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Modeling Health Risks Associated with Second-hand Tobacco Smoke Exposure

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Abstract

A non-smoker who is exposed to second-hand tobacco smoke is in danger of suffering from diseases such as coronary heart disease, asthma attacks, stroke and lung cancer. This exposure occurs in various settings, including living with smokers at home, visiting bars and casinos, public places, and transport vehicles. Additionally, individuals exposed to second-hand tobacco smoke may experience severe health risks including deaths. To gain insights about the dynamics of second-hand tobacco smoke exposure and its associated health risks to non-smokers, a deterministic mathematical model is developed and analysed. Such a model is developed by using non-linear first order ordinary differential equations and the analysis was carried out analytically and numerically. Numerical simulation results in this study confirm that, 90% increase in interaction between smokers and non-smokers can increase health risks to non-smokers by 7%. Additionally, the formulated system exhibits backward bifurcation implying the possibility of having large outbreaks of health risks related to second-hand tobacco smoke even in communities with a relatively small number of smokers. The study underscores the importance of interventions to mitigate the health risks associated with second-hand tobacco smoke. Specifically, efforts should focus on reducing interactions between smokers and non-smokers during smoking or providing robust support mechanisms to help smokers quit.

Keywords: Second-hand smoke, smokers, non-smokers, health risks, basic reproduction number.
2010 MSC: Epidemiology, Ordinary differential equations, Partial differential equation, Numerical linear algebra.

1. Introduction

Second-hand tobacco smoke is a mixture of mainstream smokes produced by burning tobacco products such as cigarettes, pipes and side stream smokes exhaled by tobacco consumers [23]. According to Fekede and Mebrate [7], second-hand tobacco smoke exposure is reluctantly inhalation of tobacco smoke that occurs to people who get into contact with smokers. Exposure to second-hand tobacco smoke poses health challenges to hu-

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Figure 1: Children exposure to second-hand tobacco smoke [Source: Mohamed et al. [18]]

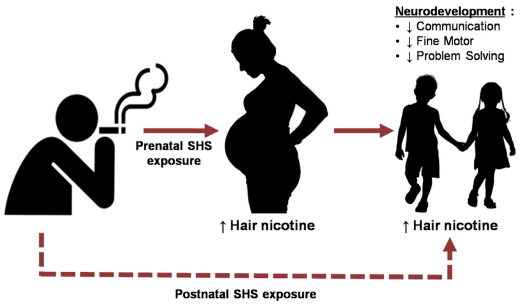

Figure 2: Pregnant women exposure to second-hand tobacco smoke [Source: Mohamed et al. [18]]

man beings including irritation of the eyes, nose, throat, lungs and sometimes nausea, headaches, dizziness as well as triggering asthma attacks. Moreover, long-term effects of exposure to second-hand tobacco smoke may include the risk of developing lung cancer, coronary heart disease, stroke, sudden death syndrome and cardiac deaths [14, 23, 26].

Second-hand tobacco smoking is one of the leading causative agents of deaths in the world [30]. It kills more than 1.3 million people in the world yearly [9], translating into nearly three deaths in every minute. Moreover, Global Adults Tobacco Survey (GATS) report of 2018 indicates that, on a monthly basis, 13.8% of people are exposed to second-hand tobacco smoke at home (see Fig. 1 and Fig. 2), 32.9% at work, 9.4% in the government buildings and offices, 4.7% at health care services, 31.1% at restaurants and 7.9% in public transport systems [19]. Statistics further reveal that a substantial number of individuals worldwide, including in Tanzania, face health risks associated with exposure to second-hand tobacco smoke. This calls for an immediate intervention to assess the extent to which second-hand tobacco smoke exposure poses health risks to non-smokers as a result of their unavoidable interactions with smokers, at the time of smoking.

Several initiatives have been taken by international organizations such as the World Health Organization (WHO), International Agency for Research and Cancer (IARC) as well as Centre for Disease Control (CDC) to minimize health risks related to second-hand tobacco smoke exposure. For instance, WHO [29] recommends six control strategies, popularly known by the acronym *MPOWER*, to minimize the current health risks related to second-hand tobacco smoke exposure. The strategies are: Monitoring tobacco consumption and the effectiveness of control measures; Protecting people from tobacco smoke; Offering help to quit tobacco use; Warning about the dangers of tobacco; Enforcing bans on tobacco advertising; promotion and sponsorship; and Raising taxes on tobacco [29].

According to Abdo et al. [1], Abdulwasaa et al. [2] and Mayengo [15], behavioral dynamics can be modeled by considering the interactions between individuals exhibiting a specific behavior and those susceptible to it, as these interactions can facilitate the transfer of behavior from one group to another. In such a situation, individuals exhibiting the

behavior act as transmission agents of such behavior to the others. In view of this, Mathematical models are useful tools in addressing the problems associated with health risks attributed to both direct smoking and second-hand tobacco smoke exposure. For instance, mathematical modeling approach is used by Fekede and Mebrate [7] to study the sensitivity analysis on second-hand smoking tobacco by utilizing forward sensitivity approach. The study findings identify that, increase in number of smokers exaggerates health risks in the community. However, health risks attributed to proximity between smokers and non-smokers was not detailed enough to capture the infected second-hand smoker's class.

On the other hand, both Pulecio-Montoya et al. [21] and Rezapour et al. [22] confirm that, increased contact between smokers and non-smokers can trigger smoking habit among non-smokers. While Rezapour et al. [22] used the power law type Kernel to conduct analysis on the new fractal-fractional model of second-hand smokers, Pulecio-Montoya et al. [21] analyzed non-linear ODEs in three compartments mathematical model to reach that conclusion. The effects of second-hand tobacco smoke exposure can be felt in the number of cigarettes smoked indoor, house air exchange rate, house volume and second-hand smoke emission factors [12]. Additionally, Adhana and Mekonnen [3] and Hussain et al. [11] in different occasions modeled second-hand smoking to gain more insights about the behavior.

Despite the fact that existing mathematical models have addressed the problems related with tobacco smoking, the problem still persists to the levels that calls for more interventions. Therefore, this study aims at developing and analyzing a deterministic model to assess the extent to which second-hand tobacco smoke exposure poses health risks to non-smokers taking into consideration the interaction between smokers and non-smokers at the time of smoking. Moreover, the deaths attributed by second-hand tobacco smoke exposure related illness and bifurcation analysis are incorporated in this study in order to address the existing gap. It is worthy note that, our study is driven by the research question: To what extent does the interaction between smokers and non-smokers during smoking accelerate health risks to non-smokers?

The current proposed model offers several contributions when it comes to modelling dynamics of second-hand tobacco smoke exposure. Some of the contributions include

(i) The study proposes a deterministic mathematical model by using non-linear first order ordinary differential equations while incorporating infected non-smokers class.

(ii) The study has noticed the backward bifurcation where by it establishes that, R_0 being less than a unity does not always guarantee extinction of the health risks related to second-hand tobacco smoke exposure from the community.

