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Research article

A mathematical model with control strategies for marijuana smoking prevention

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Abstract: Our goal of this study is to prevent marijuana smoking in the human population. In this manuscript, an updated mathematical model was established by incorporating two additional compartments: The hospitalized class and the prisoner's class. The updated model was validated, and it was shown to be novel compared to the non-user, experimental, recreational, and addicted (NERA) users' model. This distinction was crucial as it was challenging to prevent marijuana usage without these realistic classes. The entire population was split into six primary groups, including these new classes: non-users, experimental, recreational, addicted, hospitalized, and prisoners' class. Additionally, control techniques for marijuana prevention in the population were addressed with the aid of sensitivity analysis. The important point at which we may have determined the preliminary transmission rate of marijuana smoking was the basic reproductive number $\mathbb{R}_0$. Utilizing MATLAB, the Runge-Kutta method of order four was employed for the numerical simulation of the updated model to investigate the impact of control measures on marijuana smoking prevention.

Keywords: mathematical model; marijuana prevention; reproduction number; sensitivity analysis; control strategies

1. Introduction

Marijuana stands as one of the most widely misused substances globally, primarily derived from the buds of the hemp plant with a lesser proportion originating from other plant components. Comprising dried leaves, seeds, and flowers exhibiting a blue-black hue marijuana represents a composite product sourced from the hemp plant [1]. Hash oil manifests as a dense, dark fluid, while hashish is a highly potent, adhesive variant. Various terms such as Mary Jane, Smoke, Cannabis, Ganja, Bud, Wheezy, Grass, Reefer, Herb, Weed, Endo, Dope, Chiba, Green, Pot, and Buddha are employed to refer to marijuana [2].

Marijuana contains approximately 400 different chemicals with tetrahydrocannabinol (THC) identified as the most hazardous substance, leading to user intoxication. THC induces psychoactive effects, categorizing marijuana as a psychoactive drug. Intoxication results in alterations both in physiological functions and cognitive processes. There exists a potential for addiction or dependency, compelling individuals to seek continued use despite potential harm to their well-being [3].

Following only a few instances of consumption, discernible psychological and physical effects of marijuana emerge. These include impaired memory and learning, challenges in problem-solving and reasoning [4] diminished academic or occupational performance [5], strained interpersonal relationships with family or friends, compromised driving safety, irregular heart rate, and heightened levels of nervousness [6].

Beyond its short-term effects, marijuana usage is associated with enduring consequences for the user. Long-term use has been correlated with elevated risks of respiratory and cognitive disorders [7], heart-related issues, compromised immune system function, and indications of depression [8]. Moreover, the prolonged use of marijuana is linked to the development of abuse and dependence, exerting detrimental impacts on driving proficiency, memory, learning, and both academic and occupational performance [9].

In various countries such as Japan, Spain, Ireland, France, and China, the legal cultivation of hemp is permitted. Commercially available products derived from hemp, such as hemp milk and other cannabinoid-free items, are legally produced. It is noteworthy that marijuana is sourced from the buds of the hemp plant, and the cannabinoids in marijuana contain carcinogenic compounds comparable to those found in cigarette smoke. The dried leaves, flowers, stems, and seeds of the Cannabis sativa/Cannabis indica plant can be amalgamated to produce marijuana. Additionally, the cannabis sativa plants harbor over 100 molecules chemically identical to THC, the principal intoxicating compound in marijuana, along with more than 400 other substances [10].

As more states legalize these items for both pharmaceutical and social reasons, marijuana consumption and its variations are on the rise. The American Heart Association has issued remarks in response to the legalization of smoking and the increased use of new tobacco products, including electronic cigarettes and hookahs (waterpipes) [11,12]. Furthermore, an escalating number of reports indicate the proliferation of lung diseases and pulmonary hypertension, both of which pose health risks associated with smoking [13]. As the prevalence of marijuana use increases, patients are increasingly inquiring about its impact on cardiovascular health, especially when used concurrently with regularly

prescribed heart treatments. Nonetheless, the circulatory effects of marijuana remain inadequately understood, and the cardiovascular community lacks comprehensive research findings and guidance on this matter [14].

Marijuana smokers can be explained as:

Experimental Smokers: This demographic initiate marijuana use as an experimental endeavor for the first time, primarily due to a lack of sufficient understanding regarding the use of marijuana and its implications for the future.

Recreational Smokers: Given their ability to regulate the type of marijuana, the quantity consumed, and the specific setting for consumption, it can be asserted that these individuals infrequently partake in marijuana use, or more precisely, engage in controlled consumption. They typically exercise discretion in deciding whether to consume marijuana, often opting to do so within social settings.

Addicted Smokers: This user category encompasses individuals characterized by habitual marijuana consumption. These individuals engage in regular and frequent use, developing a dependency on the substance resulting in both physical and psychological challenges. The compulsive need for marijuana surpasses other fundamental necessities, indicating a perceived inability to navigate daily life without it, seeking solace or escape from routine existence. While encouraging alternative and enriching lifestyles may be effective for other users, those classified as addicts exhibit a persistent desire to perpetuate drug use indefinitely. The deleterious effects of drugs intensify in this group as they pursue frequent and escalating drug consumption, emphasizing quantity over quality. For these individuals the act of smoking marijuana assumes paramount importance [15].

The concept suggesting that the most frequent marijuana users often initiate their substance use with less harmful drugs forms the foundation of the gateway hypothesis for drugs. This hypothesis likens the progression to a staircase, transitioning from lighter substances such as tobacco to progressively more potent drugs like heroin, ice, and beer [16,17].

A significant proportion of women engage in marijuana use, with approximately half of these users indicating an inability to cease consumption during pregnancy [18]. This prevalence has the potential to impact up to 34% of births, signifying a noteworthy percentage. Factors contributing to the increased incidence of maternal marijuana consumption include the perceived stability and safety of marijuana during pregnancy, the ongoing trend toward drug prohibition, and its appeal as a potential remedy for common pregnancy-related ailments such as nausea. Given the expression of cannabinoid receptors in the developing womb and brain, coupled with the ease with which the active component in marijuana, THC, traverses the uterus [19,20] causes concerns regarding potential adverse and lethal effects. Current available data on marijuana exposure during pregnancy suggest a potential for prenatal disturbance associated with maternal marijuana smoking. Adverse outcomes linked to marijuana usage during pregnancy encompass mortality, severe growth limitations, and potentially fatal neurological effects. Limited well-designed research exists to thoroughly investigate the impact of marijuana use during pregnancy on parental and foetal outcomes. This paucity of research is partly attributed to historical perceptions of marijuana as an illicit substance, hindering comprehensive exploration of its health consequences during pregnancy [21,22].

The assessment of perceived risks associated with medicinal marijuana was adjusted based on research examining factors influencing opinions on marijuana legalization in Michigan [23]. Historically, the Malaysian government had contemplated legalization within a dual-policy framework, with a predominant focus on hard narcotics like heroin and opium, primarily administered through

intramuscular injection, posing a heightened risk of HIV transmission. In the case of individuals with drug abuse issues (PWID), access to free needles was facilitated through the nationwide needle exchange program (NNEP) or enrollment in the marijuana substitution treatment program (MSTP) [24]. Regarding marijuana, the consideration of legalizing it for medicinal purposes revolved around granting individuals' access to treatment through medical marijuana when supported by appropriate scientific and clinical evidence [25]. The dissemination of medical cannabis-related information online may influence the current perception of cannabis in terms of its medicinal hazards, potential harm from usage, and the legalization debate, potentially resulting in underreporting to the National Anti-Drug Agency (NADA). Since 1975 [26,27], these trends have been observed among many U.S. adolescents as part of a national survey on drug use, encompassing the consumption of other illicit substances. The survey findings revealed an increasing perception of marijuana as having a low risk, low likelihood of harm, and high acceptability [28].

Founded on historical biological evidence and classical literature on medicinal herbs, marijuana has traditionally served as a remedy for alleviating symptoms associated with illnesses, notably severe pain and epilepsy, showcasing specific medicinal attributes [29]. Despite its historical medicinal use, contemporary associations with criminal activities, social problems, and recreational use prevail in the context of cannabis usage. Consequently, within the scope of this research, the term "medical marijuana" is employed specifically to denote the use of marijuana for medicinal purposes [30].

