{aligned} \Phi'(E_*) &= -\frac{\sigma(\gamma + \tau + \mu)}{\mu} \frac{\partial f(P_*, A_*)}{\partial P} + \frac{\sigma\gamma}{\xi + \mu} \frac{\partial f(P_*, A_*)}{\partial A} - \frac{\sigma\gamma f(P_*, A_*)}{(\xi + \mu)A_*} \\ &= -\frac{\sigma(\gamma + \tau + \mu)}{\mu} \frac{\partial f(P_*, A_*)}{\partial P} + \frac{\sigma\gamma}{\xi + \mu} \left(\frac{\partial f(P_*, A_*)}{\partial A} - \frac{f(P_*, A_*)}{A_*} \right) \\ &< 0. \end{aligned}$$

To establish the uniqueness of drug-persistent equilibrium $T_*(P_*, E_*, A_*)$, we employ a proof by contradiction. Suppose there exists another drug-persistent equilibrium $T_{**}(P_{**}, E_{**}, A_{**})$. By the Intermediate Value Theorem and geometric properties of continuous functions, this implies that $\Phi'(E_{**}) > 0$, which contradicts the previous finding that $\Phi'(E_*) < 0$. This contradiction completes the proof of Theorem 3.4. $\square$

3.3.3. Local stability

Before proceeding with the local stability analysis, it is essential to clarify the stability criteria for fractional-order systems. According to Matignon's stability theorem [25], a fractional linear time-invariant system ${}_0^C D_t^\alpha X = JX$ with $0 < \alpha \leq 1$ is locally asymptotically stable if and only if all eigenvalues λ_i of the Jacobian matrix J evaluated at the equilibrium satisfy the condition $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$.

The classical Routh-Hurwitz criterion (see [24]) ensures that all roots of a characteristic polynomial have strictly negative real parts, which is equivalent to the condition $|\arg(\lambda_i)| > \frac{\pi}{2}$. Since the fractional order α is bounded by $0 < \alpha \leq 1$, the inequality $\frac{\pi}{2} \geq \frac{\alpha\pi}{2}$ invariably holds. Consequently, any system that satisfies the classical Routh-Hurwitz criterion inherently satisfies Matignon's condition. Therefore, applying the classical Routh-Hurwitz criterion provides a strict sufficient condition for the local asymptotic stability of fractional-order systems. In the subsequent theorems, we utilize this criterion to establish the stability of the equilibria for model (3.3).

We present the local stability analysis for the drug-free equilibrium T_0 and the drug-persistent equilibrium T_* of model (3.3). The Jacobian matrix of the system, evaluated at an arbitrary point $X = (P, E, A)$, is derived as follows:

$$J(P, E, A) = \begin{bmatrix} -\sigma \frac{\partial f(P, A)}{\partial P} - \mu & 0 & -\sigma \frac{\partial f(P, A)}{\partial A} \\ \sigma \frac{\partial f(P, A)}{\partial P} & -(\gamma + \tau + \mu) & \sigma \frac{\partial f(P, A)}{\partial A} \\ 0 & \gamma & -(\xi + \mu) \end{bmatrix}.$$

Theorem 3.5. *If $R_0 < 1$, the drug-free equilibrium T_0 is locally asymptotically stable for all $\alpha \in (0, 1]$.*

Proof. The Jacobian matrix of model (3.3) evaluated at the drug-free equilibrium $T_0 \left(\frac{\Lambda}{\mu}, 0, 0 \right)$ is

$$J(T_0) = \begin{bmatrix} -\mu & 0 & -\sigma \frac{\partial f(P_0, 0)}{\partial A} \\ 0 & -(\gamma + \tau + \mu) & \sigma \frac{\partial f(P_0, 0)}{\partial A} \\ 0 & \gamma & -(\xi + \mu) \end{bmatrix}.$$

Considering the characteristic equation $|\lambda I - J(T_0)| = 0$, we find it factors as $(\lambda + \mu)P(\lambda) = 0$, where

$$\begin{aligned} P(\lambda) &= \lambda^2 + x_1\lambda + x_2 = 0, \\ x_1 &= \gamma + \tau + 2\mu + \xi, \\ x_2 &= (\xi + \mu)(\gamma + \tau + \mu) - \gamma\sigma \frac{\partial f(P_0, 0)}{\partial A}. \end{aligned}$$

It follows from the condition $R_0 < 1$ that $\gamma\sigma \frac{\partial f(P_0, 0)}{\partial A} < (\xi + \mu)(\gamma + \tau + \mu)$, thereby yielding $x_1 > 0$ and $x_2 > 0$. Thus, according to the Routh-Hurwitz criteria (see [24]), the local asymptotic stability of the drug-free equilibrium T_0 for the fractional-order model (3.3) is ensured under the condition $R_0 < 1$. $\square$

Theorem 3.6. *If $R_0 > 1$, the drug-persistent equilibrium T_* is locally asymptotically stable for all $\alpha \in (0, 1]$.*

Proof. The Jacobian matrix of model (3.3) evaluated at the drug-persistent equilibrium $T_* (P_*, E_*, A_*)$ is

$$J(P_*, E_*, A_*) = \begin{bmatrix} -\sigma \frac{\partial f(P_*, A_*)}{\partial P} - \mu & 0 & -\sigma \frac{\partial f(P_*, A_*)}{\partial A} \\ \sigma \frac{\partial f(P_*, A_*)}{\partial P} & -(\gamma + \tau + \mu) & \sigma \frac{\partial f(P_*, A_*)}{\partial A} \\ 0 & \gamma & -(\xi + \mu) \end{bmatrix}.$$

Expanding the characteristic equation $|\lambda I - J(P_*, E_*, A_*)| = 0$ yields the polynomial form: $\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0$, where

$$b_1 = \sigma \frac{\partial f(P_*, A_*)}{\partial P} + \gamma + \tau + 3\mu + \xi,$$

$$b_2 = (\gamma + \tau + 2\mu + \xi) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) + (\xi + \mu)(\gamma + \tau + \mu) - \gamma \sigma \frac{\partial f(P_*, A_*)}{\partial A},$$

$$b_3 = (\xi + \mu)(\gamma + \tau + \mu) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) - \gamma \sigma \mu \frac{\partial f(P_*, A_*)}{\partial A}.$$

Next, we prove the positivity of the coefficients b_1, b_2, b_3 and the expression $b_1 b_2 - b_3$. Substituting the drug-persistent equilibrium $T_*(P_*, E_*, A_*)$ into the equations of model (3.5) and applying hypothesis (H_4) yields

$$\gamma \sigma \frac{\partial f(P_*, A_*)}{\partial A} \leq \gamma \sigma \frac{f(P_*, A_*)}{A_*} = \frac{\gamma(\gamma + \tau + \mu) E_*}{\frac{\gamma E_*}{\xi + \mu}} = (\xi + \mu)(\gamma + \tau + \mu).$$

Consequently,

$$\begin{aligned} b_2 &= (\gamma + \tau + 2\mu + \xi) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) + (\xi + \mu)(\gamma + \tau + \mu) - \gamma \sigma \frac{\partial f(P_*, A_*)}{\partial A} \\ &\geq (\gamma + \tau + 2\mu + \xi) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) + (\xi + \mu)(\gamma + \tau + \mu) - (\xi + \mu)(\gamma + \tau + \mu) \\ &= (\gamma + \tau + 2\mu + \xi) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right), \\ b_3 &= (\xi + \mu)(\gamma + \tau + \mu) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) - \gamma \sigma \mu \frac{\partial f(P_*, A_*)}{\partial A} \\ &\geq (\xi + \mu)(\gamma + \tau + \mu) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) - \mu(\xi + \mu)(\gamma + \tau + \mu) \\ &= (\xi + \mu)(\gamma + \tau + \mu) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} \right), \\ b_1 b_2 - b_3 &= \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \gamma + \tau + 3\mu + \xi \right) \left[(\gamma + \tau + 2\mu + \xi) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) \right. \\ &\quad \left. + (\xi + \mu)(\gamma + \tau + \mu) - \gamma \sigma \frac{\partial f(P_*, A_*)}{\partial A} \right] \\ &\quad - \left[(\xi + \mu)(\gamma + \tau + \mu) \left(\sigma \frac{\partial f(P_*, A_*)}{\partial P} + \mu \right) - \gamma \sigma \mu \frac{\partial f(P_*, A_*)}{\partial A} \right] \\ &= \sigma(\gamma + \tau + 2\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \frac{\partial f(P_*, A_*)}{\partial P} \\ &\quad + (\gamma + \tau + 2\mu + \xi)(\gamma + \tau + 3\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \\ &\quad + (\xi + \mu)(\gamma + \tau + \mu)(\gamma + \tau + 2\mu + \xi) - \gamma \sigma(\gamma + \tau + 2\mu + \xi) \frac{\partial f(P_*, A_*)}{\partial A} \\ &\quad - \gamma \sigma^2 \frac{\partial f(P_*, A_*)}{\partial P} \frac{\partial f(P_*, A_*)}{\partial A} \\ &\geq \sigma(\gamma + \tau + 2\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \frac{\partial f(P_*, A_*)}{\partial P} \\ &\quad + (\gamma + \tau + 2\mu + \xi)(\gamma + \tau + 3\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \\ &\quad + (\xi + \mu)(\gamma + \tau + \mu)(\gamma + \tau + 2\mu + \xi) - (\gamma + \tau + 2\mu + \xi)(\xi + \mu)(\gamma + \tau + \mu) \end{aligned}$$

$$\begin{aligned}
& -\sigma(\xi + \mu)(\gamma + \tau + \mu) \frac{\partial f(P_*, A_*)}{\partial P} \\
& = \sigma(\gamma + \tau + 2\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \frac{\partial f(P_*, A_*)}{\partial P} \\
& \quad + (\gamma + \tau + 2\mu + \xi)(\gamma + \tau + 3\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \\
& \quad - \sigma(\xi + \mu)(\gamma + \tau + \mu) \frac{\partial f(P_*, A_*)}{\partial P} \\
& = \sigma(\gamma + \tau + 2\mu + \xi) \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \frac{\partial f(P_*, A_*)}{\partial P} \\
& \quad + \left[(\gamma + \tau + \mu)^2 + (\mu + \xi)(2\mu + \xi) \right] \left(\mu + \sigma \frac{\partial f(P_*, A_*)}{\partial P} \right) \\
& \quad + \mu(3\mu + 2\xi)(\gamma + \tau + \mu) + \sigma(2\mu + \xi)(\gamma + \tau + \mu) \frac{\partial f(P_*, A_*)}{\partial P}.
\end{aligned}$$

Therefore, hypothesis (H_3) ensures that the coefficients satisfy $b_1, b_2, b_3, b_1 b_2 - b_3 > 0$, which, according to the Routh-Hurwitz criteria (see [24]), implies the local asymptotic stability of the drug-persistent equilibrium T_* of model (3.3) when $R_0 > 1$. $\square$

3.3.4. Global stability

In this subsection, we analyze the global stability of both the drug-free equilibrium T_0 and the drug-persistent equilibrium T_* by employing Lyapunov theory.

