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Modelling the Dynamics of Smoking Epidemic

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Authors' contributions

This work was carried out in collaboration between both authors. Authors MARENO and IKA assisted in developing the model equations, writing the draft, numerical simulations and the review of the final draft. Both authors read and approved the final manuscript.

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Abstract

Our study is made up two sections: Non-smokers, problem- smokers, smokers-in-treatment and counselling, and removed-smokers (SPT_cR) mathematical model that explains the dynamics of smoking epidemic without considering the recovery class to susceptible class transferring followed by modelling smoking epidemic where the recovery class is considered to revert to susceptible class to become problem smokers again after treatment and recovery respectively. We discussed the existence and stability of the smoking-free and endemic equilibria of both models. Our mathematical analysis of both models establish that the global dynamics of smoking epidemic transmission can be determined by the basic reproductive number. The smoking-free equilibrium was locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ in both models. Global stability of smoking-free and endemic equilibria was also discussed in our first model, using Lassalle's invariance principle of Lyapunov functions. Numerical simulations were conducted using Matlab software to confirm our analytic results in both models. Our findings were that reducing the contact rate between the non-smokers and problem smokers, increasing the number of smokers that go into treatment and educating smokers to refrain from smoking can be useful in combating the smoking epidemic.

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1 Introduction

Smoking not only affects health of individuals but also creates burden on society as well as on economy of a country [1]. Almost 6 million people die from tobacco use each year, both from direct tobacco use and second-hand smoking. It is estimated that by the year 2020, the annual tobacco related deaths will be increased to 7.5 million which will account for 10% of total deaths worldwide [1,2].

Smoking is the primary cause of lungs diseases such as Lungs Cancer and Chronic Obstructive Pulmonary diseases(COPD) [3]. It has been reported that there is a relation between tobacco use and Pulmonary Tuberculosis (TB), and there is increasing evidence of this association. Smoking has been positively associated with the development of TB infection, active TB-relapse and related mortality rates [4]. Smoking is also a cause of heart disease, stroke, peripheral vascular disease and other respiratory diseases and low-birth weight in babies [5,7]. Among adolescents, smoking is also connected to social factors. Adolescents whose families and friends smoke are more likely to start smoking earlier than their counterparts [5]. Figs. 1 and 2 indicate comparisons of smoking prevalence among adults in some selected five countries of five continents in the world. Due to unavailability of continuous data on smoking for the selected countries and for simplicity, we decided to use one country from each continent and 2013 data [6] respectively for our comparisons.

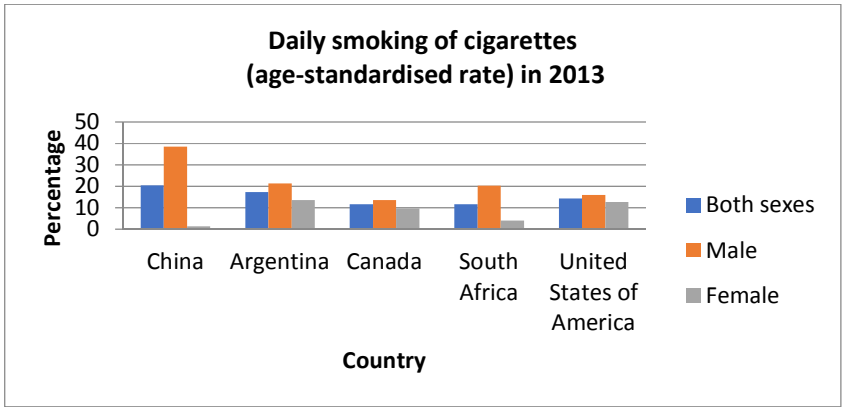


Fig. 1. Prevalence of tobacco use among adults in selected five countries—males and females, age-standardised [6]

Tobacco use is considered as a disease that can spread through social contact in a way similar to the spread of infectious diseases. Mathematical models can be used to understand the spread of smoking and predict the impact of smoking on the community in order to help reduce the number of smokers [8].

In 2015, [1] proposed mathematical models to study the dynamics of smoking behaviour under the influence of educational programs and also individual determination to quit smoking. They divided their total population into three classes: potential smokers (P), smokers (S) and quitters (Q). They performed stability analysis of smoking free and endemic equilibria, sensitivity analysis and numerical simulations of their model. According to their results, determination and education play an important role in reducing the smoking prevalence but determination alone cannot eradicate it.

van Voorn et al. [3], presented a simple but dynamical eco-epidemiological model on smoking. Their model formulation consists of a resource-population dynamic part coupled with an epidemiological part similar to

an SIR type model for the three compartments: non-smokers, smokers and ex-smokers. According to their model, the coupling is via birth of non-smokers and death of the three classes with different death rates. They used brute force simulations for the short-term dynamics and bifurcation analysis for the long-term dynamics to study the final four-dimensional systems of Ordinary Differential Equations in their model. Due to a feed-back mechanism of the two coupling terms there is a codim-two tangent transcritical bifurcation. This leads to bi-stability of one smoker epidemic interior equilibrium and a smoker-free boundary equilibrium. They concluded that changing parameters beyond the emerging tangent bifurcation leads on the short-term to eradicate smoking.

Lahrouz et al. [7], studied a deterministic and stochastic stability of mathematical model of smoking. They constructed a Lyapunov function to prove the global stability of the unique smoking-present equilibrium state of a mathematical model of smoking. They incorporated random noise into their deterministic model. They also proved that the stochastic model established in their paper possesses non-negative solutions. They then used a Stochastic Lyapunov method to obtain the sufficient conditions for mean square and asymptotic stability in probability of the stochastic model. Their analysis indicate that the stochastic stability of the smoking present equilibrium state depends on the magnitude of the intensities of the noise as well as the parameters involved within the model system.

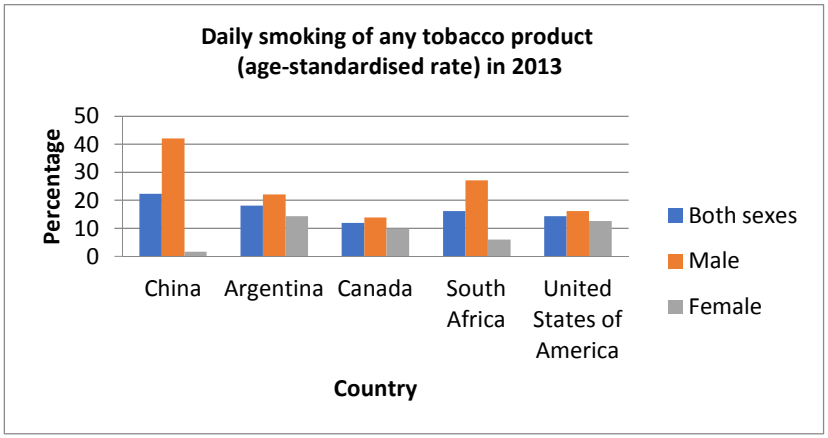


Fig. 2. Prevalence of tobacco use among adults in selected five countries—males and females, age-standardised [6]

Another model which is related to this model is that of Alkhudhari et al. [8], they presented a model to investigate the stability analysis of giving up smoking, in which smoking can be temporary or permanent. In their model, they studied a population with peer pressure effect on temporary quitters and they considered also the possibility of temporary quitters becoming permanent quitters and the impact of this transformation on the existence and stability equilibrium points. Their results show that the number of smokers may be controlled by reducing the contact rate between the potential smokers and smokers.

Also Zeb et al. [9], developed a square-root dynamics of given up smoking. Their model is made up of four compartments: potential smokers, occasional smokers, smokers and quit smokers. In their model, they considered the interaction between the potential smokers and occasional smokers in the form of a square-root followed by a finite difference scheme using the non-standard finite difference(NSFD). According to the results of their model, the NSFD method gives a highly accurate and valid approximate solution for a long time. Also the reliability of the method and reduction in the size of computational domain give this method wider applicability.

Although the previous related research discussed above considered the various ways by which smoking epidemic can be minimised, they did not consider the possibility of smokers under treatment becoming problem smokers again due to inadequate counselling. They also fail to consider recovered smokers to potential smokers compartment transfer.