In Thailand, a parallel situation exists, where the utilization of marijuana for medical purposes is sanctioned under the condition of having a prescription from a licensed member of the Thai traditional medical community, a physician, or a dentist. We aim to gauge the willingness to decriminalize medicinal cannabis among individuals in Selangor Malaysia, while also examining the prevalence of such acceptance and the influencing factors. The outcomes can inform the formulation of policies that strike a delicate balance between instigating reform and the potential expansion of healthcare coverage, aiming to grant patients in need access to medical marijuana treatment, akin to the approaches adopted by Thailand, Canada, and Germany [31].

Between 2018 and 2019, Malaysia experienced an 8% increase in the count of drug users and addicts [32,33]. Notably, Selangor exhibits the lowest percentage of substance abusers and drug addicts relative to the estimated total population, distinguishing it from other districts. This observation may be attributed to heightened awareness among individuals residing in metropolitan areas regarding the hazards and risks associated with drug misuse and addiction [34].

Residents of Selangor Malaysia, aged 18 or older were required to meet the inclusion criteria to participate in this study [35]. Despite limited scientific evidence supporting the therapeutic efficacy of medical marijuana for various symptoms, public interest in its purported health benefits remains substantial. Notably, awareness of diverse national policies on marijuana uses in other countries, particularly in industrialized nations, has triggered public discourse. These comparisons compel the population to assess the existing governmental approach, which treats medical marijuana issues as criminal offenses. In Malaysia as of 2017, there were 1122 individuals on death row with 71% convicted for drug smuggling, including marijuana-related offenses [36].

An enhanced iteration of the NERA model, incorporating the traditional predator-prey concept, was subsequently developed by Ginoux et al. In this updated model, drug users are analogized to predators, while non-users are regarded as prey. The stochastic mathematical model accounts for fluctuations in the numbers of each group, influenced by interactions with other groups. These interactions are defined by the impact of an individual from one class on an individual from another class.

This phenomenon elucidates why individuals transition from one group to another, indicating that some individuals shift from their original group to join another. Following the predator-prey concepts, the factors contributing to fluctuations in group membership can be conceptualized as one group exerting influence on another. In this framework, non-smokers serve as prey for three other groups: Experimental smokers (E), recreational smokers (R), and addicted smokers (A). Addicted smokers (A) act as predators to all recreational users (R), experimental users (E), and non-smokers (N), while recreational users (R) act as predators to both experimental smokers (E) and non-users (N).

Two distinct types of "functional responses" are employed to model the dynamics of prey (non-users) and predators (experimental, recreational, and addicted users). In the absence of a predator population, the growth of prey (N) is intentionally slowed for stability reasons, while the saturation of the predator percentage (indicating the influence of one predator group on the others) must be taken into account [37] has a destabilizing effect.

Previous research has established a mathematical framework for marijuana consumption. However, a significant oversight in these studies involves the exclusion of crucial classes, specifically the hospitalized class (comprising individuals under treatment) and the prisoner's class. We address this limitation by adapting the existing model to incorporate these essential classes. The inclusion of these realistic classes is imperative for effective control measures in the context of marijuana smoking. The NERA model is the system of mathematical equations that follows, and Figure 1 is a geometric representation of the previous model.

$$\begin{aligned} \frac{dN}{dt} &= \beta - (\beta + r_1E + r_1R)N + r_3E + r_5R + r_6A, \\ \frac{dE}{dt} &= -(\beta + r_3 - r_1N + r_2R)E + r_1NR, \\ \frac{dR}{dt} &= -(\beta + r_4 + r_5 - r_2E)R, \\ \frac{dA}{dt} &= r_4R - (\beta + r_6)A. \end{aligned} \tag{1}$$

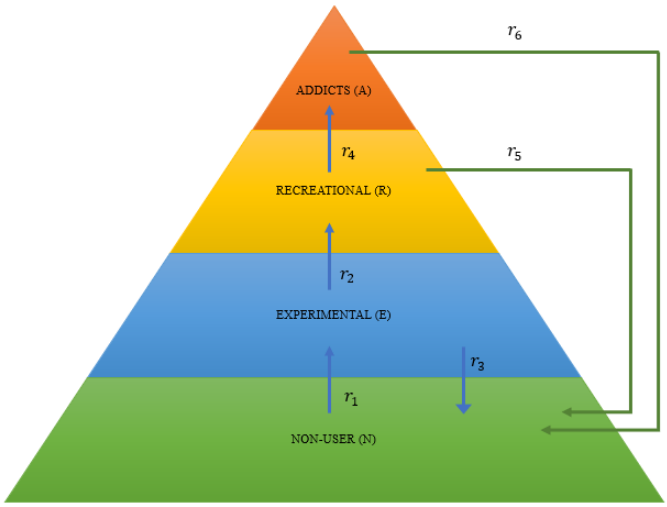

Figure 1. A graphical depiction of the previous model [38].

2. Model formulation

The process of formulating a model is a critical step in translating our comprehension of a natural system into a mathematical representation. It involves two pivotal stages: Constructing a conceptual model and subsequently converting this conceptual model into mathematical equations.

Initially, the identification of primary components, termed state variables, and the dynamics defining flows is crucial. Applying the principle of conservation, the equations of the conceptual model articulate the rate of change of state variables, reflecting the sum of all incoming flows minus the outgoing flows from each compartment. This can be visually represented through a conceptual diagram or flow chart, where state variables are depicted by boxes connected by arrows representing the flows. In the subsequent stage, the flows are precisely defined and expressed as mathematical equations.

In this section, we will introduce an enhanced iteration of the NERA model, emphasizing the understanding of the dynamics of a social epidemic. The entire community under examination is categorized into two principal groups: Marijuana smokers and non-smokers. Smokers are further subdivided into five distinct stages, each representing different progressions toward addiction. The individuals are categorized into distinct classes: S_{Nu}, representing non-users; E_{Mu}, denoting the experimental class; C_{Mu}, signifying casual smokers; A_{Mu}, characterizing addicts; and H_{Mu}, representing the hospitalized category. Additionally, there is a specific class for prisoners, denoted as J_{Mu}. The total population $T_p(t)$ at any given time "t" is described by Eq (2):

$$T_p(t) = S_{Nu}(t) + E_{Mu}(t) + C_{Mu}(t) + A_{Mu}(t) + H_{Mu}(t) + J_{Mu}(t). \tag{2}$$

Individuals (non-smokers) to the influence of smoke are designated as susceptible and are assigned to the susceptible class S_{Nu}. The ratio of recruitment in this class is denoted as Γ_p, with recruited individuals having an average age of approximately fourteen years. Some individuals naturally exit the class due to mortality. Moreover, certain individuals are swayed by interactions with casual smokers, leading them to initiate experimental smoking and be categorized into the experimental category. Another subset of susceptible individuals adopts casual smoking because of interactions with addicts, placing them in the casual smokers' class. The quantity of individuals within this group is contingent on their level of interaction with addicts. A heightened rate of interaction corresponds to an increased influx of individuals into the experimental class E_{Mu}, and the casual smokers' class C_{Mu}. The term $a_1 C_{Mu} S_{Nu}$ represents the interaction between susceptible individuals S_{Nu} and casual smokers C_{Mu}, while the term $a_4 A_{Mu} S_{Nu}$ signifies the interaction between susceptible individuals S_{Nu} and addicts A_{Mu}. Consequently, we express the dynamics through the following differential Eq (3) to delineate the alterations in the non-smoker's population.

$$\dot{S_{Nu}} = \Gamma_p - a_1 C_{Mu} S_{Nu} - a_4 A_{Mu} S_{Nu} - \mu S_{Nu} + a_3 E_{Mu} + a_5 C_{Mu} + a_8 H_{Mu} + a_{10}(1-\alpha) J_{Mu}. \tag{3}$$

The contact rate between members of the susceptible class and the casual class is denoted as "a_1", while the contact rate between susceptible individuals and the addicts are expressed as "a_4". Other contact rates include "a_3", "a_5", "a_8", and "a_{10}", which signify the ratios of individuals from the experimental class E_{Mu}, casual smokers' class C_{Mu}, hospitalized class H_{Mu}, and prisoners class J_{Mu} respectively, who cease smoking and transition to the susceptible class due to various reasons. The mortality ratio within the susceptible class is denoted as "μS_{Nu}". The term $\dot{S_{Nu}}$ represents the temporal change in the susceptible class S_{Nu}. Within the susceptible class, $a_1 C_{Mu} S_{Nu}$ individuals migrate to the experimental class. Consequently, the evolution of the experimental class E_{Mu} is

described by the ensuing differential Eq (4).