Theorem 3.7. *If $R_0 < 1$, then the drug-free equilibrium T_0 is globally asymptotically stable for all $\alpha \in (0, 1]$.*

Proof. We define a Lyapunov function as follows:

$$\varphi(t) = E(t) + \frac{(\gamma + \tau + \mu)}{\gamma} A(t).$$

Applying the Caputo fractional derivative to $\varphi(t)$, we have

$${}_0^c D_t^\alpha \varphi(t) = {}_0^c D_t^\alpha E(t) + \frac{(\gamma + \tau + \mu)}{\gamma} {}_0^c D_t^\alpha A(t).$$

After straightforward calculations, it follows that

$$\begin{aligned}
{}_0^c D_t^\alpha \varphi(t) & = \sigma f(P, A) - \gamma E - \tau E - \mu E + \frac{(\gamma + \tau + \mu)}{\gamma} (\gamma E - \xi A - \mu A) \\
& = \sigma f(P, A) - \frac{(\xi + \mu)(\gamma + \tau + \mu)}{\gamma} A \\
& = \sigma f(P, A) - \sigma \frac{1}{R_0} \frac{\partial f(P_0, 0)}{\partial A} A.
\end{aligned}$$

From the first equation of model (3.3), we deduce that ${}_0^c D_t^\alpha P(t) \leq \Lambda - \mu P$. According to Lemma 2.2, it implies that $P(t) \leq \left(P(0) - \frac{\Lambda}{\mu} \right) E_\alpha(-\mu t^\alpha) + \frac{\Lambda}{\mu}$. Since the Mittag-Leffler function

$E_\alpha(-\mu t^\alpha)$ tends to zero as $t \rightarrow \infty$ [9], we obtain $\limsup_{t \rightarrow +\infty} P(t) \leq \frac{\Lambda}{\mu} = P_0$. Finally, under hypotheses (H_1) , (H_3) , (H_5) and by applying Lagrange's Mean Value Theorem, we derive

$$f(P, A) \leq \frac{\partial f(P_0, 0)}{\partial A} A.$$

Thus,

$$\begin{aligned} {}^c_0 D_t^\alpha \varphi(t) &\leq \sigma \frac{\partial f(P_0, 0)}{\partial A} A - \sigma \frac{1}{R_0} \frac{\partial f(P_0, 0)}{\partial A} A \\ &= \sigma \frac{\partial f(P_0, 0)}{\partial A} A \left(1 - \frac{1}{R_0}\right). \end{aligned}$$

So, when $R_0 < 1$ holds and hypothesis condition (H_4) is considered, we have ${}_0^c D_t^\alpha \varphi(t) \leq 0$. Furthermore, it is easy to verify that the single point set $\{T_0\}$ is the largest compact invariant set of $\{(P, E, A) \in \mathbb{R}_+^3 : {}_0^c D_t^\alpha \varphi(t) = 0\}$. According to the fractional LaSalle's invariance principle, we obtain the following conclusion: If $R_0 < 1$, then for any $\alpha \in (0, 1]$, the drug-free equilibrium T_0 is globally asymptotically stable. $\square$

Theorem 3.8. *If $R_0 > 1$ and the following inequality (H_6) holds true, then for any $\alpha \in (0, 1]$, the drug-persistent equilibrium T_* is globally asymptotically stable.*

$$(H_6) \left(1 - \frac{f(P, A_*)}{f(P, A)}\right) \left(\frac{f(P, A)}{f(P, A_*)} - \frac{A}{A_*}\right) \leq 0, \quad \forall P, A > 0.$$

Proof. Define the Lyapunov function $\Psi(t)$ as follows:

$$\begin{aligned} \Psi(t) &= \left(P(t) - P_* - \int_{P_*}^{P(t)} \frac{f(P_*, A_*)}{f(\theta, A_*)} d\theta\right) + \left(E(t) - E_* - \int_{E_*}^{E(t)} \frac{E_*}{\theta} d\theta\right) \\ &\quad + \frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(A(t) - A_* - \int_{A_*}^{A(t)} \frac{A_*}{\theta} d\theta\right). \end{aligned}$$

According to Lemma 2.4, we can see that

$${}_0^c D_t^\alpha \Psi(t) \leq \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) {}_0^c D_t^\alpha P(t) + \left(1 - \frac{E_*}{E}\right) {}_0^c D_t^\alpha E(t) + \frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(1 - \frac{A_*}{A}\right) {}_0^c D_t^\alpha A(t).$$

By utilizing the first equation of model (3.3), we derive that

$$\left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) {}_0^c D_t^\alpha P(t) = \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (\Lambda - \sigma f(P, A) - \mu P). \quad (3.7)$$

Substituting the expression $\Lambda = \sigma f(P_*, A_*) + \mu P_*$ into (3.7), we obtain

$$\begin{aligned} \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) {}_0^c D_t^\alpha P(t) &= \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (\sigma f(P_*, A_*) + \mu P_* - \sigma f(P, A) - \mu P) \\ &= \mu \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (P_* - P) \\ &\quad + \sigma f(P_*, A_*) \left(1 - \frac{f(P, A)}{f(P_*, A_*)} - \frac{f(P_*, A_*)}{f(P, A_*)} + \frac{f(P, A)}{f(P, A_*)}\right). \end{aligned} \quad (3.8)$$

Recalling the second equation of model (3.3), we can see that

$$\left(1 - \frac{E_*}{E}\right) {}^c_0D_t^\alpha E(t) = \left(1 - \frac{E_*}{E}\right) [\sigma f(P, A) - (\gamma + \tau + \mu) E].$$

On the basis of the established equation $\gamma + \tau + \mu = \frac{\sigma f(P_*, A_*)}{E_*}$, we derive that

$$\begin{aligned} \left(1 - \frac{E_*}{E}\right) {}^c_0D_t^\alpha E(t) &= \left(1 - \frac{E_*}{E}\right) \left(\sigma f(P, A) - \sigma f(P_*, A_*) \frac{E}{E_*}\right) \\ &= \sigma f(P_*, A_*) \left(1 - \frac{E}{E_*} - \frac{E_* f(P, A)}{E f(P_*, A_*)} + \frac{f(P, A)}{f(P_*, A_*)}\right). \end{aligned} \quad (3.9)$$

In view of the third equation of model (3.3), we obtain

$$\frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(1 - \frac{A_*}{A}\right) {}^c_0D_t^\alpha A(t) = \frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(1 - \frac{A_*}{A}\right) [\gamma E - (\xi + \mu) A]. \quad (3.10)$$

By substituting $\xi + \mu = \frac{\gamma E_*}{A_*}$ into (3.10), we deduce that

$$\begin{aligned} \frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(1 - \frac{A_*}{A}\right) {}^c_0D_t^\alpha A(t) &= \frac{\sigma f(P_*, A_*)}{\gamma E_*} \left(1 - \frac{A_*}{A}\right) \left(\gamma E - \frac{\gamma E_* A}{A_*}\right) \\ &= \sigma f(P_*, A_*) \left(1 + \frac{E}{E_*} - \frac{A}{A_*} - \frac{A_* E}{A E_*}\right). \end{aligned} \quad (3.11)$$

Combined with (3.8), (3.9) and (3.11), one can obtain

$$\begin{aligned} & {}^c_0D_t^\alpha \Psi(t) \\ & \leq \mu \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (P_* - P) + \sigma f(P_*, A_*) \left(1 - \frac{f(P, A)}{f(P_*, A_*)} - \frac{f(P_*, A_*)}{f(P, A_*)} + \frac{f(P, A)}{f(P, A_*)}\right) \\ & \quad + \sigma f(P_*, A_*) \left(1 - \frac{E}{E_*} - \frac{E_* f(P, A)}{E f(P_*, A_*)} + \frac{f(P, A)}{f(P_*, A_*)}\right) + \sigma f(P_*, A_*) \left(1 + \frac{E}{E_*} - \frac{A}{A_*} - \frac{A_* E}{A E_*}\right) \\ & = \mu \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (P_* - P) + \sigma f(P_*, A_*) \left(3 - \frac{f(P_*, A_*)}{f(P, A_*)}\right. \\ & \quad \left. + \frac{f(P, A)}{f(P, A_*)} - \frac{E_* f(P, A)}{E f(P_*, A_*)} - \frac{A}{A_*} - \frac{A_* E}{A E_*}\right) \\ & = \mu \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (P_* - P) + \sigma f(P_*, A_*) \left(1 - \frac{f(P, A_*)}{f(P, A)}\right) \left(\frac{f(P, A)}{f(P, A_*)} - \frac{A}{A_*}\right) \\ & \quad + \sigma f(P_*, A_*) \left(4 - \frac{f(P_*, A_*)}{f(P, A_*)} - \frac{E_* f(P, A)}{E f(P_*, A_*)} - \frac{A f(P, A_*)}{A_* f(P, A)} - \frac{A_* E}{A E_*}\right). \end{aligned}$$

From hypothesis (H_3) , since $f(P, A)$ is a monotonically increasing function of P , it follows that

$$\begin{aligned} \frac{f(P_*, A_*)}{f(P, A_*)} &\geq 1, \forall P_* \geq P(t), \\ \frac{f(P_*, A_*)}{f(P, A_*)} &\leq 1, \forall P_* \leq P(t). \end{aligned}$$

Hence, $\mu \left(1 - \frac{f(P_*, A_*)}{f(P, A_*)}\right) (P_* - P) \leq 0$. By hypothesis (H_6) and the arithmetic-geometric mean inequality, we obtain ${}^c_0D_t^\alpha \Psi(t) \leq 0$. Consequently, according to the LaSalle invariance principle, the drug-persistent equilibrium T_* of model (3.3) is globally asymptotically stable. $\square$

4. Numerical results and discussion

Drug education and early intervention programs play a critical role in shaping individuals' perceptions of substance abuse and their understanding of its consequences, thereby influencing their subsequent choices and behaviors regarding drug use.

For potential users ($P(t)$), who are at risk of initiation, comprehensive education can heighten awareness of the dangers of addiction, the risk of overdose, and the long-term health and social impacts. This strengthened awareness helps build resilience and reduces the likelihood of progressing to experimentation.

Experimental users ($E(t)$), who are in a critical and volatile stage of initial use, are highly susceptible to developing dependence. Targeted interventions at this stage can educate them about the rapid transition from recreational use to addiction and encourage help-seeking behavior before a severe substance use disorder develops.

For those who are already addicted ($A(t)$), proactive and sustained education within treatment frameworks is essential. It improves their understanding of relapse triggers, promotes adherence to treatment plans, and supports the adoption of harm-reduction strategies—all of which are vital for recovery and for mitigating the negative impacts on their lives and communities.

To validate the established results of this study, we adopt a nonlinear saturated incidence rate $f(P, A) = \frac{\beta PA}{1 + \omega A}$, which captures both the psychological alertness among the general population as the number of addicts A increases (saturation effect) and the limited social circles of addicts that prevent the contact rate from growing linearly without bound. This improvement renders the model more consistent with realistic population dynamics in predicting large-scale drug epidemics compared with the original model (3.1). Here, β is the transmission coefficient and ω is the saturation constant. We introduce these parameters to analyze the impact of self-initiated preventive measures within the three population groups: Potential users, experimental users, and addicted individuals. The corresponding model is formulated as follows:

$$\begin{cases} {}^c_0D_t^\alpha P = \Lambda - \sigma \frac{\beta PA}{1 + \omega A} - \mu P, \\ {}^c_0D_t^\alpha E = \sigma \frac{\beta PA}{1 + \omega A} - \gamma E - \tau E - \mu E, \\ {}^c_0D_t^\alpha A = \gamma E - \xi A - \mu A, \\ {}^c_0D_t^\alpha R = \tau E + \xi A - \mu R. \end{cases} \quad (4.1)$$

We now verify that $f(P, A) = \frac{\beta PA}{1 + \omega A}$ satisfies the hypotheses (H_1) to (H_6) . Direct calculation gives:

$$\begin{aligned} \frac{\partial f(P, A)}{\partial P} &= \frac{\beta A}{1 + \omega A}, \\ \frac{\partial f(P, A)}{\partial A} &= \frac{\beta P}{(1 + \omega A)^2}, \\ \frac{\partial^2 f(P, A)}{\partial A^2} &= \frac{-2\beta\omega P}{(1 + \omega A)^3}. \end{aligned}$$

From these expressions, it is straightforward to confirm that the hypotheses (H_1) and (H_2) are satisfied.

Since $0 < \frac{\partial f(P, A)}{\partial P} = \frac{\beta A}{1 + \omega A} \leq \frac{\beta}{\omega}$, it follows that hypothesis (H_3) holds.

Since $0 < \frac{\partial f(P,A)}{\partial A} = \frac{\beta P}{(1+\omega A)^2} \leq \frac{\beta P}{1+\omega A} = \frac{f(P,A)}{A}$, hypothesis (H_4) holds.