In this paper, the following assumptions were made in the first model: that the problem smoker under treatment may either recover from smoking or become a problem smoker again during treatment; a problem smoker in treatment who recovers from smoking will not become a smoker again. In our second model of this paper we considered a problem smoker in treatment who recovers from smoking to become a smoker again. The paper is organized as follows: In section 2, we present the model description and the basic reproduction number. Model analysis consisting of the stability analysis of smoking- free and endemic equilibria is discussed in section 3. In Section 4, we use numerical example to show the dynamical behaviour of our results in the first model. In Section 5, we discuss smoking model with temporary immunity. In section 6, we discuss the numerical simulations and sensitivity analysis of our second model, Section 7 is made up discussion of our results. We end the paper with a conclusion in section 8.

2 Mathematical Model

2.1 Model Description

The Population of our model is divided into four compartments: non-smokers (S), problem- smokers (P), smokers-in-treatment and counselling (T_c), and removed-smokers (R). The schematic diagram below shows the interaction between the four smoking states mentioned above.

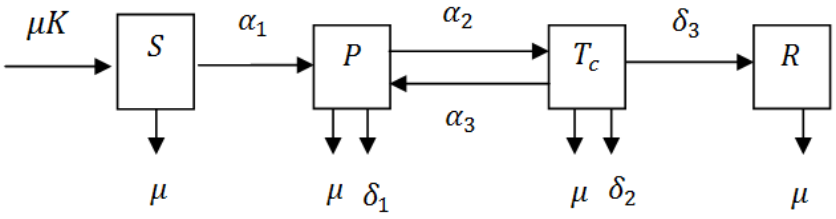


Fig. 3. Schematic diagram of the four smoking classes in the model

2.2 Model Assumptions

The following assumptions were made in the model:

(i) The smoking epidemic occurs in a closed environment.
(ii) Problem smoking is transmitted to non-smokers when they are in contact with problem Smokers.
(iii) The members of the population mix homogeneously (have the same interactions with one another to the same degree).
(iv) Smokers-in-treatment may become problem smokers again due to inadequate counselling.

The smoking epidemic is modelled using the system of nonlinear Differential Equations below:

$$\begin{aligned} \frac{dS}{dt} &= \mu K - \alpha_1 PS - \mu S \\ \frac{dP}{dt} &= \alpha_1 PS + \alpha_2 T_c - (\mu + \delta_1 + \alpha_3)P \\ \frac{dT_c}{dt} &= \alpha_3 P - \alpha_2 T_c - (\mu + \delta_2 + \delta_3)T_c \end{aligned} \tag{1}$$

$$\frac{dR}{dt} = \delta_3 T_c - \mu R$$

with $S > 0, P \geq 0, T_c \geq 0$ and $R \geq 0$.

where, μK is the recruitment rate of the population, K is the number of all individuals in the population, α_1 is the transmission rate from S to P, μ is the natural death rate, δ_1 is the smoking induced death rate of P, δ_2 is the smoking induced death rate of T_c, α_2 is the proportion of smokers entering T_c, α_3 is the proportion of smokers entering P from T_c and δ_3 is the recovered rate of T_c.

Table 1. Model parameters

Parameter	Description	Value	Source
$\boldsymbol{K}$	Number of all individuals in the population	100	**Assumption**
$\boldsymbol{\alpha_1}$	Transmission rate from S to P	0.003	**Assumption**
$\boldsymbol{\alpha_2}$	The proportion of smokers entering T_c,	0.01	**Assumption**
$\boldsymbol{\alpha_3}$	The proportion of smokers entering P from T_c	0.025	**Assumption**
$\boldsymbol{\mu}$	The natural death rate	0.02	**[1]**
$\boldsymbol{\delta_1}$	Smoking induced death rate of P	0.44	**Assumption**
$\boldsymbol{\delta_2}$	Smoking induced death rate of T_c	0.03	**[9]**
$\boldsymbol{\delta_3}$	Recovered rate of T_c.	0.02	**Assumption**
$\boldsymbol{\eta}$	Recurrence rate coefficient	0.001	**Assumption**

Since the variable R of the system (1) does not appear in the first three equations in the subsequent analysis, we only consider the system

$$\begin{aligned} \frac{dS}{dt} &= \mu K - \alpha_1 PS - \mu S \\ \frac{dP}{dt} &= \alpha_1 PS + \alpha_2 T_c - (\mu + \delta_1 + \alpha_3)P \\ \frac{dT_c}{dt} &= \alpha_3 P - \alpha_2 T_c - (\mu + \delta_2 + \delta_3)T_c \end{aligned} \tag{2}$$

We also consider the following equations:

$$N(t) = S(t) + P(t) + T_c(t)$$

and this implies that

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dP}{dt} + \frac{dT_c}{dt} \\ (S + P + T_c)' &= \mu[K - (S + P + T_c)] - \delta_1 P - (\delta_2 + \delta_3)T_c \\ &\leq \mu[K - (S + P + T_c)] \end{aligned} \tag{3}$$

From (3), it follows that:

$$\lim_{t \to \infty} Sup\ (S + P + T_c) \leq K.$$

Thus the feasible region of the system (2) is

$$\Omega = \{(S, P, T): S + P + T_c \leq K, S > 0, P \geq 0,\ T_c \geq 0\}$$

is positively invariant.

2.3 Basic reproduction number

The basic reproduction number is defined as the number of new infective individuals produced by a single infective individual during his or her infective infectious period when introduced into susceptible population [15].

Next, we investigate the basic reproduction number of the system (2) by using the next generation matrix approach [10]. It is obvious to see that the system (2) has the smoking-free equilibrium $E_0 = (K, 0, 0)$.

Let $y = (S, P, T)^T$, then system (2) can be written as

$$y' = F(y) - V(y),$$

Where

$$F(y) = \begin{bmatrix} \alpha_1 PS \\ 0 \\ 0 \end{bmatrix} \text{ and } \quad V(y) = \begin{bmatrix} (\mu + \delta_1 + \alpha_3)P \\ -\alpha_3 P + \alpha_2 T_c + (\mu + \delta_2 + \delta_3)T_c \\ -\mu K + \alpha_1 PS + \mu S \end{bmatrix}$$

The Jacobian matrices of $F(y)$ and $V(y)$ at the smoking-free equilibrium, E_0 are respectively

$$DF(E_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, DV(E_0) = \begin{bmatrix} V & 0 \\ \alpha_1 K & 0 \end{bmatrix}$$

Where

$$F = \begin{bmatrix} \alpha_1 K & 0 \\ 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu + \delta_1 + \alpha_3 & 0 \\ -\alpha_3 & \mu + \delta_2 + \delta_3 \end{bmatrix}$$

The reproduction number is given by the spectral radius of FV^{-1} and that is

$$R_0 = \frac{\alpha_1}{(\mu + \delta_1 + \alpha_3)} \tag{4}$$

Theorem 1: The smoking-free equilibrium $E_0(K, 0, 0,)$ of the system (2) is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$

3 Model Analysis

3.1 Smoking-free Equilibrium

In this section, we investigate the local geometrical properties of the smoking-free equilibrium $E_0 = (K, 0, 0)$ by considering the linearised system of ODE's (2), taking the Jacobian matrix and obtained

$$J(S, P, T) = \begin{bmatrix} -(\alpha_1 P + \mu) & -\alpha_1 S & 0 \\ 0 & \alpha_1 S - (\mu + \delta_1 + \alpha_3) & \alpha_2 \\ 0 & \alpha_3 & -\alpha_2 - (\mu + \delta_1 + \delta_3) \end{bmatrix} \tag{5}$$

The local stability of the equilibrium may be determined from the Jacobian matrix (5). This implies that the Jacobian matrix for the smoking-free equilibrium is given by

$$J(E_0) = \begin{bmatrix} -\mu & -\alpha_1 & 0 \\ 0 & \alpha_1 - (\mu + \delta_1 + \alpha_3) & \alpha_2 \\ 0 & \alpha_3 & -\alpha_2 - (\mu + \delta_2 + \delta_3) \end{bmatrix} \tag{6}$$