$$\dot{E_{Mu}} = a_1 C_{Mu} S_{Nu} - a_2 E_{Mu} - a_3 E_{Mu} - \mu E_{Mu}. \tag{4}$$

The rate of recruitment from the non-smokers to the experimental smokers E_{Mu}, is given by the term $a_1 C_{Mu} S_{Nu}$. Additionally, the rate at which experimental smokers join the casual smoker's class C_{Mu} is expressed as a_2. A subset of individuals discontinues smoking in the experimental class, transitioning back to the susceptible class. The natural death ratio within this experimental class is denoted as μE_{Mu}. The temporal evolution of the experimental class E_{Mu} is captured by the differential Eq (5), reflecting the changes occurring over time.

$$\dot{C_{Mu}} = a_4 A_{Mu} S_{Nu} + a_2 E_{Mu} - a_6 C_{Mu} - a_5 C_{Mu} - \mu C_{Mu}. \tag{5}$$

The rate at which individuals are recruited from the experimental class E_{Mu} into the casual smokers' class C_{Mu} is denoted as $a_2 E_{Mu}$, while the rate of recruitment from non-smokers class S_{Nu} is expressed as $a_4 A_{Mu} S_{Nu}$. The natural death ratio within the casual smokers' class is represented by μC_{Mu}. A segment of individuals in this class transitions to the addicted class after a certain duration in C_{Mu}, occurring at the rate $a_6 C_{Mu}$. Additionally, within the casual smokers' class a proportion of individuals becomes susceptible with an increase in the percentage denoted by a_5. The dynamics of the addicted smokers' class are encapsulated in the subsequent nonlinear system of differential Eq (6).

$$\dot{A_{Mu}} = a_6 C_{Mu} + a_{10} \alpha J_{Mu} - a_7 A_{Mu} - a_9 A_{Mu} - e A_{Mu} - \mu A_{Mu}. \tag{6}$$

Upon concluding their tenure in the casual group, individuals proceed to enroll in the addicted category at a rate denoted as $a_6 C_{Mu}$. Subsequently, upon serving their prison sentence, some individuals reintegrate into the addicted class at a rate of α. The law enforcement diligently pursues gang members, apprehending them at a continuous rate represented by "a_9". Certain addicted individuals are compelled to undergo treatment at a rate denoted as a_7, subsequently being categorized in the hospitalized individuals H_{Mu}. Within this class a natural death rate is denoted as μA_{Mu}, while during police intervention, a mortality rate denoted as "e" is observed among certain individuals within the specified group. The dynamics of the hospitalized class are governed by the ensuing system of non-linear differential Eq (7).

$$\dot{H_{Mu}} = a_7 A_{Mu} - a_8 H_{Mu} - \mu H_{Mu}. \tag{7}$$

Individuals from the hospitalized class H_{Mu} rejoin the susceptible (non-users) class S_{Nu} with a percentage of a_8. Some individuals in this family pass away naturally at a rate of μH_{Mu}. Individuals transitioning from the addicted class to the incarcerated class occur at a rate of "a_9" and their departure from this class transpires at a rate of $a_{10}(1-\alpha)$. Within this class, a natural mortality rate of μJ_{Mu} is attributed to certain individuals. The dynamics of the jail class are delineated by the differential equation as stipulated in Eq (8) below.

$$\dot{J_{Mu}} = a_9 A_{Mu} - a_{10} J_{Mu} - \mu J_{Mu}. \tag{8}$$

The ensuing non-linear differential equations encapsulate the entirety of marijuana smoking behaviors, while Figure 2 serves as its graphical representation.

$$\begin{cases} \dot{S_{Nu}} = \Gamma_p - a_1 C_{Mu} S_{Nu} - a_4 A_{Mu} S_{Nu} - \mu S_{Nu} + a_3 E_{Mu} + a_5 C_{Mu} \\ \qquad\qquad + a_8 H_{Mu} + a_{10}(1-\alpha) J_{Mu}, \\ \dot{E_{Mu}} = a_1 C_{Mu} S_{Nu} - a_2 E_{Mu} - a_3 E_{Mu} - \mu E_{Mu}, \\ \dot{C_{Mu}} = a_4 A_{Mu} S_{Nu} + a_2 E_{Mu} - a_6 C_{Mu} - a_5 C_{Mu} - \mu C_{Mu}, \\ \dot{A_{Mu}} = a_6 C_{Mu} + a_{10} \alpha J_{Mu} - a_7 A_{Mu} - a_9 A_{Mu} - e A_{Mu} - \mu A_{Mu}, \\ \dot{H_{Mu}} = a_7 A_{Mu} - a_8 H_{Mu} - \mu H_{Mu}, \\ \dot{J_{Mu}} = a_9 A_{Mu} - a_{10} J_{Mu} - \mu J_{Mu}. \end{cases} \tag{9}$$

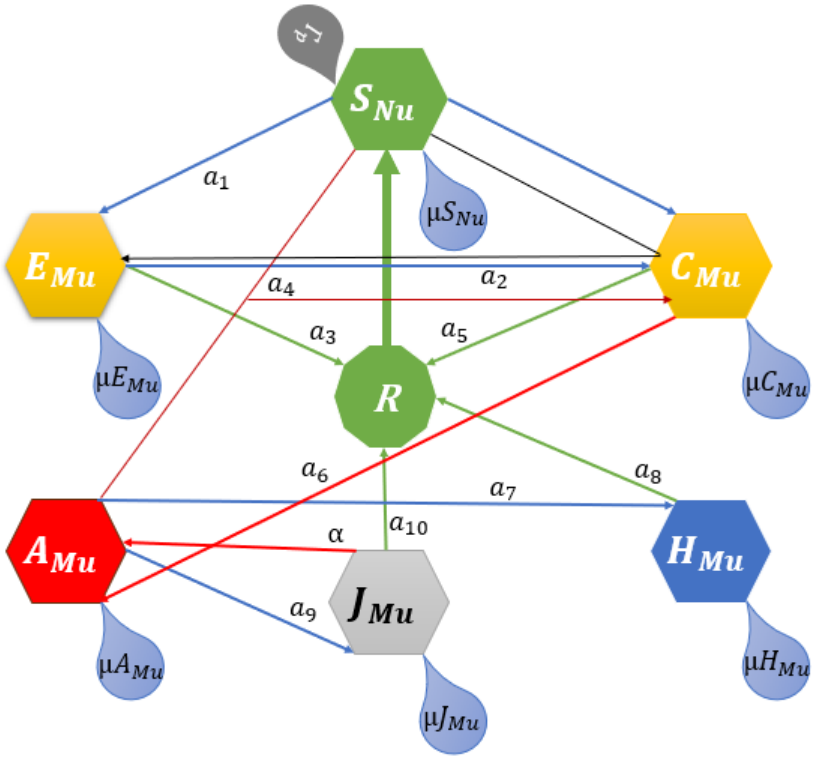

Figure 2. A geometric depiction of the modified model [39].

Table 1 provides a compilation of descriptions for the parameters involved in the current model.

Table 1. Description of the parameters.