Since $\frac{\partial^2 f(P,A)}{\partial A^2} = \frac{-2\beta\omega P}{(1+\omega A)^3} \leq 0$, hypothesis (H_5) holds.

Since

$$\begin{aligned} \left(1 - \frac{f(P, A_*)}{f(P, A)}\right) \left(\frac{f(P, A)}{f(P, A_*)} - \frac{A}{A_*}\right) &= \frac{A(1 + \omega A_*)}{A_*(1 + \omega A)} + \frac{1 + \omega A}{1 + \omega A_*} - \frac{A}{A_*} - 1 \\ &= \frac{-\omega(A_* - A)^2}{A_*(1 + \omega A)(1 + \omega A_*)} \\ &\leq 0, \end{aligned}$$

it is obvious that hypothesis (H_6) holds.

The basic reproduction number for model (4.1) is given by $R_0 = \frac{\gamma\sigma\Lambda\beta}{\mu(\xi+\mu)(\gamma+\tau+\mu)}$.

4.1. Stability analysis of drug-free equilibrium

This section explores the stability of the drug-free equilibrium under different initial conditions. The following parameter values are selected for model (4.1) to ensure that $R_0 \approx 0.8569 < 1$: $\alpha = 0.998, \Lambda = 0.9, \sigma = 0.25, \mu = 0.045, \gamma = 0.22, \tau = 0.32, \xi = 0.35, \beta = 0.18, \omega = 0.4$. The initial values adopted for the numerical simulations denote the proportion of each compartment relative to the total population, with detailed values presented in Table 2.

Table 2. Initial compartment proportions for model (4.1).

Case	$P(0)$	$E(0)$	$A(0)$	$R(0)$
1	0.882	0.010	0.010	0.098
2	0.827	0.019	0.029	0.125
3	0.780	0.029	0.039	0.152
4	0.736	0.039	0.049	0.176
5	0.684	0.049	0.068	0.199
6	0.635	0.059	0.088	0.218
7	0.581	0.077	0.116	0.226

As illustrated in Figure 1, when the basic reproduction number is successfully controlled below unity ($R_0 \approx 0.8569 < 1$), the temporal trajectories of all population compartments exhibit a consistent and definitive convergence pattern, completely independent of their diverse initial conditions. The proportion of potential users $P(t)$ robustly approaches its maximum capacity (Figure 1a), while the active drug-using populations—both experimental and addicted ($E + A$)—steadily decline and ultimately vanish (Figure 1b). Consequently, the recovered population $R(t)$ also safely dissipates over time (Figure 1c), visually confirming the global asymptotic stability of the drug-free equilibrium T_0 . Moreover, the convergence behavior is independent of the initial conditions, with all variables eventually tending to the drug-free equilibrium $T_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$. From a dynamical systems perspective, $R_0 < 1$ mathematically guarantees that the overall rate of individuals exiting the addiction cycle (via recovery or natural mortality) strictly exceeds the rate at which new users are recruited. Translated into public health policy, this result defines the ultimate criterion for successful addiction containment. It profoundly

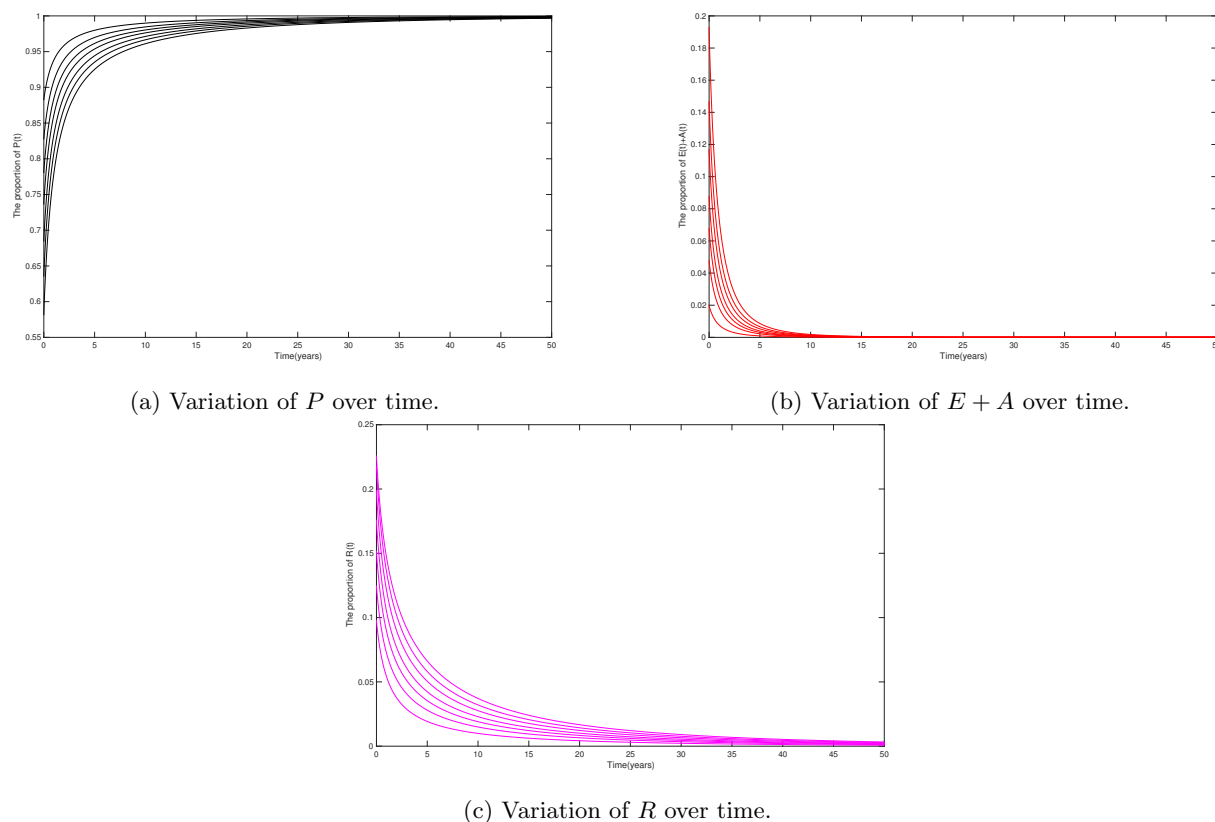


Figure 1. Variation of the proportion of P , $E + A$ and R over time with different initial values, $R_0 \approx 0.8569 < 1$.

demonstrates that the long-term eradication of a drug epidemic does not necessitate reducing the initial number of addicts to zero overnight. Instead, policymakers must focus their resources on constructing a comprehensive intervention ecosystem—combining robust primary prevention to lower incidence with efficient clinical treatment to accelerate recovery—to synergistically suppress the system’s threshold parameter R_0 below 1. Once this epidemiological threshold is secured, the community effectively achieves a state of “social immunity” against substance abuse, guaranteeing the gradual, inevitable, and permanent extinction of the drug epidemic regardless of its initial scale.

4.2. Stability analysis of drug-persistent equilibrium

This section investigates the stability of the drug-persistent equilibrium when $R_0 \approx 2.2524 > 1$. The parameters of model (4.1) are set to $\alpha = 0.998$, $\Lambda = 0.6$, $\sigma = 0.5$, $\mu = 0.07$, $\gamma = 0.35$, $\tau = 0.3$, $\xi = 0.3$, $\beta = 0.4$. Various initial conditions are listed in Table 2.

Case 1. When $\omega = 0$, $f(P, A) = \beta PA$.

Case 2. When $\omega = 0.4$, $f(P, A) = \frac{\beta PA}{1 + \omega A}$.

Figures 2 and 3 illustrate the stability characteristics of the drug-persistent equilibrium under two distinct behavioral scenarios: A purely bilinear incidence without psychological deterrence ($\omega = 0$, Figure 2) and a saturated incidence incorporating societal vigilance ($\omega = 0.4$, Figure 3). The results demonstrate a profound global convergence; when the basic reproduction number exceeds unity ($R_0 \approx 2.2524 > 1$), all trajectories—regardless of their vastly different

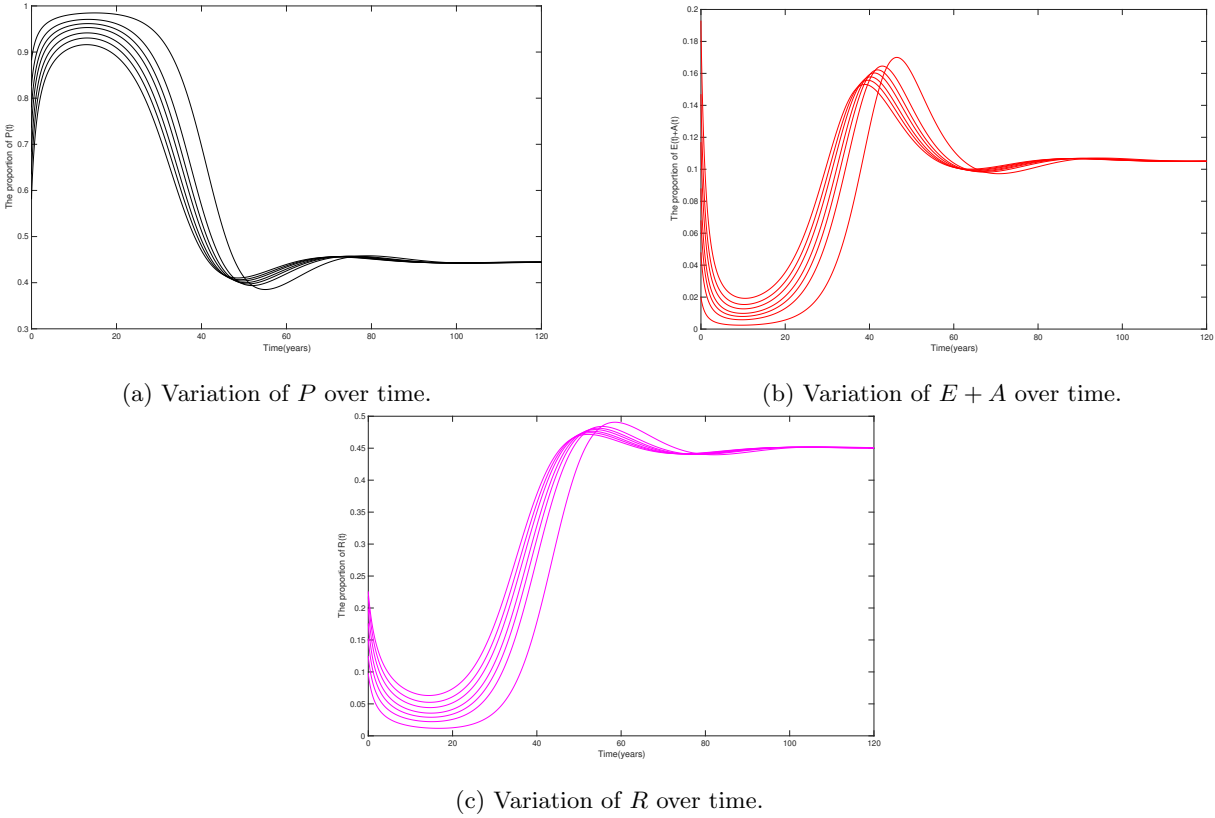


Figure 2. Variation of the proportion of P , $E + A$ and R over time with different initial values.

initial demographic distributions—inevitably approach the unique drug-persistent equilibrium T_* . Epidemiologically, this global stability signifies that the recruitment rate of new drug users structurally outpaces the combined rates of successful rehabilitation and natural mortality. From a public health and sociological perspective, this mathematical inevitability serves as a severe cautionary tale. It implies that if a community’s environmental and behavioral parameters allow R_0 to breach the threshold of 1, the drug epidemic will irreversibly entrench itself as a permanent, endemic societal fixture. Even if the initial number of experimental or addicted individuals is minuscule, the mathematical dynamics dictate that the addiction crisis will not “burn itself out” nor resolve through natural demographic turnover. Consequently, policymakers are warned that merely managing the symptoms of an outbreak is futile. To prevent a perpetual crisis, governance strategies must aggressively dismantle transmission networks (lowering β and σ) and radically expand treatment capacities (raising ξ) to force the fundamental threshold R_0 back below 1, which is the only mathematically proven avenue to break the cycle of persistent substance dependence.

