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# Curbing the Menace of Re-Cycling Smokers in a Population through Mathematical Modeling Approach

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**Abstract.** Smoking can cause or trigger many diseases that can lead to death or disability. It affects the person's financial status and therefore his way of life. Hence, its negative impact on smokers includes cancer, stroke, heart diseases, immune disorder, infertility, a complication of pregnancy, pollution, mental health, depression, aging, and anxiety. It also affected one's financial status. We formulate a SEMRE deterministic model to assess the effect of re-cycling smokers into the population. We compute the basic reproduction number of the model, its equilibrium points and their stability, the sensitivity indices of the parameters of the basic reproduction number and we perform numerical analysis of the model to see how it tallies with the result of analytical calculation. Our model exhibits a backward bifurcation for some parameters' values. By varying the recycling parameter, the model bifurcation changes from the backward bifurcation to forward bifurcation as the recycling parameter reduces.

**Keywords:** Smokers, Backward bifurcation, Stability, Recycle, Equilibrium

## Introduction

Smoking takes extra care to be managed because it is the leading preventable cause of death in the world. Smokers have between 10 and 12-year shorter life expectancy (Awan et al., 2021) and illnesses brought on by smoking can be fatal. There are around 1 billion smokers on the globe. Due to the poor quality of available treatment and long-term smoking cessation, some people still smoke (Ilmayasinta et al., 2021). When smokers and non-smokers were compared, smokers were 70% more likely to have a heart attack. The risk of developing lung cancer is ten times higher for smokers, and one in ten deaths are related to smoking (Ilmayasinta et al., 2021; Morales-Delgado et al., 2018). Even without this illness, smoking can result in cancer. All of this is not unexpected considering that cigarette smoke includes more than 4000 chemicals and poisons, all of which are extremely hazardous to human health (Ilmayasinta et al., 2021; Sharma, 2020; Veeresha et al., 2019). In some circumstances, smoking may be seen as a contagious form of agony brought on by addiction. Smoking's harmful consequences can affect practically every organ and can worsen everyone's health. Smoking is one of the many preventable causes of death, but there are many other factors as well. Sharomi and Gumel (2008) introduce the lighter class of the earlier model by outlining their advancements and the negative effects of smoking on society's health.

Smoking is a significant cause of numerous diseases, including chronic obstructive pulmonary disease, stomach ulcers, heart disease, tooth stains, asthma, vascular disorders, and high blood pressure, according to the World Health Organization. Because cigarettes contain tens of thousands of chemicals and toxins, they are the direct cause of all these dangerous diseases. Mathematicians sought to create many practical smoking models to provide the best depiction of the cigarette smoking phenomenon (Awan et al., 2021; Ilmayasinta et al., 2021; Morales-Delgado et al., 2018; Sharma, 2020; Veeresha et al., 2019).

Stress, external pressure, and curiosity are factors that can contribute to the development of a smoking habit (Anjam et al., 2022). The development of a habit takes place in stages such as preparation, the first-time use of tobacco, experimentation, intermittent but recurring use,

habitual use of nicotine, and finally the establishment of dependency and addiction (Anjam et al., 2022). It is theoretically possible to stop smoking after starting to do so voluntarily and permanently (Anjam et al., 2022; Sharomi & Gumel, 2008), and most smokers who are aware of the risks associated with tobacco desire to do so. If a smoker wants to stop, however, counseling and medication can double their chances of success.

There are five stages in the process of giving up smoking: pre-contemplation, meditation, planning, getting things done, and upkeep (Sharomi & Gumel, 2008). To understand the rise in smoking and foresee how smokers would affect society, mathematical models are developed (Singh et al., 2017; Singh et al., 2017; Zaman, 2011). Some studies on the topic have appeared in the literature in this manner. These studies have three main things in common: a constant population that is split into three groups: potential smokers, smokers, and ex-smokers. These studies don't account for cigarette-related illnesses that result in death or the potential that someone would cease using nicotine entirely. Among them, in 1997, Castillo-Garsow et al. (1997) considered a fundamental model of three ordinary nonlinear differential equations that considered cigarette usage in general among teenagers and the elements of contagion, recovery, and relapse. Four nonlinear differential equations are built into a model in (Zeb et al., 2016; Zaman, 2011) that represents three separate groups of people: potential smokers, smokers, temporary smokers and long-term ex-smokers. While Sharomi and Gumel (2008), Ham (2007), Zaman et al. (2017) develop a model using five nonlinear differential equations to categorize smokers into two groups based on how frequently they smoke, high or low. Mardlijah et al. (2019), Sun and Jia (2019), like Castillo-Garsow et al. (1997), Zaman et al. (2015), investigate a non-constant population while maintaining the other properties that significantly alter the model when changed, as suggested in this article.

