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MATHEMATICAL MODELING, ANALYSIS, AND OPTIMAL CONTROL OF THE N+7 COMPARTMENT SMOKING EPIDEMIC MODEL

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Abstract. In this study, we present a new mathematical model for the $n+7$ compartment smoking epidemic and analyze its behavior using optimal control techniques. We examine the system's basic properties and use Lyapunov functions and Routh-Hurwitz criteria to perform stability analysis. Our results show that the system is globally and locally asymptotically stable at the free equilibrium E_0 when $R_0 < 1$, and globally and locally asymptotically stable at the endemic equilibrium E^* when $R_0 > 1$. We also conduct a sensitivity analysis to identify the model parameters that significantly impact the reproduction number R_0. Our goal is to identify optimal strategies for minimizing the number of heavy smokers, maximizing the number of sick heavy smokers who receive hospital treatment, and increasing the number of rich and poor heavy smokers who seek treatment at private and public smoking treatment centers. We use Pontryagin's maximum principle in continuous time to characterize the optimal controls, and we confirm our theoretical findings through numerical simulations conducted using Matlab.

Keywords: smoking; epidemic; analysis; optimization; optimal control.

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1. INTRODUCTION

The WHO claims that smoking is a significant contributor to disease and early mortality among smokers as well as discomfort, pain, and even poor health in those who are exposed to tobacco smoke. Every year, smoking takes the lives of more than 8 million people globally, including about 1.2 million passive smokers. According to the WHO, 36.7% of men and 78% of women used tobacco in 2020, making up 22.3% of the world's population [1]. Tobacco production is responsible for the toll it takes on human lives and the irreparable damage it causes to the planet. The WHO report entitled "Tobacco: Poisoner of our planet" shows that the production of tobacco causes more than 8 million deaths annually, the extinction of 600 million trees, the clearing of 200,000 hectares of land, the depletion of 22 billion tonnes of water, and the release of 84 million tonnes of $C0_2$ [2].

According to Dr. Ruediger Krech, Director of WHO's Health Promotion Department, tobacco products that contain more than 7.5 billion tonnes of toxic substances are the largest waste product in the world. Tobacco products contain more than 7,000 toxic chemicals that enter our environment, making them the largest waste product on the planet. Additionally, every year 4,500 billion cigarette filters end up in our oceans, rivers, soils, and beaches [3]. Smoking harm extends beyond the environment and affects a variety of areas, including the economy, mental health, and—most seriously—health. In addition to lung and stomach cancer, stroke, and coronary heart disease, long-term tobacco use also leads to cancer and other chronic diseases. For instance, smoking is linked to several cancers and a higher incidence of respiratory disorders in children of smokers, as well as 90% of all cases of lung cancer, 75% of bronchitis and emphysema, and 25% of ischemic heart disease in men over 65. The practice of traditional smoking is pervasive in many developing nations. According to the IARC (International Agency for Research on Cancer), tobacco use causes 39% of bladder cancer cases in women and 53% of cases in men. Additionally, it causes 75% of ductal cancers. It is believed that chewing tobacco causes about 90% of oral cancer deaths in Southeast Asia. These facts have solid documentation [4, 14].

A study conducted in Morocco in 2021 evaluated the epidemiological and economic impact of smoking. The results showed that in 2019, smoking was responsible for 74,000 prevalent

cases of ischemic heart disease, 4,227 new cases of lung cancer, and 12,800 premature deaths. The economic cost of smoking in Morocco in 2019 was over 5 billion Dirhams, with direct medical costs accounting for 60.9%, mortality costs at 33%, and productivity losses due to morbidity at 6.1% [5].

Numerous studies have focused on the mathematical modeling of smoking to help reduce the number of small and large smokers in continuous time, as described by differential equations [6, 7, 8], and in discrete time, as described by differential equations [9, 10]. M. LABZAI, for example, developed a discrete time study of smoker dynamics and introduced a saturated incidence rate in article [11], he added two elements that have been overlooked in most studies. These two components are a group of light smokers who quit smoking and a group of heavy smokers who died as a result of diseases caused by excessive smoking. In [12] Nur Emayasanita made use of a mathematical simulation of the smoking epidemic created by Gul Zaman and colleagues. They suggest the following four best preventative measures: vaccinations, treatment, and rehabilitation. [13] used a derived fractional Caputo operator to work on a new non-integer time order. [27] is interested in a delayed smoking model with users represented as value objects, by taking delay into consideration as a bifurcation parameter and examining the corresponding characteristic transcendental equation, its dynamics are examined in terms of local stability and Hopf bifurcation. In order to generate smoking effects that are not seen in deterministic form, [28] is connected to the stochastic smoking model.

We present a mathematical model of the smoking epidemic in this paper for the continuous case, where we are interested in tracing the progression of the number with and without the disease. We simulate the smoking epidemic in n+7 compartments that don't treat potential smokers (P), moderate smokers (A), heavy smokers (D), heavy smokers (S_k) with smoking-related diseases ($k = 1, ..., n$), rich heavy smokers who join private smoking treatment centers (C_{pv}), poor heavy smokers who join public smoking treatment centers (C_{pb}), sick smokers who join hospitals (H), and those who have quit smoking (R). Throughout this research, we examine the model's local and global stabilities in order to identify optimal strategies for reducing the number of heavy smokers while increasing the number of heavy smokers who join private treatment centers and hospitals to treat these smoking-caused diseases, as well as the number

of rich and poor heavy smokers who join private and public treatment centers. To accomplish this goal, we employ optimal control strategies associated with two types of control. The first is represented by the media and efforts to encourage wealthy heavy smokers to attend private treatment centers and poor people to attend public treatment centers. The second represents disease-specific treatment by characterizing the index k (where k=1...n), the effort to encourage heavy smokers to quit smoking with diseases.

The structure of this paper is as follows: in section 1, we introduce the suggested model and list some fundamental properties. Following that, in Section 2, we examine regional and global activities, parameter sensitivity issues, and a few numerical simulations. We present the optimal control problem for the suggested model in Section 3 and provide some findings regarding the existence of optimal controls. We also use the Portrygian maximum principle to describe this optimal control. Finally, Section 4 includes a MATLAB-based numerical simulation. Finally, Section 5 brings the essay to a close.

The population under investigation is divided into n+7 compartments in the continuous model of $P(t), A(t), D(t)$, $S_k(t)$ for all $k = 1, ..., n$, $C_{pv}(t)$,$C_{pb}(t)$,$H(t), R(t)$ disease that is presented in this section:

2. Mathematical Model Formulation and Properties of Base

2.1. Mathematical model.

The Compartment P: Potential smokers who do not use tobacco or use it only occasionally, in a hidden way. This compartment is influenced by the rate of recruitment noted in Λ and is influenced negatively by natural death μ and actual contact with moderate smokers in rate β as well. It is assumed that actual contact with heavy smokers during specific social occasions, such as those with parents, school, friends, and ceremonies, can help potential smokers pick up smoking behavior and turn into heavy smokers.

$$\dot{P}(t) = \Lambda - \beta \frac{P(t)A(t)}{N} - \mu P(t)$$

The Compartment A: is made up of light smokers. It is increased at rate β by potential smokers who become moderate smokers, and it decreases at rate $\alpha, \alpha_1, .., \alpha_n$ by smokers who become heavy smokers with and without disease, as well as natural death μ.

$$\dot{A}(t) = \beta \frac{P(t)A(t)}{N} - \sum_{k=1}^{n} \alpha_k A(t) - (\alpha + \mu)A(t)$$

The Compartment D: includes heavy smokers who do not have disease; the number of heavy smokers increases at rate α and decreases at rate γ_1 (γ_1 is the rate of heavy smokers who enter private treatment centers) and also decreases by rate γ_2 (γ_2 is the rate of heavy smokers join public treatment centers). Additionally, natural death X causes this compartment to shrink μ.

$$\dot{D}(t) = \alpha A(t) - \gamma_1 D(t) - \gamma_2 D(t) - \mu D(t)$$

The Compartment S_k: represents heavy smokers who have become ill as a result of their smoking habits, which is known to be the leading cause of death from fatal diseases such as lung cancer, mouth cancer, and stomach ulcers. Each S_k (with $k = 1, ..., n$) represents a disease, such as S_1 for lung cancer and S_2 for stomach cancer. This compartment expands as the number of heavy smokers increases at α_k (for all $k = 1, ..., n$)Furthermore, at rate θ_k. this compartment decreases due to natural death μ and deaths due to diseases caused by excessive smoking at rate δ.

$$\dot{S_k}(t) = \alpha_k A(t) - \theta_k S_k(t) - (\mu + \delta_k)S_k(t)$$

The Compartement C_{pv}: contains a large number of heavy smokers who use their wealth to access private smoking treatment centers, which are often well equipped and offer quality services. This compartment is increased by the γ_1 rate and decreased by the λ_1 rate representing individuals treated in private treatment centers as well as natural death at the μ rate.

$$\dot{C_{pv}}(t) = \gamma_1 D(t) - (\lambda_1 + \mu)C_{pv}(t)$$

The Compartement C_{pb}:contains people who cannot afford to join private centers and represents the number of heavy smokers who have joined public smoking treatment centers, which do not always offer advanced treatment and are marked by a lack of poor quality facilities and services, especially in developing countries. This compartment is increased by the γ_2 rate and decreases at the λ_2 rate which represents people who have been treated in public treatment centers as well as people who died naturally at the μ rate.

$$\dot{C_{pb}}(t) = \gamma_2 D(t) - (\lambda_2 + \mu)C_{pb}(t)$$

The Compartment H: the number of sick heavy smokers who go to the hospital for treatment of tobacco addiction-related illnesses. It is increased by the θ_k rate and decreases by the λ_3 rate which represents individuals hospitalized for illness as well as natural death at the μ rate.

$$\dot{H}(t) = \sum_{k=1}^{n} \theta_k S_k(t) - (\lambda_3 + \mu)H(t)$$

The Compartment R: includes individuals who have quit smoking permanently. It increases at λ_1 and λ_2 rates,, which corresponds to the recruitment of those who received treatment in tobacco treatment centers, and decreases at the μ rate due to natural deaths.

$$\dot{R}(t) = \lambda_1 C_{pv}(t) + \lambda_2 C_{pb}(t) + \lambda_3 H(t) - \mu R(t).$$

The population size is represented by $N(t)$, which:

$$N(t) = P(t) + A(t) + D(t) + S_k(t) + C_{pv}(t) + C_{pb}(t) + H(t) + R(t),$$

and it is assumed to be Constant.