4.3. Sensitivity analysis of R_0

To guide drug control strategies and understand the influence of system parameters on the basic reproduction number R_0 , Theorem 3.7 establishes that eradicating drug addiction requires interventions that adjust system parameters so that $R_0 < 1$. Conversely, Theorem 3.8 demonstrates that if existing measures are insufficient to reduce R_0 below unity, alternative strategies must be

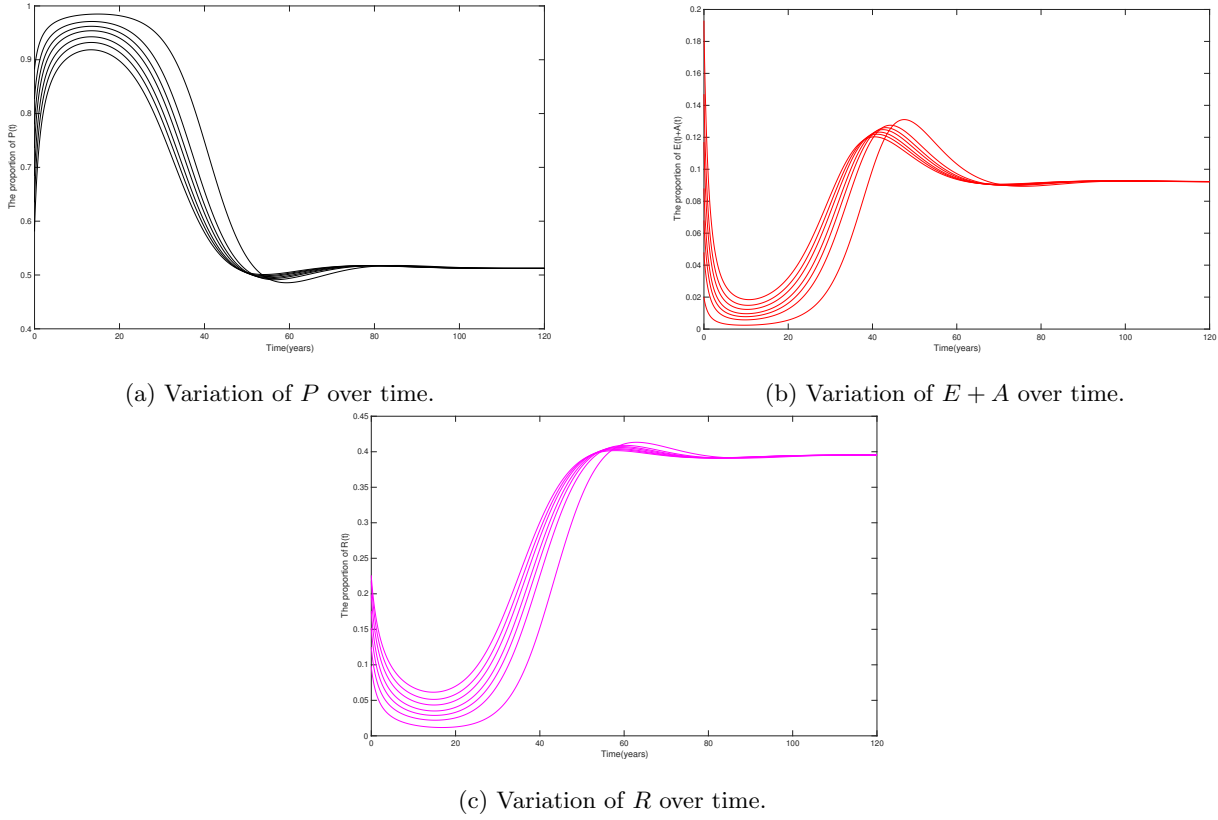


Figure 3. Variation of the proportion of P , $E + A$ and R over time with different initial values.

implemented to lower the prevalence of addicted individuals, thereby steering the system toward a drug-persistent equilibrium. This section therefore performs a sensitivity analysis on R_0 to evaluate how variations in key parameters affect this threshold.

This section analyzes the sensitivity of the threshold parameter R_0 using the normalized sensitivity index defined as $\gamma_{y_i}^{R_0} = \frac{\partial R_0}{\partial y_i} \times \frac{y_i}{R_0}$ [38]. This index quantifies the relative influence of each system parameter y_i on R_0 . By evaluating these indices, key parameters can be identified and strategically adjusted to control R_0 . Sensitivity indices for a selection of system parameters are computed under fixed values $\Lambda = 380$ and $\mu = 0.031$ presented in [16], with the corresponding experimental configurations provided in Table 3.

$$\begin{aligned}\gamma_{\sigma}^{R_0} &= 1 > 0, \\ \gamma_{\gamma}^{R_0} &= \frac{\tau + \mu}{\gamma + \tau + \mu} > 0, \\ \gamma_{\xi}^{R_0} &= -\frac{\xi}{\xi + \mu} < 0, \\ \gamma_{\tau}^{R_0} &= -\frac{\tau}{\gamma + \tau + \mu} < 0, \\ \gamma_{\beta}^{R_0} &= 1 > 0.\end{aligned}$$

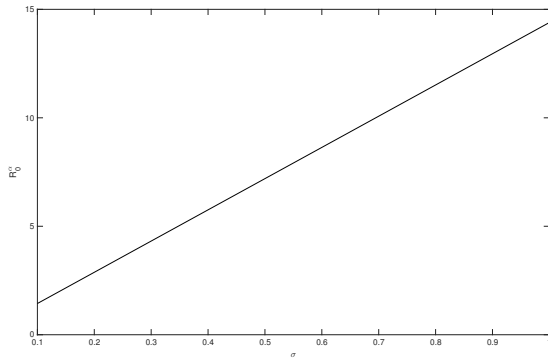
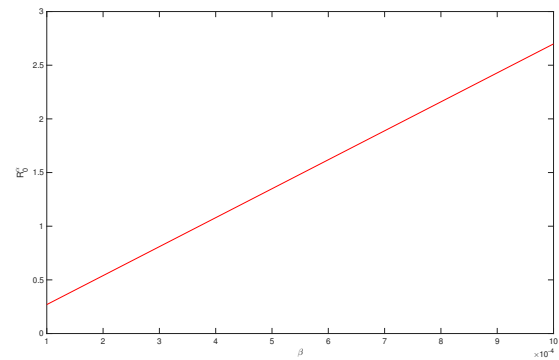
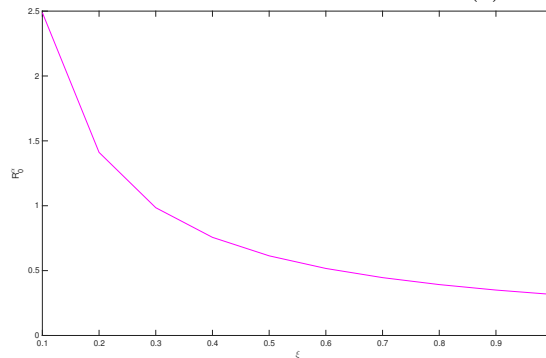
Based on Table 3, we graphically visualize R_0 to explore the impact of parameters σ , β , and ξ on its variation, as shown in Figure 4.

Table 3. Parameter values used in estimating the sensitivity indices of R_0 .

Parameter	σ	γ	ξ	τ	β
Value 1	0.15	0.08	0.12	0.25	0.0008
Value 2	0.20	0.12	0.08	0.15	0.0012
Value 3	0.10	0.05	0.15	0.30	0.0005
Value 4	0.25	0.15	0.05	0.10	0.0015
Value 5	0.08	0.10	0.10	0.20	0.0006

Table 4. The sensitivity indices of R_0 .

Group	$\gamma_{\sigma}^{R_0}$	$\gamma_{\gamma}^{R_0}$	$\gamma_{\xi}^{R_0}$	$\gamma_{\tau}^{R_0}$	$\gamma_{\beta}^{R_0}$
Set 1	1.0000	0.7784	-0.7947	-0.6925	1.0000
Set 2	1.0000	0.6013	-0.7207	-0.4983	1.0000
Set 3	1.0000	0.8688	-0.8287	-0.7874	1.0000
Set 4	1.0000	0.4662	-0.6173	-0.3559	1.0000
Set 5	1.0000	0.6979	-0.7634	-0.6042	1.0000

(a) Variation of R_0 with σ .(b) Variation of R_0 with β .(c) Variation of R_0 with ξ .**Figure 4.** The evolution of R_0 with σ , β and ξ .

Based on the sensitivity analysis, the basic reproduction number R_0 exhibits a positive

correlation with the experimentation rate σ , the transmission coefficient β , and the addiction progression rate γ . As quantified in Table 4, R_0 is most profoundly sensitive to σ and β . Given the unitary sensitivity indices ($\gamma_\sigma^{R_0} = \gamma_\beta^{R_0} = 1$), any percentage reduction in these parameters yields an exactly proportional decrease in the epidemic threshold. Epidemiologically, these parameters govern the initial “social contagion” and drug availability, making them the most efficient leverage points for intervention. In contrast, γ exerts a relatively weaker influence on R_0 , which biologically and sociologically indicates that once an individual transitions into the experimental phase, the neurological and behavioral memory of drug use heavily entrenches their trajectory. This observation complements the strong “memory effects” governed by the fractional-order dynamic framework, demonstrating that downstream interventions to alter γ are mathematically and practically less efficient than preemptive prevention.

Conversely, R_0 is negatively correlated with the treatment-induced recovery rate ξ and the spontaneous recovery rate τ . Among these, R_0 demonstrates a substantially higher sensitivity to variations in ξ , as illustrated by the variation curves in Figure 4. Even minor enhancements in ξ can significantly depress R_0 , a phenomenon uniquely captured by the fractional-order derivative, which accounts for the long-term, cumulative memory effects inherent in the rehabilitation process. In addiction medicine, this reflects the reality that recovery is not an instantaneous state change but a historically dependent journey where sustained medical and psychosocial support cumulatively reinforces sobriety. Meanwhile, the spontaneous early recovery rate τ has the least overall impact, underscoring that without structured clinical intervention, relying solely on an individual’s unassisted cessation is insufficient to curb the broader epidemic.

Guided by these sensitivity profiles, public health authorities can strategically optimize the allocation of anti-drug resources. High-impact strategies must prioritize “demand reduction” via aggressive educational deterrence to lower σ , alongside strict “supply reduction” and law enforcement to minimize drug accessibility and lower β . Moderate-efficiency measures should focus on expanding long-term clinical rehabilitation infrastructure to boost ξ , while early identification programs targeting γ serve as a necessary secondary defense. Furthermore, the distinct insights provided by the fractional-order structure emphatically demonstrate that addiction dynamics are heavily governed by temporal memory and historical dependence. Consequently, conventional integer-order strategies that assume memoryless transitions must be fundamentally adapted; public health interventions must be sustained, historically informed, and preemptive to successfully dismantle the complex temporal mechanisms of substance dependence.