(iii) The study performs stability analysis by using Lyapunov function under the condition that the risk-persistence equilibrium point ($\Omega^\star$) is globally asymptotically stable if it satisfies the condition $\frac{dV}{dt} \leqslant 0$.

(iv) The study quantifies the extent to which smokers and non-smokers interaction accelerates health risks related to second-hand tobacco smoke exposure on non-smokers.

The remaining parts of this paper are organized as follows: Section 2 presents model formulation while section 3 presents model analysis where by basic properties of the model

such as positivity and boundedness of its solutions, stability analysis of equilibrium points, basic reproduction number calculation and bifurcation analysis are described. Additionally, section 3 presents simulation results and discussion while section 4 presents conclusion and recommendations of the study.

2. Model Development

The mathematical model for the health risks associated with second-hand tobacco smoke exposure is formulated by dividing human population into six distinct compartments namely Susceptible ($S(t)$), Exposed ($E(t)$), Infected ($I(t)$), Recovered ($R(t)$), Smokers ($M(t)$) and Quitters ($Q(t)$). In this context, ($S(t)$) denotes non-smokers who are vulnerable to exposure to second-hand tobacco smoke and, as a result, are at risk of experiencing health complications associated with such exposure; $E(t)$ represents non-smokers who interact with smokers in various occasions; $I(t)$ represents non-smokers who are suffering from second-hand smoke exposure related illness; $R(t)$ represents individuals who have recovered from second-hand smoke exposure related illness. On the other hand, $M(t)$ represents individuals who smoke tobacco cigarettes; and $Q(t)$ represents individuals who quit from smoking tobacco cigarettes.

Susceptible individuals, $S(t)$ are recruited at the rate $\pi\Lambda$ where π represents the proportion of individuals who enter into susceptible population class yearly and Λ represents recruitment rate of susceptible population yearly. This group become exposed after interacting with smokers $M(t)$ at the rate β. Exposed individuals, $E(t)$ may either progress into infected class $I(t)$ at the rate α or become smokers at the rate ρ. Infected individuals, $I(t)$ may either die at the rate σ_2 or recover at the rate γ. Recovered individuals do not acquire permanent immunity, they become susceptible again at the rate λ. Moreover, smokers $M(t)$ are recruited at the rate $(1-\pi)\Lambda$ and they increase in number through exposed individuals who join smoking group at the rate ρ. Smokers die due to health risks associated with their smoking habit at the rate σ_1. They may quit from smoking at the rate ϕ and enter into quitters' compartment $Q(t)$. Moreover, quitters become susceptible at the rate ν.

A deterministic model for the health risks associated with second-hand tobacco smoke exposure was formulated based on the following assumptions: non-smokers are exposed to health risks associated with second-hand tobacco smoke exposure through interaction with smokers only; health risks due to exposure to second-hand tobacco smoke are proportional to interaction between smokers and non-smokers; mixing of individuals in the population is homogeneous; smoking pattern is not used to classify smokers; natural mortality rate is smaller than recruitment rate. The interactions between smokers and non-smokers human populations represented by variables and parameters in Figure 3, can be

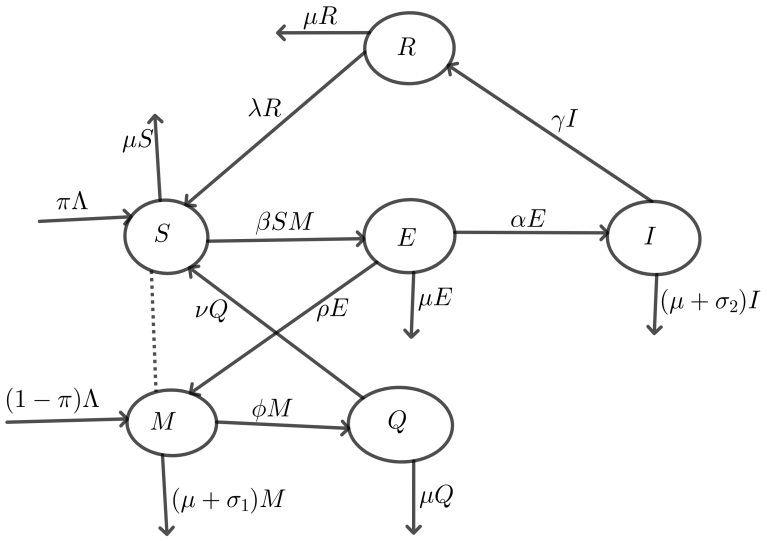

Figure 3: Second-hand Smoking Model

illustrated mathematically by using the following non-linear differential equations

$$\begin{cases} \dfrac{dS}{dt} = \pi\Lambda + \lambda R + \nu Q - (\mu + \beta M)\, S \\ \dfrac{dE}{dt} = \beta SM - (\alpha + \rho + \mu)\, E \\ \dfrac{dI}{dt} = \alpha E - (\mu + \sigma_2 + \gamma)\, I \\ \dfrac{dR}{dt} = \gamma I - (\mu + \lambda)\, R \\ \dfrac{dM}{dt} = (1-\pi)\,\Lambda + \rho E - (\phi + \mu + \sigma_1)\, M \\ \dfrac{dQ}{dt} = \phi M - (\mu + \nu)\, Q \end{cases} \tag{2.1}$$

with initial conditions $S(0) > 0,\ E(0) \geqslant 0,\ I(0) \geqslant 0,\ R(0) \geqslant 0,\ M(0) \geqslant 0$ and $Q(0) \geqslant 0$.

3. Model analysis

3.1. Positivity of the model solutions

In order for the model system (2.1) to be epidemiologically meaningful, all state variables must be non-negative. Non-negativity of state variables for this model can be assessed by using Theorem 3.1.

Theorem 3.1. *Let* $S(0) > 0,\ E(0) \geqslant 0,\ I(0) \geqslant 0,\ R(0) \geqslant 0,\ M(0) \geqslant 0$ *and* $Q(0) \geqslant 0$ *, be non-negative initial conditions, then the solutions* $S(t),\ E(t),\ I(t),\ R(t),\ M(t)$ *and* $Q(t)$ *for model system* (2.1) *remain non-negative for all time* $t \geqslant 0$.

Proof. Consider equation for susceptible population, clearly

$$\frac{dS}{dt} \geqslant -(\mu + \beta M)\, S, \tag{3.1}$$

$$S(t) \geqslant S(0)e^{-\left(\mu t + \beta \int_0^t M(t)\,dt\right)} > 0. \tag{3.2}$$

Following similar pattern, it can be proved that the rest of variables are positive for all non-negative initial conditions. □

3.2. Boundedness of the model solution

Theorem 3.2. *There is a unique solution to non-linear ordinary differential equation of system Eq. 2.1 in $\mathbb{R}^6_+$ with non-negative initial conditions, solution enters a region* $\psi = \{S(t), E(t), I(t), R(t), M(t), Q(t)\} \geqslant 0 \in \mathbb{R}^6_+, \forall t \geqslant 0$.

Proposition 3.3. *The solutions of the proposed system Eq. 2.1 starting in $\mathbb{R}^6_+$ are uniformly bounded for all* $t \geqslant 0$.