The directions of individuals among compartments are represented by directed arrows in Figure 1 as shown in the following diagram:

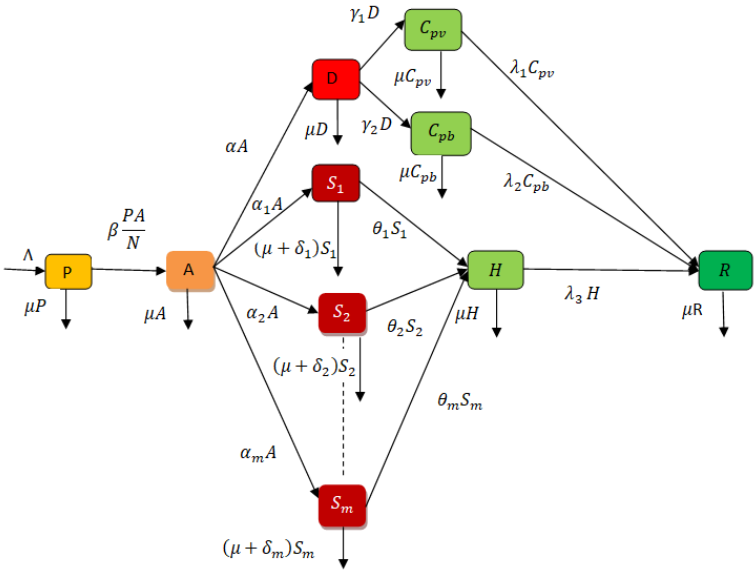

FIGURE 1 - Relations between the $7+n$ compartments $P(t), A(t), D(t), S_k(t), C_{pv}(t), C_{pb}(t), H(t), R(t)$

The dynamics of the model is governed by the differential equation system given by:

$$
\left\{
\begin{array}{rcl}
\dot{P}(t) &=& \Lambda - \beta \frac{P(t)A(t)}{N} - \mu P(t) \\
\dot{A}(t) &=& \beta \frac{P(t)A(t)}{N} - \sum_{k=1}^{n} \alpha_k A(t) - (\alpha + \mu)A(t) \\
\dot{D}(t) &=& \alpha A(t) - \gamma_1 D(t) - \gamma_2 D(t) - \mu D(t) \\
\dot{S_k}(t) &=& \alpha_k A(t) - \theta_k S_k(t) - (\mu + \delta_k) S_k(t) \\
\dot{C_{pv}}(t) &=& \gamma_1 D(t) - (\lambda_1 + \mu) C_{pv}(t) \\
\dot{C_{pb}}(t) &=& \gamma_2 D(t) - (\lambda_2 + \mu) C_{pb}(t) \\
\dot{H}(t) &=& \sum_{k=1}^{n} \theta_k S_k(t) - (\lambda_3 + \mu) H(t) \\
\dot{R}(t) &=& \lambda_1 C_{pv}(t) + \lambda_2 C_{pb}(t) + \lambda_3 H(t) - \mu R(t)
\end{array}
\right. \quad (1)
$$

Where $P(0) \geq 0, A(0) \geq 0, D(0) \geq 0, S_k(0) \geq 0, C_{Pv}(0) \geq 0, C_{Pb}(0) \geq 0, H(0) \geq 0, R(0) \geq 0$ the given initial states.

2.2. Basic Properties: To establish that all solutions of the system with positive initial data will remain positive for all time $t > 0$ and are bounded, the following theorem and lemma are introduced.

a) Positivity of the model solutions:

Theorem 1. *If $P(0) \geq 0$, $A(0) \geq 0$, $D(0) \geq 0$, $S_k(0) \geq 0$, $C_{Pv}(0) \geq 0, C_{Pb}(0) \geq 0, H(0) \geq 0$, $R(0) \geq 0$. The Solutions $P(t), A(t), D(t), S_k(t), C_{pv}(t), C_{pb}(t), H(t), R(t)$ of system (1) are positive for all $t > 0$.*

Proof: It follows from the first equation of system (1) that:

$\dot{P} = \frac{dP}{dt} = \Lambda - \beta \frac{P(t)A(t)}{N} - \mu P(t)$

$\Longrightarrow \frac{dP(t)}{dt} + \beta \frac{P(t)A(t)}{N} + \mu P(t) = \Lambda \geq 0$

$\Longrightarrow \frac{dP(t)}{dt} + (\beta \frac{A(t)}{N} + \mu)p(t) \geq 0$

We multiply the inequality by

$$exp(\int_0^t \beta \frac{A(s)}{N} + \mu ds)$$

We obtain:

$\frac{dP(t)}{dt} exp(\int_0^t \beta \frac{A(s)}{N} + \mu ds) + (\beta \frac{A(t)}{N} + \mu) exp(\int_0^t \beta \frac{A(s)}{N} + \mu ds) P(t) \geq 0$

so

$$\frac{d}{dt}(P(t) exp(\int_0^t \beta \frac{A(s)}{N} + \mu ds)) \geq 0$$

Let's integrate this inequality

$$P(t) \geq P(0) exp(-\int_0^t \beta \frac{A(s)}{N} + \mu ds)$$

Then $P(t)$ is positive. Similarly for the other equations we find

$$A(t) = A(0) exp(-\int_0^t \beta \frac{P(s)}{N} - \sum_{k=1}^{n} (\alpha_k) - \mu ds) \geq 0;$$

$$D(t) \geq D(0) e^{-(\gamma_1 + \gamma_2 + \mu)t} \geq 0;$$

$$S_k \geq S_k(0) e^{-(\theta_k + \mu + \delta_k)t} \geq 0;$$

$$C_{pv}(t) \geq C_{pv}(0) e^{-(\lambda_1 + \mu)t} \geq 0;$$

$$C_{pb}(t) \geq C_{pb}(0) e^{-(\lambda_2 + \mu)t} \geq 0;$$

$$H(t) \geq H(0) e^{-(\lambda_3 + \mu)t} \geq 0;$$

$$R(t) \geq R(0) e^{-\mu t} \geq 0;$$

b) Invariant region:

Lemma: The feasible region Ω defined by:

$\Omega = \{P(t), A(t), D(t), S_k(t), C_{pv}(t), C_{pb}(t), H(t), R(t), P + A + D + S_k + C_{pv} + C_{pb} + H + R \leq \frac{\Lambda}{\mu}\}$

With the conditions $P(0) \geq 0, A(0) \geq 0, D(0) \geq 0, S_k(0) \geq 0, C_{Pv}(0) \geq 0, C_{Pb}(0) \geq 0, H(0) \geq 0,$ $R(0) \geq 0$.

Proof: We add the system equations (1) we find:

$$\dot{N} = \frac{dN}{dt} = \dot{P} + \dot{A} + \dot{D} + \dot{S_k} + \dot{C_{pv}} + \dot{C_{pb}} + \dot{H} + \dot{R}$$

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

We integrate, we get:

$$N(t) \leq N(0) + \Lambda t + \int_0^t -\mu N(s) ds$$

According to Gronwall's lemma we have:

$$N(t) \leq N(0) exp(-\mu t) - \frac{\Lambda}{\mu}(1 - exp(-\mu t))$$

Where N(0) represents the initial values of the total population. So $\limsup_{t \longrightarrow +\infty} N = \frac{\Lambda}{\mu}$. This implies that the region Ω a positively invariant set for system (1). So we only need to consider the dynamics of the system on the set Ω. The first four equations of system (1) are independent of the variables C_{pb}, C_{pv}, H and R. Therefore, the dynamics of the system of equations (1) is equivalent to the dynamics of the system of equations:

$$2 \begin{cases} \dot{P}(t) &= \Lambda - \beta \frac{P(t)A(t)}{N} - \mu P(t) \\ \dot{A}(t) &= \beta \frac{P(t)A(t)}{N} - \sum_{k=1}^{n} \alpha_k A(t) - (\alpha + \mu) A(t) \\ \dot{D}(t) &= \alpha A(t) - \gamma_1 D(t) - \gamma_2 D(t) - \mu D(t) \\ \dot{S}_k(t) &= \alpha_k A(t) - \theta_k S_k(t) - (\mu + \delta_k) S_k(t) \end{cases}$$

3. Stability and Sensitivity Analysis of Model Parameters

In the next section, we will study system's stability behavior at equilibrium with and without tabac.

3.1. Local stability analysis.

We analyze the local stability of the equilibrium points E^0_{eq} and E^*_{eq}

3.1.1. *Smoking-Free Equilibrium Point:*

Theorem 2. *Equilibrium points without Smoking* $E^0_{eq}(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0)$ *of the system (2) is asymptotically stable* $R_0 < 1$ *and is unstable* $R_0 > 1$

Proof: The Jacobian matrix at E^0_{eq} is given by:

$$J(E^0_{eq}) = \begin{pmatrix} -\mu & -\frac{\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & \frac{\beta\Lambda}{N\mu} - (\sum_{k=1}^{n}\alpha_k + (\mu+\alpha)) & 0 & 0 \\ 0 & \alpha & -(\gamma_1+\gamma_2+\mu) & 0 \\ 0 & \alpha_k & 0 & -(\theta_k+\delta_k+\mu) \end{pmatrix}$$

The characteristic equation of this matrix is given by $det(J(E^0_{eq}) - \zeta I_4) = 0$, where I_4 is an identity matrix of order 4

$det(J(E^0_{eq}) - \zeta I_4) = -(\mu+\zeta)[(\frac{\beta\Lambda}{N\mu} - (\sum_{k=1}^{n}\alpha_k + (\mu+\alpha)) - \zeta)(\gamma_1+\gamma_2+\mu+\zeta)(\theta_k+\delta_k+\mu+\zeta)] = 0$ so,the eigenvalues of the characteristic equation of $J(E^0_{eq})$ are:

$$\zeta_1 = \mu$$

$$\zeta_2 = -(\mu+\alpha+\sum_{k=1}^{n}\alpha_k)[1-R_0]$$

$$\zeta_3 = -(\gamma_1+\gamma_2+\mu)$$

$$\zeta_4 = -(\theta_k+\delta_k+\mu)$$

where,

$$R_0 = \frac{\beta\Lambda}{\mu N((\sum_{k=1}^{n}\alpha_k)+\alpha+\mu)}$$

Hence, if $R_0 < 1$, all eigenvalues of the characteristic equation $J(E^0_{eq})$ are negative real numbers. So, we conclude that Equilibrium point without disease $E^0_{eq}(\frac{\Lambda}{\mu},0,0,0,0,0,0,0)$ of system (2) is asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

Remark: The value of R_0 determines the possibility of an epidemic occurrence, which can be calculated using the next generation matrix method as described in [26].