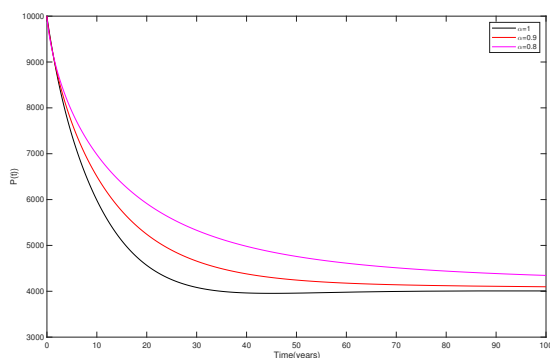
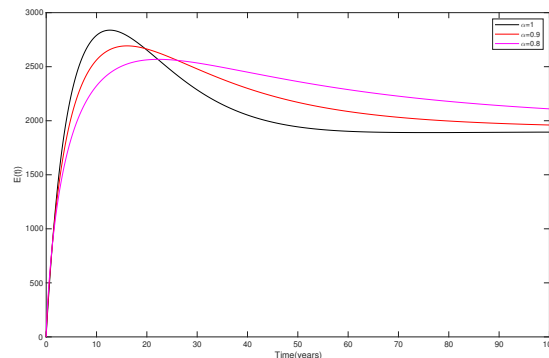
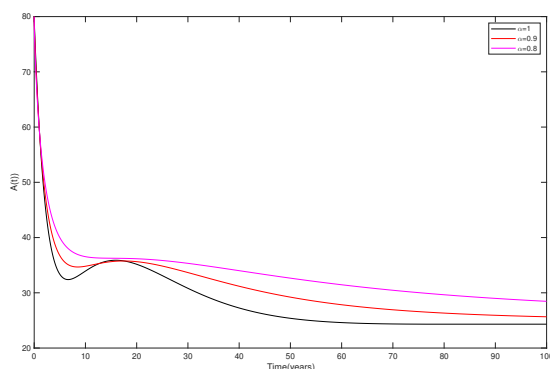
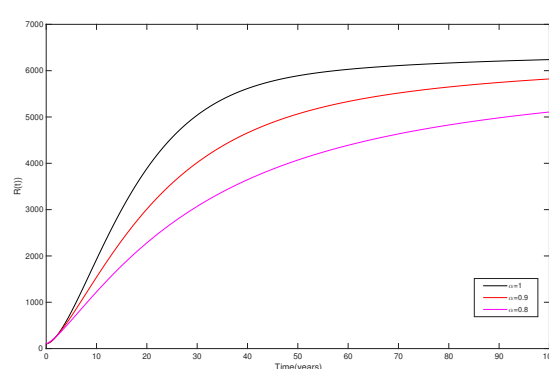
4.4. Model prediction

This section undertakes numerical simulations using MATLAB to elucidate the influence of system parameters on the dynamics of drug addiction. The selected parameter values, presented in Table 5, are principally informed by established studies, supplemented by necessary assumptions where prior data was unavailable. All simulations are conducted based on the initial conditions: $P(0) = 10,000$, $E(0) = 0$, $A(0) = 80$, $R(0) = 100$.

To explicitly elucidate the role of the fractional order α , we conduct a numerical comparison between the classical integer-order model ($\alpha = 1$) and our fractional-order models ($\alpha = 0.9, 0.8$). The results, depicted in Figure 5, clearly demonstrate the profound impact of the fractional index. As α decreases, the system exhibits a stronger “memory effect.” As shown, the integer-order model ($\alpha = 1$) overestimates both the initial outbreak velocity of drug transmission (Figure 5b) and the long-term recovery efficiency (Figure 5d), suggesting an unrealistically rapid resolution of the drug epidemic. By contrast, the fractional-order models reveal a “long-tail” character-

Table 5. Parameter values used for predictions in model (4.1).

Parameter	Value	Source	Parameter	Value	Sources
Λ	380	[16]	σ	0.1	Assumed
μ	0.031	[16]	γ	0.004	Assumed
τ	0.1	[16]	ξ	0.28	[16]
β	0.09	Assumed	ω	0.1	Assumed

(a) Variation of P over time.(b) Variation of E over time.(c) Variation of A over time.(d) Variation of R over time.**Figure 5.** Forecasting of the future drug addiction in the context of model (4.1) with $\alpha = 1, 0.9$ and 0.8 , respectively.

istic in the addiction dynamics; lower values of α lead to a slower decline in the number of addicts and a higher residual addicted population over time. Biologically, this confirms that the deeply ingrained neurological memory of addiction makes rehabilitation a protracted, historically dependent process. This comparison justifies the necessity of the fractional-order framework: Integer-order models may mislead public health authorities into prematurely withdrawing interventions, whereas fractional models accurately capture the stubborn persistence of substance dependence.

As shown in Figure 6, the transition rate γ from experimental drug users to addicted individuals plays a decisive role in the overall dynamics of the system. Numerical simulations indicate that as γ increases from 0.004 to 0.044, the steady-state levels of the potential population

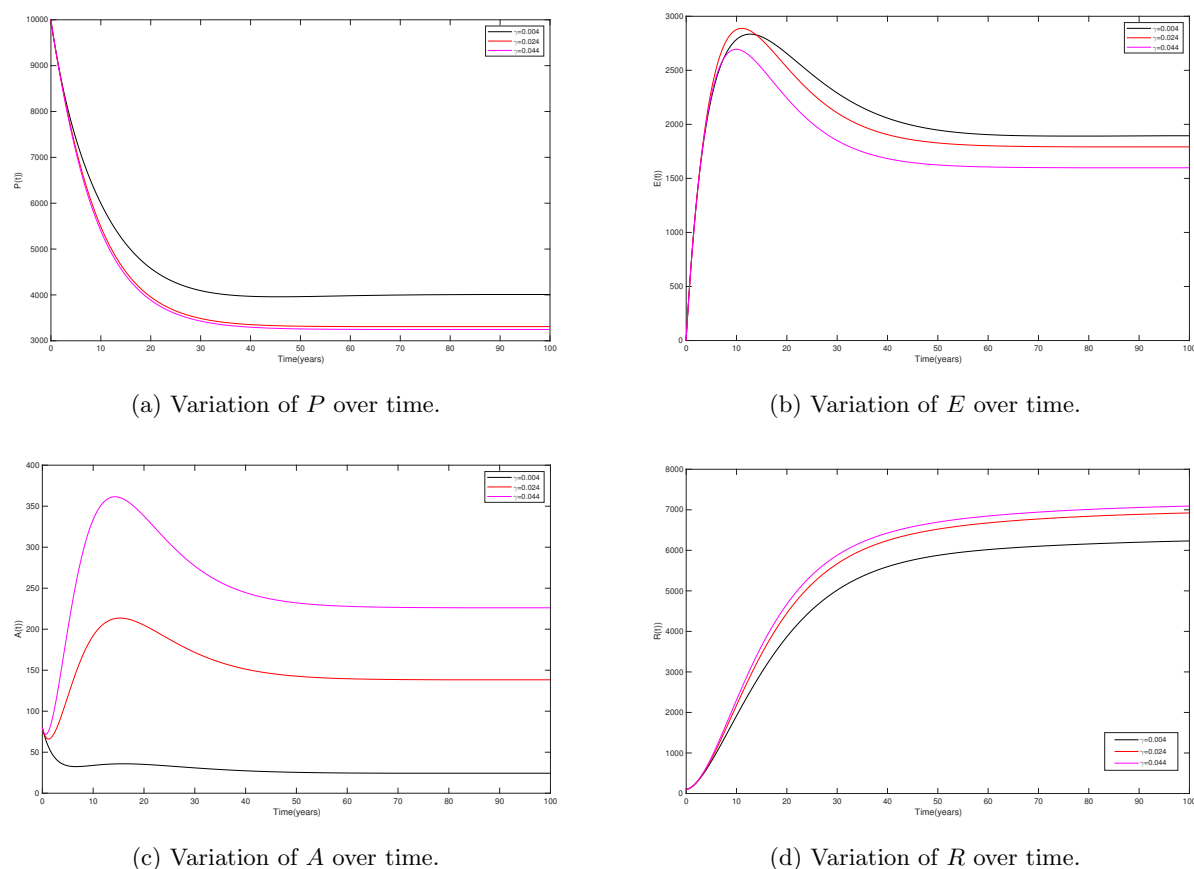


Figure 6. Forecasting of the future drug addiction in the context of model (4.1) with $\gamma = 0.004, 0.024$ and 0.044 , respectively.

$P(t)$ and the experimental population $E(t)$ decline significantly, while the size of the addicted population $A(t)$ expands sharply, accompanied by a consequent increase in the total recovered population $R(t)$. From the perspective of dynamical mechanisms, an elevated γ not only directly accelerates the flow of experimenters into the addicted compartment, but also—owing to the large addicted population acting as a primary driver of transmission—accelerates the assimilation and depletion of the potential population through the nonlinear saturated incidence function $f(P, A) = \frac{\beta PA}{1 + \omega A}$. This result profoundly reveals that the experimental stage constitutes a critical intervention window for blocking the spread of drug dependence. In practical public health policy formulation, this suggests that decision-makers should not rely solely on the post-addiction rehabilitation system, but must adopt targeted upfront interventions for early drug-exposed groups—such as establishing mandatory early psychological intervention and behavioral correction mechanisms, and strengthening community peer intervention networks—to substantially reduce the transition rate γ , thereby breaking the vicious cycle of drug transmission at its source.

As illustrated in Figure 7, enhancing the treatment and recovery rate ξ yields a profound suppressing effect on the spread of drug addiction. Numerical simulations reveal that as ξ increases from 0.28 to 0.48, the steady-state size of the potential user population $P(t)$ expands, whereas both the experimental population $E(t)$ and the addicted population $A(t)$ undergo a substantial contraction. Counterintuitively, the long-term recovered population $R(t)$ also stabi-

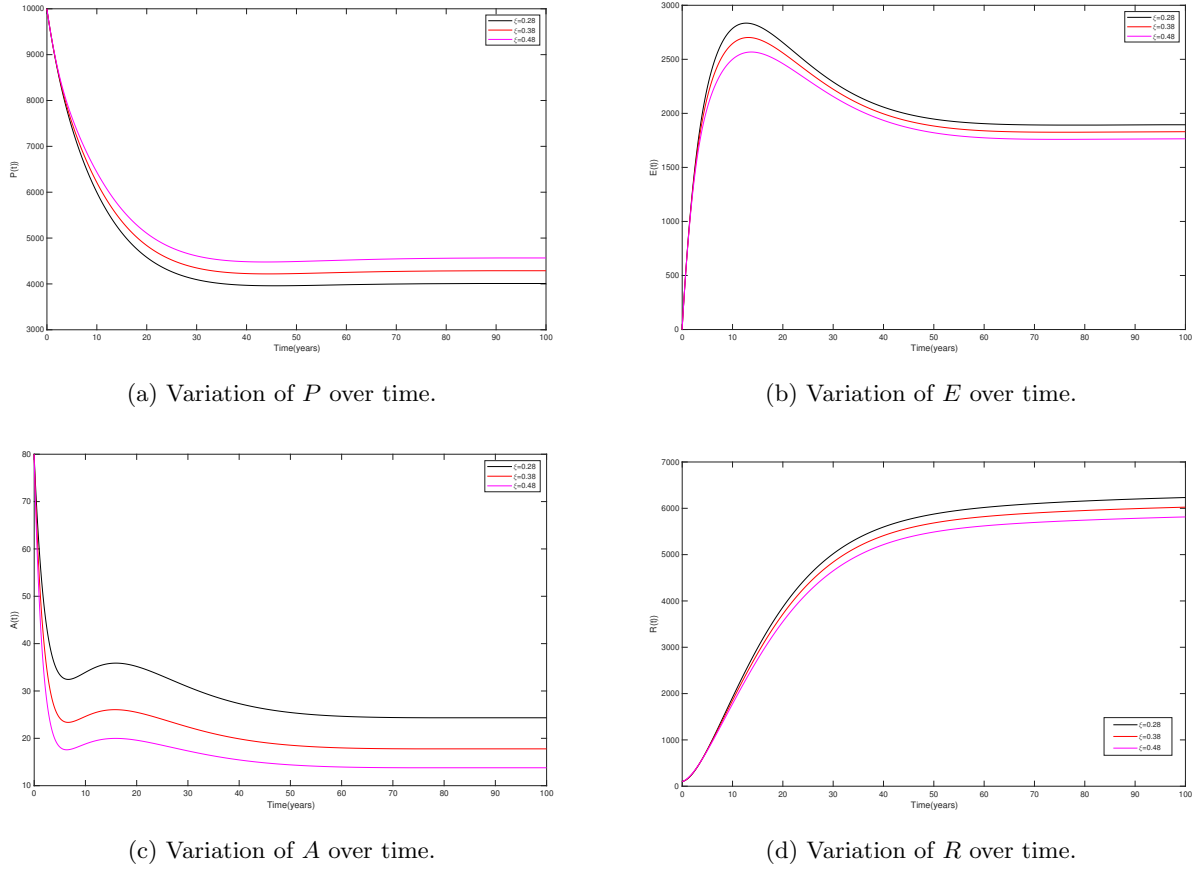


Figure 7. Forecasting of the future drug addiction in the context of model (4.1) with $\xi = 0.28, 0.38$ and 0.48 , respectively.