The aim of this study is to assess the effect of re-cycling smokers back into the population on the bifurcation phenomena of the model.

**Formation of the Model**

We grouped the population into five different variables. People that are liable to smoke when they are exposed to smoking. We represent them with a letter (S), people that have been exposed to smoking called (E), people that smoke called (M) and those individuals that have recovered from smoking called (R). The recruitment rate of the susceptible compartment is assumed to be $\pi$, the susceptible class when mixed up with smokers become exposed to smoking and move to the exposed class at the rate $\beta$ which we term recruitment rate. Individuals that are exposed to smoking may decide to start smoking at the rate $\xi$. It is also assumed that there are people who are exposed to smoking but do not indulge in smoking and join the recovered compartment at the rate $\tau$ and there are people who have recovered from smoking but due to temptation and other reasons like frustration, bad company, bad environment and nature of their work have become exposed to smoking, but at a rate less than or equal to the recruitment rate. We assumed it to be $(1-\rho)\beta$ where $\rho$ represent the scaling factor and its value is assumed to be between 0 and 1. When $\rho = 0$, the rate at which the recovered individual joined the exposed class is equal to the contact rate, $\beta$ while if $\rho = 0.5$ then the rate at which recovered people move to exposed class is $\frac{1}{2}\beta$ and if $\rho = 1$, then no recovered individual move to the exposed class. People who join the smokers may stop smoking base on some reasons like ill-health, or when they are aware of the disadvantages of smoking or due to religion's reason and other reasons and join the recovered class at the rate $\Upsilon$. We also considered that everyone in the population could pass away naturally at the rate $\mu$, while some smokers could pass away at the rate d from a variety of diseases brought on by smoking. Figure 1 illustrates this procedure.

**Figure 1. The flow diagram**

The system of ordinary differential equation

$$\frac{dS}{dt} = \pi - \beta SM - \mu S$$
$$\frac{dE}{dt} = \beta SM + (1-\rho)\beta MR - (\tau + \mu + \xi)E$$
$$\frac{dM}{dt} = \xi E - (\gamma + \mu + d)M$$
$$\frac{dR}{dt} = \tau E + \gamma M - (\mu + (1-\rho)\beta M)R$$

**Table 1. The parameters of the model and their explanation**

| S/no | Parameter | Description |
|---|---|---|
| 1 | π | Recruitment rate |
| 2 | μ | Natural death rate |
| 3 | β | Contact rate of smoker and susceptible |
| 4 | d | Death rate due to diseases caused by smoking |
| 5 | τ | Recovering rate of the exposed individuals |
| 6 | $\rho$ | Rate of loss of smoking resistivity |
| 7 | ξ | Rate of becoming a smoker |
| 8 | γ | Rate of given up smoking |

**Table 2. The model variables and their description**

| S/no | Variables | Description |
|---|---|---|
| 1 | S | Susceptible population |
| 2 | E | Exposed population |
| 3 | M | Smoking population |
| 4 | R | Recovered population |

With the initial conditions
$S(0) = S_0 \geq 0,, E(0) = E_0 \geq 0, M(0) = M_0 \geq 0, R(0) = R_0 \geq 0$
For all time t > 0, it is assumed that the system (1)'s parameters are all positive.

**The SEMRE Model's Basic Properties of Invariant Region**

In this section, we go over the characteristics of the suggested model.

**Lemma:** The initial conditions of the solutions of the system (1) are contained in the region $B \in \Re_+^4$, defined by

$$B = \{(S, E, M, R) \in \Re^4 : 0 \leq N \leq \frac{\pi}{\mu}\}$$

**Proof:** The summation of all equations of model system (1) gives $N = S + E + M + R$

So, if we differentiate N with respect to time and substitute the values of $\frac{dS}{dt}, \frac{dE}{dt}, \frac{dM}{dt}$ and $\frac{dR}{dt}$ and simplify, we get

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dM}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \pi - \beta SM - \mu S + \beta SM + (1-\rho)\beta MR - (\tau + \mu + \xi)E + \xi E - (\gamma + \mu + d)M + \tau E + \gamma M - (\mu + (1-\rho)\beta M)R$$

$$\frac{dN}{dt} = \pi - \mu(S + E + M + R) - dM$$

If there is no smoking related death, then the equation becomes

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

Solving this inequality, gives

$$B = \{(S, E, M, R) \in \Re^4 : 0 \leq N \leq \frac{\pi}{\mu}\}$$

Hence, region B is where the model equation's solution is constrained.