The determinant of (6) is given by

$$|J(E_0) - \lambda I| = \begin{vmatrix} -\mu - \lambda & -\alpha_1 & 0 \\ 0 & \alpha_1 - (\mu + \delta_1 + \alpha_3) - \lambda & \alpha_2 \\ 0 & \alpha_3 & -\alpha_2 - (\mu + \delta_2 + \delta_3) - \lambda \end{vmatrix} = 0 \tag{7}$$

It follows that the characteristic equation of $J(E_0)$ is computed from equation (7) and is given by

$$\begin{gathered} \lambda^3 + (3\mu + \delta_1 + \delta_2 + \delta_3 + \alpha_2 + \alpha_3 - \alpha_1)\lambda^2 + \\ (3\mu^2 + 2\alpha_2\mu + 2\alpha_3\mu + 2\delta_1\mu + 2\delta_2\mu + 2\delta_3\mu - 2\alpha_1\mu + \alpha_3\delta_2 + \alpha_3\delta_3 + \alpha_2\delta_1 + \delta_1\delta_2 + \delta_1\delta_3 \\ -\alpha_1\alpha_2 - \alpha_1\delta_2 - \alpha_1\delta_3)\lambda + (\mu^3 + \alpha_2\mu^2 + \delta_3\mu^2 + \delta_2\mu^2 + \alpha_3\mu^2 + \delta_1\mu^2 - \alpha_1\mu^2 + \alpha_3\delta_2\mu + \alpha_3\delta_3\mu \\ +\alpha_2\delta_1\mu + \delta_1\delta_2\mu + \delta_1\delta_3\mu - \alpha_1\alpha_2\mu - \alpha_1\delta_2\mu - \alpha_1\delta_3\mu) = 0 \end{gathered}$$

We can write the characteristic equation above as:

$$\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0 \tag{8}$$

Where

$$\begin{gathered} \boldsymbol{b_1} = 3\mu + \delta_1 + \delta_2 + \delta_3 + \alpha_2 + \alpha_3 - \alpha_1 \\ \boldsymbol{b_2} = 3\mu^2 + 2\alpha_2\mu + 2\alpha_3\mu + 2\delta_1\mu + 2\delta_2\mu + 2\delta_3\mu - 2\alpha_1\mu + \alpha_3\delta_2 + \alpha_3\delta_3 + \alpha_2\delta_1 + \delta_1\delta_2 \\ +\delta_1\delta_3 - \alpha_1\alpha_2 - \alpha_1\delta_2 - \alpha_1\delta_3 \\ \boldsymbol{b_3} = \mu^3 + \alpha_2\mu^2 + \delta_3\mu^2 + \delta_2\mu^2 + \alpha_3\mu^2 + \delta_1\mu^2 - \alpha_1\mu^2 + \alpha_3\delta_2\mu + \alpha_3\delta_3\mu + \alpha_2\delta_1\mu + \delta_1\delta_2\mu \\ +\delta_1\delta_3\mu - \alpha_1\alpha_2\mu - \alpha_1\delta_2\mu - \alpha_1\delta_3\mu \\ \boldsymbol{b_1b_2 - b_3} = (9 + 8\alpha_2 + 8\alpha_3 + 8\delta_1 + 8\delta_2 + 8\delta_3 - 8\alpha_1 - \mu)\mu^2 + (6\alpha_3\delta_2 + 6\alpha_3\delta_3 + 6\alpha_2\delta_1 + 6\delta_1\delta_2 \\ +6\delta_1\delta_3 - 6\alpha_1\alpha_2 - 6\alpha_1\delta_2 - 6\alpha_1\delta_3 + 4\alpha_3\delta_1 + {\delta_1}^2 - 4\alpha_1\delta_1 + 4\alpha_2\delta_2 + 2{\delta_2}^2 + 4\delta_2\delta_3 + 4\alpha_2\delta_3 + \\ 2{\delta_3}^2 + 2{\alpha_2}^2 + 4\alpha_2\alpha_3 + 2{\alpha_3}^2 - 4\alpha_1\alpha_3 + 2{\alpha_1}^2)\mu + (2\delta_1\delta_2\alpha_3 + 2\delta_1\delta_3\alpha_3 + {\delta_1}^2\alpha_2 + {\delta_1}^2\delta_2 + {\delta_1}^2\delta_3 \\ -2\alpha_1\alpha_2\delta_1 - 2\alpha_1\delta_1\delta_2 + \delta_1{\delta_2}^2 + 2\delta_1\delta_2\delta_3 - 2\alpha_1\alpha_2\delta_2 - \alpha_1{\delta_2}^2 - 2\alpha_1\delta_2\delta_3 + \alpha_3\delta_2\delta_3 + \alpha_3{\delta_3}^2 \\ +2\alpha_2\delta_1\delta_3 + \delta_1{\delta_3}^2 - 2\alpha_1\alpha_2\delta_3 - \alpha_1{\delta_3}^2 + \alpha_2\alpha_3\delta_2 + \alpha_2\alpha_3\delta_3 + {\alpha_2}^2\delta_1 - \alpha_1{\alpha_2}^2 + {\alpha_3}^2\delta_2 + {\alpha_3}^2\delta_3 + \alpha_2\alpha_3\delta_1 \\ -\alpha_1\alpha_2\alpha_3 - 2\alpha_1\alpha_3\delta_2 - 2\alpha_1\alpha_3\delta_3 + {\alpha_1}^2\alpha_2 + {\alpha_1}^2\delta_2 + {\alpha_1}^2\delta_3) \end{gathered}$$

Using the Routh-Hurwitz criterion [14], it can be seen that all the eigenvalues of the characteristic equation (8) have negative real part if and only if:

$$b_1 > 0,\ b_2 > 0, b_3 > 0,\ b_1b_2 - b_3 > 0 \tag{9}$$

Theorem 2: E_0 is asymptotically stable if and only if inequalities (9) is satisfied.

3.2 Existence of endemic equilibrium

In this section, we consider a situation in which all the smoking states coexist in the equilibrium. We denote $E^* = (S^*, P^*, {T_c}^*)$ as the endemic equilibrium of the system (2). We also obtain

$$S^* = \frac{(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)-\alpha_2\alpha_3}{\alpha_2(\alpha_2+\mu+\delta_2+\delta_3)}$$
$$P^* = \frac{\mu K\alpha_1(\alpha_2+\mu+\delta_2+\delta_3)-\mu[(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)-\alpha_2\alpha_3]}{(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)[(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)-\alpha_2\alpha_3]}$$
$$T_c{}^* = \frac{\alpha_3[\mu K\alpha_1(\alpha_2+\mu+\delta_2+\delta_3)-\mu[(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)-\alpha_2\alpha_3]]}{(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)[(\mu+\alpha_3+\delta_1)(\alpha_2+\mu+\delta_2+\delta_3)-\alpha_2\alpha_3]}$$

from system of ODE's (2) and linearized the same system to obtained:

$$J(E^*) = \begin{bmatrix} -(\alpha_1P^*+\mu) & -\alpha_1S^* & 0 \\ 0 & \alpha_1S^*-(\mu+\delta_1+\alpha_3) & \alpha_2 \\ 0 & \alpha_3 & -\alpha_2-(\mu+\delta_1+\delta_3) \end{bmatrix} \tag{10}$$

We determine the local stability of the positive equilibrium E^*, by using the following lemma.

Lemma 1 [11,14]: Let M be a 3×3 real matrix. If $tr(M)$, $det\ (M)$ and $det\ (M^{[2]})$ are all negative, then all the eigenvalues of M have negative real part.

Definition 1 [8,11,14] (Second additive compound matrix). Let A be a real $m\times m$ matrix. The second additive compound matrix of $A=(a_{ij})$ for $m=3$ is defined as

$$A^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix} \tag{11}$$

Theorem 3: The positive equilibrium E^* of the system (2) is locally asymptotically stable if $R_0>1$.