Notations	Definitions of the parameters	Values	References
Γ_p	The individual's recruitment ratio in susceptible class	0.0015875 day^{-1}	[40]
a_1	The contact rate between members of the susceptible class and the casual class	0.446 day^{-1}	[38]
a_2	The rate at which experimental smokers join the casual smoker's class	0.5 day^{-1}	[38]
a_3	Proportion of experimental users who quit smoking because of counselling	0.17 day^{-1}	[38]
a_4	The rate at which susceptible individuals interact with addicts and subsequently become casual smokers	0.001201 day^{-1}	[39]
a_5	Proportion of occasional marijuana users quitting consumption due to a constrained environment	0.002 day^{-1}	[38]
a_6	Post-adjustment period, the rate at which occasional users transition to addiction	0.025 day^{-1}	[38]
a_7	Rate at which addict's moving transition to the hospital because of heavy usage	0.22 day^{-1}	Assumed
a_8	Addicts' recovery rate after proper treatment	0.2010 day^{-1}	Assumed
a_9	Proportion of addict's entering the penal system	0.0157871 day^{-1}	Assumed
a_{10}	The rate at which prisoners undergo rehabilitation	0.0331 day^{-1}	Assumed
α	The likelihood of individuals rejoining the addicted class after serving their prison term	0.03 day^{-1}	[41]
e	The ratio of police encounters with addict's	0.0005 day^{-1}	[41]
μ	Individuals' natural death ratio	0.006 day^{-1}	[40]

2.1. Invariant region

Given the mathematical formulation describing the dynamics of the human population, it is presumed that all state variables and parameters within this framework possess non-negative values at the initial time point, denoted as t = 0 [42]. This assumption extends to encompass the collective behaviors of the entire human population, as elucidated by the differential equation representing the total population [43,44]:

$$T_p = S_{Nu} + E_{Mu} + C_{Mu} + A_{Mu} + H_{Mu} + J_{Mu}. \tag{10}$$

Upon solving Eq (10), the resulting solution yields:

$$\dot{T_p} = \Gamma_p - \mu T_p - eA_{Mu} \text{ [41]}. \tag{11}$$

Upon resolving Eq (11), the derived solution is:

$$T_p \leq T_p(0)e^{-\mu t} + \frac{\Gamma_p}{\mu}(1 - e^{-\mu t}) \Rightarrow T_p \leq \frac{\Gamma_p}{\mu} \; when \; t \dashrightarrow \infty \; [41]. \tag{12}$$

The mathematical model is explicitly delineated from both mathematical and epidemiological perspectives [40,45], constituting a positively invariant domain wherein all pathway borders exclusively progress in the forward direction. In essence, this implies a restriction on the population. In accordance with the prior examination, we posit an additional assertion:

The suggested model's region is specified by

$$\Omega = [(S_{Nu}, E_{Mu}, C_{Mu}, A_{Mu}, H_{Mu}, J_{Mu}) \in \mathbb{R}_+^6, T_p \leq \frac{\Gamma_p}{\mu}]. \tag{13}$$

is a positively invariant set of T_p. Furthermore, we chose the initial data from Ω to study the dynamics of the system (9) [46].

2.2. Basic reproduction number

This section addresses the initial propagation of marijuana within the population. When an individual who smokes marijuana integrates into a population entirely susceptible to the drug, it elicits the attraction of other individuals towards substance use, thereby leading to an augmentation in the number of drug users. The term "reproduction number" denoted as ($\mathbb{R}_0$), signifies the initial rate of such dissemination. The determination of the initial transmission rate is achieved through a well-established technique known as the "next generation MATRIX method". $\mathbb{R}_0 = \rho(\dot{F}\tilde{V}^{-1})$ [40,47] as:

$$\mathbb{R}_0 = \rho(\dot{F}\tilde{V}^{-1}) \; [48, 49]. \tag{14}$$

In this context, the spectral radius is given by ρ. The Jacobian matrix of "f̀"represented as $\dot{F}$ and defined as $\dot{F} = \mathcal{J}_{\grave{f}}$.

$$\grave{f} = \begin{pmatrix} \grave{f}_1 \\ \grave{f}_2 \\ \grave{f}_3 \end{pmatrix} = \begin{pmatrix} a_1 C_{Mu} S_{Nu} \\ a_4 A_{Mu} S_{Nu} \\ 0 \end{pmatrix}, \tag{15}$$

Those people who develop addiction are symbolized by the column specified in Eq (15).

$$\dot{F} = \begin{pmatrix} \dot{F}_{11} & \dot{F}_{12} & \dot{F}_{13} \\ \dot{F}_{21} & \dot{F}_{22} & \dot{F}_{23} \\ \dot{F}_{31} & \dot{F}_{32} & \dot{F}_{33} \end{pmatrix} = \begin{pmatrix} 0 & a_1 S_{Nu} & 0 \\ 0 & 0 & a_4 S_{Nu} \\ 0 & 0 & 0 \end{pmatrix} \; [48]. \tag{16}$$

For the sake of simplicity, we express Eq (16) in the following manner:

$$\dot{F} = \begin{pmatrix} 0 & \mathfrak{p}_1 & 0 \\ 0 & 0 & \mathfrak{p}_2 \\ 0 & 0 & 0 \end{pmatrix}_{(MFE)} \; [48, 50, 51]. \tag{17}$$

where $\mathfrak{p}_1 = a_1 S_{Nu}$ and $\mathfrak{p}_2 = a_4 S_{Nu}$

Similarly, the Jacobian matrix of "ṽ" is denoted as $\tilde{V}$ and is defined as $\tilde{V} = \mathcal{J}_{\tilde{v}}$; where,

$$\tilde{\mathrm{V}} = \begin{pmatrix} \tilde{\mathrm{v}}_1 \\ \tilde{\mathrm{v}}_2 \\ \tilde{\mathrm{v}}_3 \end{pmatrix} = \begin{pmatrix} -(a_2 + a_3 + \mu)E_{Mu} \\ a_2 E_{Mu} - (a_6 + a_5 + \mu)C_{Mu} \\ a_6 C_{Mu} - (a_7 + a_9 + e + \mu)A_{Mu} \end{pmatrix}, \tag{18}$$

The individuals entering or leaving the addicted category, excluding those originating from the non-smokers category, are illustrated in the column of the matrix $\tilde{\mathrm{V}}$, as specified in Eq (18).

$$\tilde{\mathrm{V}} = \begin{pmatrix} \tilde{\mathrm{V}}_{11} & \tilde{\mathrm{V}}_{12} & \tilde{\mathrm{V}}_{13} \\ \tilde{\mathrm{V}}_{21} & \tilde{\mathrm{V}}_{22} & \tilde{\mathrm{V}}_{23} \\ \tilde{\mathrm{V}}_{31} & \tilde{\mathrm{V}}_{32} & \tilde{\mathrm{V}}_{33} \end{pmatrix}, \tag{19}$$

$$\tilde{\mathrm{V}} = \begin{pmatrix} -(a_2 + a_3 + \mu) & 0 & 0 \\ a_2 & -(a_6 + a_5 + \mu) & 0 \\ 0 & a_6 & -(a_7 + a_9 + e + \mu) \end{pmatrix}_{(MFE)} \quad [48,52], \tag{20}$$

For the sake simplicity, we express Eq (20) in the following manner:

$$\tilde{\mathrm{V}} = \begin{pmatrix} -\text{Ђ}_1 & 0 & 0 \\ a_2 & -\text{Ђ}_2 & 0 \\ 0 & a_6 & -\text{Ђ}_3 \end{pmatrix}_{(MFE)}. \tag{21}$$

The principal eigenvalue of the product of $\dot{\mathrm{F}}$ and the inverse of $\tilde{\mathrm{V}}$, denoted as $\dot{\mathrm{F}}\tilde{\mathrm{V}}^{-1}$ and consequently the quantity $\mathbb{R}_0$ is as follows:

$$\mathbb{R}_0 = \sqrt{((a_1 * \text{Ң} * a_2)/(\text{Ђ}_1 * \text{Ђ}_2))}, \tag{22}$$

$$\mathbb{R}_0 = \sqrt{\frac{a_1 a_2 \Gamma_p}{\mu(a_2 + a_3 + \mu)(a_6 + a_5 + \mu)}}. \tag{23}$$

2.2.1. Reproduction number in biological perspective

In a biological context, the term "reproduction number" typically refers to a key epidemiological parameter denoted as $\mathbb{R}_0$ and read as ($\mathbb{R}$-naught). In the realm of infectious diseases or addiction, particularly in epidemiology, the reproduction number is crucial for assessing the potential for an outbreak to become an epidemic or pandemic. It serves as an indicator of the infectiousness or addictiveness and transmissibility of a pathogen, influencing the dynamics of disease spread within a given population. A reproduction number greater than 1 suggests the potential for sustained transmission, while a value below 1 indicates that the disease is likely to decline over time. Understanding the reproduction number is fundamental in formulating effective public health strategies and interventions to control the spread of infectious diseases or addiction.