Proof. To prove proposition 3.3, we use the function $N(t)$ to represent the total human populations such that,

$$N(t) = S(t) + E(t) + I(t) + R(t) + M(t) + Q(t). \tag{3.3}$$

Consequently, we have

$$\frac{dN}{dt} \leqslant \Lambda - \mu N. \tag{3.4}$$

Solving the differential inequality Eq. 3.4 by integrating factor, yields:

$$N(t) \leqslant \frac{\Lambda}{\mu} - \left[\frac{\Lambda}{\mu} - N(0)\right] e^{-(\mu t)}. \tag{3.5}$$

As $t \to \infty$, $\left[\frac{\Lambda}{\mu} - N(0)\right] e^{-(\mu t)}$ approaches to 0. Thus, inequality Eq. 3.5 becomes $N(t) \leqslant \frac{\Lambda}{\mu}$. Thus, the solution is bounded for $0 \leqslant N(t) \leqslant \frac{\Lambda}{\mu}$, and the solutions of the proposed model system Eq. 2.1 in $\mathbb{R}^6_+$ are in the region $\psi \in \mathbb{R}^6_+$ such that $0 \leqslant N(t) \leqslant \frac{\Lambda}{\mu}, \forall t \geqslant 0$.

Therefore, the feasible solution set of the proposed model system Eq. 2.1 is positively invariant in the region ψ, biologically meaningful and mathematically well-posed. □

3.3. Risk-free equilibrium point and Basic reproduction number, $\mathcal{R}_0$

3.3.1. Risk-free equilibrium point

Risk-free equilibrium point of the model system Eq. 2.1, refers to the situation where smokers and non-smokers are not interacting. This may also translate into a minimum interaction between them which is insufficient to expose a non-smoker at risk of second-hand smoking. Additionally, when there are no smokers, this point can also be reached in the long run. In this study, the risk-free equilibrium point of the model system Eq. 2.1 is given by

$$\Omega^0 = \left(\frac{\pi}{\mu}\Lambda, 0, 0, 0, 0, 0\right). \tag{3.6}$$

3.3.2. Basic reproduction number, $\mathcal{R}_0$

Basic reproduction number $(\mathcal{R}_0)$ is the number of secondary infections produced by a single infected individual [5, 28]. In our context, $\mathcal{R}_0$ is defined as an average number of second-hand smokers produced by a single smoker introduced to entirely susceptible population. Utilizing the next generation matrix approach by Fekede and Mebrate [7] and Sofia et al. [25], the value of $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \frac{\pi\Lambda\beta\rho}{\mu a_1 a_2}. \tag{3.7}$$

where, $a_1 = \alpha + \rho + \mu$, and $a_2 = \phi + \mu + \sigma_1$ are simplifying factors.

3.4. Risk-persistence equilibrium point

When smokers and non-smokers co-exist and freely interact at the time of smoking, a risk-persistent point is acquired. In this study, risk-persistence equilibrium point is denoted by $\Omega^* = (S^*, E^*, I^*, R^*, M^*, Q^*)$. Expressing each state variables in terms of M^*, the following results are established

$$S^* = \frac{a_1(a_2M^* - a_4)}{\rho\beta M^*}, \quad E^* = \frac{a_2M^* - a_4}{\rho}, \quad I^* = \frac{\alpha(a_2M^* - a_4)}{a_3\rho},$$

$$R^* = \frac{\alpha\gamma(a_2M^* - a_4)}{a_3\rho(\mu+\lambda)}, \quad Q^* = \frac{\phi M^*}{\mu+\nu}$$

for more simplifying factors $a_3 = \mu + \sigma_2 + \gamma$, $a_4 = \mu + \lambda$ and $a_5 = \mu + \nu$. Substituting the values of S^*, E^*, I^*, R^* and Q^* into the first equation of system Eq. 2.1, quadratic equation on M^* is formed, such that

$$\tau_2 M^{*2} + \tau_1 M^* + \tau_0 = 0 \tag{3.8}$$

where $\tau_2 = \frac{a_2\alpha\beta\gamma\lambda}{a_3a_4} + \frac{\nu\phi\rho\beta}{a_5} + a_1a_2\beta$, $\tau_1 = \mu a_1a_2(\mathcal{R}_0 - 1) - \frac{\alpha\beta\gamma\lambda}{a_3} + \beta a_1a_4$ and $\tau_0 = -\mu a_1a_2(\mathcal{R}_0 + 1)\left(\mathcal{R}_0 - \frac{a_4}{a_2}\right)$. Clearly $\tau_2 > 0$, and upon satisfying condition (3.9) we have $\tau_1 > 0$ and $\tau_0 < 0$,

$$\begin{cases} \mathcal{R}_0 > \max\left(\frac{a_4}{a_2}, 1\right) \\ \frac{\mu a_2(\mathcal{R}_0 - 1) + a_4\beta}{\mu a_2} > \frac{\alpha\gamma\Lambda\beta}{\mu a_1a_2a_3} \end{cases}. \tag{3.9}$$

We now utilize Descartes' rule of signs to conclude that equation (3.8) has a unique positive real root M^*. Since M^* exists, the values of S^*, E^*, I^*, R^* and Q^* can be obtained. Thus Ω^* exists under specified conditions.

3.5. Stability of equilibrium points

3.5.1. Local stability of risk-free equilibrium

In this subsection, we analyze the local dynamics of system Eq. 2.1 around its equilibrium points by calculating the Jacobian matrix (J) for each point. The eigenvalues of J are then used to assess the stability of the risk-free equilibrium point (Ω^0).

Theorem 3.4. *The risk-free equilibrium Ω^0 of the model system Eq. 2.1 is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.*

Proof. To prove Theorem 3.4, we show that the Jacobian matrix Eq. 3.10 has real negative eigenvalues. Jacobian matrix of system Eq. 2.1 evaluated at risk free equilibrium is given by:

$$J(\Omega^0) = \begin{bmatrix} -\mu & 0 & 0 & \lambda & -\dfrac{a_1 a_2 \mathcal{R}_0}{\rho} & \nu \\ 0 & -a_1 & 0 & 0 & \dfrac{a_1 a_2 \mathcal{R}_0}{\rho} & 0 \\ 0 & \alpha & -a_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -a_4 & 0 & 0 \\ 0 & \rho & 0 & 0 & -a_2 & 0 \\ 0 & 0 & 0 & 0 & \phi & -a_5 \end{bmatrix}. \tag{3.10}$$

The eigenvalues of matrix Eq. 3.10 are $-\frac{1}{2}\left((a_1+a_2) \pm \sqrt{(a_1+a_2)^2 + 4a_1a_2(\mathcal{R}_0-1)}\right)$, $-\mu$, $-a_5$, $-a_4$, and $-a_3$. Clearly, matrix Eq. 3.10 has negative real eigenvalues whenever $\mathcal{R}_0 < 1$ suggesting that the risk-free equilibrium point Ω^0 of model system Eq. 2.1 is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$. □

3.5.2. Bifurcation analysis

In this section, we examine the the possibility of having backward bifurcation. The model state variables S, E, I, R, M and Q are renamed as x_1, x_2, x_3, x_4, x_5 and x_6 respectively. Thus, the vector $X = [x_1, x_2, x_3, x_4, x_5, x_6]^T$ transforms the model system Eq. 2.1 into the form $\frac{dX}{dt} = F(X)$ such that $F(X) = [f_1, f_2, f_3, f_4, f_5, f_6]^T$.