3.1.2. *Point of equilibrium with smoking.*

Theoreme 3. *Equilibrium points with smoking $E^*_{eq}(P^*,A^*,D^*,S^*_k,C^*_{pv},C^*_{pb},H^*,R^*)$ of system (2) is asymptotically stable if $R_0 > 1$ and is unstable $R_0 < 1$.*

Proof: The Jacobian matrix at E^*_{eq} is given by:

$E^*_{eq}(\frac{\Lambda}{\mu R_0}, \frac{\mu N(R_0-1)}{\beta}, \frac{\alpha\mu N(R_0-1)}{\beta(\gamma_1+\gamma_2+\mu)}, \frac{\alpha_k\mu N(R_0-1)}{\beta(\theta_k+\delta_k+\mu)}, \frac{\gamma_1\mu N(R_0-1)}{\beta(\lambda_1+\mu)(\gamma_1+\gamma_2+\mu)}, \frac{\gamma_2\mu N(R_0-1)}{\beta(\lambda_2+\mu)(\gamma_1+\gamma_2+\mu)}, \frac{\sum_{k=1}^{n}\theta_k S_k}{\lambda_3+\mu}, \frac{\lambda_1 C^*_{pv}+\lambda_2 C^*_{pb}\lambda_3 H^*}{\mu})$

where

$$R_0 = \frac{\beta\Lambda}{\mu N((\sum_{k=1}^{n}\alpha_k)+\alpha+\mu)}$$

R_0 is the basic reproduction number which measures the average number of newly infected (smoker) individuals generated by a single infected (heavy smoker) individual in a population of susceptible individuals. Then, the Jacobian matrix at E is given by E^*_{eq}

$$J(E^*_{eq}) = \begin{pmatrix} -\mu R_0 & -(\sum_{k=1}^{n}\alpha_k+(\alpha+\mu)) & 0 & 0 \\ \mu(R_0-1) & 0 & 0 & 0 \\ 0 & \alpha & -(\gamma_1+\gamma_2+\mu) & 0 \\ 0 & \alpha_k & 0 & -(\theta_k+\delta_k+\mu) \end{pmatrix}$$

We notice that the characteristic equation $\varphi(\zeta)$ of $J(E^*_{eq})$

$$\varphi(\zeta) = \zeta^4 + a_1\zeta^3 + a_2\zeta^2 + a_3\zeta + a_4$$

where,

$$a_1 = \gamma_1+\gamma_2+\theta_k+\delta_k+\mu(R_0+2),$$

$$a_2 = (\gamma_1+\gamma_2+\mu)(\theta_k+\delta_k+\mu)+(\gamma_1+\gamma_2+2\mu)\mu R_0+\frac{\beta\Lambda(R_0-1)}{NR_0},$$

$$a_3 = \mu R_0(\gamma_1+\gamma_2+\mu)(\theta_k+\delta_k+\mu)+\frac{(\gamma_1+\gamma_2+\theta_k+2\mu)\beta\Lambda(R_0-1)}{NR_0},$$

$$a_4 = \frac{(\gamma_1+\gamma_2+\mu)(\theta_k+\mu)\beta\Lambda(R_0-1)}{NR_0}.$$

applying the Routh-Hurwitz criterion [24, 25],we can determine the stability of the system. Specifically, the system is locally asymptotically stable if $a_1>0, a_2>0, a_3>0, a_4>0$, and $a_1a_2>a_3a_4$. Therefore, the equilibrium point $E^*_{eq}(P^*, A^*, D^*, S^{k*}, C^*_{pv}, C^*_{pb}, H^*, R^*)$ of system (2) is asymptotically stable if $R_0>1$.

4. GLOBAL STABILITY

4.1. global stability without smoking. To prove that system (2) is globally asymptotically stable, we use the Lyapunov function theory for equilibrium points with and without tobacco. We show here the global stability of the tobacco-free equilibrium E^0_{eq}.

Theorem 4. *Equilibrium points without smoking E^0_{eq} of system (2) is globally asymptotically stable if $R_0 \leq 1$ and is unstable $R_0 > 1$.*

Proof: We consider the following Lyapunov function:

$$V \ : \Gamma \longrightarrow \mathbb{R}$$

$$V(P,A,D,S_k) = A$$

where $\Gamma = \{(P,A,D,S_k) \in \Gamma / P > 0, A > 0, D > 0, S_k > 0\}$.
Then the derivative of the Lyapunov function is given by:

$$\frac{dV}{dt} = \frac{dA}{dt} = (\frac{\beta\Lambda}{\mu N} - (\sum_{k=1}^{n}(\alpha_k) + \alpha + \mu))A$$

$$\frac{dV}{dt} = (\sum_{k=1}^{n}(\alpha_k) + \alpha + \mu)(R_0 - 1)A$$

So, $\frac{dV}{dt} \leq 0$ if $R_0 \leq 1$ also $\frac{dV}{dt} = 0$ if $A = 0$.
Using Lasalle's invariance principle [23], E^0_{eq} is globally asymptotically.

4.2. global stability with smoking.

Theorem 5. *Equilibrium point with smoking E^*_{eq} of system (2) is globally asymptotically stable if $R_0 > 1$.*

Proof: We consider the following Lyapunov function:

$$V \ : \Gamma \longrightarrow \mathbb{R}$$

$$V(P,A) = P - P^* \ln(\frac{P}{P^*}) + A - A^* \ln(\frac{A}{A^*})$$

where $\Gamma = \{(P,A,D,S_k) \in \Gamma / P > 0, A > 0\}$ Then, the derivative of the Lyapunov function is given by:

$$\frac{dV(P,A)}{dt} = (-\frac{\Lambda(P-P^*)}{PP^*} - \frac{\beta}{N}(A-A^*))(P-P^*) + \frac{\beta}{N}(P-P^*)(A-A^*)$$

then,

$$\frac{dV(P,A)}{dt} = -\frac{\Lambda(P-P^*)^2}{PP^*} \leq 0$$

and also,

$$\frac{dV(P,A)}{dt} = 0 \quad if \quad P = P^*.$$

Using Lasalle's invariance principle [24], we can show that E^*_{eq} is globally asymptotically stable.

4.3. Sensitivity analysis of R_0. Sensitivity analysis can help identify the parameters that have the most significant impact on the reproduction number R_0, considering potential errors in data collection and assumed parameter values. We use the approach of Chitnis et al [15] to calculate the normalized forward sensitivity indices of R_0 defined as:

$$\gamma_n^{R_0} = \frac{\partial R_0}{\partial n} * \frac{n}{R_0}.$$

We can note the sensitivity index of R_0 with respect to the parameter n, which we can obtain using the following equation:

$$R_0 = \frac{\beta\Lambda}{\mu N((\sum_{k=1}^{n}\alpha_k) + \alpha + \mu)}$$

$$\gamma_\beta^{R_0} = 1$$

$$\gamma_\alpha^{R_0} = -\frac{\alpha}{(\sum_{k=1}^{n}\alpha_k) + \alpha + \mu}$$

$$\gamma_{\sum_{k=1}^{n}\alpha_k}^{R_0} = -\frac{\sum_{k=1}^{n}\alpha_k}{(\sum_{k=1}^{n}\alpha_k) + \alpha + \mu}$$

$$\gamma_\mu^{R_0} = -\frac{\mu}{(\sum_{k=1}^{n}\alpha_k) + \alpha + \mu} - 1.$$

We note that the basic reproduction number R_0 is the most sensitive to changes in β. Indeed, if β increases, R_0 will also increase in the same proportion, and if β decreases, R_0 will also decrease. But μ, $\sum_{k=1}^{n}\alpha_k$, and α will have an inversely proportional relation with R_0. Therefore, an increase in one of them will lead to a decrease in R_0.

4.4. numerical simulations: In this section, we present numerical simulations of model (1) withdifferent parameter values and initial conditions. The total population is fixed at 17500, by setting $k = \{1,2\}$.

4.4.1. *Smoking-free equilibrium:* We use and present some numerical simulations of the system (1) to illustrate our results, choosing $\Lambda = 1500, \mu = 0.04, \beta = 0.03, \gamma_1 = 0.05, \gamma_2 = 0.05, \alpha = 0.01, \alpha_1 = 0.02, \alpha_2 = 0.02, \delta_1 = 0.07, \delta_2 = 0.07, \lambda_1 = 0.5, \lambda_2 = 0.5, \lambda_3 = 0.7 \theta_1 = 0.05, \theta_2 = 0.05$, and different initial values for each state variable, we have the smoking-free equilibrium $R_0 = 0.7143 < 1$.

In this instance, according to theorem (4), the smoking-free equilibrium E_0 of system (1) is globally asymptotically stable on Ω. (See figures)