Proof: We first construct a second additive compound matrix $J^{[2]}(E^*)$ of $J(E^*)$ and obtain

$$J^{[2]}(E^*) = \begin{bmatrix} \alpha_1(S^*-P^*)-2\mu-\delta_1-\alpha_3 & \alpha_2 & 0 \\ \alpha_3 & -(\alpha_1P^*+\mu)-\alpha_2+c_1 & -\alpha_1S^* \\ 0 & 0 & \alpha_1S^*-c_2-\alpha_2+c_1 \end{bmatrix} \tag{12}$$

Where $c_1=\mu+\delta_1+\delta_3$ and $c_2=\mu+\alpha_3+\delta_1$.

It follows that:

$$tr(J(E^*)) = -(\alpha_1P^*+\mu)+\alpha_1S^*-(\mu+\delta_1+\alpha_3)-\alpha_2-(\mu+\delta_1+\delta_3)<0,$$

If $(\mu+\alpha_3+\delta_1)>\alpha_1S^*$.

$$\det(J(E^*)) = -(\alpha_1P^*+\mu)\big[(\alpha_1S^*-(\mu+\alpha_3+\delta_1))\big(-\alpha_2-(\mu+\delta_1+\delta_2)\big)-\alpha_2\alpha_3]<0$$

If $\big[(\alpha_1S^*-(\mu+\alpha_3+\delta_1))\big(-\alpha_2-(\mu+\delta_1+\delta_2)\big)\big]>\alpha_2\alpha_3$.

Next, we compute the determinant of $J^{[2]}(E^*)$ in (12) and obtained

$$\det[J^{[2]}(E^*)] = -[\alpha_1P^*-\alpha_1S^*+2\mu+\alpha_3+\delta_1][\alpha_1P^*+2\mu+\alpha_2+\delta_1+\delta_3][-\alpha_1S^*+\alpha_2+c_1+c_2]$$

$\det[J^{[2]}(E^*)]<0$, if $(2\mu+\alpha_3+\delta_1)>\alpha_1S^*$ and $(\alpha_2+c_1+c_2)>\alpha_1S^*$. Note that if $R_0>1$, then $(2\mu+\alpha_3+\delta_1)>\alpha_1S^*$ and $(\alpha_2+c_1+c_2)>\alpha_1S^*$. Thus $\det[J^{[2]}(E^*)]<0$. This completes the proof.

3.3 Global stability of the equilibrium points

3.3.1 Global stability of the smoking free equilibrium

We prove the global stability when $\alpha_1 \leq \mu$.

Theorem 4: The global stability of the smoking free equilibrium, E_0 is asymptotically stable in the region $\Omega = \{(S,P,T): S+P+T_c \leq K, S>0, P \geq 0, T \geq 0\}$ if $\alpha_1 \leq \mu$ (note that $\alpha_1 \leq \mu$ implies $R_0 < 1$).

Proof: it should be noted that $S < 1$ in Ω for time $(t) > 1$. Consider the Lyaponov function:

$$\begin{aligned} L &= P + T_c \\ \frac{dL}{dt} &= \alpha_1 PS - (\mu+\delta_1)P - (\mu+\delta_2+\delta_3)T_c \\ &\leq (\alpha_1 - \mu + \delta_1)P - (\mu+\delta_2)T_c \end{aligned} \tag{13}$$

$\frac{dL}{dt} < 0$ for $\alpha_1 \leq \mu$ and $\frac{dL}{dt} = 0$ if $(\alpha_1 - \mu + \delta_1)P - (\mu+\delta_2)T_c = 0$. Therefore, the only trajectory of the system in which $\frac{dL}{dt} = 0$ is E_0. Hence, Lasalle's invariance principle, E_0 is globally asymptotically stable in Ω [8,12].

3.3.2 Global stability analysis of endemic equilibrium (E^*)

We investigate the global stability of the endemic equilibrium E^* in this section, by using **Lemma 2** to prove that the system (2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region.

Lemma 2: Let $g(S,P,T_c) = \{g_1(S,P,T_c), g_2(P,S,T_c), g_3(S,P,T_c)\}$ be a vector field on Ω^* and which satisfies the conditions $f = 0, (\nabla \times g).\vec{n} < 0$, in the interior of Ω^*, where $\vec{n}$ is the normal vector to Ω^* and $f = (f_1, f_2, f_3)$ is a Lipschitz continuous field in the interior of Ω^*, and

$$\nabla \times g = \begin{vmatrix} i & j & k \\ \frac{\partial}{\partial S} & \frac{\partial}{\partial P} & \frac{\partial}{\partial T_c} \\ g_1 & g_2 & g_3 \end{vmatrix}$$

$$\nabla \times g = \begin{vmatrix} \frac{\partial}{\partial P} & \frac{\partial}{\partial T_c} \\ g_2 & g_3 \end{vmatrix} \underline{i} - \begin{vmatrix} \frac{\partial}{\partial S} & \frac{\partial}{\partial T_c} \\ g_1 & g_3 \end{vmatrix} \underline{j} + \begin{vmatrix} \frac{\partial}{\partial S} & \frac{\partial}{\partial P} \\ g_1 & g_2 \end{vmatrix} \underline{k}$$

$$= \left(\frac{\partial g_3}{\partial P} - \frac{\partial g_2}{\partial T_c}\right)\underline{i} - \left(\frac{\partial g_3}{\partial S} - \frac{\partial g_1}{\partial T_c}\right)\underline{j} + \left(\frac{\partial g_2}{\partial S} - \frac{\partial g_1}{\partial P}\right)\underline{k} \tag{14}$$

Thus, the differential equation of the system $S = f_1, P = f_2, T_c = f_3$ has no periodic solutions, homoclinic loops and oriented phase polygons inside Ω^*.

We consider $\Omega^* = \{(S,P,T_c): S + \left(\frac{\mu+\delta_1}{\mu}\right)P + \left(\frac{\mu+\delta_2+\delta_3}{\mu}\right)T_c = K, S>0, P \geq 0, T_c \geq 0\}$. Then $\Omega^* \subset \Omega$, Ω^* is positively invariant and Ω^* and $E^* \in \Omega^*$.

Theorem 5: The system (2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Ω^*

Proof: Let f_1, f_2 and f_3 represent the right-hand side of equations in model (2), respectively. We use $S + \frac{(\mu+\delta_1)}{\mu} + \frac{(\mu+\delta_2+\delta_3)}{\mu} = K$ to rewrite f_1, f_2 and f_3 inequivalent forms and obtain:

$$f_1(S,P) = \mu K - \alpha_1 PS - \mu S \tag{15}$$

$$f_1(S,T_c) = \mu K - \alpha_1 S\left[K - S - \left(\frac{\mu+\delta_2+\delta_3}{\mu}\right)T_c\right]\left(\frac{\mu}{\mu+\delta_1}\right) - \mu S \tag{16}$$

$$f_2(S,P) = \alpha_1 PS + \alpha_2\left[K - S - \left(\frac{\mu+\delta_1}{\mu}\right)P\right]\left(\frac{\mu}{\mu+\delta_2+\delta_3}\right) - (\mu+\delta_1+\alpha_3)P \tag{17}$$

$$f_2(P,T_c) = \alpha_1 P\left[K - \left(\frac{\mu+\delta_1}{\mu}\right)P - \left(\frac{\mu+\delta_2+\delta_3}{\mu}\right)T_c\right] + \alpha_2 T_c - (\mu+\delta_1+\alpha_3)P \tag{18}$$

$$f_3(S,T_c) = \alpha_3\left[K - S - \left(\frac{\mu+\delta_2+\delta_3}{\mu}\right)T_c\right]\left(\frac{\mu}{\mu+\delta_1}\right) - \alpha_2 T_c - (\mu+\delta_2+\delta_3)T_c \tag{19}$$

$$f_3(P,T_c) = \alpha_3 P - \alpha_2 T_c - (\mu+\delta_2+\delta_3)T_c \tag{20}$$

Let $g = (g_1, g_2, g_3)$, denote a vector field, where

$$g_1 = \frac{f_3(S,T_c)}{ST_c} - \frac{f_2(S,P)}{SP} =$$
$$= \frac{\alpha_3\mu K}{ST_c(\mu+\delta_1)} - \left[\frac{\mu+\delta_2+\delta_3}{S(\mu+\delta_1)} + \frac{\alpha_2}{S} + \frac{(\delta_2+\delta_3)}{S} - \frac{\alpha_2(\mu+\delta_1)}{S(\mu+\delta_2+\delta_3)} - \frac{(\delta_1+\alpha_3)}{S}\right]$$
$$-\frac{\alpha_2\mu K}{(\mu+\delta_2+\delta_3)SP} + \frac{\alpha_2\mu}{(\mu+\delta_2+\delta_3)P} - \left(\frac{\alpha_3\mu}{\mu+\delta_1} + \alpha_1\right) \tag{21}$$