Re-writing model system Eq. 2.1 in the form of $\frac{dX}{dt} = F(X)$, gives the following Jacobian matrix evaluated at risk-free equilibrium point (Ω^0) and bifurcation parameter $\beta^* = \frac{\mu a_1 a_2}{\rho \Lambda \pi}$:

$$J(\Omega^0, \beta^*) = \begin{bmatrix} -\mu & 0 & 0 & \lambda & -\dfrac{a_1\, a_2}{\rho} & \nu \\ 0 & -a_1 & 0 & 0 & \dfrac{a_1\, a_2}{\rho} & 0 \\ 0 & \alpha & -a_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -a_4 & 0 & 0 \\ 0 & \rho & 0 & 0 & -a_2 & 0 \\ 0 & 0 & 0 & 0 & \phi & -a_5 \end{bmatrix}. \tag{3.11}$$

Following Theorem 4.1 in Castillo-Chavez and Song [6], the nature of bifurcation is determined by the signs of coefficients a and b for

$$a = \sum_{k,i,j=1}^{n} V_k W_i W_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\Omega_0, \beta^*), \quad b = \sum_{k,i=1}^{n} V_k W_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(\Omega_0, \beta^*) \tag{3.12}$$

where, V is the left eigenvector, W is the right eigenvector, f is the linearlized system at equilibrium point Ω_0, x is the equilibrium point of the linearlized system.

To analyze bifurcation, we consider the Jacobian matrix Eq. (3.11) whose eigenvalues are $-\mu, -(a_1+a_2), -a_3, -a_4, -a_5$ and 0. Since the system has a zero as a simple eigenvalue while the remaining eigenvalues are negative, center manifold theory can be applied to analyze the stability associated with the simple eigenvalue [24]. The essence of using this theorem is to find left and right eigenvectors of the Jacobian matrix Eq. (3.11) associated with a simple zero eigenvalue. To get right eigenvector (W), we solve the equation $J(\Omega^0)\cdot W = \mathbf{0}$, where $W = [W_1 \dots W_6]^T$ and $\mathbf{0}$ is a zero matrix. Expressing each term in terms of W_5 as a free variable, we obtain $W_1 = \left(\dfrac{\alpha a_2 a_5 \gamma\lambda + a_3 a_4 \nu\rho\phi - a_3 a_5 a_6 a_4 \rho}{a_3 a_4 a_5 \mu\rho}\right) W_5, W_2 = \dfrac{a_2}{\rho} W_5, W_3 = \dfrac{\alpha a_2}{a_3\rho} W_5$
$W_4 = \dfrac{\alpha a_2 \gamma}{a_3 a_4 \rho} W_5$ and $W_6 = \dfrac{\phi}{a_5} W_5$. We need to show that $W_5 > 0$. Finding left eigenvector V by solving the equation $J^T \cdot V = 0$, we get: $V_1 = V_3 = V_4 = V_6 = 0$ and $V_2 = \dfrac{\rho}{a_1} V_5$. Without loss of generality, we set $V_5 = 1$ and solve the equation $V_i \cdot W_i = 1$ where $i = 1, 2, 3 \dots 6$. Solving the equation of left and right eigenvectors $V_i \cdot W_i = 1$, we get $W_5 = \dfrac{a_1}{a_1 + a_2} > 0$. Then, using formulas of a and b in equation (3.12) , we get:

$$a = \frac{\beta^* \left[\alpha\,\gamma\,\lambda\,a_2 a_5 + \rho a_3 a_4 (\nu\,\phi - a_5 a_6)\right]}{\mu (a_1 + a_2)^2 a_3 a_4 a_5}, \tag{3.13}$$

$$b = \frac{\rho\,\pi\,\Lambda}{\mu(a_1 + a_2)}. \tag{3.14}$$

From the two equations, Eq. (3.13) and Eq. (3.14) above, it can be observed that, $a, b > 0$ provided that $\nu\phi > a_5 a_6$. According to Shirima Sabini et al. [24], this condition implies that, the model system Eq. (2.1) undergoes backward bifurcation at $\beta^* = \dfrac{\mu a_1 a_2}{\rho\Lambda\pi}$. Epidemiological implication of the existence of backward bifurcation is that, $\mathcal{R}_0 < 1$ does not guarantee extinction of the health risks attributed to second-hand tobacco smoke exposure from the community. That is to say, introduction of small number of smokers in the community may cause large out breaks of health risks related with second-hand tobacco smoke exposure to non-smokers who interact with smokers at the time of smoking. A schematic diagram of backward bifurcation is illustrated in Figure 4.

3.5.3. Global stability of risk-free equilibrium

Following Asamoah et al. [4] and Mayengo [16] , we utilize Metzler matrix approach to perform global stability analysis of risk-free equilibrium points for the model system (2.1). Let X_s be non-transmitting class, X_i be transmitting class, $X_{s,RFE}$ be risk-free equilibrium. Re-writing system (2.1) in terms of X_s and X_i, yield

$$\begin{cases} \dfrac{dX_s}{dt} = A\,(X_s - X_{s,\,RFE}) + A_1 X_i \\[2ex] \dfrac{dX_i}{dt} = A_2 X_i \end{cases} \tag{3.15}$$