In Eq (23), the term "a_1" signifies the impact rate of experimental marijuana smokers on non-smokers, while "a_2" shows the influence ratio of casual smokers on experimental users. Consequently, the expression "$a_1 a_2 \Gamma_p$" within the context of $\mathbb{R}_0$ indicates that a subset of individuals in the

susceptible (non-users) category will initiate marijuana use, subsequently joining the user's group due to the influence of marijuana smokers on susceptible individuals. This results in the propagation of marijuana from users to susceptible individuals. It is important to note that the additional terms (parameters) incorporated in $\mathbb{R}_0$ contribute solely to determining the magnitude of $\mathbb{R}_0$.

2.3. Sensitivity analysis of $\mathbb{R}_0$

Alterations in certain parameters induce changes in interconnected variables. This proportional variation is termed "sensitivity" with respect to the parameters [53]. The sensitivity of the given function (ĸ) concerning a specific parameter (n) is established under the condition that the function is differentiable with respect to that parameter.

$$\check{\Upsilon}_{\mathrm{n}}^{\mathrm{\kappa}} = \frac{\partial \mathrm{\kappa}}{\partial \mathrm{n}} \frac{\mathrm{n}}{\mathrm{\kappa}} \text{ [48].} \tag{24}$$

Table 2 presents the sensitivity indexes associated with the parameters, and Figure 3 graphically depicts the visual representation of these sensitivity indexes.

Table 2. Indexes reflecting the sensitivity of parameters.

Parameters	Values	Sensitivity indexes
a_1	0.446	+0.5000
a_2	0.5	+0.1309
a_3	0.17	−0.1258
a_5	0.002	−0.0303
a_6	0.025	−0.3788
μ	0.006	−0.5954
Γ_p	0.0015875	+0.5000

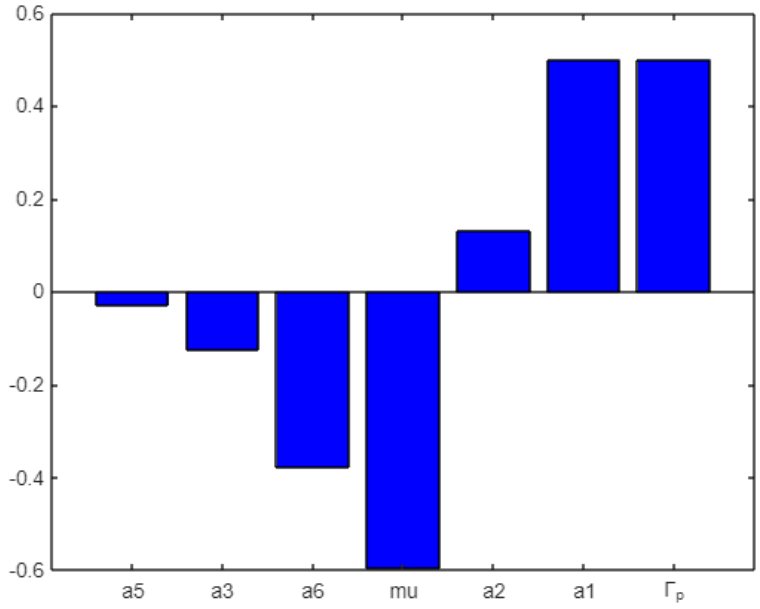

Figure 3. The visual depiction of the sensitivity indexes associated with the parameters.

3. Control strategies

When a parameter exhibits a positive sensitivity index, as exemplified by the human birth rate's (Γ_p) sensitivity index of +0.5000, its value directly correlates with the initial rate of transmission $\mathbb{R}_0$. Conversely, if the parameter's index is negative, as seen in the sensitivity index of μ at –0.5954, the parameter's value is inversely proportional to $\mathbb{R}_0$. A higher sensitivity index for a parameter implies a more substantial influence on marijuana transmission. Nevertheless, certain attributes, such as the natural mortality ratio and the human birth rate, remain unavoidable despite possessing high sensitivity indices. We modify five parameters, including "a_1" the impact level of the experimental class (E_{Mu}) on the non-user's group (S_{Nu}) and "a_2" the impact level of the casual group (C_{Mu}) on the experimental group (E_{Mu}). "a_3" the rate at which the affected class recovers by obtaining the counselling of elders, due to the restricted environment, "a_5" represents the rate of recovery from casual smokers, while "a_6" represents the probability of transmission that the casual smokers will become addicted. The maximum values of these interventions are represented by the control strategies shown in Table 3.

Table 3. The parameter values corresponding to different strategies are detailed as follows.

Strategies	a_5	a_6	a_2	a_3	a_1
Strategy-1	0.002	0.025	0.5	0.17	0.446
Strategy-2	0.0621	0.215	0.002	0.367	0.000511

4. Results and discussion

The anticipated results of the proposed strategies are depicted in the ensuing figures. We have considered initial values for each state variable in our projections. $S_{Nu}(0) = 1000$, $E_{Mu}(0) = 20$, $C_{Mu}(0) = 20$, $A_{Mu}(0) = 20$, $H_{Mu}(0) = 10$, and $J_{Mu}(0) = 10$ Utilizing MATLAB, we employ the fourth-order Runge-Kutta (RK4) method to numerically simulate the designed strategies. The RK4 method, renowned for its multiple advantages, stands as a widely adopted numerical technique for solving ordinary differential equations (ODEs). In the numerical simulation of the ten first-order ODEs with boundary conditions, RK4 proves to be the preeminent approach, recognized for its efficiency and reliability across diverse domains, as indicated by comparative data in [54]. In contrast, findings from [55] underscore that RK5 and RK8 exhibit lower efficiency compared to RK4, attributed to the latter's ability to achieve comparable accuracy with reduced computational time in calculating the truncation global error within the numerical solution.

RK4 consistently yields precise solutions, particularly for ODEs characterized by intricate behaviors. Its general stability surpasses that of several alternative numerical methods employed in ODE solving. The method's robustness extends across a wide spectrum of ODEs, demonstrating resilience against stability challenges and rendering it suitable for diverse applications. RK4's adaptability is evident in its efficacy for both stiff and non-stiff systems. Notably, in certain contexts, RK4 has exhibited superior energy conservation compared to simpler methods, a critical consideration in simulations of physical systems where energy preservation holds paramount importance.