$$g_2 = \frac{f_1(S,P)}{SP} - \frac{f_3(P,T_c)}{PT_c} = \frac{\mu K}{PS} - \frac{\alpha_3}{T_c} + \frac{1}{P}[\alpha_2+\delta_2+\delta_3] - \alpha_1 \tag{22}$$

$$g_3 = \frac{f_2(P,T_c)}{PT_c} - \frac{f_1(S,T_c)}{ST_c} = \frac{\alpha_1 K}{T_c} - \frac{\alpha_1(\mu+\delta_1)P}{\mu T_c} - \frac{\alpha_1(\mu+\delta_2+\delta_3)}{\mu} + \frac{\alpha_2}{P} - \left(\frac{\mu+\delta_1+\alpha_3}{T_c}\right)$$
$$-\frac{\mu K}{ST_c} - \frac{\alpha_1\mu K}{(\mu+\delta_1)T_c} - \frac{\alpha_1\mu S}{(\mu+\delta_1)T_c} - \frac{\alpha_1(\mu+\delta_2+\delta_3)}{\mu+\delta_1} - \frac{\mu}{T_c} \tag{23}$$

$$g.f = g_1 f_1 + g_2 f_2 + g_3 f_3$$
$$g.f = \left(\frac{f_3}{ST_c} - \frac{f_2}{SP}\right)f_1 + \left(\frac{f_1}{SP} - \frac{f_3}{PT_c}\right)f_2 + \left(\frac{f_2}{PT_c} - \frac{f_1}{ST_c}\right)f_3$$
$$= \frac{f_3 f_1}{ST_c} - \frac{f_2 f_1}{SP} + \frac{f_1 f_2}{SP} - \frac{f_3 f_2}{PT_c} + \frac{f_2 f_3}{PT_c} - \frac{f_1 f_3}{ST_c} = 0 \tag{24}$$

on Ω^*. Since the alternate forms of f_1, f_2 and f_3 are equivalent in Ω^*.

From **lemma 2**, it is easy to see that

$$\nabla \times g = \left(-\frac{\alpha_1(\mu+\delta_1)}{\mu T_c} - \frac{\alpha_2}{P^2} - \frac{\alpha_3}{{T_c}^2}\right)\underline{i} - \left(\frac{\mu K}{S^2 T_c} - \frac{\alpha_1\mu}{(\mu+\delta_1)T_c} + \frac{\alpha_3 K\mu}{{T_c}^2(\mu+\delta_1)}\right)\underline{j}$$
$$+\left(-\frac{\mu K}{PS^2} - \frac{\alpha_2 K\mu}{(\mu+\delta_2+\delta_3)SP^2} + \frac{\alpha_3\mu}{(\mu+\delta_2+\delta_3)P^2}\right)\underline{k}. \tag{25}$$

Using $\vec{n} = \left(\frac{1}{K}, \frac{\mu+\delta_1}{\mu K}, \frac{\mu+\delta_2+\delta_3}{\mu K}\right)$ to Ω^*, it can be shown that,

$$(\nabla \times g).\vec{n} = -\frac{1}{K}\left(\frac{\alpha_1(\mu+\delta_1)}{\mu T_c}+\frac{\alpha_2}{P^2}+\frac{\alpha_3}{{T_c}^2}\right)-\left(\frac{K}{S^2T_c}-\frac{\alpha_1}{(\mu+\delta_1)T_c}+\frac{\alpha_3 K}{S{T_c}^2(\mu+\delta_1)}\right)\left(\frac{\mu+\delta_1}{K}\right)$$
$$-\left(\frac{K}{PS^2}-\frac{\alpha_2 K}{(\mu+\delta_2+\delta_3)SP^2}+\frac{\alpha_2}{(\mu+\delta_2+\delta_3)P^2}\right)\left(\frac{\mu+\delta_2+\delta_3}{K}\right)<0. \quad (26)$$

Since $S+\left(\frac{\mu+\delta_1}{\mu}\right)P+\left(\frac{\mu+\delta_2+\delta_3}{\mu}\right)T_c=K$. Thus, by lemma 2, the system (2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Ω^*[8,12].

Theorem 5: The endemic equilibrium point E^* of model (2) is globally asymptotically stable if $R_0>1$(This means that $\alpha_2 \leq \alpha_1$).

Proof: From **theorem 1**, if $R_0>1$ in Ω^*, then E_0 is unstable. Also $\Omega^*$is positively invariant subset of Ω and the ω-limit set of each solution of model (2) is a single point in Ω^*since there is no periodic solutions, homoclinic loops and oriented phase polygons inside Ω^*if $\alpha_2 \leq \alpha_1$. Therefore E^* is globally asymptotically stable [8].

4 Numerical Simulations

In this section, we use numerical simulations to show the dynamical behavior of our model. Then we carry out some sensitivity analysis of the basic reproduction number using the model parameters. The parameter values used in this section are displayed in Table 1.

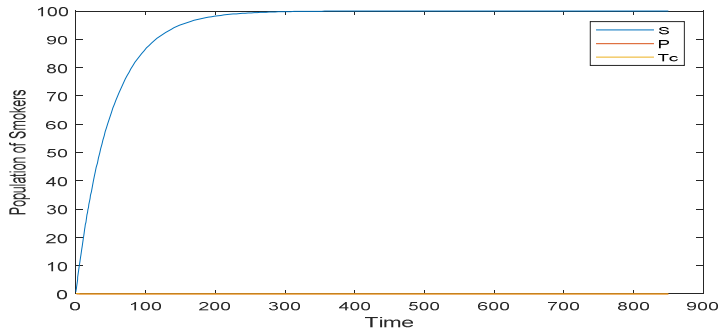


Fig. 4. Time series plot of model 2, with initial parameter values $K=100, \alpha_1=0.003, \alpha_2=0.01, \mu=0.02, \delta_1=0.44, \delta_2=0.03, \delta_3=0.02, \alpha_3=0.025$ when $R_0<1$. Only non-smokers are present in the population. The populations of problem smokers and smokers in treatment approach zero and reach disease free equilibrium.

4.1 Sensitivity of the reproduction number

We study how the R_0 depends on the model parameters especially α_1 and α_3, that is the transmission rate coefficient and the coefficient of the proportion of smokers entering P from T_c. We consider the following cases:

i. At Fig. 4 (which depicts the graph of disease free equilibrium), we increased the value of $\alpha_1$from 0.003 to 0.55 and obtained Fig. 5 (which depicts graph of endemic equilibrium).
ii. At Fig. 5 we increased the value of α_3 from 0.025 to 48 and obtained the graph in Fig. 6 (which depicts graph of disease free- equilibrium).