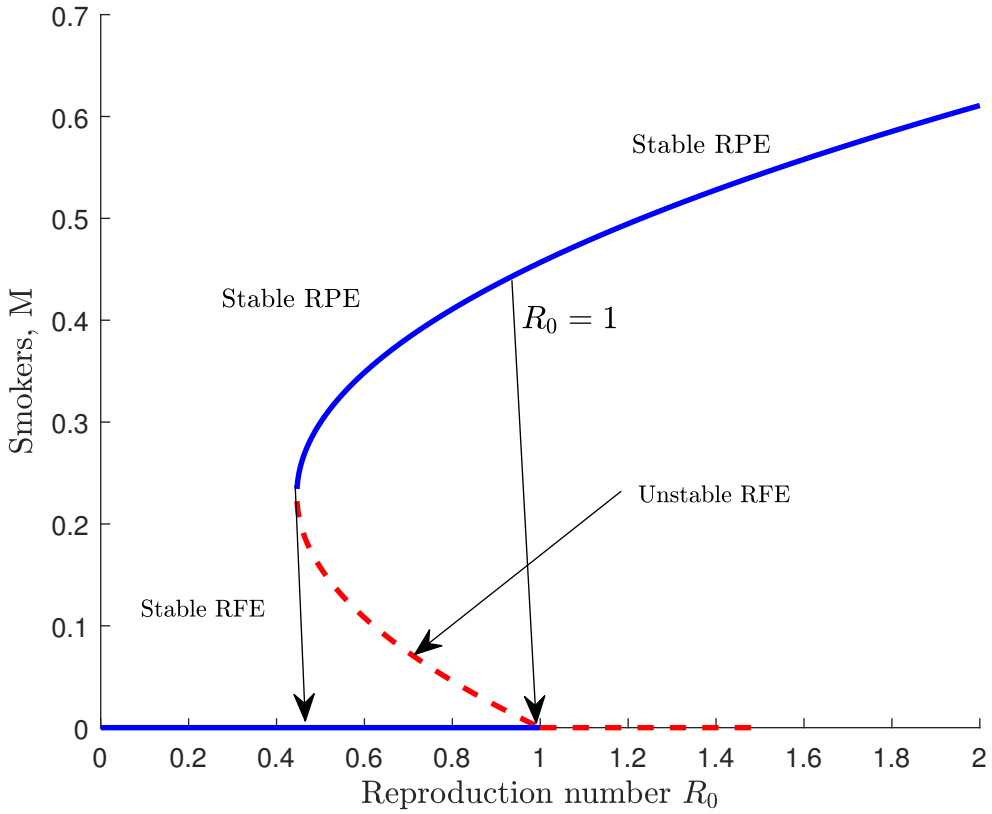

Figure 4: Backward bifurcation diagram

where A is a Jacobian matrix of X_s at risk-free equilibrium, A_1 is a Jacobian matrix of X_i at risk-free equilibrium, A_2 is a Jacobian matrix of X_i in the presence of E, I and M variables in the community. Based on model system Eq. (2.1), it can be deduced that $X_s = (S, R, Q)^T$ and $X_i = (E, I, M)^T$. For risk-free equilibrium of system Eq. (2.1) to be globally asymptotically stable, the following conditions must be satisfied in combination:

Condition 1

Matrix A must have real negative eigenvalues and

Condition 2

Matrix A_2 must be a Metzler matrix.

Checking if condition 1 holds: Matrix A is given by

$$A = \begin{bmatrix} -\mu & \lambda & \nu \\ 0 & -\mu-\lambda & 0 \\ 0 & 0 & -\mu-\nu \end{bmatrix}. \tag{3.16}$$

The eigenvalues of matrix A are $-\mu$, $-(\mu+\nu)$ and $-(\mu+\lambda)$ which is the prerequisite of condition 1. Moreover, all off-diagonal elements of matrix A_2 are non-negative which shows that matrix A_2 is a Metzler matrix. Therefore, the risk-free equilibrium (Ω^0) of

model system Eq. (2.1) is globally asymptotically stable.

3.5.4. Global stability of risk-persistence equilibrium

This subsection presents the global behavior of system Eq. (2.1) in the presence of health risks related to second-hand tobacco smoke exposure. In order to analyze global stability of the system, the following theorem can be applied

Theorem 3.5. *The risk-persistence equilibrium point Ω^* of model system Eq. (2.1) is globally asymptotically stable in the invariant region $\psi \in \mathbb{R}^6_+$ if and only if $\mathcal{R}_0 > 1$, otherwise it is unstable.*

Proof. To prove Theorem 3.5, we use the Lyapunov function as used in Korobeinikov and Wake [13] and Nyerere et al. [20]. Let V be a Lyapunov function such that

$$V = \sum_{i=1}^{6} (y_i - y_i^* \ln y_i)$$

where y_i is a population in the i^{th} compartment and y_i^* is a risk-persistence equilibrium point. For the model system Eq. (2.1), the Lyapunov function V is defined as

$$\begin{aligned} V &= (S - S^* \ln S) + (E - E^* \ln E) + (I - I^* \ln I) \\ &\quad + (R - R^* \ln R) + (M - M^* \ln M) + (Q - Q^* \ln Q)\,. \end{aligned} \tag{3.17}$$

Differentiating Lyapunov function Eq. (3.17) with respect to time (t) and substituting all the required values of the derivatives, yield

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{S^*}{S}\right)(\pi\Lambda + \lambda R + \nu Q - (\mu + \beta M)\, S) \\ &\quad + \left(1 - \frac{E^*}{E}\right)(\beta S M - (\alpha + \rho + \mu)\, E) \\ &\quad + \left(1 - \frac{I^*}{I}\right)(\alpha E - (\mu + \sigma_2 + \gamma)\, I) \\ &\quad + \left(1 - \frac{R^*}{R}\right)(\gamma I - (\mu + \lambda)\, R) \\ &\quad + \left(1 - \frac{M^*}{M}\right)((1 - \pi)\,\Lambda + \rho E - (\phi + \mu + \sigma_1)\, M) \\ &\quad + \left(1 - \frac{Q^*}{Q}\right)(\phi M - (\mu + \nu)\, Q)\,. \end{aligned} \tag{3.18}$$

At risk-persistence equilibrium point Ω^*, we have

$$\pi\Lambda = (\mu + \beta M^*)\, S^* - \lambda R^* - \nu Q^*,\ \alpha + \rho + \mu = \frac{\beta M^* S^*}{E^*},\ \mu + \sigma_2 + \gamma = \frac{\alpha E^*}{I^*},$$
$$\mu + \lambda = \frac{\gamma I^*}{R^*},\ (1 - \pi)\,\Lambda = \frac{(\phi + \mu + \sigma_1)\, M^*}{\rho E^*},\ \mu + \nu = \frac{\phi M^*}{Q^*}.$$

Then equation (3.18) becomes

$$\begin{aligned}\frac{dV}{dt} &= -\mu S\left(1-\frac{S^*}{S}\right)^2-(\phi+\mu+\sigma_1)\left(1-\frac{M^*}{M}\right)^2\\ &+\lambda R\left(1-\frac{S^*}{S}\right)\left(1-\frac{R^*}{R}\right)+Q\nu\left(1-\frac{S^*}{S}\right)\left(1-\frac{Q^*}{Q}\right)\\ &-\beta MS\left(1-\frac{S^*}{S}\right)\left(1-\frac{M^*S^*}{MS}\right)\\ &+\beta MS\left(1-\frac{E^*}{E}\right)\left(1-\frac{S^*M^*E}{E^*SM}\right)\\ &+\alpha E\left(1-\frac{I^*}{I}\right)\left(1-\frac{E^*I}{EI^*}\right)+\gamma I\left(1-\frac{R^*}{R}\right)\left(1-\frac{I^*R}{R^*I}\right)\\ &+\rho E\left(1-\frac{M^*}{M}\right)\left(1-\frac{E^*}{E}\right)+\phi M\left(1-\frac{Q^*}{Q}\right)\left(1-\frac{M^*Q}{Q^*M}\right).\end{aligned} \tag{3.19}$$