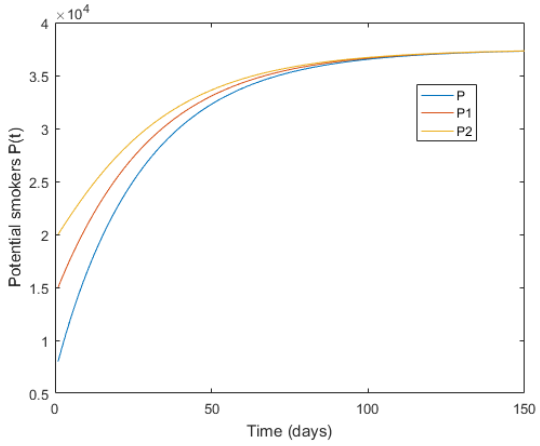

Figure a

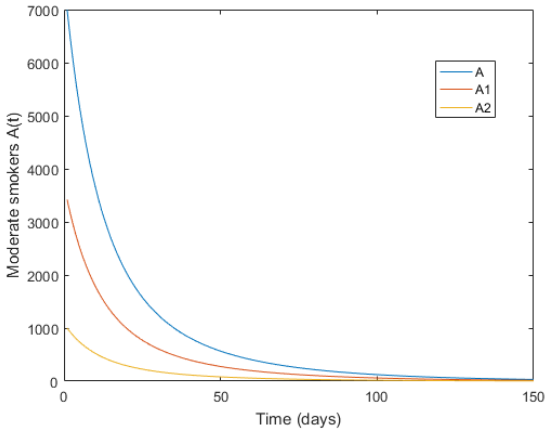

Figure b

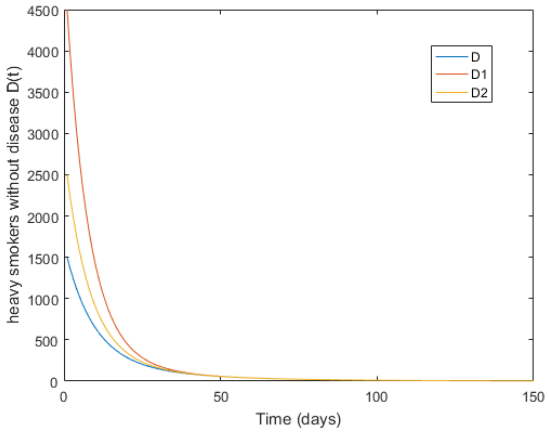

Figure c

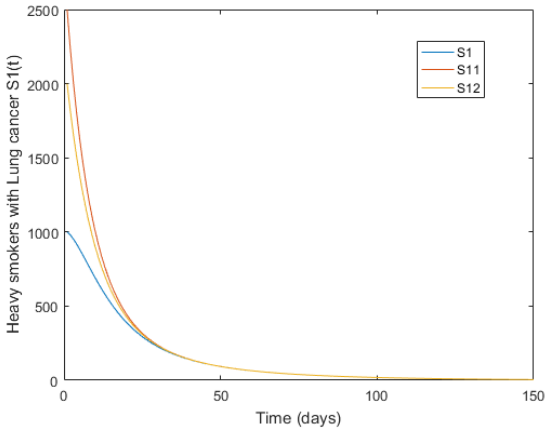

Figure d

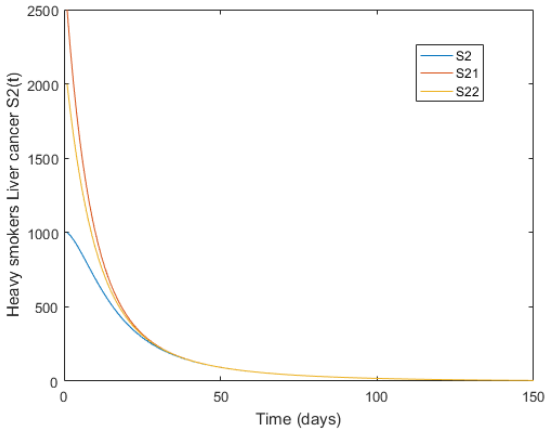

Figure e

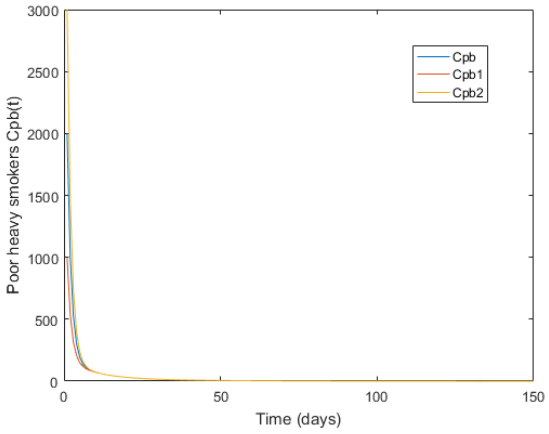

Figure f

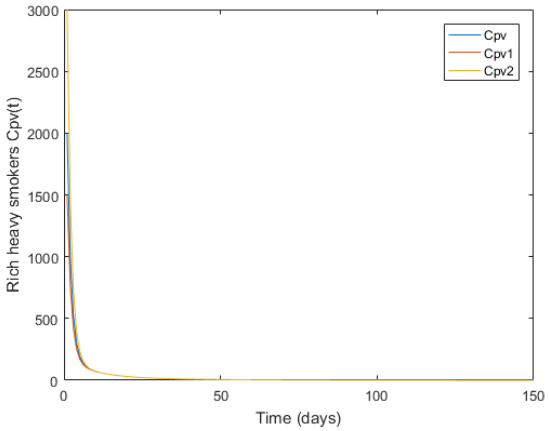

Figure g

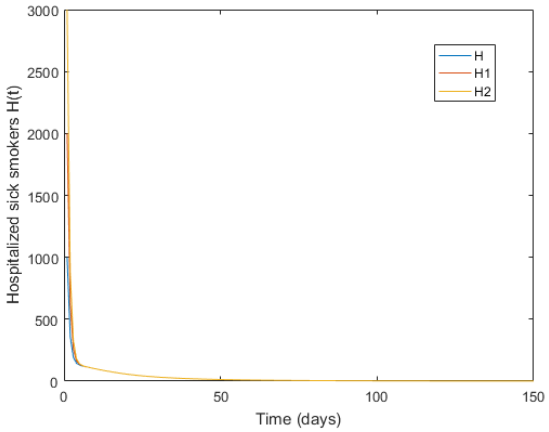

Figure h

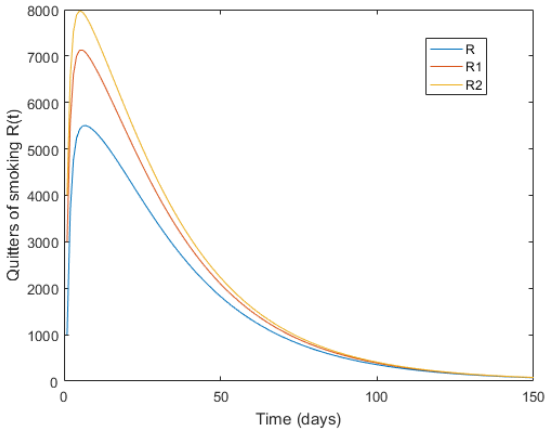

Figure i

From these Figures, using the different values of the initial variables $P_0, A_0, D_0, S_{k,0}$, $C_{pv,0}$,$C_{pb,0}$,H_0 and R_0, we obtained the following remarks:

Remarks:

-The number of potential smokers is increasing and approaching that of the population $P_0 \simeq 3700$ (see figure a)

-The number of moderate smokers decreases and converges to zero (see figure b)

-The number of heavy smokers is decreasing and approaching zero (see figure c)

-The number of heavy smokers who suffer from diseases such as lung cancer S1 and liver cancer S2 decreases and approaches zero (see figures d and e).

- The number of wealthy heavy smokers who join private smoking treatment centers decreases from the beginning and converges to 0 (see figure f).

- Similarly, the number of poor heavy smokers who join public smoking treatment centers decreases at the beginning and converges to 0 (see figure g).

- The number of sick heavy smokers who reach the hospitals of decreases towards 0 (see figure h).

-The number of recovered cases increases and then decreases and approaches zero (see figure i)

Therefore, the solution curves toward the equilibrium $E^0_{eq}(P_0,0,0,0,0,0,0)$ when $R_0 < 1$. Thus, model (1) is globally asymptotically stable.

4.4.2. *Point of equilibrium with smoking:* Also, for $\Lambda = 1500, \mu = 0.04, \beta = 0.09, \gamma_1 = 0.05, \gamma_2 = 0.05, \alpha = 0.02, \alpha_1 = 0.02, \alpha_2 = 0.02, \delta_1 = 0.07, \delta_2 = 0.07, \lambda_1 = 0.5, \lambda_2 = 0.5, \lambda_3 =$

$0.7, \theta_1 = 0.05, \theta_2 = 0.05$,we have equilibrium point with smoking E^*_{eq}, and $R_0 = 1.9286 > 1$ In this case, according to theorem (5), the equilibrium with smoking E^* of system (1) is globally asymptotically stable on Ω.(See Figures)