**Positivity of the Solution**

**Theorem:** Let D = $\{(S, E, M, R) \in \Re^4 : S(0) = S_0 > 0, E(0) = E_0 > 0, M(0) = M_0 > 0, R(0) = R_0 > 0\}$; consequently, for t ≥ 0, all the solutions $\{S, E, M, R\}$ are positive.

**Proof:** From equation (1) of the system of differential equation (1), we get

$$\frac{dS}{dt} \geq -\mu S$$

$$\frac{dS}{S} \geq -\mu dt$$

Solving this equation and applying the initial condition yields,

$$S(t) \geq S_0 e^{-\mu t} \geq 0$$

Similarly, from the second, third and fourth equations we get

$$E(t) \geq E_0 e^{-(\tau+\mu+\xi)t} \geq 0, \quad M(t) \geq M_0 e^{-(\gamma+\mu+d)t} \geq 0, R(t) \geq R_0 e^{-(\mu+\rho)t} \geq 0$$

The theorem's proof is now complete. As a result, the model's solution is positive for t > 0.

**The Smoking-Free Equilibrium Point**

The smoking-free equilibrium (*MFE*) state, $E^0_{smok}$, of the system model (1) is obtained by setting the right-hand side of the equation equal to zero and let $E = M = R = 0$, we solve for S, and we get:

$$\pi - \mu S = 0$$

$$S = \frac{\pi}{\mu}$$

$$E^0_{smok} = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

**Effective Reproduction Number ($\Re_{smok}$)**

We applied the next generation matrix approach (Ham, 2007)to the system in order to determine the effective reproduction number, where F is the matrix of the new infection terms and V is the matrix of the transition terms.

Thus:

$$F = \begin{pmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial M} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial M} \end{pmatrix}\Bigg|_{E^0_{smok}} = \begin{pmatrix} 0 & \frac{\beta\pi}{\mu} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial M} \\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial M} \end{pmatrix}\Bigg|_{E^0_{smok}} = \begin{pmatrix} (\tau+\mu+\xi) & 0 \\ -\xi & (\gamma+\mu+d) \end{pmatrix}$$

$$|FV^{-1} - \lambda I_{2\times 2}| = \frac{\xi\beta\pi}{\mu(\tau+\mu+\xi)(\mu+\gamma+d)}$$

$\lambda$ is an eigenvalue of the matrix $(FV^{-1})$ and $I_{2\times 2}$ is a 2 by 2 identity matrix

Therefore, the basic reproduction number $\Re_{smok}$ is the spectral radius of the matrix $(FV^{-1})$. Hence,

$$\Re_{smok} = \frac{\xi\beta\pi}{\mu(\tau+\mu+\xi)(\mu+\gamma+d)}$$

**Sensitivity Indices of the Basic Reproduction Number's Parameters**

Due to uncertainty in the calculation of some parameter values, it is useful to perform a sensitivity analysis to evaluate how sensitive the basic reproductive number is with respect to these parameters. As a result, we will be able to determine which control (constant) causes the biggest reduction in $\Re_{smok}$ and, hence, which control measure is most effective at halting the spread of smoking habit. To do this, we calculate the reproduction number's normalized forward sensitivity index regarding these parameters. This index evaluates the relationship between a variable's relative change and changes to its parameters.

The formula used for the computation of forward normalized sensitivity index as described by Zaman (2011) and Ham (2007) is

$$\Lambda_{\theta}^{\Re_{smok}} = \frac{d\Re_{smok}}{d\theta} X \frac{\theta}{\Re_{smok}}$$

Table 2 below displays the results of computing the sensitivity indices of each basic reproduction parameter regarding the basic reproduction number.