Let $\frac{S}{S^*}=a$, $\frac{E}{E^*}=b$, $\frac{I}{I^*}=c$, $\frac{R}{R^*}=d$, $\frac{M}{M^*}=e$, $\frac{Q}{Q^*}=f$ and $\phi+\mu+\sigma_1=\eta$, upon proper substitutions Eq. (3.19) becomes

$$\begin{aligned}\frac{dV}{dt} &= -\mu S\left(1-\frac{1}{a}\right)^2-\eta\left(1-\frac{1}{e}\right)^2+\lambda R\left(1-\frac{1}{a}\right)\left(1-\frac{1}{d}\right)\\ &+\nu Q\left(1-\frac{1}{a}\right)\left(1-\frac{1}{f}\right)-\beta MS\left(1-\frac{1}{a}\right)\left(1-\frac{1}{ae}\right)\\ &+\beta MS\left(1-\frac{1}{b}\right)\left(1-\frac{b}{ae}\right)+\alpha E\left(1-\frac{1}{c}\right)\left(1-\frac{c}{b}\right)\\ &+\gamma I\left(1-\frac{1}{d}\right)\left(1-\frac{d}{c}\right)+\rho E\left(1-\frac{1}{e}\right)\left(1-\frac{1}{b}\right)\\ &+\phi M\left(1-\frac{1}{f}\right)\left(1-\frac{f}{e}\right).\end{aligned} \tag{3.20}$$

□

Definition 3.6. Consider the function $\epsilon(y)=1-y+\ln(y)\leqslant 0$, for any $y>0$ with equality holds if $y=1$. Therefore, $1-y\leqslant -\ln(y)$ [17].

Proof. Consider the expression $\left(1-\frac{1}{a}\right)\left(1-\frac{1}{d}\right)=1-\frac{1}{d}-\frac{1}{a}+\frac{1}{ad}$ from Eq. (3.20) which can be written as

$$\left(1-\frac{1}{a}\right)\left(1-\frac{1}{d}\right)=\left(1-\frac{1}{d}\right)+\left(1-\frac{1}{a}\right)-\left(1-\frac{1}{ad}\right). \tag{3.21}$$

Utilizing definition 3.6 we have,

$$\begin{aligned}\left(1-\frac{1}{d}\right)+\left(1-\frac{1}{a}\right)-\left(1-\frac{1}{ad}\right) &\leqslant -\ln\left(\frac{1}{d}\right)-\ln\left(\frac{1}{a}\right)+\ln\left(\frac{1}{ad}\right)\\ &\leqslant -\ln\left(\frac{1}{ad}\right)+\ln\left(\frac{1}{ad}\right)=0.\end{aligned}$$

So, we have already shown that $\forall a, d > 0$,

$$\left(1-\frac{1}{d}\right)+\left(1-\frac{1}{a}\right)-\left(1-\frac{1}{ad}\right)=0.$$

Following similar pattern all other expressions in Eq. (3.20) can be proved that they are equal to zero $\forall a, b, c, d, e, f > 0$. By using LaSalle's principle, the limit of each solution belongs to the largest invariant set for which $S^* = S$, $E^* = E$, $I^* = I$, $R^* = R$, $M^* = M$, $Q^* = Q$ which is a singleton $\{\Omega^*\}$ [17]. Thus, the risk-persistence equilibrium point of the model system Eq. (2.1) is globally asymptotically stable on $\psi \in \mathbb{R}^6_+$ when $\mathcal{R}_0 > 1$. □

Epidemiologically, this implies that health risks related to second-hand tobacco smoke exposure will continue to affect the community at a relatively constant level without extinction or causing massive effects to a large population.

3.6. Numerical simulation results and Discussion

In this section we simulate and discuss the behaviour of the model state variables based on the selected values of model parameters from Rezapour et al. [22]. In order to ensure the stability of analytical solutions, numerical simulations for the model system (2.1) is presented by using parameter values obtained from the work done by Rezapour et al. [22]. In this study, a hypothetical population of 7000 individuals is considered with initial conditions $S(0) = 7000$, $E(0) = 200$, $I(0) = 20$, $R(0) = 10$, $M(0) = 400$ and $Q(0) = 10$. All parameter values used for simulation in this study are presented in Table 1 as follows: Using the initial conditions of each state variables and the parameter values in

Table 1: Parameter values for second-hand exposure model

Par.	Value	Dimension	Source	Par.	Value	Dimension	Source
Λ	100	$Year^{-1}$	Assumed	β	9.6×10^{-5}	$Year^{-1}$	[22]
μ	0.0142	$Year^{-1}$	[27]	α	0.04	$Year^{-1}$	Assumed
γ	0.2	$Year^{-1}$	[22]	ρ	0.02	$Year^{-1}$	[22]
σ_1	0.01	$Year^{-1}$	[22]	π	0.3	$Year^{-1}$	[22]
σ_2	0.01	Day^{-1}	[22]	λ	0.25	$Year^{-1}$	[22]
ϕ	0.3	$Year^{-1}$	[22]	ν	0.01	$Year^{-1}$	[22]

Table 1, we plot the corresponding simulation graphs with the help of MATLAB packages.

Figure 5 presents the phase portraits which proves the global stability of risk-persistence equilibrium point Ω^* by illustrating the convergence of various trajectories originating from different initial values (see Figs. 5(a) – 5(d)). This convergence stands as the manifestation of globally asymptotically stability of Ω^*. Moreover, the simulation results of the model state variables are presented in Fig. 6. Specifically, Fig. 6(a) shows a significant decrease in number of susceptible individuals $S(t)$ due to their interaction with smokers over time. Similarly, Fig. 6(e) shows an increase in number of smokers $M(t)$ as a results of such interactions. An increase in a number of individuals who are likely to suffer from health risks related to second-hand tobacco smoke exposure as illustrated in Fig. 6(b). Figure 6(f) manifests a rapid increase in number of quitters $Q(t)$ due to smoking cessation.

The study further revels that parameters β, ϕ and ρ play a vital roles in accelerating health risks related to second-hand tobacco smoke exposure. Due to their exceptional role play in this study, they are varied to study their effects in smokers, exposed and infected populations (see Fig. 7). An increase in interaction between smokers and non-smokers by 90% lead to an increase in number of exposed individuals by 11% which eventually results into an increase in health risks attributed by second-hand tobacco smoke exposure by 7%. This result aligns with findings presented by Flouris et al. [8], Hoe et al. [10] and Laverty et al. [14]. Further more, Fig. 7 shows that exposure to second-hand tobacco smoke increases smokers population by 12% which aligns with the findings reported, in different occasions, by Pulecio-Montoya et al. [21] and Rezapour et al. [22].

On the other hand, Fig. 8 shows that when 90% of smokers quit smoking, the exposed population decreases by 40%, representing a substantial reduction. This change also translates into a 36% decrease in health risks associated with second-hand tobacco smoke exposure and a 24% reduction in recruitment effects into smoking. Additionally, Fig. 9 reveals that, as smokers population increases by 50% the health risks associated with exposure to second-hand tobacco smoke is averted by 22%. This result is supported by some previous literature including studies by Pulecio-Montoya et al. [21] and Rezapour et al.[22].