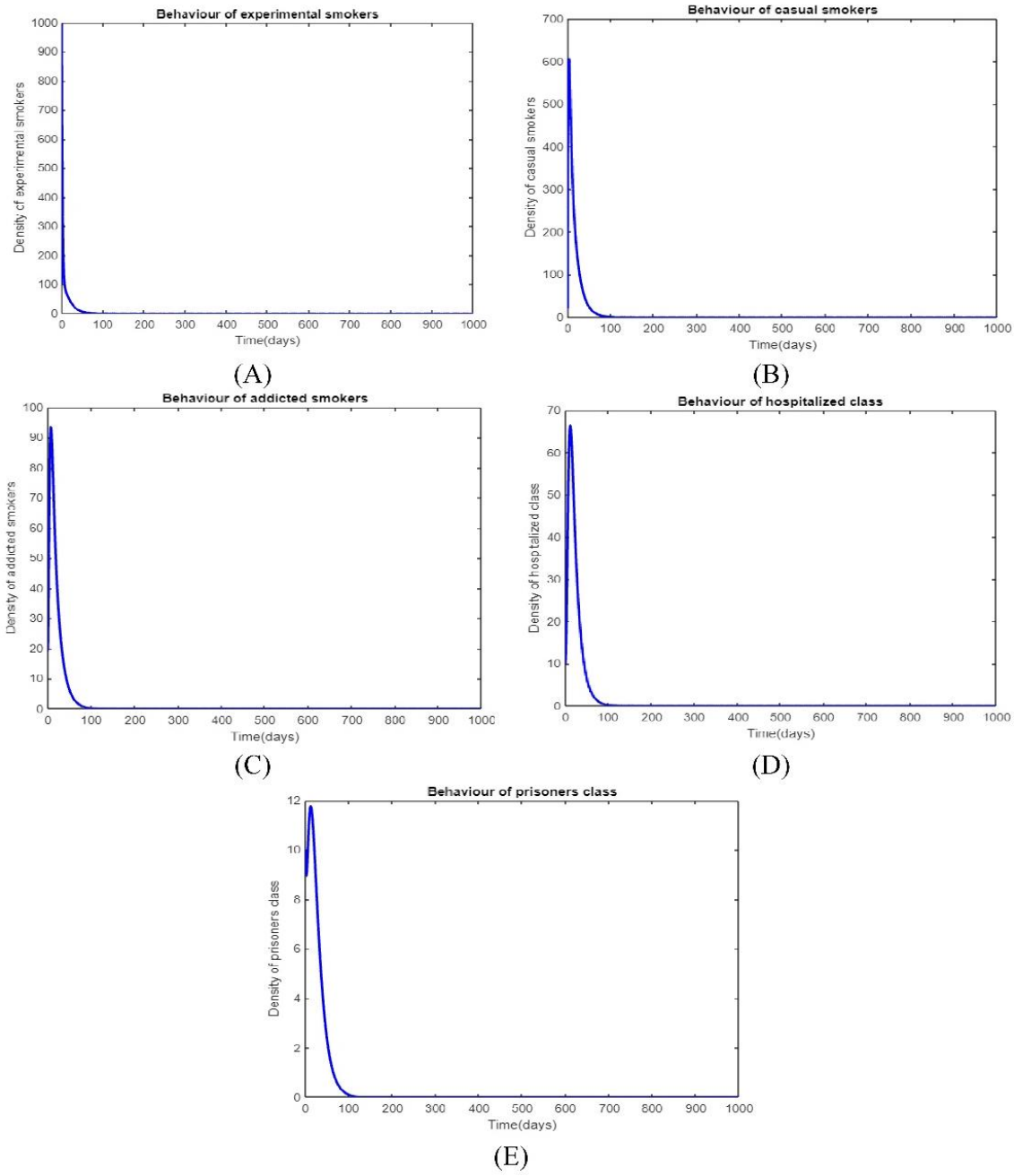

Figure 4. The numerical findings present a comparison of the effectiveness of various strategies employed to discourage marijuana use within the experimental, recreational, addicted, hospitalized, and prisoner groups (referred to as Strategy 1).

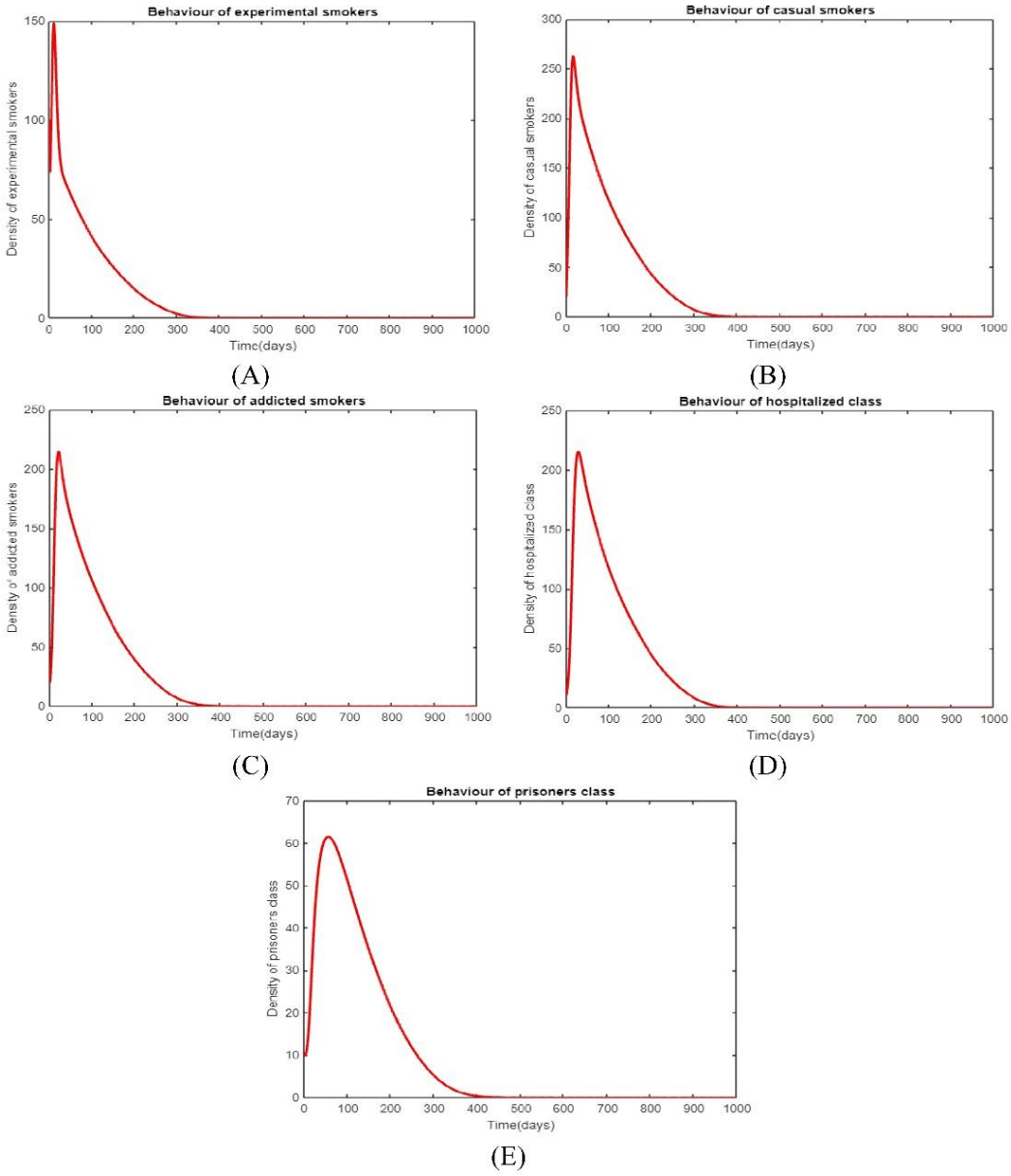

Figure 5. The numerical findings present a comparison of the effectiveness of various strategies employed to discourage marijuana use within the experimental, recreational, addicted, hospitalized and prisoner groups (referred to as Strategy 2).

4.1. Outcomes of strategy 1

Upon implementing Strategy 1, the regulation of marijuana smoking becomes achievable within a nineteen-month timeframe. Figure 4A illustrates the gradual reduction of the experimental cohort to zero over 80 days, resulting in the recovery of 996 individuals. In Figure 4B, the casual smokers' group diminishes to zero within 109 days, accompanied by the recovery of 600 individuals. Similarly, Figure 4C portrays the addicted class converging to zero in 123 days, with the restoration of 92 persons. In Figure 4D, the hospitalized category reaches zero over 135 days, leading to the recovery of 66 individuals. The prisoners' class follows suit in Figure 4E, attaining zero over 149 days, and witnessing the recovery of 11 individuals.

4.2. Outcomes of strategy 2

Through the implementation of this approach, a decline in marijuana utilization can be realized over a duration of approximately 66 months. In 356 days, the prevalence of experimental smokers as portrayed in Figure 5A, decreases to zero. Figure 5B depicts the reduction of casual smokers to zero within 385 days, and Figure 5C emphasizes the gradual decrease of addicted smokers to zero over 400 days. Additionally, Figure 5D denotes the hospitalization category reaching zero in 416 days, while Figure 5E indicates the prisoners class reaching zero in 442 days. Concurrently, the conclusion of these categories is marked by the recovery of 148, 258, 212, 213, and 62 individuals respectively.

4.3. Comparison of the proposed strategies

The evaluation of implemented strategies for controlling marijuana smoking reveals a significant distinction in their impact on usage reduction. Strategy 1 proves effective within a nineteen-month timeframe, gradually decreasing marijuana consumption across various classes. In contrast, Strategy 2 extends the intervention period to around 66 months, resulting in a more prolonged timeline for achieving reduction objectives. Despite both strategies yielding recoveries in affected categories, Strategy 1 accomplishes this in a comparatively shorter duration, emphasizing a swifter approach to curbing marijuana consumption. In comparing the suggested approaches, Strategy 1 exhibits a notably higher daily recovery rate of 19.26 individuals, whereas Strategy 2 achieves a more modest recovery of 2.27 individuals per day. This numerical contrast underscores the distinct efficiency levels of the two strategies in mitigating marijuana smoking, highlighting their unique characteristics and effectiveness in usage reduction. As a result, Strategy 1 is more effective than Strategy 2.