lizes at a lower level. This dynamic outcome occurs because a higher ξ accelerates the removal of individuals from the addicted compartment. While this directly rehabilitates existing users, it more importantly shrinks the core “infectious pool”. Through the mechanism of the nonlinear incidence function $f(P, A) = \frac{\beta PA}{1 + \omega A}$, the depleted addicted population significantly reduces the contact and transmission risk for potential users, thereby limiting the overall scale of the drug epidemic. The subsequent reduction in the total number of people entering the addiction cycle explains the ultimate decrease in the cumulative recovered population $R(t)$. From a public health policy perspective, these mathematical findings highlight a crucial biological interpretation: Medical treatment and rehabilitation for addicts are not merely “backend” remedial measures, but possess a powerful “secondary prevention” effect. Decision-makers should prioritize funding for accessible rehabilitation infrastructure and clinical addiction treatments, as efficiently curing existing addicts is a highly proactive strategy to sever the transmission network and protect the broader susceptible population from initial exposure.

As depicted in Figure 8, increasing the early recovery rate of experimental users (τ) exerts a cascading protective effect on the entire population dynamics. Numerical results indicate that elevating τ from 0.1 to 0.2 leads to a notable expansion of the potential population $P(t)$ and a swift, significant reduction in both the experimental $E(t)$ and addicted $A(t)$ populations, alongside a corresponding rise in the recovered population $R(t)$. From a dynamical perspective, τ acts as a critical interception mechanism within the addiction pipeline: By successfully diverting

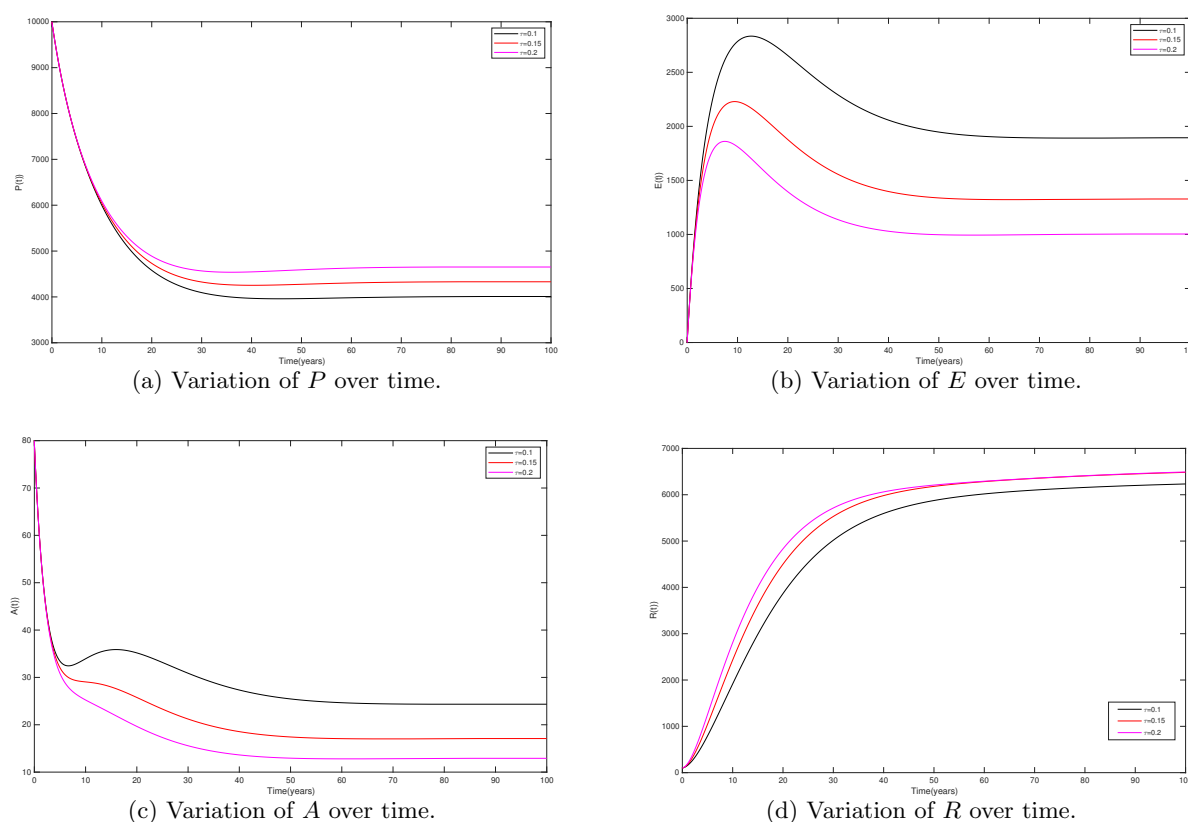


Figure 8. Forecasting of the future drug addiction in the context of model (4.1) with $\tau = 0.1, 0.15$ and 0.2 , respectively.

individuals directly from the experimental phase to recovery, it prevents them from progressing into severe physiological and psychological dependence. Consequently, this preemptive diversion fundamentally shrinks the future addicted pool $A(t)$, which in turn weakens the nonlinear transmission feedback loop $f(P, A)$ and shields a broader base of potential users from initial exposure. In the context of public health policy, these mathematical insights strongly advocate for a strategic emphasis on early intervention. Rather than waiting for severe addiction to manifest, intervention strategies must target the fragile window of experimentation. Implementing proactive screening in educational and community settings, providing accessible psychological counseling for early-stage users, and offering targeted educational diversion programs can effectively “short-circuit” the trajectory of addiction, thereby mitigating the epidemic’s spread at a significantly lower systemic and social cost.

Figure 9 illustrates the profound impact of varying the effective contact rate σ on the population-level dynamics. Numerical outcomes demonstrate that an increase in σ from 0.1 to 0.3 dramatically depletes the potential population $P(t)$, while propelling both the experimental $E(t)$ and addicted $A(t)$ populations to significantly higher prevalence levels. The recovered population $R(t)$ also experiences a consequent rise purely due to the massive influx of individuals moving through the addiction pipeline. Mechanistically, σ acts as the primary catalyst for the entire epidemic outbreak; it dictates the intensity of “social contagion” governed by the incidence function $f(P, A)$. A higher σ implies that the presence of existing addicts triggers a stronger peer-influence effect, rapidly pulling susceptible individuals into the experimental phase

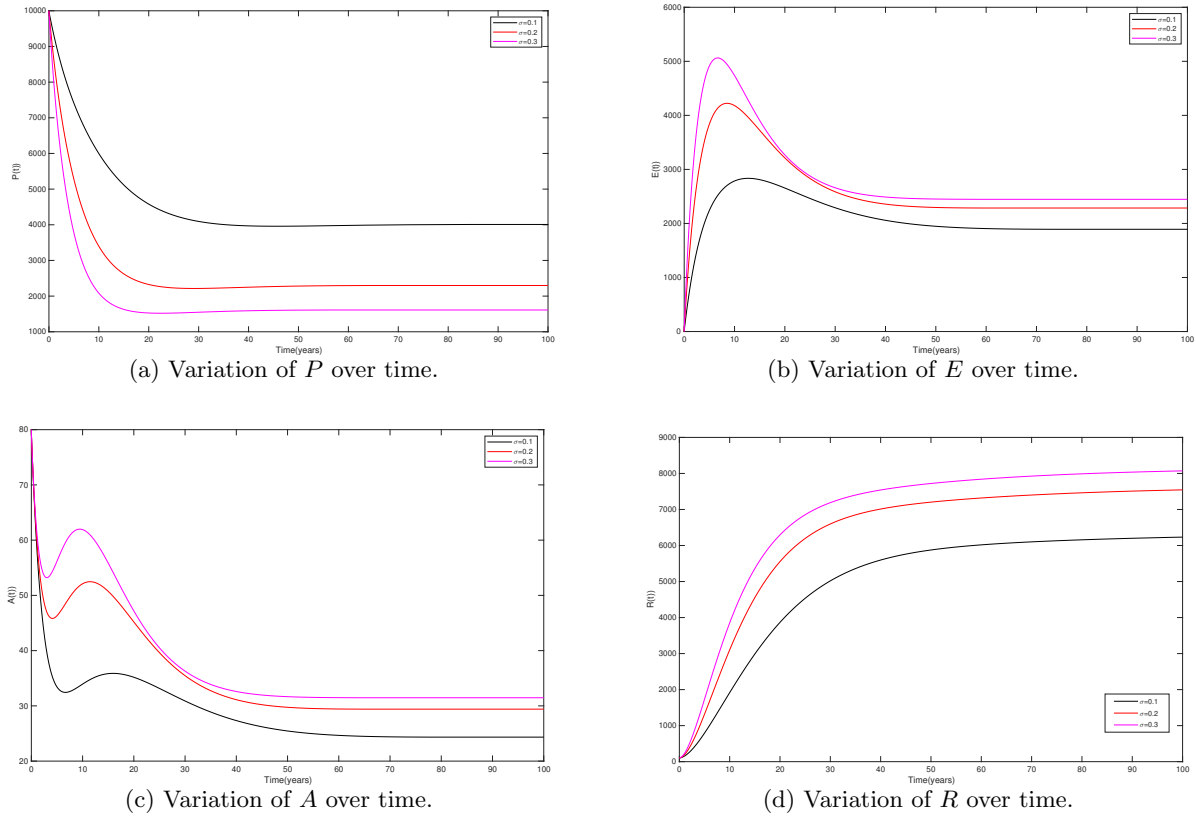


Figure 9. Forecasting of the future drug addiction in the context of model (4.1) with $\sigma = 0.1, 0.2$ and 0.3 , respectively.

and igniting a chain reaction of substance dependence. Translating these dynamical insights into public health strategies, managing σ is tantamount to executing robust primary prevention. To fundamentally curb the spread of drug abuse, policymakers must prioritize macro-level interventions that neutralize the risk of initial exposure before the first use ever occurs. This mandates implementing comprehensive, evidence-based drug resistance education in schools, fortifying community policing to restrict drug availability, and dismantling the social pathways through which addictive behaviors are socially transmitted.