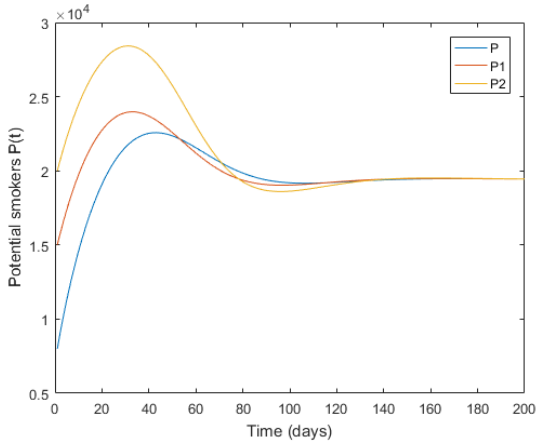

Figure a

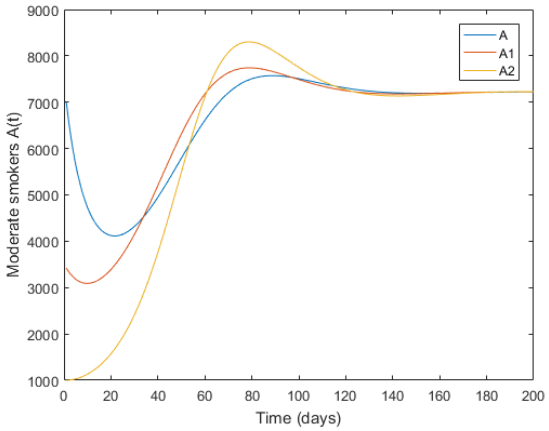

Figure b

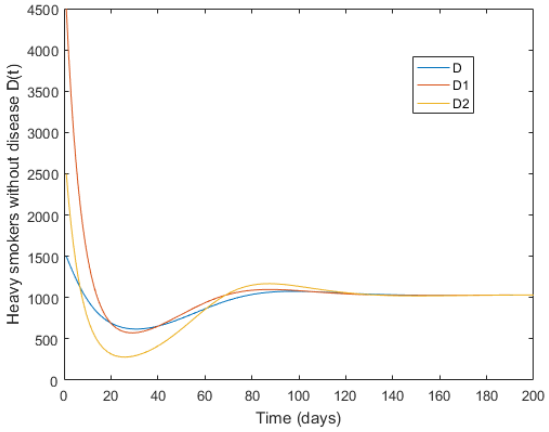

Figure c

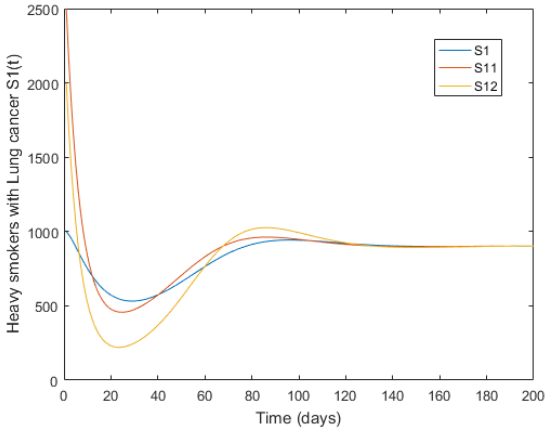

Figure d

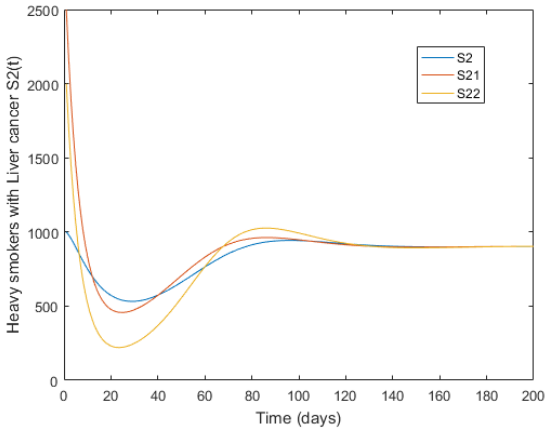

Figure e

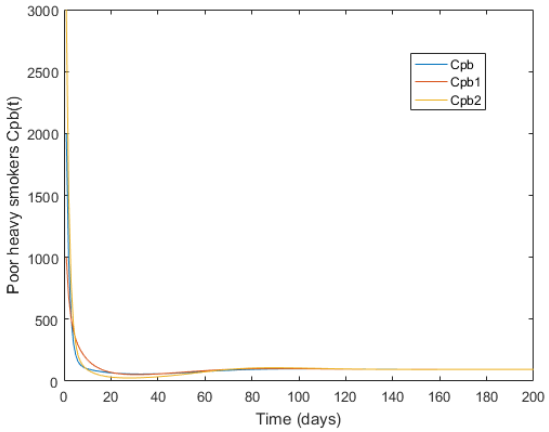

Figure f

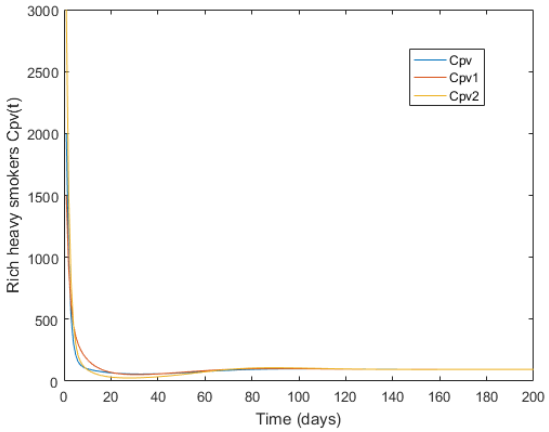

Figure g

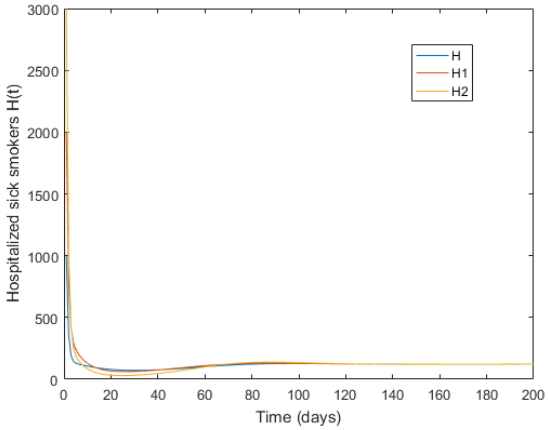

Figure h

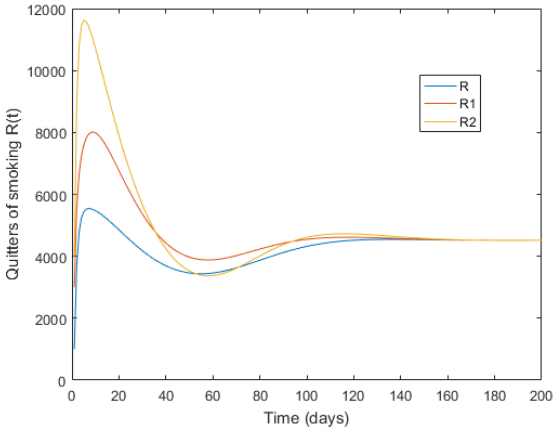

Figure i

Remarks:

-The number of potential smokers increases and then decreases towards $P^* \simeq 1900$ (see figure a).

-The number of moderate smokers increases towards $A^* \simeq 7200$ (see figure b).

-The number of heavy smokers decreases and increases at $D^* \simeq 1050$ (see figures c)

-The number of heavy smokers who suffer from diseases such as lung cancer S_1 and liver cancer S_2 decreases and then increases towards $S_1^* \simeq 800$ and $S_2^* \simeq 800$ respectively (see figures d and e).

- The number of wealthy heavy smokers who join private smoking treatment centers decreases in the early stages and converges to $C_{pv}^* \simeq 100$ (See figure f)

- Similarly, the number of poor heavy smokers who join public smoking treatment centers decreases in the early stages and converges toward $C_{pb}^* \simeq 100$ (See figure g)

- The number of sick heavy smokers who reach the hospitals decreases towards $H^* \simeq 100$ (see figure h).

-The number of recovered cases decreases slightly and then increases towards $R^* \simeq 4300$ (see figure i).

Therefore, the solution curves toward the equilibrium $E_{eq}^*(P^*, A^*, D^*, S_1^*, S_2^*, C_{pv}^*, C_{pb}^*, H^*, R^*)$ when $R_0 > 1$. Thus, model (1) is globally asymptotically stable.

5. Optimal Control

The control strategy we adopt consists of a media and education awareness program, treatment, and psychological support with follow-up. Our principal objective in adopting these strategies is to reduce the number of smokers during the time steps t from 0 to T and also minimize the cost expenditure in applying the three strategies in In this model, we include the two controls $u(t)$, and $v_k(t)$ such that $u(t)$ consecutively represents the awareness program through the media and the effort to encourage rich (poor) heavy smokers to turn to private (public) treatment centers and $v_k(t)$ represents the disease-specific treatment by characterizing by the index k, the effort to encourage heavy smokers diseases to join to hospitals, and the psychological support with follow-up measures at time t. The controlled mathematical system is thus given by the following system of differencial equations:

$$3\begin{cases} \dot{P}(t) &= \Lambda - \beta\frac{P(t)A(t)}{N} - \mu P(t) + \frac{\varepsilon}{3}uD(t) \\ \dot{A}(t) &= \beta\frac{P(t)A(t)}{N} - \sum_{k=1}^{n}\alpha_k A(t) - (\alpha+\mu)A(t) \\ \dot{D}(t) &= \alpha A(t) - \gamma_1 D(t) - \gamma_2 D(t) - \mu D(t) - \varepsilon u D(t) \\ \dot{S_k}(t) &= \alpha_k A(t) - \theta_k S_k(t) - (\mu+\delta_k)S_k(t) - V_k S_k \\ \dot{C_{pv}}(t) &= \gamma_1 D(t) - (\lambda_1+\mu)C_{pv}(t) + \frac{\varepsilon}{3}uD(t) \\ \dot{C_{pb}}(t) &= \gamma_2 D(t) - (\lambda_2+\mu)C_{pb}(t) + \frac{\varepsilon}{3}uD(t) \\ \dot{H}(t) &= \sum_{k=1}^{n}\theta_k S_k(t) - (\lambda_3+\mu)H(t) + \sum_{k=1}^{m} V_k S_k \\ \dot{R}(t) &= \lambda_1 C_{pv}(t) + \lambda_2 C_{pb}(t) + \lambda_3 H(t) - \mu R(t). \end{cases}$$

Where $P(0)\geq 0, A(0)\geq 0, D(0)\geq 0, S_k(0)\geq 0, C_{pv}(0)\geq 0, C_{pb}(0)\geq 0, H(0)\geq 0$ and $R(0)\geq 0$. The optimal control problem for minimizing the objective function is given by:

$$J(u,v_k) = B_1 D(T) + B_2\sum_{k=1}^{m} S_k(T) + \int_0^{T-1}(B_1 D(t) + B_2\sum_{k=1}^{m} S_k(t) + \frac{B_3 u^2}{2} + \frac{\sum_{k=1}^{m} C_k v_k^2}{2})dt$$

where $B_1(t)\geq 0, B_2(t)\geq 0,\ B_3(t)\geq 0, C_k(t)\geq 0$ are selected to assess the relative importance of the cost of awareness programs, treatment programs, and follow-up counseling, respectively. The goal is to find an optimal control u^* and v_k^* such that:

$$J(u^*, v_k^*) = \min_{u,v_k\in U_{ad}} J(u,v_k)$$

where U_{ad} is the set of admissible controls defined by:
$U_{ad} = \{(u(t), v_k(t)), 0\leq u(t)\leq 1, 0\leq v_k(t)\leq 1, \text{for all } t\in[0,T] \text{ and } k = 1,..,n\}$.

5.1. Existence of optimal controls: The existence of optimal controls can be obtained by using the result of Fleming and Rishel [16] (see Corollary 4.1).

Theorem 6. *We consider the control problem with the system (2). There exists an optimal control* $u^*, v_k^* \in U_{ad}$*, with* $k = 1,..,n$ *such as:*

$$J(u^*, v_k^*) = \min_{u, v_k \in U_{ad}} J(u, v_k)$$

If the following conditions are met:

(1) The set of controls and state variables is not empty.

(2) The set of controls U_{ad} *is convex and closed.*

(3) The right-hand side of the system is bounded by a linear function in the state variables and control variables. and control variables.

(4) Lagrange $L(P,A,D,S_k,C_{pv},C_{pb},H,R,u,v_k)$ *for* $k = 1,..,n$ *of the objective function is convex on* U_{ad} *and there are constants* $a_1, a_2 \geq 0$ *et* $\eta > 1$ *such as:*

$$L(P,A,D,S_k,C_{pv},C_{pb},H,R,u,v_k) \geq a_1 + a_2(|u|^2, |v_k|^2)^{\frac{\eta}{2}}$$

Proof:

(1) To show that the set of control and state variable is not empty, we use a simple version of an existence result in the simple version of existence result ([17] theorem 7.1.1).