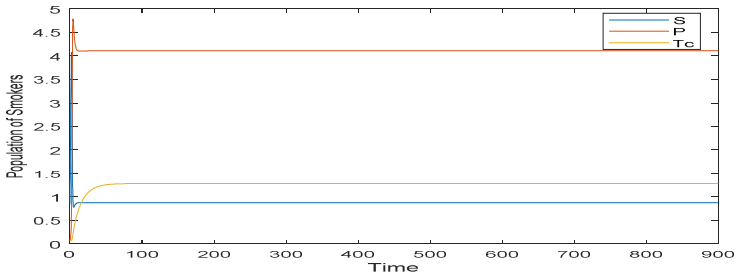


Fig. 5. Time series plot of model 2, with parameter values $K = 100, \alpha_1 = 0.55,\ \alpha_2 = 0.01, \mu = 0.02, \delta_1 = 0.44,\ \delta_2 = 0.03, \delta_3 = 0.02, \alpha_3 = 0.025$ when $R_0 = 1.13402$. All the distinct smoking classes coexist and therefore approach endemic equilibrium

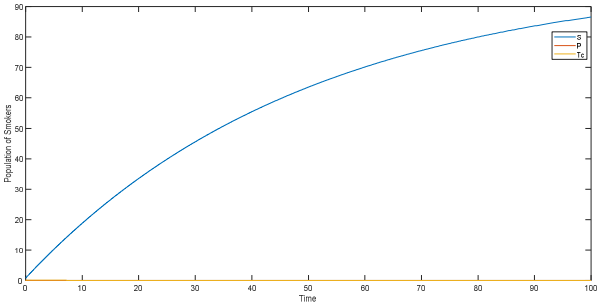


Fig. 6. Time series plot of model 2, with parameter values $K = 100, \alpha_1 = 0.55,\ \alpha_2 = 0.01, \mu = 0.02, \delta_1 = 0.44,\ \delta_2 = 0.03, \delta_3 = 0.02,\ \alpha_3 = 48$, when $R_0 = 0.01135$. Only non-smokers are present in the population. The populations of problem-smokers and smokers-in-treatment approach zero and reach disease free equilibrium.

5 SPT$_c$R Smoking Model with Temporal Immunity

In this section, we assume that problem smokers who have stopped smoking enter into recovery compartment after treatment and become problem smokers again.

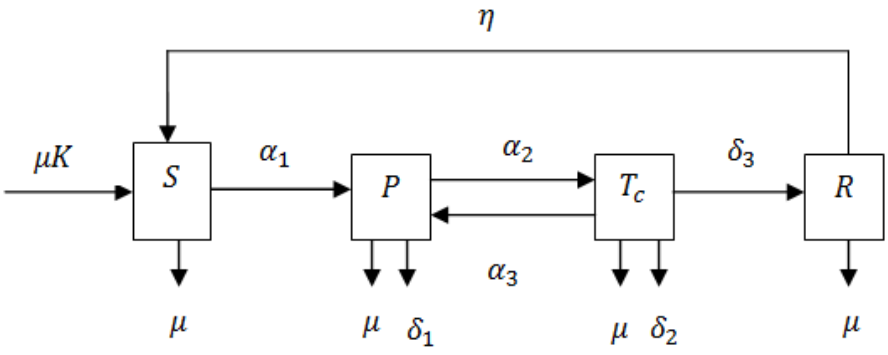


Fig. 7. Schematic diagram of the four-smoking model with temporal immunity

Therefore, our model equations become:

$$\begin{aligned}
\frac{dS}{dt} &= \mu K + \eta R - \alpha_1 PS - \mu S \\
\frac{dP}{dt} &= \alpha_1 PS + \alpha_2 T_c - (\mu + \delta_1 + \alpha_3)P \\
\frac{dT_c}{dt} &= \alpha_3 P - \alpha_2 T_c - (\mu + \delta_2 + \delta_3)T_c \\
\frac{dR}{dt} &= \delta_3 T_c - (\mu + \eta)R
\end{aligned} \tag{27}$$

with initial conditions $S > 0$, $P \geq 0$, $T_c \geq 0$ and $R \geq 0$ and

$$\begin{aligned}
\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dP}{dt} + \frac{dT_c}{dt} + \frac{dR}{dt} \\
&(S + P + T_c + R)' = \mu[K - (S + P + T_c)] - \delta_1 P - (\delta_2 + \delta_3)T_c \\
&\leq \mu[K - (S + P + T_c + R)]
\end{aligned} \tag{28}$$

From (28), it follows that:

$$\lim_{t \to \infty} Sup\ (S + P + T_c + R) \leq K.$$

Thus, the feasible region of the system (27) is

$$\Omega^{**} = \{(S, P, T_c, R): S + P + T_c + R \leq K, S > 0, P \geq 0,\ T_c \geq 0, R \geq 0\}$$

is positively invariant.

5.1 Model Analysis

5.1.1 Smoking- free equilibrium and the basic reproduction number

In this section, we study the basic properties of the model (27). We first find the smoking- free equilibrium and then continue with the reproduction number. We denote the smoking-free equilibrium by $E^0 = (S, 0,0,0)$ and consider the linearized system of the system of ODE's (27), by taking the Jacobian matrix under E^0 and obtain

$$J(E^0) = \begin{bmatrix} -\mu & -\alpha_1 & 0 & \eta \\ 0 & \alpha_1 - (\mu + \delta_1 + \alpha_3) & \alpha_2 & 0 \\ 0 & \alpha_3 & -\alpha_3 - (\mu + \delta_2 + \delta_3) & 0 \\ 0 & 0 & \delta_3 & -(\mu + \eta) \end{bmatrix} \tag{29}$$

The eigenvalues of the characteristic equation of $J(E^0)$ are $\lambda_1 = -\mu$, $\lambda_2 = -(\mu + \eta)$ and the solution of the cubic equation

$$\lambda^2 + a_1\lambda + a_2 = 0 \tag{30}$$

where

$$\begin{aligned}
a_1 &= 2\alpha_3 + 2\mu + \delta_2 + \delta_3 + \delta_1 - \alpha_1, \\
a_2 &= 2\alpha_3\mu + \alpha_3\delta_1 + {\alpha_3}^2 + \mu^2 + \mu\delta_1 + \mu\delta_2 + \mu\delta_3 + \delta_1\delta_2 + \delta_1\delta_3 + \delta_2\alpha_3 + \alpha_3\delta_3 - \alpha_1\alpha_3 \\
&\quad -\alpha_1\delta_2 - \alpha_1\delta_3 - \alpha_2\alpha_3, \\
a_1 a_2 &= 4{\alpha_3}^2\mu + 2{\alpha_3}^2\delta_1 + 2{\alpha_3}^3 + 2\alpha_3\mu^2 + 2\alpha_3\mu\delta_1 + 2\alpha_3\delta_2\mu + 2\alpha_3\delta_3\mu + 2\alpha_3\delta_1\delta_2
\end{aligned}$$

$$+2\alpha_3\delta_1\delta_3 + 2\alpha_3{}^2\delta_2 + 2\alpha_3{}^2\delta_3 - 2\alpha_1\alpha_3{}^2 - 2\alpha_1\alpha_3\delta_2 - 2\alpha_1\alpha_3\delta_3 - 2\alpha_2\alpha_3{}^3 + 2\alpha_3\mu^2$$
$$+2\mu\alpha_3\delta_1 + 2\mu\alpha_3{}^2 + 2\mu^3 + 2\mu^2\delta_1 + 2\delta_2\mu^2 + 2\delta_3\mu^2 + 2\mu\delta_1\delta_2 + 2\mu\delta_1\delta_3 + 2\mu\delta_2\alpha_3$$
$$+2\mu\alpha_3\delta_3 - 2\mu\alpha_1\alpha_3 - 2\mu\alpha_1\delta_2 - 2\mu\alpha_1\delta_3 - 2\mu\alpha_2\alpha_3 + 2\mu\delta_2\alpha_3 + \delta_1\delta_2\alpha_3 + \delta_2\alpha_3{}^2 + \delta_2\mu^2$$
$$+\delta_1\delta_2\mu + \delta_2{}^2\mu + \delta_2\delta_3\mu + \delta_1\delta_2{}^2 + 2\delta_1\delta_2\delta_3 + \delta_2{}^2\alpha_3 + \delta_2\delta_3\alpha_3 - \delta_2\alpha_1\alpha_3 - \delta_2{}^2\alpha_1 - \alpha_1\delta_2\delta_3$$
$$-\delta_2\alpha_2\alpha_3 + 2\delta_3\alpha_3\mu + \alpha_3\delta_1\delta_3 + \delta_3\alpha_3{}^2 + \delta_3\mu^2 + \delta_1\delta_3\mu + \delta_2\delta_3\mu + \delta_3\mu + \delta_1\delta_3{}^2 + \delta_2\delta_3\alpha_3$$
$$+\delta_3{}^2\alpha_3 - \alpha_1\alpha_3\delta_3 - \alpha_1\delta_2\delta_3 - \alpha_1\delta_3{}^2 - \alpha_2\alpha_3\delta_3 + 2\delta_1\alpha_3\mu + \delta_1{}^2\alpha_3 + \delta_1\alpha_3{}^2 + \delta_1\mu^2 + \delta_1{}^2\mu$$
$$+\delta_1\delta_2\mu + \delta_1\delta_3\mu + \delta_1{}^2\delta_2 + \delta_1{}^2\delta_3 + \delta_1\delta_2\alpha_3 + \delta_1\delta_3\alpha_3 - \delta_1\alpha_1\alpha_3 - \delta_1\delta_2\alpha_1 - \delta_1\delta_3\alpha_1 - \delta_1\alpha_2\alpha_3$$
$$-2\alpha_1\alpha_3\mu - \alpha_1\alpha_3\delta_1 - \alpha_1\alpha_3{}^2 - \alpha_1\mu^2 - \alpha_1\delta_1\mu - \alpha_1\delta_2\mu - \alpha_1\delta_3\mu - \alpha_1\delta_1\delta_2 - \alpha_1\delta_1\delta_3 - \alpha_1\alpha_3\delta_2$$
$$-\alpha_1\alpha_3\delta_3 + \alpha_1{}^2\alpha_3 + \alpha_1{}^2\delta_2 + \alpha_1{}^2\delta_3 + \alpha_1\alpha_2\alpha_3.$$