As illustrated in Figure 10, an escalation in the drug transmission coefficient β significantly exacerbates the scale of the addiction epidemic. Numerical simulations show that as β increases from 0.09 to 0.15, the steady-state level of the potential population $P(t)$ drops markedly, while the experimental $E(t)$ and addicted $A(t)$ populations surge to substantially higher levels. The recovered population $R(t)$ correspondingly increases, driven passively by the inflated baseline of addicted individuals entering the treatment phase. From a mathematical epidemiology perspective, β dictates the per-contact probability of “infection” within the nonlinear incidence term $\frac{\beta PA}{1+\omega A}$. A higher β implies a highly contagious drug environment where interactions between addicts and potential users are more likely to result in new drug use, thereby accelerating the depletion of the susceptible pool. In reality, this transmission coefficient reflects environmental and societal facilitators, such as the market availability of illicit substances, the purity and addictiveness of the drugs, and the structural density of high-risk social networks. To address this from a public health and law enforcement standpoint, reducing β necessitates robust environ-

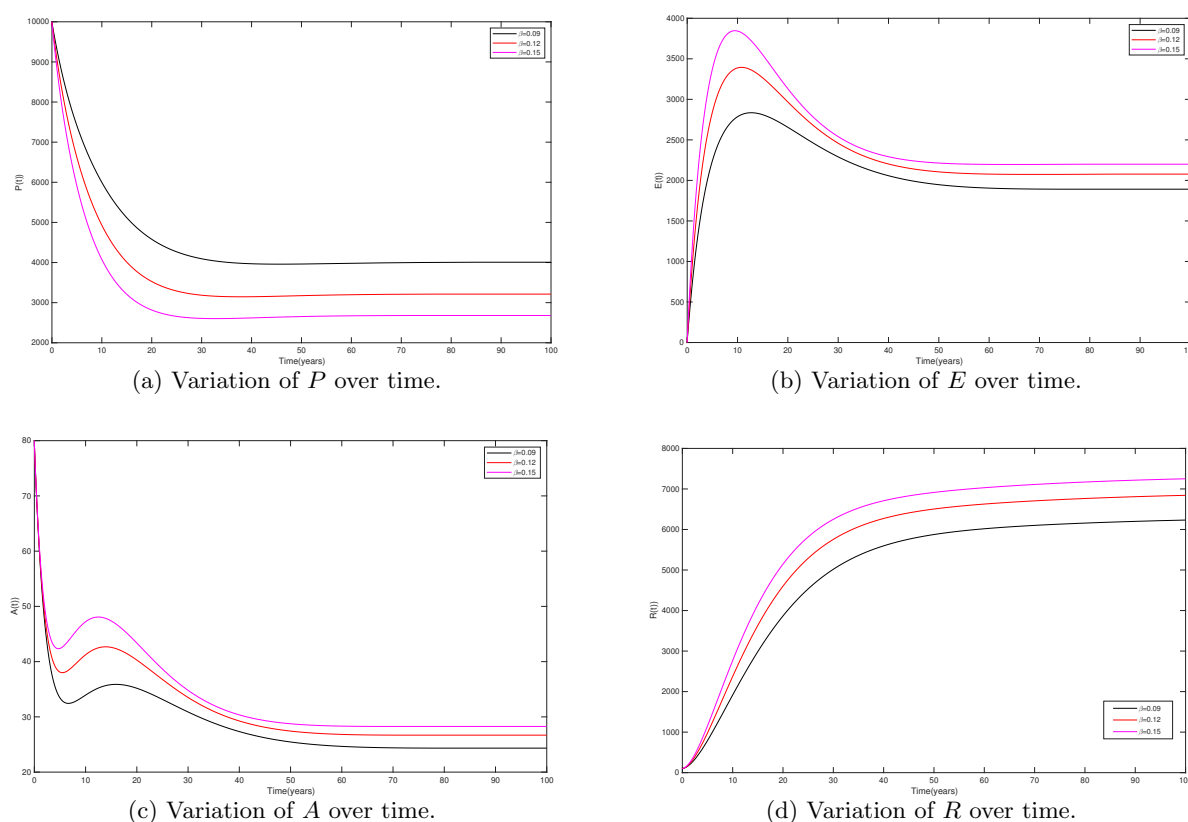


Figure 10. Forecasting of the future drug addiction in the context of model (4.1) with $\beta = 0.09, 0.12$ and 0.15 , respectively.

mental controls and supply reduction strategies. Policymakers must deploy aggressive measures to disrupt drug supply chains, crack down on drug-trafficking networks, and eliminate neighborhood “hotspots” where drug use clusters. By strictly restricting physical access to illicit substances and dismantling the illicit infrastructure that enables transmission, authorities can effectively lower the transmission coefficient β , thus insulating the broader community from the pervasive reach of addiction.

As depicted in Figure 11, although the saturation constant ω does not alter the theoretical basic reproduction number R_0 , it profoundly modulates the ultimate magnitude of the drug epidemic. Numerical simulations demonstrate that an increase in ω from 0.1 to 0.3 effectively preserves the potential population $P(t)$, while significantly suppressing both the peak and steady-state prevalence of experimental $E(t)$ and addicted $A(t)$ users. Consequently, the recovered population $R(t)$ also diminishes in size, reflecting a fundamental contraction of the overall addiction pool. Mechanistically, ω governs the saturation effect within the nonlinear incidence function $f(P, A) = \frac{\beta PA}{1 + \omega A}$. It mathematically captures the psychological alertness and self-protective behavioral adaptations of the public: As the visible number of addicts (A) grows, it triggers a heightened community-level perception of risk, which inherently deters susceptible individuals from initiating drug use. Translating this mathematical mechanism into practical addiction control strategies suggests that policymakers should actively seek to enhance societal vigilance (thereby increasing ω). This can be achieved by launching transparent public health campaigns that expose the severe physiological and socio-economic realities of substance

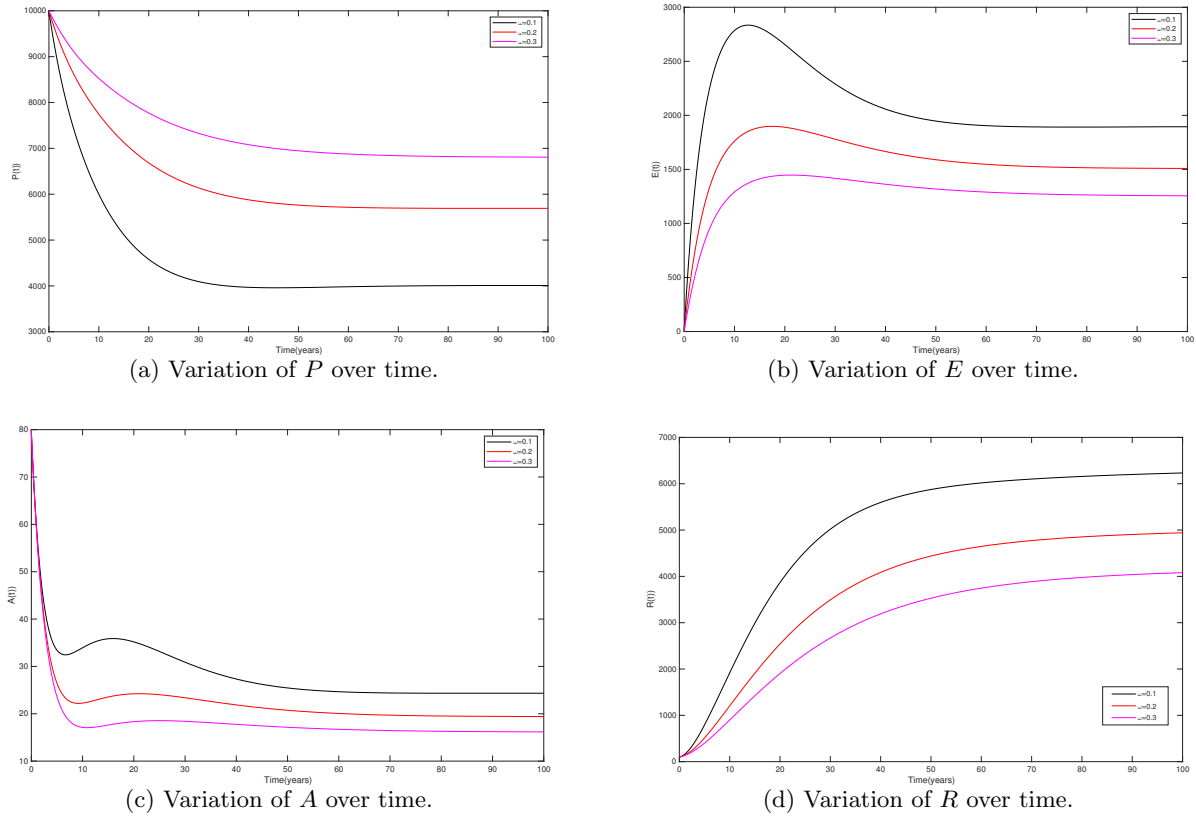


Figure 11. Forecasting of the future drug addiction in the context of model (4.1) with $\omega = 0.1, 0.2$ and 0.3 , respectively.

abuse, integrating impactful anti-drug education into school curricula, and establishing community warning systems. By transforming the harsh consequences of existing addiction into a powerful societal deterrent, authorities can build a robust psychological “firewall” that naturally restricts the drug epidemic’s rampant spread, providing a vital complement to traditional supply-reduction and medical-treatment measures.

Based on the numerical simulations of model (4.1), each parameter critically influences the temporal dynamics of drug addiction, offering profound insights for designing highly targeted intervention strategies. Unlike classical integer-order frameworks, the fractional-order approach mathematically encapsulates the persistent “memory effects” and historical dependence inherent in substance abuse. Analyzing these parameter variations allows us to understand not only the immediate behavioral shifts but also the long-term, systemic trajectory of the drug epidemic. This deeply mechanistic understanding is vital for public health authorities to formulate evidence-based policies that transcend short-term fixes, directly addressing the temporal complexity of addiction to facilitate sustainable population-level recovery.

A comprehensive public health strategy must systematically modulate these key parameters to disrupt the addiction cycle before the neurological and social “memory” of drug use irrevocably solidifies. To suppress the experimentation rate σ and the effective transmission coefficient β —the fundamental drivers of the drug epidemic—policymakers must prioritize aggressive “primary prevention” and “supply reduction.” This requires implementing evidence-based school prevention programs and restrictive legislation to lower σ , coupled with robust law enforce-

ment, online monitoring, and the dismantling of illicit supply networks to reduce β . Simultaneously, mitigating the progression rate γ from experimental use to full addiction demands swift “secondary prevention”; authorities must deploy early psychological intervention services, risk assessment tools, and targeted peer support to intercept users before deep physiological dependence takes root.

On the backend of the drug epidemic, enhancing the treatment-induced recovery rate ξ is paramount, necessitating the widespread expansion of clinical rehabilitation infrastructure, accessible psychosocial therapies, and long-term follow-up systems that account for the protracted nature of addiction recovery. Supplementing this, the spontaneous early recovery rate τ can be bolstered by destigmatization campaigns and 24/7 digital self-help networks. Furthermore, maximizing the saturation constant ω —which acts as a societal “psychological firewall”—requires transparent public warning campaigns and responsible media reporting to artificially elevate community vigilance against the severe consequences of drug use. Ultimately, successfully navigating the history-dependent dynamics of addiction requires a multi-sectoral, culturally adapted approach. By synergistically integrating strict supply disruption, proactive early intervention, and sustained medical rehabilitation, policymakers can decisively alter the drug epidemic’s trajectory and secure lasting population-wide immunity.

By examining these simulation outcomes, we gain deeper insight into the intrinsic mechanisms underlying addiction and how different interventions influence population-level trends. Successful implementation of these strategies depends on multi-sectoral collaboration, culturally adapted approaches grounded in local epidemiological data, continuous monitoring and evaluation, and optimal resource allocation prioritizing high-impact interventions. Such an integrated approach can effectively alter the dynamics of drug addiction and support population-level recovery. This knowledge is indispensable for designing evidence-based policies and approaches aimed at preventing drug abuse and supporting the recovery of affected individuals.

5. Conclusion

This paper establishes and rigorously analyzes a novel fractional-order drug addiction model incorporating a generalized nonlinear incidence rate $f(P, A)$. To ensure the model’s validity and applicability, a set of mathematical assumptions, labeled (H_1) to (H_6) , is introduced to constrain and rationalize generalized incidence rates. During model construction, we examine the existence, non-negativity, and boundedness of solutions and define the basic reproduction number R_0 as a key metric for assessing the addiction potential of the drug. Building on this foundation, we establish criteria for the local asymptotic stability of both the drug-free equilibrium T_0 and the drug-persistent equilibrium T_* , providing a rigorous framework for understanding the dynamic progression of drug addiction.