Let $\dot{P} = F_P(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$,

$\dot{A} = F_A(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$,

$\dot{D} = F_D(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$,

$\dot{S_k} = F_{S_k}(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$,

$\dot{C_{pv}} = F_{C_{pv}}(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$

$\dot{C_{pb}} = F_{C_{pb}}(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$

$\dot{H} = F_H(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$

et $\dot{R} = F_R(t,P,A,D,S_k,C_{pv},C_{pb},H,R)$

where $F_P, F_A, F_D, F_{S_k} F_{C_{pv}}, F_{C_{pb}}, F_H, F_R$ form the right-hand side of the system of equations. Let $u(t) = q_1$, $v_k(t) = p_k$, for some constants, and since all parameters are constants and $(P,A,D,S_k,C_{pv},C_{pb},H,R)$ are continuous, then $F_P, F_A, F_D, F_{S_k} F_{C_{pv}}, F_{C_{pb}}, F_H, F_R$ are also continuous.

Moreover the partial derivatives $\frac{F_P}{\partial P}$, $\frac{F_P}{\partial A}$, $\frac{F_P}{\partial D}$, $\frac{F_P}{\partial S_k}$, $\frac{F_P}{\partial C_{pv}}$, $\frac{F_P}{\partial C_{pb}}$, $\frac{F_P}{\partial H}$, $\frac{F_P}{\partial R}$, $\frac{F_A}{\partial P}$, $\frac{F_A}{\partial A}$, $\frac{F_A}{\partial D}$, $\frac{F_A}{\partial S_k}$, $\frac{F_A}{\partial C_{pv}}$, $\frac{F_A}{\partial C_{pb}}$, $\frac{F_A}{\partial H}$, $\frac{F_A}{\partial R}$, $\frac{F_D}{\partial P}$, $\frac{F_D}{\partial A}$, $\frac{F_D}{\partial D}$, $\frac{F_D}{\partial S_k}$, $\frac{F_D}{\partial C_{pv}}$, $\frac{F_D}{\partial C_{pb}}$, $\frac{F_D}{\partial H}$, $\frac{F_D}{\partial R}$, $\frac{F_{S_k}}{\partial P}$, $\frac{F_{S_k}}{\partial A}$, $\frac{F_{S_k}}{\partial D}$, $\frac{F_{S_k}}{\partial S_k}$, $\frac{F_{S_k}}{\partial C_{pv}}$, $\frac{F_{S_k}}{\partial C_{pb}}$, $\frac{F_{S_k}}{\partial H}$, $\frac{F_{S_k}}{\partial R}$, $\frac{F_{C_{pb}}}{\partial P}$, $\frac{F_{C_{pb}}}{\partial A}$, $\frac{F_{C_{pb}}}{\partial D}$, $\frac{F_{C_{pb}}}{\partial S_k}$, $\frac{F_{C_{pb}}}{\partial C_{pv}}$, $\frac{F_{C_{pb}}}{\partial C_{pb}}$, $\frac{F_{C_{pb}}}{\partial H}$, $\frac{F_{C_{pb}}}{\partial R}$, $\frac{F_{C_{pv}}}{\partial P}$, $\frac{F_{C_{pv}}}{\partial A}$, $\frac{F_{C_{pv}}}{\partial D}$, $\frac{F_{C_{pv}}}{\partial S_k}$, $\frac{F_{C_{pv}}}{\partial C_{pv}}$, $\frac{F_{C_{pv}}}{\partial C_{pb}}$, $\frac{F_{C_{pv}}}{\partial H}$, $\frac{F_{C_{pv}}}{\partial R}$, $\frac{F_H}{\partial P}$, $\frac{F_H}{\partial A}$, $\frac{F_H}{\partial D}$, $\frac{F_H}{\partial S_k}$, $\frac{F_H}{\partial C_{pv}}$, $\frac{F_H}{\partial C_{pb}}$, $\frac{F_H}{\partial H}$, $\frac{F_H}{\partial R}$, $\frac{F_R}{\partial P}$, $\frac{F_R}{\partial A}$, $\frac{F_R}{\partial D}$, $\frac{F_R}{\partial S_k}$, $\frac{F_R}{\partial C_{pv}}$, $\frac{F_R}{\partial C_{pb}}$, $\frac{F_R}{\partial H}$, $\frac{F_R}{\partial R}$, are all continuous.

So there exists a unique solution $(P,A,D,S_k,C_{pv},C_{pb},H,R)$ satisfying the initial conditions. Then the set of controls and state variables is nonempty.

(2) By definition, U_{ad} is closed. For all controls $u, v_k, \in U_{ad}$ with $k = 1,..,n$ and $\lambda \in [0,1]$, then $\lambda u + (1-\lambda)v_k \geq 0$. Moreover, we notice that $\lambda u \leq \lambda$ and $(1-\lambda)v_k \leq (1-\lambda)$ so

$$\lambda u + (1-\lambda)v_k \leq \lambda + (1-\lambda) = 1$$

therefore

$$0 \leq \lambda u + (1-\lambda)v_k \leq 1$$

thus U_{ad} is convex.

(3) From the system of differential equations, we have:

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

then

$$\limsup_{t \longrightarrow +\infty} N(t) = \frac{\Lambda}{\mu}.$$

Therefore, all solutions of model (2) are bounded. So there are positive constants $B_1, B_2, B_3, B_4\ B_5\ B_6\ B_7$ *and* B_8 such that:

$\forall t \in [0,T]$, $P(t) \leq B_1$, $A(t) \leq B_2$, $D(t) \leq B_3$, $S_k(t) \leq B_4$ for all $k = 1,...,n$, $C_{pv}(t) \leq B_5, C_{pb}(t) \leq B_6, H(t) \leq B_7, R(t) \leq B_8$.

We consider:

$$(3)\begin{cases} F_P &= \dot{P}(t) \leq \Lambda + \frac{\varepsilon}{3}uD \\ F_A &= \dot{A}(t) \leq \frac{\beta}{N}P(t) \\ F_D &= \dot{D}(t) \leq \alpha A(t) - \varepsilon u B_3 \\ F_{S_k} &= \dot{S_k}(t) \leq \alpha_k A(t) - v_k S_k \\ F_{C_{pv}} &= \dot{C_{pv}}(t) \leq \gamma_1 D(t) + \frac{\varepsilon}{3}uD \\ F_{C_{pb}} &= \dot{C_{pb}}(t) \leq \gamma_2 D(t) + \frac{\varepsilon}{3}uD \\ F_H &= \dot{H}(t) \leq \sum_{k=1}^{n} \theta_k S_k(t) + \sum_{k=1}^{n} v_k S_k(t) \\ F_R &= \dot{R}(t) \leq \lambda_1 C_{pv}(t) + \lambda_2 C_{pb}(t) + \lambda_3 H(t). \end{cases}$$

So we can write system (3) in matrix form:

$$F(t,P,A,D,S_k,C_{pv},C_{pb},H,R) \leq \bar{\Lambda} + AX(t) + BU(t)$$

Where,

$$F(t,P,A,D,S_k,C_{pv},C_{pb},H,R) = [F_P, F_A, F_D, F_{S_k} F_{C_{pv}}, F_{C_{pb}}, F_H, F_R]^T$$

$$\bar{\Lambda} = [\Lambda\ 0\ 0\ 0\ 0\ 0\ 0\ 0]^T$$

$$X(t) = [P(t)\ A(t)\ D(t)\ S_k(t)\ C_{pv}(t)\ C_{pb}(t)\ H(t)\ R(t)]$$

$$U(t) = [u, v_k]$$

$$A = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta}{N} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_k & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sum_{k=1}^{n} \theta_k & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda_1 & \lambda_2 & \lambda_3 & 0 \end{bmatrix}$$

$$B = \begin{bmatrix} \frac{\varepsilon}{3}D & 0 \\ 0 & 0 \\ -\frac{\varepsilon}{3}D & 0 \\ 0 & -S_k \\ \frac{\varepsilon}{3}D & 0 \\ \frac{\varepsilon}{3}D & 0 \\ 0 & \sum_{k=1} S_k \\ 0 & 0 \end{bmatrix}$$

It gives a linear function of the control vector and the state variable vector, so it is possible to write:

$$\begin{aligned} ||F(t,P,A,H,T^r,T^p,Q)|| &\leq ||\bar{\Lambda}|| + ||A||||X(t)|| + ||B||||U(t)|| \\ &\leq \varphi + \phi(||X(t)|| + ||U(t)||), \end{aligned}$$

Where $\varphi = ||\bar{\Lambda}||$ et $\phi = \max(||A||, ||B||)$.

Therefore we see the right side is augmented by a sum of state vectors and control vectors. So condition 3 is satisfied.

(4) It is clear that Lagrange $L(P,A,D,S_k,C_{pv},C_{pb},H,R,u,v_k)$ of the objective function is convex on U_{ad}, it remains to see that there are constants $a_1, a_2 \geq 0$ *et* $\delta > 1$ such as: $L(P,A,D,S_k,C_{pv},C_{pb},H,R,u,v_k) = B_1 D(T) + B_2 \sum_{k=1}^{m} S_k(T) + \frac{B_3 u^2}{2} + \frac{\sum_{k=1}^{n} C_k v_k^2}{2} \geq a_1 + a_2(|u|^2, |v_k|^2)^{\frac{\eta}{2}}$.

The state variables are bounded, that is: $a_1 = 2\sup_{t\in[0,T]}\{B_1 D(t) + B_2 \sum_{k=1}^{m} S_k(t)\}$,

$a_2 = \inf_{t\in[0,T]}\{\frac{B_3(t)}{2} + \frac{\sum_{k=1}^{n} C_k(t)}{2}$ and $\eta = 2$.

Therefore it follows that

$$L(P,A,D,S_k,C_{pv},C_{pb},H,R,u,v_k) \geq a_1 + a_2(|u|^2,|v_k|^2)^{\frac{\eta}{2}}.$$

5.2. Characterization of optimal controls. In this subsection, we apply the Pontryagin's maximum principle [18, 19, 20, 21, 22].We introduce the adjoint function to relate the system of differential equations to the resulting in the formation of a Hamiltonian function.

This principle converts the problem of finding the control to optimize the objective function with the initial condition, to finding the control to optimize Hamiltonian point by point.