Using the Routh-Hurwitz criterion [13], it can be seen that all eigenvalues of the characteristics equation (30) has negative real part if and only if

$$a_1 > 0,\ a_2 > 0,\ \ a_3 > 0,\ a_1a_2 - a_3 > 0 \tag{31}$$

Theorem 1: E^0 is locally asymptotically stable if and only if inequalities (31) is satisfied.

The basic reproduction number of the Smoking Model with Temporal Immunity (SPT_cR) is the same as that of our first model and is given by

$$R_0 = \frac{\alpha_1}{(\mu+\delta_1+\alpha_3)}.$$

5.1.2 Endemic equilibrium

We denote the endemic equilibrium, $E^1 = (S^1, P^1, T_c{}^1, R^1)$ and evaluate the equilibrium points of the model by setting the left-hand side of system (28), equal to zero, solve for the equilibrium points and obtain the following

$$S^1 = \frac{\mu k(\mu+\eta)(\alpha_2+\mu+\delta_2+\delta_3) + \eta\delta_3\alpha_3)}{(\mu+\eta)(\alpha_2+\mu+\delta_2+\delta_3)(\alpha_1P^1+\mu)}$$
$$T_c{}^1 = \frac{\alpha_3P^1}{(\alpha_2+\mu+\delta_2+\delta_3)}$$
$$R^1 = \frac{\delta_3}{(\mu+\eta)}\frac{\alpha_3P^1}{(\alpha_2+\mu+\delta_2+\delta_3)}$$

The local stability of the endemic equilibrium is determined from the Jacobian matrix $J(E^1)$ below

$$J(E^1) = \begin{bmatrix} -\mu & -\alpha_1S^1 & 0 & \eta \\ \alpha_1P^1 & \alpha_1S^1 - (\mu+\delta_1+\alpha_3) & \alpha_2 & 0 \\ 0 & \alpha_3 & -\alpha_2-(\mu+\delta_2+\delta_3) & 0 \\ 0 & 0 & \delta_3 & -(\mu+\eta) \end{bmatrix} \tag{32}$$

The characteristic $J(E^1)$ is given by

$$\lambda^4 + B_1\lambda^3 + B_2\lambda^2 + B_3\lambda + B_4 = 0 \tag{33}$$

Where

$$D_1 = -a_{11} - a_{22} - a_{33} - a_{44},$$
$$D_2 = a_{11}a_{22} + a_{11}a_{33} + a_{11}a_{44} + a_{33}a_{44} + a_{22}a_{44} - a_{12}a_{21},$$
$$D_3 = a_{12}a_{21}a_{33} + a_{12}a_{21}a_{44} - a_{11}a_{22}a_{44} - a_{22}a_{33}a_{44},$$
$$D_4 = -a_{12}a_{21}a_{33}a_{44}.$$

And

$$a_{11} = -\mu,\ a_{12} = -\alpha_1 S^1,\ a_{14} = \eta,\ a_{21} = \alpha_1 P^1,\ a_{22} = \alpha_1 S^1 - (\mu + \delta_1 + \alpha_3),\ a_{23} = \alpha_2,$$
$$a_{32} = \alpha_3, a_{33} = -\alpha_2 - (\mu + \delta_2 + \delta_3), a_{43} = \delta_3, a_{44} = -(\mu + \eta).$$

It follows from Routh-Hurtwitz criteria [14] that all the eigenvalues associated to $J(E^1)$ have negative real parts if only if $D_i > 0$ for $i = 1, 2, 3, 4$ and

$$D_1 D_2 D_3 > {D_3}^2 + {D_1}^2 D_4 \tag{34}$$

Thus, the system (27) is locally asymptotically stable if $R_0 > 1$ and the condition (34) is satisfied.

6 Numerical Simulations and Sensitivity Analysis

In order to see the dynamical behaviour of our model, we performed numerical Simulation in this section using the parameters in Table 1. Sensitivity Analysis of model (27) and that of the basic reproduction number were also performed.

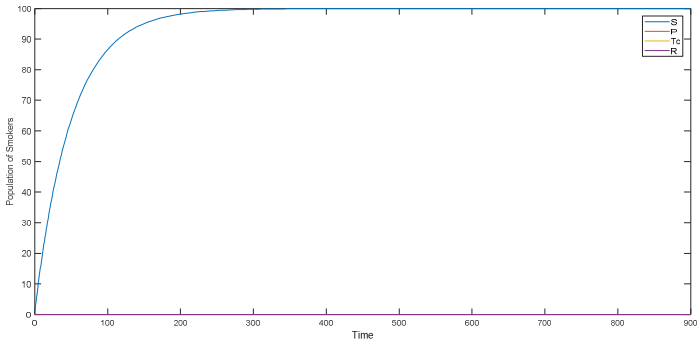


Fig. 8. Time series plot of model (28) with parameter values $K = 100, \mu = 0.02, \alpha_1 = 0.003,\ \alpha_2 = 0.01, \alpha_3 = 0.025, \delta_1 = 0.44, \delta_2 = 0.03, \delta_3 = 0.02, \eta = 0.001$ when $R_0 < 1$. Only non-smokers are present in the population. The populations of problem smokers and smokers in treatment approach zero and reach disease free equilibrium.

6.1 Sensitivity analysis

Sensitivity Analysis of system (28) with respect to $\alpha_1 = 0.003$, $\delta_1 = 0.44$ and $\eta = 0.001$ are displayed in Figs. 9 and 10 respectively.

7 Discussion

We studied two simple mathematical models capturing the transmission dynamics of smoking epidemic. The existence and stability of smoking-free and endemic equilibria and the sensitivity analysis of the reproductive number of both models were performed. Based on our parameter values, the basic reproductive number of the smoking-free equilibrium is estimated to be $R_0 = 0.006186 < 1$. This implies that only non-smokers population is present and the problem smokers and smokers-in- treatment population reduces to zero in both models. This means that our models are asymptotically stable at $R_0 < 1$ and satisfies Theorem1. This has been verified numerically in Figures 4 and 8. At the sensitivity analysis of the basic reproductive number, if the value of α_1 is increased from 0.003 to 0.55 and 0.003 to 2 in our first and second models respectively, $R_0 > 1$. This indicates the existence of smoking problem in the population. People with

smoking problem will continue to transform more non-smokers into problem-smokers and the smoking-free equilibrium becomes unstable at $R_0 > 1$. This situation has been verified numerically in Fig. 5. Also, if the value of α_3 is increased from 0.025 to 48, and μ, δ_1, α_1 maintained the same in our first model, $R_0 < 1$ and the situation is reversed. This situation is also in line with our numerical results in Fig. 6 in our first model. It was observed in our second model that $R_0 < 1$ whenever the value of η is either increased or decreased. This implies that η has no significant impact on the model. However, when the value of δ_1 is reduced from 0.44 to 0.1 the situation is reversed in Fig. 11 and all the distinct smoking classes reappear in the population. Fig. 12 shows the relationship between our basic reproduction number with respect to the parameters α_1, μ, δ_1 and α_3 in the models. A Lyapunov function is used to prove the global stability of the smoking-free equilibrium when the transmission rate between non-smokers and problem smokers is less than or equal to the natural death rate ($\alpha_1 \leq \mu$) in our first model. This indicates that the smoking epidemic can be controlled by reducing α_1 to be less than μ. On the other hand, if $\alpha_1 \geq \mu$ and $\mu \geq \delta_3$ then the endemic equilibrium state is locally asymptotically stable. In order to show that our model has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Ω^*, we used our first model to prove that the global asymptotic stability of the endemic equilibrium for $\alpha_1 \geq \alpha_2$. This indicates that the smoking epidemic will persist in the population if $\alpha_1 \geq \alpha_2$.