5. Conclusions

In this work, the author enhanced the NERA model by introducing two additional compartments: The prisoner's class and hospitalized class (smokers under treatment). The inclusion of these classes is deemed realistic, and their absence posed challenges in mitigating and recovering the prevalence of marijuana smokers within the population. The mathematical formulation employed in this study utilized a system of first-order non-linear ordinary differential equations. We addressed various aspects, including the basic reproduction number, invariant region, and sensitivity analysis, each serving distinct purposes. The invariant region was solved to validate the modified model. The basic reproduction number was computed for the initial rate of marijuana smoking transmission, and sensitivity analysis identified the most crucial parameters influencing marijuana transmission. To curtail marijuana smoking, two strategies were derived based on the most sensitive (targeted) parameters. Through numerical simulation, the results indicated that Strategy 1 proves to be more efficacious than Strategy 2 in controlling marijuana smoking. Moreover, the modified model exhibited more rapid convergence compared to the previous model, leading to the conclusion that the modified model holds greater significance. We suggest potential future research directions, including the exploration of novel techniques such as “optimal control problems” and “threshold conditions”, to optimize the control of marijuana smoking with minimized costs and time investment.

Use of AI tools declaration

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

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Conflicts of interest

We declare that this work is original, and the content has not been published elsewhere or submitted for publication elsewhere. All the authors contributed significantly and agree with the content of the manuscript. We declare that there are no conflicts of interest in this research.

Author contributions

Atta Ullah: conceptualization, methodology, investigation, and visualization. Writing an original draft, reviewing, and editing; Hamzah Sakidin: supervision, conceptualization, investigation, review, and editing; Kamal Shah: supervision, conceptualization, investigation visualization, review, and editing; Yaman Hamed: supervision, conceptualization, methodology, investigation, visualization, review, and editing; Thabet Abdeljawad: investigation, visualization, and review.

References

1. D. S. Timberlake, A comparison of drug use and dependence between blunt smokers and other cannabis users, *Subst. Use Misuse*, **44** (2009), 401–415. https://doi.org/10.1080/10826080802347651
2. L. E. Zimmer, J. P. Morgan, Marijuana myths, marijuana facts: A review of the scientific evidence, *Lindes Center NY*, (1997).
3. A. Berenson, Tell your children: The truth about marijuana, mental illness, and violence, *Free Press*, (2020).
4. C. L. Hart, W. van Gorp, M. Haney, R. W. Foltin, M. W. Fischman, Effects of acute smoked marijuana on complex cognitive performance, *Neuropsychopharmacol*, **25** (2001), 757–765. https://doi.org/10.1016/S0893-133X(01)00273-1
5. R. L. Pacula, J. Ringel, Does marijuana use impair human capital formation?, *NBERC*, (2003). https://doi.org/10.3386/w9963
6. W. H. Jeynes, Adolescent religious commitment and their consumption of marijuana, cocaine, and alcohol, *J. Health Soc. Policy*, **21** (2006), 1–20. https://doi.org/10.1300/J045v21n04_01
7. S. F. Tapert, A. D. Schweinsburg, S. A. Brown, The influence of marijuana use on neurocognitive functioning in adolescents, *Curr. Drug Abuse Rev.*, **1** (2008), 99–111. https://doi.org/10.2174/1874473710801010099

8. G. Thomas, R. A. Kloner, S. Rezkalla, Adverse cardiovascular, cerebrovascular, and peripheral vascular effects of marijuana inhalation: What cardiologists need to know, *AM. J. Cardiol.*, **113** (2014), 187–190. https://doi.org/10.1016/j.amjcard.2013.09.042
9. H. Wen, J. Hockenberry, J. R. Cummings, The effect of medical marijuana laws on marijuana, alcohol, and hard drug use, *NBER*, (2014). https://doi.org/10.1016/j.amjcard.2013.09.042
10. M. A. Alsherbiny, C. G. Li, Medicinal cannabis—potential drug interactions, *Medicines*, **6** (2018), 3. https://doi.org/10.3390/medicines6010003
11. A. Bhatnagar, W. Maziak, T. Eissenberg, K. D. Ward, G.Thurston, B. A. King, et al., Water pipe (hookah) smoking and cardiovascular disease risk: A scientific statement from the American Heart Association, *Circulation*, **139** (2019), e917–e936. https://doi.org/10.1161/CIR.0000000000000671
12. Aruni Bhatnagar, L. P. Whitsel, K. M. Ribisl, C. Bullen, F. Chaloupka, M. R. Piano, et al., Electronic cigarettes: A policy statement from the American Heart Association, *Circulation*, **130** (2014), 1418–1436. https://doi.org/10.1161/CIR.0000000000000107
13. J. E. Layden, I. Ghinai, I. Pray, A. Kimball, M. Layer, M. W. Tenforde, et al., Pulmonary illness related to e-cigarette use in Illinois and Wisconsin, *N. Eng. J. Med.*, **382** (2020), 903–916. https://doi.org/10.1056/NEJMoa1911614
14. T. M. Kaufman, S. Fazio, M.D. Shapiro, Brief commentary: Marijuana and cardiovascular disease—what should we tell patients? *Ann. Intern. Med.*, **170** (2019), 119–120. https://doi.org/10.7326/M18-3009
15. S. Mushayabasa, G. Tapedzesa, Modeling illicit drug use dynamics and its optimal control analysis, *Comput. Math. Methods Med.*, **2015** (2015).
16. A. L. Bretteville-Jensen, H. O. Melberg, A. M. Jones, Sequential patterns of drug use initiation-Can we believe in the gateway theory?, *BE. J. Econ. Anal. Policy*, **8** (2008). https://doi.org/10.2202/1935-1682.1846
17. A. Ghaffar, M. Iqbal, M. Bari, S. M. Hussain, R. Manzoor, K. S. Nisar, et al., Construction and application of nine-tic B-spline tensor product SS, *Math*, **7** (2019), 675. https://doi.org/10.3390/math7080675
18. M. E. Passey, R. W. Sanson-Fisher, C. A. D'Este, J. M. Stirling, Tobacco, alcohol and cannabis use during pregnancy: clustering of risks, *Drug. Alcohol. Dep.*, **134** (2014), 44–50. https://doi.org/10.1016/j.drugalcdep.2013.09.008
19. L. Cristino, V. Di Marzo, Fetal cannabinoid receptors and the "dis-joint-ed" brain, *EMBO J.*, **33** (2014), 665–667. https://doi.org/10.1002/embj.201488086
20. A. Shah, H. Khan, M. De la Sen, J. Alzabut, S. Etemad, C. T. Deressa, et al., On non-symmetric fractal-fractional modeling for ice smoking: Mathematical analysis of solutions, *Symmetry*, **15** (2022), 87. https://doi.org/10.3390/sym15010087
21. T. D. Metz, E. H. Stickrath, Marijuana use in pregnancy and lactation: A review of the evidence, *Am. J. Obstet. Gynecol.*, **213** (2015), 761–778. https://doi.org/10.1016/j.ajog.2015.05.025
22. A. Alkhazzan, J. Wang, Y. Nie, H. Khan, J. Alzabut, A stochastic SIRS modeling of transport-related infection with three types of noises, *Alexandria Eng. J.*, **76** (2023), 557–572. https://doi.org/10.1016/j.aej.2023.06.049
23. J. D. Ellis, S. M. Resko, K. Szechy, R. Smith, T. J. Early, Characteristics associated with attitudes toward marijuana legalization in Michigan, *J. Psychoact. Drugs*, **51** (2019), 335–342. https://doi.org/10.1080/02791072.2019.1610199