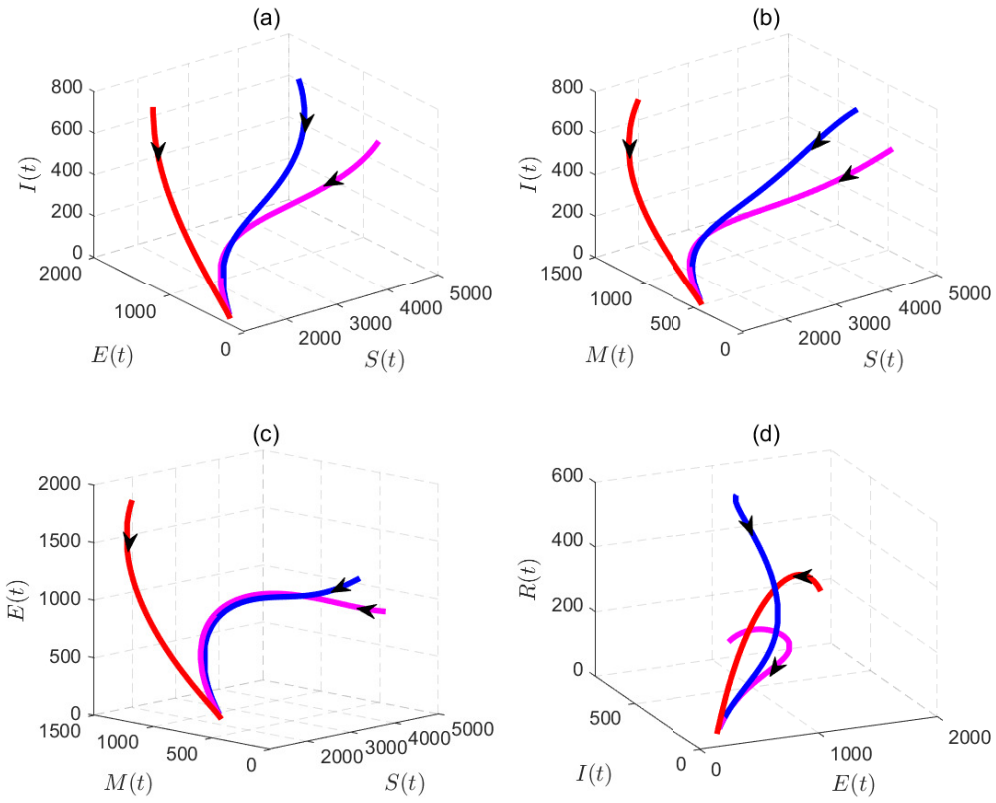

Figure 5: Global stability of risk-persistence equilibrium points

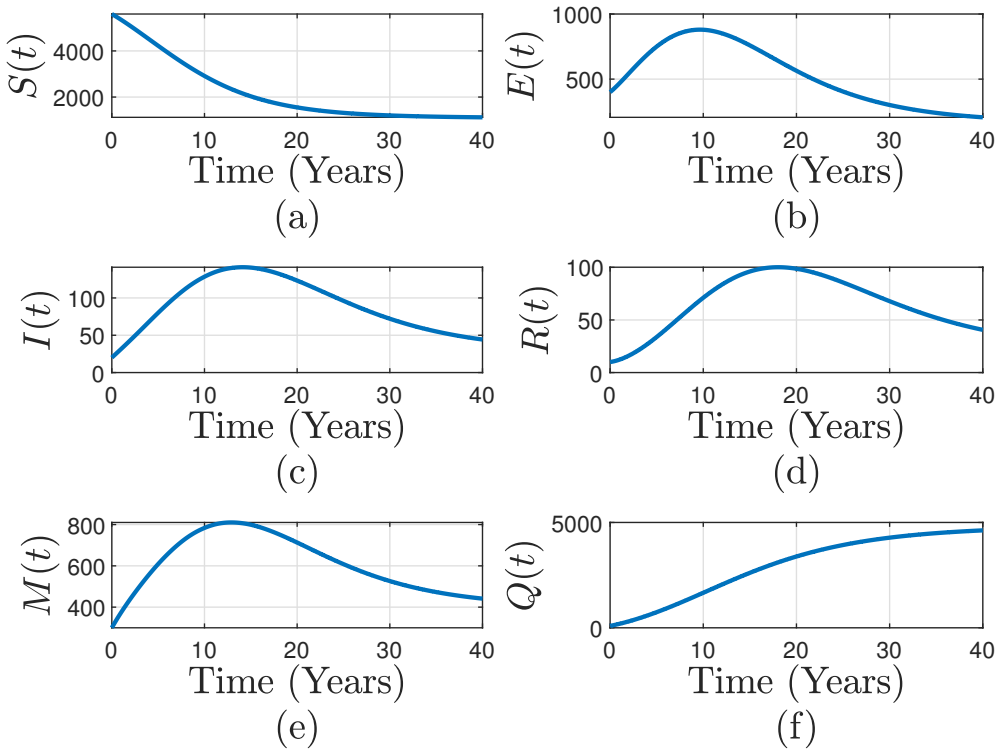

Figure 6: Dynamics of second-hand smoke exposure model state variables in relation to time