A cornerstone finding of this analytical framework is the global asymptotic stability of the system based on the threshold R_0 . Notably, when $R_0 < 1$, the drug-free equilibrium T_0 is proven to be globally asymptotically stable. From a public health perspective, this mathematically guarantees that if comprehensive interventions can systematically suppress the reproduction threshold below unity, the drug epidemic will inevitably be eradicated, regardless of the initial severity of the crisis. Conversely, when $R_0 > 1$, the drug-persistent equilibrium T_* becomes globally asymptotically stable. This serves as a severe cautionary tale for policymakers: Without adequate structural interventions to break the transmission chain, the addiction crisis will not naturally dissipate but will irreversibly entrench itself as a permanent societal endemic.

Finally, utilizing the saturated incidence rate $\frac{\beta PA}{1+\omega A}$, our sensitivity and predictive analyses translate these mathematical thresholds into actionable policy directives. We demonstrate that to effectively force $R_0 < 1$, public health authorities must deploy high-impact “primary and secondary prevention” to lower the experimentation rate σ , transmission coefficient β , and progression rate γ , while simultaneously expanding “tertiary medical rehabilitation” to enhance the recovery rates ξ and τ . Furthermore, although the saturation constant ω does not mathematically alter the R_0 threshold, our simulations reveal that actively enhancing this parameter through transparent public awareness campaigns acts as a crucial “psychological firewall,” significantly curbing the drug epidemic’s maximum magnitude. Ultimately, this fractional-order framework underscores that combating drug addiction requires sustained, history-informed, and multi-sectoral strategies.

The dynamic mechanisms of drug addiction are undoubtedly far more complex and diverse than can be adequately captured by existing mathematical formulations. Consequently, this study possesses inherent limitations to some extent. Potential directions for future research are as follows:

- **Optimal Control:** The systematic integration of optimal control theory into fractional-order drug addiction models offers a promising methodological advance, enabling the design of dynamic, time-dependent intervention strategies. By adapting control measures in response to evolving drug abuse patterns, such an approach seeks to simultaneously minimize both addiction prevalence and associated economic costs—ultimately identifying efficient pathways toward sustainable addiction containment.
- **Parameter Estimation and Model Validation:** A critical direction for future research involves calibrating the model parameters using real-world drug addiction data via advanced statistical techniques, such as Bayesian inference or maximum likelihood estimation. Subsequent validation against longitudinal datasets, together with comprehensive sensitivity analysis, would help evaluate the model’s predictive accuracy and robustness. This rigorous calibration–validation framework is essential to enhance the model’s reliability and ensure its utility in guiding public health policies.

Author contributions

Sijie Yu: Writing-original draft, Writing-review & editing; Dapeng Gao: Supervision, Writing-review & editing; Shiqiang Feng: Writing-original draft & Writing-review; Qiang Chen: Writing-original draft & Writing-review.

Conflict of interest

All authors declare no conflicts of interest in this paper.

References

- [1] B. A. Ahmad, J. S. Rossen and M. S. Sutton, *Opioid-Involved Overdose Deaths in the United States, 2019*, CDC, Atlanta, 2021.
- [2] A. Allahamou, E. Azroul, Z. Hammouch and A. L. Alaoui, *Modeling and numerical investigation of a conformable co-infection model for describing hantavirus of the european moles*, Math. Methods Appl. Sci., 2022, 45(5), 2736–2759.

- [3] M. R. S. Ammi, M. Tahiri and D. F. M. Torres, *Global stability of a caputo fractional SIRS model with general incidence rate*, Math. Comput. Sci., 2021, 15(1), 91–105.
- [4] R. M. Anderson and R. M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, Oxford, 1991.
- [5] M. Boukhobza, A. Debbouche, L. Shangerganes and D. F. Torres, *Modeling the dynamics of the hepatitis B virus via a variable-order discrete system*, Chaos Soliton Fract., 2024, 184, 114987.
- [6] A. Boukhouima, K. Hattaf and N. Yousfi, *Dynamics of a fractional order HIV infection model with specific functional response and cure rate*, Int. J. Differ. Equ., 2017, 2017(1), 8372140.
- [7] V. Capasso and G. Serio, *A generalization of the Kermack-McKendrick deterministic epidemic model*, Math. Biosci., 1978, 42(1–2), 43–61.
- [8] J. Danane, Z. Hammouch, K. Allali, S. Rashid and J. Singh, *A fractional-order model of coronavirus disease 2019 (COVID-19) with governmental action and individual reaction*, Math. Methods Appl. Sci., 2023, 46(7), 8275–8288.
- [9] K. Diethelm, *The Analysis of Fractional Differential Equations: An Application-Oriented Exposition Using Differential Operators of Caputo Type*, Springer, Berlin, 2010.
- [10] P. Van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci., 2002, 180(1–2), 29–48.
- [11] M. Farman, R. Sarwar and A. Akgul, *Modeling and analysis of sustainable approach for dynamics of infections in plant virus with fractal fractional operator*, Chaos Soliton Fract., 2023, 170, 113373.
- [12] P. Goswami, B. Pokhriyal and K. Kumar, *On the solution of two-dimensional local fractional LWR model of fractal vehicular traffic flow*, Math. Comput. Sci., 2025, 6(2), 43–63.
- [13] H. W. Hethcote, *The mathematics of infectious diseases*, SIAM Rev., 2000, 42(4), 599–653.
- [14] M. Izadi, P. Veeresha and W. Adel, *Mathematical and computational analysis of a fractional-order drug abuse model with nonlinear incidence and logistic growth*, Math. Comput. Simul., 2026, 240, 451–472.
- [15] H. Joshi, M. Yavuz and N. Özdemir, *Analysis of novel fractional order plastic waste model and its effects on air pollution with treatment mechanism*, J. Appl. Anal. Comput., 2024, 14(6), 3078–3098.
- [16] M. Kamal, M. Ahmed and M. A. H. Sarkar, *Optimal control strategies for mitigating drug addiction: A mathematical modeling approach*, Results Control Optim., 2025, 19, 100580.
- [17] B. Khaminsou, C. Thaiprayoon, W. Sudsutad and S. A. Jose, *Qualitative analysis of a proportional caputo fractional pantograph differential equation with mixed nonlocal conditions*, Nonlinear Funct. Anal. Appl., 2021, 26(1), 197–223.
- [18] S. Kundu, N. Kumari, S. Kouachi and P. Kundu, *Stability and bifurcation analysis of a heroin model with diffusion, delay and nonlinear incidence rate*, Model. Earth Syst. Environ., 2022, 8(1), 1351–1362.

- [19] A. Lahrouz, L. Omari, D. Kiouach and A. Belmaâti, *Complete global stability for an SIRS epidemic model with generalized non-linear incidence and vaccination*, Appl. Math. Comput., 2012, 218(11), 6519–6525.
- [20] C. Vargas-De-León, *On the global stability of SIS, SIR and SIRS epidemic models with standard incidence*, Chaos Soliton Fract., 2011, 44(12), 1106–1110.
- [21] H. L. Li, L. Zhang, C. Hu, Y. L. Jiang and Z. Teng, *Dynamical analysis of a fractional-order predator-prey model incorporating a prey refuge*, J. Appl. Math. Comput., 2017, 54, 435–449.
- [22] Z. Li, Z. Teng and H. Miao, *Modeling and control for HIV/AIDS transmission in China based on data from 2004 to 2016*, Comput. Math. Methods Med., 2017, 2017(1), 8935314.
- [23] N. Madani, Z. Hammouch and H. Jafari, *Fractal-fractional modeling of drug addiction dynamics: Capturing memory-driven effects*, Math. Methods Appl. Sci., 2026, 49(3), 2114–2128.
- [24] S. Mangal, O. P. Misra and J. Dhar, *Fractional-order deterministic epidemic model for the spread and control of HIV/AIDS with special reference to Mexico and India*, Math. Comput. Simul., 2023, 210, 82–102.
- [25] D. Matignon, *Stability results for fractional differential equations with applications to control processing*, Comput. Eng. Syst. Appl., 1996, 2(1), 963–968.
- [26] F. Matonya, *Mathematical Modeling on the Effects of Drug Abuse to the Societies*, Ph.D. thesis, NM-AIST, 2022.
- [27] B. M. Mayosi, J. E. Lawn, A. Van Niekerk, D. Bradshaw, S. S. A. Karim and H. M. Coovadia, *Health in South Africa: Changes and challenges since 2009*, Lancet, 2012, 380(9858), 2029–2043.
- [28] A. Mouaouine, A. Boukhouima, K. Hattaf and N. Yousfi, *A fractional order SIR epidemic model with nonlinear incidence rate*, Adv. Differ. Equ., 2018, 2018(1), 1–9.
- [29] M. Naim, F. Lahmidi, A. Namir and A. Kouidere, *Dynamics of an fractional SEIR epidemic model with infectivity in latent period and general nonlinear incidence rate*, Chaos Soliton Fract., 2021, 152, 111456.
- [30] Z. M. Odibat and N. T. Shawagfeh, *Generalized Taylor's formula*, Appl. Math. Comput., 2007, 186, 286–293.
- [31] I. Podlubny, *Fractional Differential Equations: An Introduction to Fractional Derivatives, Fractional Differential Equations, to Methods of their Solution and Some of their Applications*, Academic Press, San Diego, 1998.
- [32] S. G. Samko, A. A. Kilbas and O. I. Marichev, *Fractional Integrals and Derivatives: Theory and Applications*, Gordon and Breach Science Publishers, Yverdon, 1993.
- [33] M. Shams, N. Kausar and B. Carpentieri, *A class of high-order fractional parallel iterative methods for nonlinear engineering problems: Convergence, stability, and neural network-based acceleration*, Chaos Soliton Fract., 2025, 199, 116646.
- [34] C. Sun, Y. Lin and S. Tang, *Global stability for an special SEIR epidemic model with nonlinear incidence rates*, Chaos Soliton Fract., 2007, 33(1), 290–297.
- [35] H. Tang, M. Li, X. Yan, Z. Lu and Z. Jia, *Modeling the dynamics of drug spreading in China*, Int. J. Environ. Res. Public Health, 2021, 18(1), 288.

- [36] P. Trucillo, R. Campardelli, M. Scognamiglio and E. Reverchon, *Control of liposomes diameter at micrometric and nanometric level using a supercritical assisted technique*, J. CO2 Util., 2019, 32, 119–127.
- [37] M. F. Uddin, M. G. Hafez, Z. Hammouch and D. Baleanu, *Traveling wave with beta derivative spatial-temporal evolution for describing the nonlinear directional couplers with meta-materials via two distinct methods*, Alex. Eng. J., 2021, 60(1), 1055–1065.
- [38] R. K. Upadhyay and P. Singh, *Modeling and control of computer virus attack on a targeted network*, Physica A, 2020, 538, 122617.
- [39] L. Wang, Z. Liu and X. Zhang, *Global dynamics for an age-structured epidemic model with media impact and incomplete vaccination*, Nonlinear Anal. Real World Appl., 2016, 32, 136–158.
- [40] L. Wang, X. Zhang and Z. Liu, *An SEIR epidemic model with relapse and general nonlinear incidence rate with application to media impact*, Qual. Theory Dyn. Syst., 2018, 17(2), 309–329.
- [41] Y. Wei, J. Zhan and J. Guo, *Asymptotic behaviors of a heroin epidemic model with nonlinear incidence rate influenced by stochastic perturbations*, J. Appl. Anal. Comput., 2024, 14, 1060–1077.
- [42] K. Witkiewitz and K. E. Vowles, *Alcohol and opioid use, co-use, and chronic pain in the context of the opioid epidemic: A critical review*, Alcohol. Clin. Exp. Res., 2018, 42(3), 478–488.
- [43] Z. Yaagoub and K. Allali, *Fractional HBV infection model with both cell-to-cell and virus-to-cell transmissions and adaptive immunity*, Chaos Soliton Fract., 2022, 165, 112855.
- [44] Z. Yaagoub, J. Danane, Z. Hammouch and K. Allali, *Mathematical analysis of a fractional order two strain SEIR epidemic model*, Result. Nonlinear Anal., 2024, 7(1), 156–175.

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