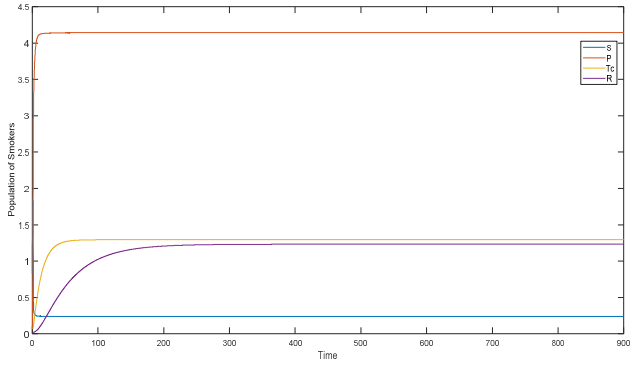


Fig. 9. Time series plot of model (28) with parameter values $K = 100, \mu = 0.02, \alpha_1 = 2, \alpha_2 = 0.01, \alpha_3 = 0.025, \delta_1 = 0.44, \delta_2 = 0.03, \delta_3 = 0.02, \eta = 0.001$. All the distinct smoking classes are present in the population and therefore the model approach endemic equilibrium.

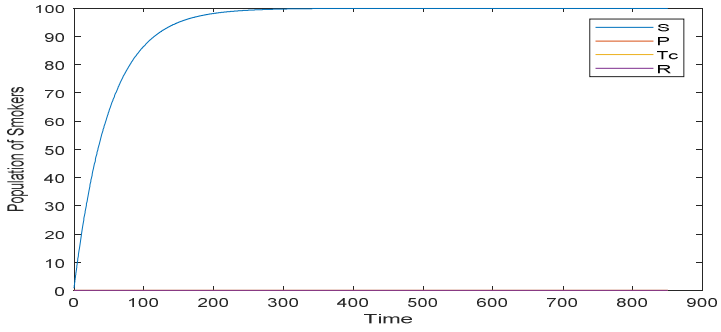


Fig. 10. Time series plot of model (28) with parameter values $= 100, \mu = 0.02, \alpha_1 = 0.003, \alpha_2 = 0.01, \alpha_3 = 0.025, \delta_1 = 0.44, \delta_2 = 0.03, \delta_3 = 0.02, \eta = 2$.

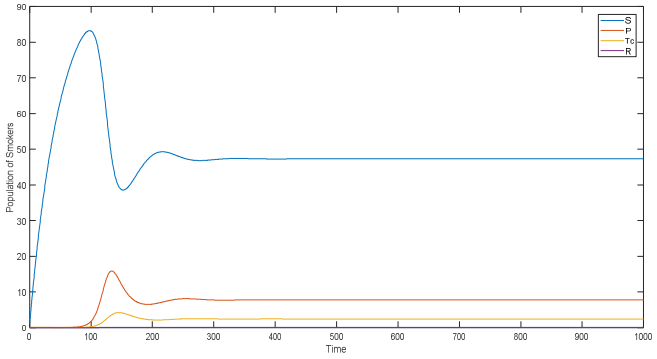
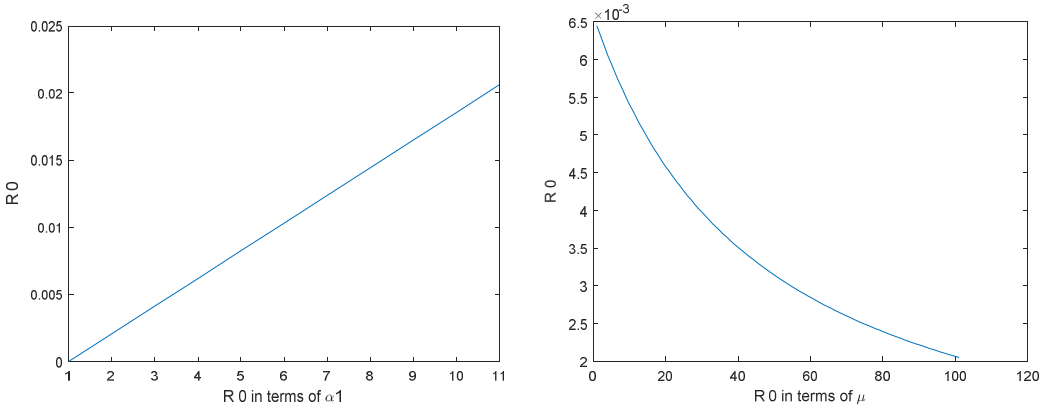
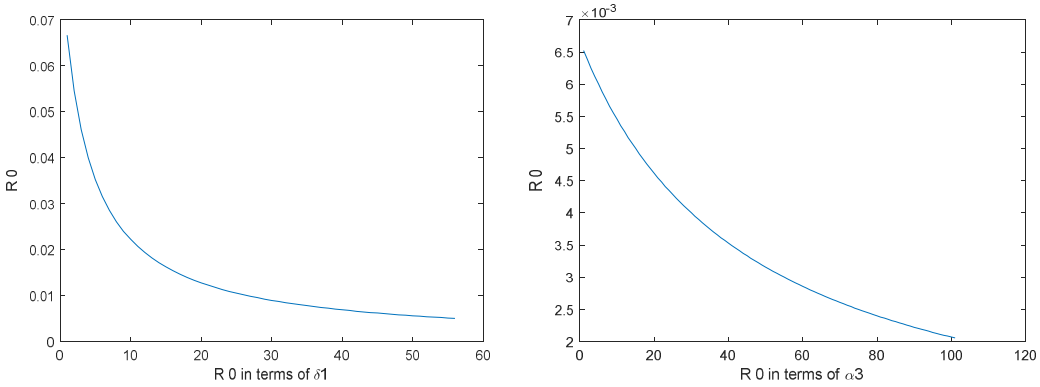


Fig. 11. Time series plot of model (28) with parameter values $= 100, \mu = 0.02, \alpha_1 = 0.003, \alpha_2 = 0.01, \alpha_3 = 0.025, \delta_1 = 0.1, \delta_2 = 0.03, \delta_3 = 0.02, \eta = 2$



(A) The relationship among R_0 and α_1. **(B) The relationship among R_0 and μ.**



(C) The relationship among R_0 and δ_1. **(D) The relationship among R_0 and α_3**

Fig. 12. Sensitivity analysis of R_0 with respect to (A) α_1, (B) μ, (C) δ_1, (D) α_3, other parameter values are in Table 1.

8 Conclusion

In this paper, we presented two mathematical models using a deterministic system of Ordinary Differential Equations. These are the that SPT_cR that explains the dynamics of smoking epidemic without considering the transfer of removed smokers to non-smokers compartment followed by an SPT_cRS model where recovered smokers are considered to re-join the non-smokers to become problem smokers again after treatment and recovery respectively. We discussed the existence and stability of smoking-free and endemic equilibria, performed sensitivity analysis and conducted numerical simulations of both models respectively. We established that our models are asymptotically stable when the associated reproduction numbers are less than one, but unstable when they are greater than one. According to the results of the two models, smoking epidemic can be reduced by minimising the contact rate between non-smokers and problem-smokers, increasing the number of smokers that go into treatment and educating smokers to refrain from smoking. We hope to modify these models in future by including passive smokers, bifurcation analysis and optimal control.

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Competing Interests

Authors have declared that no competing interests exist.

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