24. R. Dapari, M. H. Mahfot, A. M. Nazan, M. R.Hassan, Na. C. Dom, S. S. S. A. Rahim, Acceptance towards decriminalization of medical marijuana among adults in Selangor, Malaysia, *Plos One*, **17** (2022), e0262819. https://doi.org/10.1371/journal.pone.0262819
25. H. Khan, J. Alzabut, A. Shah, S. Etemad, S. Rezapour, C. Park, A study on the fractal-fractional tobacco smoking model, *AIMS Math.*, **7** (2022), 13887–13909. https://doi.org/10.3934/math.2022767
26. A. Khan, J. F. Gómez-Aguilar, T. S. Khan, H. Khan, Stability analysis and numerical solutions of fractional order HIV/AIDS model, *Chaos Solitons Fractal*, **122** (2019), 119–128. https://doi.org/10.1016/j.chaos.2019.03.022
27. W. Shatanawi, A. Raza, M. Shoaib Arif, M. Rafiq, M. Bibi, M. Mohsin, Essential features preserving dynamics of stochastic Dengue model, *Model Eng. Sci.*, **126** (2021), 201–215. https://doi.org/10.32604/cmes.2021.012111
28. R. J. Evans-Polce, M. E. Patrick, S. E. McCabe, R. A. Miech, Prospective associations of e-cigarette use with cigarette, alcohol, marijuana, and nonmedical prescription drug use among US adolescents, *Drug Alcohol. Dep.*, **216** (2020), 108303. https://doi.org/10.1016/j.drugalcdep.2020.108303
29. S. Pisanti, M. Bifulco, Medical cannabis: A plurimillennial history of an evergreen, *J. Cell Physiol.*, **234** (2019), 8342–8351. https://doi.org/10.1002/jcp.27725
30. E. Krediet, D. G. A. Janssen, E. R. Heerdink, T. C. G. Egberts, E. Vermetten, Experiences with medical cannabis in the treatment of veterans with PTSD: Results from a focus group discussion, *Eur. Neuropsychopharmacol.*, **36** (2020), 244–254. https://doi.org/10.1016/j.euroneuro.2020.04.009
31. J. Rehm, T. Elton-Marshall, B. Sornpaisarn, J. Manthey, Medical marijuana. What can we learn from the experiences in Canada, Germany and Thailand?, *Int. J. Drug Policy*, **74** (2019), 47–51. https://doi.org/10.1016/j.drugpo.2019.09.001
32. N. R. Dash, G. Khoder, A. M. Nada, M. T. Al-Bataineh, Correction: Exploring the impact of Helicobacter pylori on gut microbiome composition, *Plos One*, **16** (2021), e0256274. https://doi.org/10.1371/journal.pone.0256274
33. A. Elmazny, S. M. Hamdy, M. Abdel-Naseer, N. M. Shalaby, H. S. Shehata, N. A. Kishk, et al., Interferon-beta-induced headache in patients with multiple sclerosis: Frequency and characterization, *J. Pain. Res.*, (2020), 537–545. https://doi.org/10.2147/JPR.S230680
34. D. C. Oshi, T. Ricketts-Roomes, S. N. Oshi, K. A. Campbell-Williams, T. Ricketts-Roomes, S. N. Oshi, et al., Factors associated with awareness of decriminalization of marijuana in Jamaica, *J. Subst. Use*, **25** (2020), 152–156. https://doi.org/10.1080/14659891.2019.1664671
35. N. R. A. Rahman, F. Mohmad, M. S. Isuan, M. R. Isa, Towards a higher percentage of recreational drugs-free among patients on methadone maintenance treatment in Larkin Health Clinic, *Q. Bull.*, **1** (2021), 60–71.
36. E. Cook, State of the death penalty in Southeast Asia, *Eureka Street*, **28** (2018), 13–15.
37. J. M. Ginoux, R. Naeck, Y. B. Ruhomally, M. Z. Dauhoo, M. Perc, Chaos in a predator–prey-based mathematical model for illicit drug consumption, *Appl. Math. Comput.*, **347** (2019), 502–513. https://doi.org/10.1016/j.amc.2018.10.089

38. M. Z. Dauhoo, B. S. N. Korimboccus, S. B. Issack, On the dynamics of illicit drug consumption in a given population *IMA J. Appl. Math.*, **78** (2013), 432–448. https://doi.org/10.1093/imamat/hxr058
39. A. Ullah, H. Sakidin, S. Gul, K. Shah, M. S. Muthuvalu, T. Abdeljawad, et al., Sensitivity analysis-based validation of the modified NERA model for improved performance, *J. Adv. Res. Appl. Sci. Eng. Technol.*, **32** (2023), 1–11. https://doi.org/10.37934/araset.32.3.111
40. M. Zamir, G. Zaman, A. S. Alshomrani, Sensitivity analysis and optimal control of anthroponotic cutaneous leishmania, *PloS One,* **11** (2016), e0160513. https://doi.org/10.1371/journal.pone.0160513
41. A. Ullah, H. Sakidin, S. Gul, K. Shah, Y. Hamed, M. Aphane, et al., Sensitivity analysis-based control strategies of a mathematical model for reducing marijuana smoking, *AIMS Bioeng*, **10** (2023), 491–510. https://doi.org/10.3934/bioeng.2023028
42. W. A. Khan, R. Zarin, A. Zeb, Y. Khan, A. Khan, Navigating food allergy dynamics via a novel fractional mathematical model for antacid-induced allergies, *J. Math. Technol. Model.*, **1** (2024), 25–51. https://orcid.org/0009-0004-0810-280X
43. M. Zamir, T. Abdeljawad, F. Nadeem, A. Wahid, A. Yousef, An optimal control analysis of a COVID-19 model, *Alexandria Eng. J.*, **60** (2021), 2875–2884. https://doi.org/10.1016/j.aej.2021.01.022
44. P. Liu, A. Din, R. Zarin, Numerical dynamics and fractional modeling of hepatitis B virus model with non-singular and non-local kernels, *Results Phys.*, **39** (2022), 105757. https://doi.org/10.1016/j.rinp.2022.105757
45. F. M. Khan, Z. U. Khan, Numerical analysis of fractional order drinking mathematical model, *J. Math. Technol. Model.*, **1** (2024), 11–24. https://doi.org/10.56868/jmtm.v1i1.4
46. J. Yang, M. Gong, G. Q. Sun, Asymptotical profiles of an age-structured foot-and-mouth disease with nonlocal diffusion on a spatially heterogeneous environment, *J. Differ. Equations*, **377** (2023), 71–112. https://doi.org/10.1016/j.jde.2023.09.001
47. M. Zamir, R. Sultana, R. Ali, W. A. Panhwar, S. Kumar, Study on the threshold conditions for infection of visceral leishmaniasis, *Surj-Sci Ser*, **47** (2015), 619–622.
48. M. Zamir, F. Nadeem, M. A. Alqudah, T. Abdeljawad, Future implications of covid-19 through mathematical modeling, *Result. Phys.*, **33** (2022), 105097. https://doi.org/10.1016/j.rinp.2021.105097
49. V. Madhusudanan, M. N. Srinivas, B. S. N. Murthy, K. J. Ansari, A. Zeb, A. Althobaiti, et al., The influence of time delay and Gaussian white noise on the dynamics of tobacco smoking model, *Chaos Soliton Fractal*, **173** (2023), 113616. https://doi.org/10.1016/j.chaos.2023.113616
50. A. Din, Y. Li, Q. Liu, Viral dynamics and control of hepatitis B virus (HBV) using an epidemic model, *Alexandria Eng. J.*, **59** (2020), 667–679. https://doi.org/10.1016/j.aej.2020.01.034
51. A. Din, Y. Li, T. Khan, G. Zaman, Mathematical analysis of spread and control of the novel corona virus (COVID-19) in China, *Alexandria Eng. J.*, **141** (2020), 110286. https://doi.org/10.1016/j.chaos.2020.110286
52. S. Bushnaq, K. Shah, S. Tahir, K. J. Ansari, M.Sarwar, T. Abdeljawad, Computation of numerical solutions to variable order fractional differential equations by using non-orthogonal basis, *AIMS Math.*, **7** (2022), 10917–10938. https://doi.org/10.3934/math.2022610

53. L. Rarità, C. D'Apice, B. Piccoli, D. Helbing, Sensitivity analysis of permeability parameters for flows on Barcelona networks, *J. Differ. Equations*, **249** (2010), 3110–3131. https://doi.org/10.3934/math.2022610
54. N. Ahmad, S. Charan, V. P. Singh, Study of numerical accuracy of Runge-Kutta second, third and fourth order method, *Int. J. Comput. Math. Sci.*, **4** (2015), 111.
55. M. A. Islam, Accurate solutions of initial value problems for ordinary differential equations with the fourth order Runge Kutta method, *J. Math. Res.*, **7** (2015), 41. https://doi.org/10.5539/jmr.v7n3p41

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