Now we have the Hamiltonian H at time t defined by:

$$\hat{H}(t) = B_1 D(T) + B_2 \sum_{k=1}^{n} S_k(T) + \frac{B_3 u^2}{2} + \frac{\sum_{k=1}^{n} C_k v_k^2}{2} + \sum_{i=1}^{8} \lambda_i f_i$$

Where f_i is the right-hand side of the system of differential equations of the i th state variable.

Theoreme 7. *given optimal controls* $U^* = (u^*,$ *et* $v_k^*)$ *and the solutions* $P^*,A^*,D^*,S_k^*,C_{pv}^*,C_{pb}^*,H^*,R^*$ *of the corresponding state system, there are adjoint functions* $\xi_1,\xi_2,\xi_3,\xi_4,\xi_5,\xi_6,\xi_7,\xi_8$ *are satisfied these conditions:*

$\forall k = 1,...,n$

$$\begin{cases} \dot{\xi_1} &= (\xi_2 - \xi_1)\frac{\beta A}{N} - \xi_1\mu \\ \dot{\xi_2} &= (\xi_2 - \xi_1)\frac{\beta P}{N} - \xi_2(\sum_{k=1}^{n}\alpha_k + \alpha + \mu) + \xi_3\alpha + \xi_4\alpha_k \\ \dot{\xi_3} &= B_1 + \xi_1\frac{\varepsilon}{3}u - \xi_3(\gamma_1 + \gamma_2 + \mu + \varepsilon u) + \xi_5(\gamma_1 + \frac{\varepsilon}{3}u) + \xi_6(\gamma_2 + \frac{\varepsilon}{3}u) \\ \dot{\xi_4} &= B_2 - \xi_4(\theta_k + \delta_k + \mu + v_k) + \xi_7(\theta_k + v_k) \\ \dot{\xi_5} &= -\xi_5(\lambda_1 + \mu) + \xi_8\lambda_1 \\ \dot{\xi_6} &= -\xi_6(\lambda_2 + \mu) + \xi_8\lambda_2 \\ \dot{\xi_7} &= -\xi_7(\lambda_3 + \mu) + \xi_8\lambda_3 \\ \dot{\xi_8} &= -\xi_8\mu \end{cases}$$

With the conditions of transversality to time T:

$$\xi_1(T) = 0$$

$$\xi_2(T) = 0$$

$$\xi_3(T) = B_1$$

$$\xi_4(T) = B_2$$

$$\xi_5(T) = 0$$

$$\xi_6(T) = 0$$

$$\xi_7(T) = 0$$

$$\xi_8(T) = 0$$

In addition, for $t \in [0,T]$, the optimal controls are given by:

$$u^* = \min(1, \max(0, \frac{(3\xi_3 - \xi_6 - \xi_1 - -\xi_5)\varepsilon D}{3B_3}))$$

$$v_k^* = \min(1, \max(0, \frac{(\xi_4 - \xi_7)S_k}{C_k}))$$

Proof:

The Hamiltonian at time t is given by:

$$\begin{aligned}
\hat{H}(t) &= B_1A(t) + A_2D(t) + \tfrac{B_1u_1^2}{2} + \tfrac{B_2u_2^2}{2} + \tfrac{B_3v^2}{2} + \xi_1(\Lambda - \beta\tfrac{P(t)A(t)}{N} - \mu P(t)) \\
&+ \xi_2(\beta\tfrac{P(t)A(t)}{N} - \textstyle\sum_{k=1}^{n}\alpha_k A(t) - (\alpha+\mu)A(t) - u_1A(t)) \\
&+ \xi_3(\alpha A(t) - \gamma_1 D(t) - \gamma_2 D(t) - \mu D(t) - u_2 D(t)) \\
&+ \xi_4(\alpha_k A(t) - \theta_k S_k(t) - (\mu+\delta_k)S_k(t)) + \xi_5(\gamma_2 D(t) - (\lambda_2+\mu)C_{pb}(t)) \\
&+ \xi_6(\gamma_2 D(t) - (\lambda_2+\mu)C_{pb}(t)) + \xi_7(\textstyle\sum_{k=1}^{n}\theta_k S_k(t) - (\lambda_3+\mu)H(t)) \\
&+ \xi_8(\lambda_1 C_{pv}(t) + \lambda_2 C_{pb}(t) + \lambda_3 H(t) - \mu R(t) + u_1 A(t) + u_2 D(t) - vR(t))
\end{aligned}$$

for $t \in [0,T]$, the adjoint equations and the transversality conditions can be obtained using

Pontryagin's maximum principle in [17], such that:

$$\begin{cases} \dot{\xi_1} = \frac{\partial \hat{H}}{\partial P}, & \xi_1(T) = 0 \\ \dot{\xi_2} = \frac{\partial \hat{H}}{\partial A}, & \xi_2(T) = 0 \\ \dot{\xi_3} = \frac{\partial \hat{H}}{\partial D}, & \xi_3(T) = B_1 \\ \dot{\xi_4} = \frac{\partial \hat{H}}{\partial S_k}, & \xi_4(T) = B_2 \\ \dot{\xi_5} = \frac{\partial \hat{H}}{\partial C_{pv}}, & \xi_5(T) = 0 \\ \dot{\xi_6} = \frac{\partial \hat{H}}{\partial C_{pb}}, & \xi_6(T) = 0 \\ \dot{\xi_7} = \frac{\partial \hat{H}}{\partial H}, & \xi_7(T) = 0 \\ \dot{\xi_8} = \frac{\partial \hat{H}}{\partial R}, & \xi_8(T) = 0 \end{cases}$$

for $t \in [0,T]$ optimal controls u^*, v_k^* can be solved from the optimality condition:

$$\frac{\partial \hat{H}}{\partial u} = 0$$

$$\frac{\partial \hat{H}}{\partial v_k} = 0$$

$$\frac{\partial \hat{H}}{\partial u} = B_3 u - \xi_1 \frac{\varepsilon}{3} D - \xi_3 \varepsilon D + \xi_5 \frac{\varepsilon}{3} D + \xi_6 \frac{\varepsilon}{3} D = 0$$

$$\frac{\partial \hat{H}}{\partial v_k} = C_i v_k - \xi_4 S_k + \xi_7 S_k = 0$$

$$u(t) = \frac{(3\xi_3 - \xi_6 - \xi_5 - \xi_1)\varepsilon D}{3B_3}$$

$$v_k(t) = \frac{(\xi_4 - \xi_7) S_k}{C_k}$$

By the boundedness in $U_{ad}$of the controls, it is easy to obtain u^*, v_k^*.

6. SIMULATION

The proposed optimal control strategy is obtained by solving the optimal system which consists of nine differential equations, we take $k = 1,2$, and boundary conditions. optimality system can be solved using an iterative method. Using an initial estimate for the control variables $u(t)$, and $v_k(t)$ with $k = 1,2$, the state variables,P,A,D,S_1,S_2, C_{pv},C_{pb},H, and R are resolved forward and the adjoint variables ζ_i pour $i = 1,2,3,4,5,6,7,8,9$ are solved backward at time steps $t = 0$ and $t = T$. If the new values of the state and adjoint variables are different from the previous values, the new values are used to update $u(t)$ et $v_k(t)$ for $k = 1,2$, and the process is repeated until the system. The numerical solution of model (1) is executed using Matlab with the following parameter values and of parameters and the initial values of the state variable in table1.

P_0 8000	A_0 7000	D_0 1500	$S1_0$ 1000	$S2_0$ 1000	Cpv_0 2000	Cpb_0 2000	H_0 1000	R_0 1000	Λ 1500
N 17500	α 0.06	α_1 0.06	α_2 0.05	γ_1 0.45	γ_2 0.45	λ_1 0.2	λ_2 0.4	λ_3 0.04	θ_1 0.2
θ_2 0.2	μ 0.04	δ_1 0.05	δ_2 0.05	ε 0.5	β 0.3				

Table 1: The parameters used for the model (1)