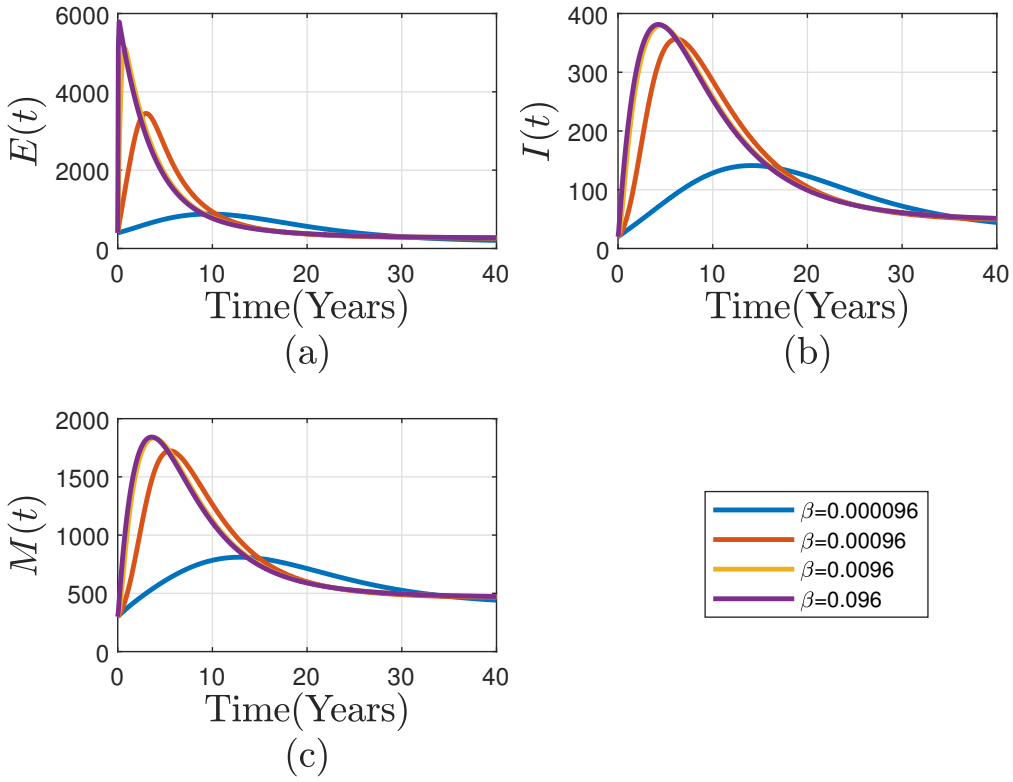

Figure 7: The effects of β on the dynamics of E(t), I(t) and M(t) populations

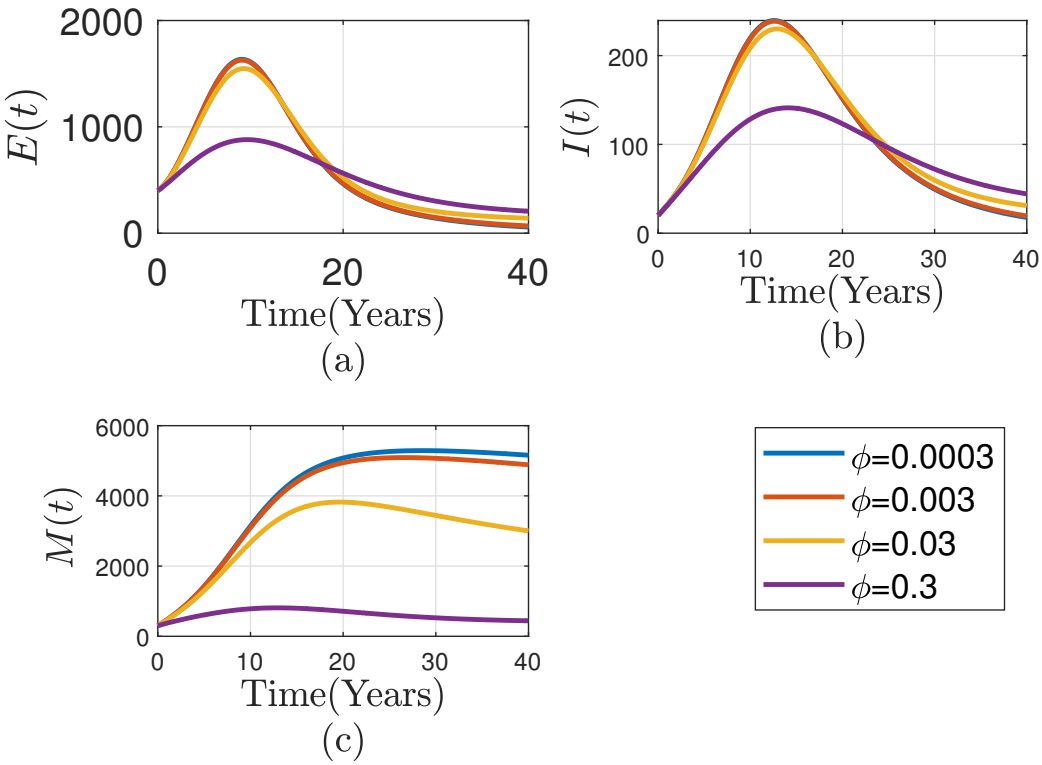

Figure 8: The effects of ϕ on the dynamics of $E(t)$, $I(t)$ and $M(t)$ populations

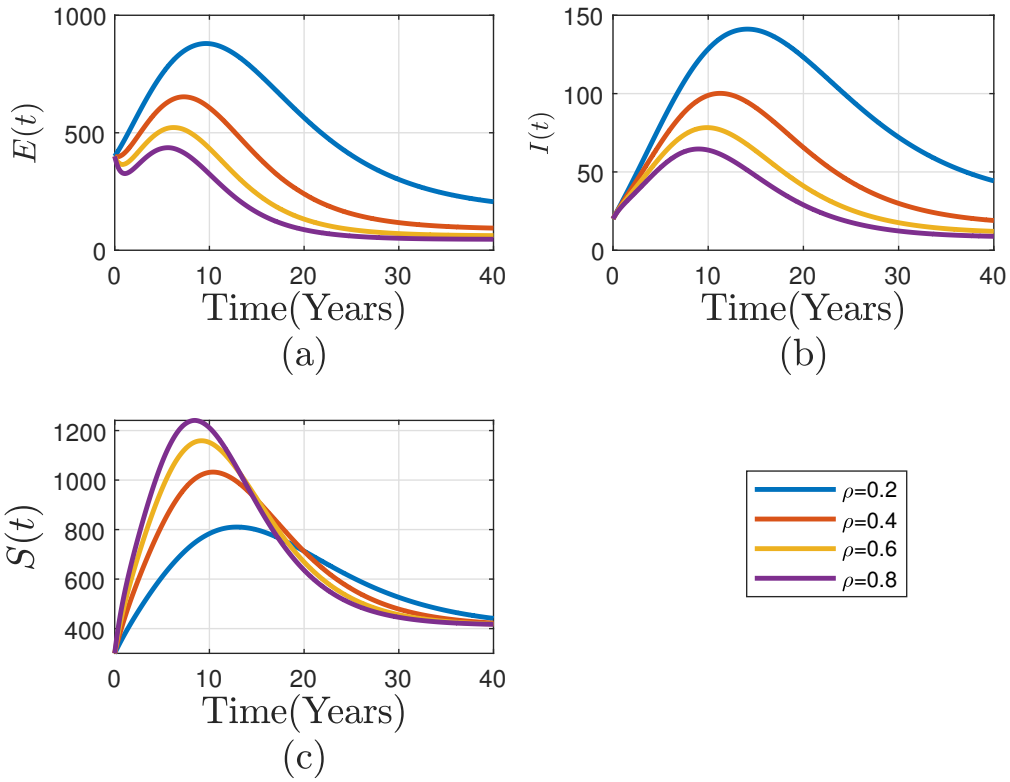

Figure 9: The effects of ρ on the dynamics of E(t), I(t) and M(t) populations

4. Conclusion and recommendations

The current study has developed a deterministic mathematical model to show the dynamics of interaction between smokers and non-smokers. Additionally, the study has explored the extent to which exposure to secondhand tobacco smoke accelerates health risks to non-smokers. Findings from this study reveal backward bifurcation of the system formulated from the developed model. Such a scenario portrays that, even if there is small number of smokers in the population, there is a possibility of having massive outbreak of health risks resulting from secondhand tobacco smoke exposure to non-smokers. Moreover, the study has found that increased interaction between smokers and non-smokers by 90% during smoking can result into increase of health risks to non-smokers by around 7%. Implying that, non-smokers who interact with smokers frequently are vulnerable to suffer from the diseases such as asthma attacks, stroke, lung cancer as well as cardio vascular infections. Consequently, the study recommends deliberate efforts to be taken while focusing at reducing interactions between smokers and non-smokers during smoking as well as supporting smokers to quit smoking.

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