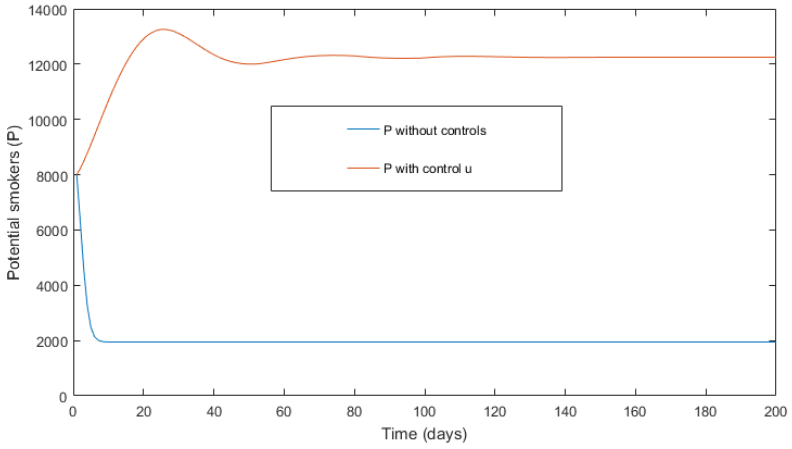

Figure a

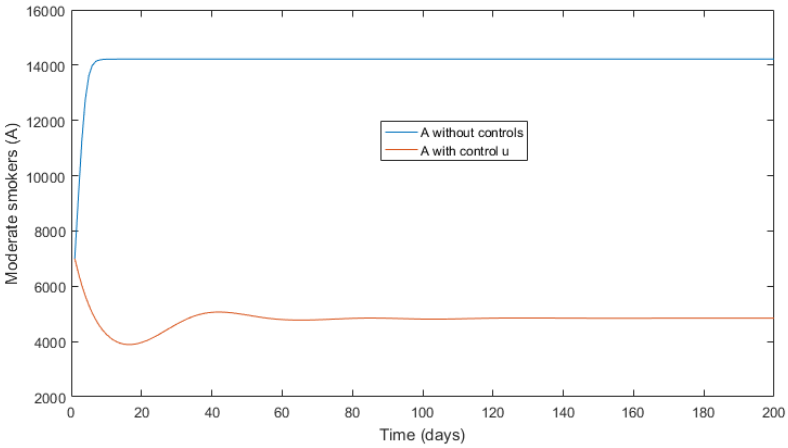

Figure b

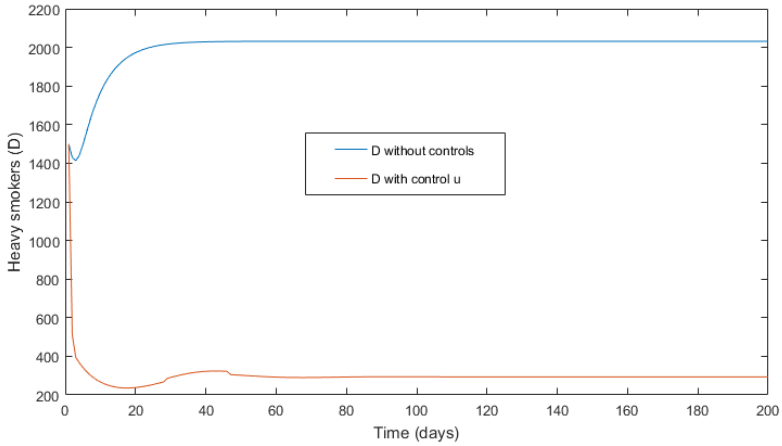

Figure c

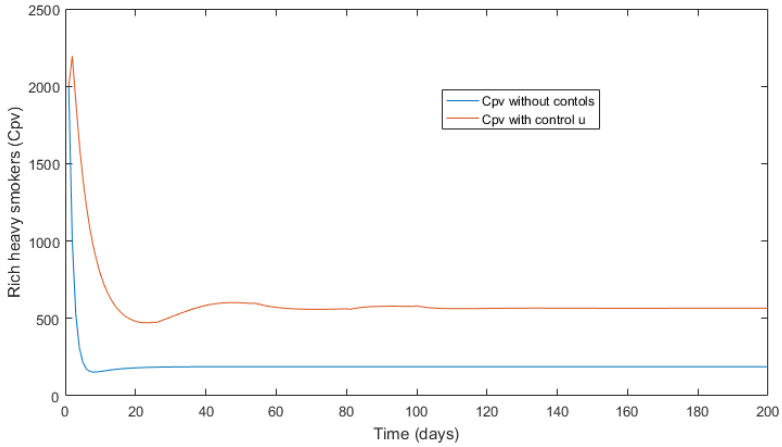

Figure d

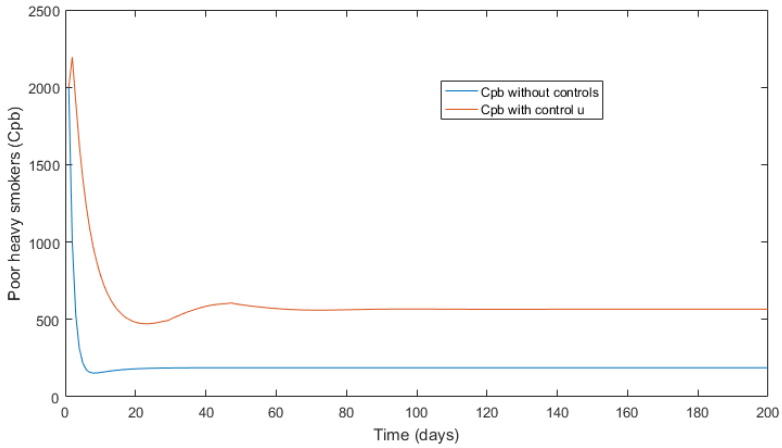

Figure e

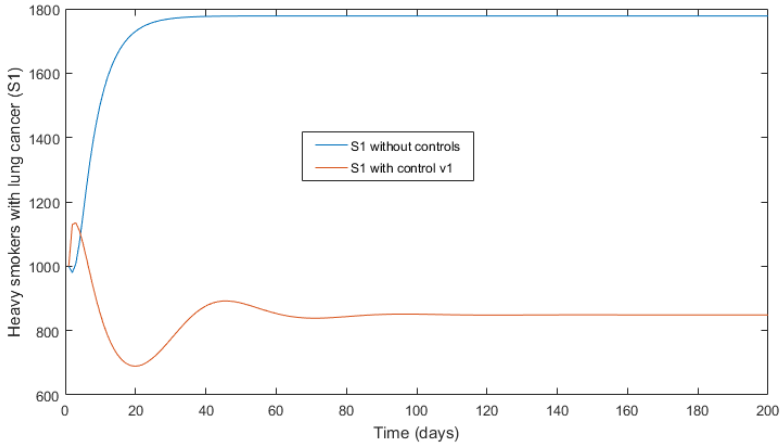

Figure f

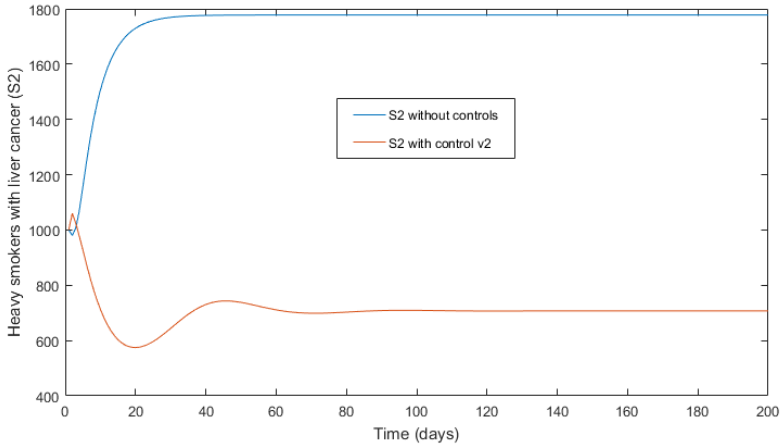

Figure g

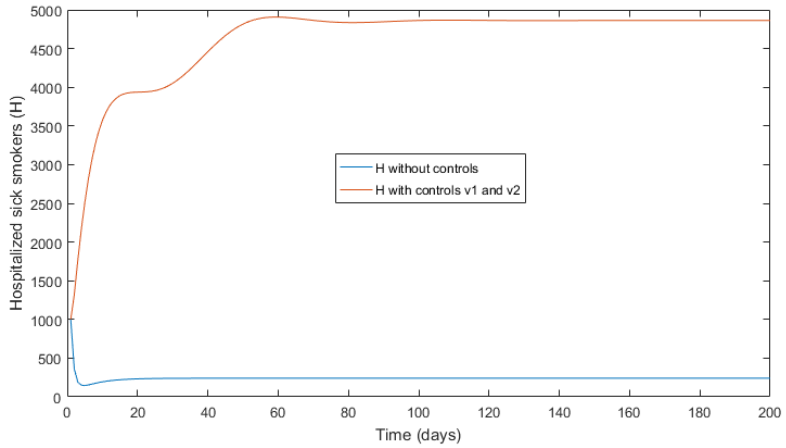

Figure h

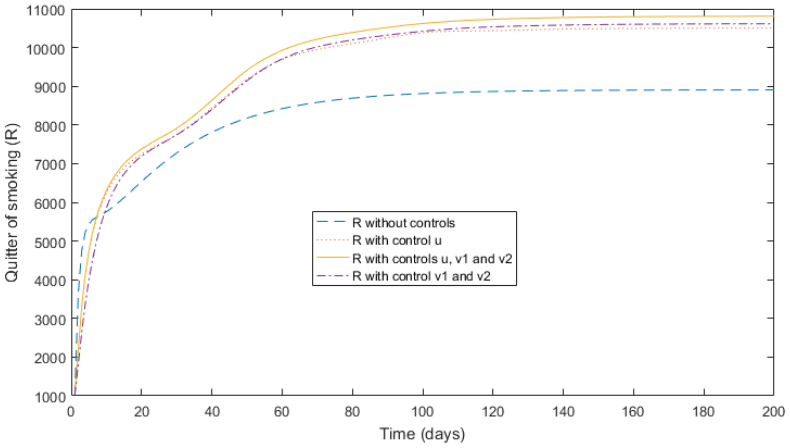

Figure i

Under Strategy A, we aim to increase the number of heavy smokers who reach smoking treatment centers and prevent them from becoming addicted to smoking. To achieve this, we apply the control u, which involves implementing awareness programs, education, and information campaigns for potential and moderate smokers to highlight the risks of smoking to their health and finances. We also encourage heavy smokers to join public and private treatment centers. Figure (a) shows a significant increase in the number of potential smokers with control, while Figure (b) shows a significant decrease in the number of moderate smokers. Figure (c) shows that the number of heavy smokers decreases from 39.8222 (without control u) to almost 2.7126 (with control u) at the end of the proposed control strategy. Figure (d) shows that the number of wealthy heavy smokers joining private treatment centers increases from 53.7600 (without

control u) to 488.2629 (with control u), while Figure (e) shows that the number of poor heavy smokers joining public treatment centers increases to 488.2625.

Under Strategy B, we aim to encourage sick heavy smokers to join tobacco treatment hospitals and relevant disease tracking programs. We choose two diseases, lung cancer and liver cancer, to test the theoretical results. We use the controls v_1 and v_2, where v_1 is for heavy smokers S_1 who suffer from lung cancer and v_2 is for heavy smokers S_2 who suffer from liver cancer. Figure (f) shows that the number of heavy smokers who carry the disease (1) "lung cancer" decreases from 83.68572 (without control v_1) to 45.5111 (with control v_1) at the end of the proposed strategy. Similarly, Figure (g) shows that the number of heavy smokers who carry the disease (2) "liver cancer" decreases from 69.73809 (without control v_2) to 45.5111 (with control v_2) at the end of the proposed strategy.

For Strategy C, we aim to prevent smoking addiction, encourage aftercare treatment, and raise awareness. To achieve this objective, we use three controls: u, v_1, and v_2. Control u promotes awareness among potential and moderate smokers, encouraging heavy smokers to join public and private aftercare treatment centers, while controls v_1 and v_2 encourage sick heavy smokers to join treatment hospitals with follow-up. Figure (h) illustrates the increase in heavy smokers joining hospitals when all three controls are applied, and Figure (i) shows a clear increase in smoking cessation from nearly 9000 (without controls) to almost 11300 (with all three controls). Consequently, the previously stated objective was successfully achieved.

Note: Based on Figure (i), it is evident that a combination of the three controls is more effective than a single control.

7. Conclusion

In the conclusion section, the continuous modeling of smokers was presented to minimize the number of heavy smokers and those suffering from smoking-related diseases. The stability analysis for the equilibrium points with and without smoking was also performed. Two controls were introduced: one involved an awareness program through education and media, treatment and psychological support with follow-up, while the second control involved encouragement and treatment. Optimum controls were characterized using the results of the control theory, and the numerical simulation confirmed the effectiveness of the proposed control strategies.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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