**Table 3. The sensitivity of the parameters of the basic reproduction number**

| Parameter | Relationship with other Parameters | Index |
|---|---|---|
| $\tau$ | $\Lambda_{\tau}^{\Re_{smok}} = \frac{-\xi}{(\tau+\mu+\xi)} < 0$ | -0.505 |
| $\beta$ | $\Lambda_{\beta}^{\Re_{smok}} = 1 > 0$ | 1.000 |
| $\pi$ | $\Lambda_{\pi}^{\Re_{smok}} = 1 > 0$ | 1.000 |
| $\xi$ | $\Lambda_{\xi}^{\Re_{smok}} = \frac{(\mu+\xi)}{(\tau+\mu+\xi)} > 0$ | 0.010 |
| $\gamma$ | $\Lambda_{\gamma}^{\Re_{smok}} = \frac{-\gamma}{(\mu+\gamma+d)} < 0$ | -0.046 |
| d | $\Lambda_{d}^{\Re_{smok}} = \frac{-d}{(\mu+\gamma+d)} < 0$ | -0.053 |
| $\mu$ | $\Lambda_{\mu}^{\Re_{smok}} = \frac{-[(\tau+\mu+\xi)(\mu+\gamma+d)+\mu(\tau+\mu+\xi)+\mu(\mu+\gamma+d)]}{(\tau+\mu+\xi)(\mu+\gamma+d)} < 0$ | -1.780 |

**Interpretation of the Sensitivity Indices of the Parameters**

The parameters in Table 3 above that have positive sensitivity indices β, π and ξ have significant impact on the spread of smoking if their values are raised while the other parameters remain the same. That is because the average cases of smoking increase when their values are increased. To remove the smokers, their values must be decreased. It is not physiologically logical to suggest that the smoking-induced death rate d and the rate at which people naturally die μ should be raised to control the smoking even though their sensitivity indices are negative. The recovery rate of exposed smokers is another potentially sensitive parameter with a negative sensitivity index that is crucial for the smoke to be well controlled when its values are increased (see Figure 2). Parameters with their bars pointing upward need to be reduced in order to reduce the smokers' population while those with their bars pointing downward need to be increased to eradicate the smoker' population in the society. But even though the bars of μ and d are pointing downward, it is not biologically reasonable to said that we should increase the natural death rate and smoking induced-death rate in other to eradicate the smoking since our aim to eradicate the smoking to save life and not to lose life.

**Figure 2. A pictorial representation of the sensitivity indices in a bar chat**

**Analysis of the Smoking-Free Equilibrium Point's Local Stability**

Applying Routh Hurwitz criteria (Chitnis et al., 2006; Saputra et al., 2023; Ucakan et al., 2021), we established the following theorem.

**Theorem:** If $\Re_{smok}$ is less than 1, the smokers-free equilibrium point is locally asymptotically stable; if $\Re_{smok}$ is greater then 1, it is unstable.

**Proof:** We got the Jacobian matrix of the system (1) at the smokers' free equilibrium $\boldsymbol{E_0}$ to compute the local stability of the smoker's free equilibrium. Hence,

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\frac{\beta\pi}{\mu} & 0 \\ 0 & -(\tau+\mu+\xi) & \frac{\beta\pi}{\mu} & 0 \\ 0 & \xi & -(\gamma+\mu+d) & 0 \\ 0 & \tau & \gamma & -\mu \end{pmatrix}$$

The Jacobian matrix's characteristic equation is provided by

$$(\lambda+\mu)^2(\mu(\lambda+d+\gamma+\mu)(\lambda+\tau+\mu+\xi)-\pi\beta\xi)=0$$

Which can be written as

$$(\lambda+\mu)^2(\mu\lambda^2+\mu(d+\tau+\gamma+2\mu+\xi)\lambda+\mu(d+\gamma+\mu)(\tau+\mu+\xi)-\pi\beta\xi)=0$$

$$(\lambda+\mu)^2\left(\mu\lambda^2+\mu(d+\tau+\gamma+2\mu+\xi)\lambda+\mu(d+\gamma+\mu)(\tau+\mu+\xi)\left(1-\frac{\pi\beta\xi}{\mu(d+\gamma+\mu)(\tau+\mu+\xi)}\right)\right)=0$$

$$(\lambda+\mu)^2\left(\mu\lambda^2+\mu(d+\tau+\gamma+2\mu+\xi)\lambda+\mu(d+\gamma+\mu)(\tau+\mu+\xi)(1-\mathfrak{R}_{smok})\right)=0$$

$$(\lambda+\mu)^2(A\lambda^2+B\lambda+C)=0$$

Where

$A=\mu$

$B=\mu(d+\tau+\gamma+2\mu+\xi)$

$C=\mu(d+\gamma+\mu)(\tau+\mu+\xi)-\pi\beta\xi$

The first two roots of the characteristic's equation are $\lambda_1=-\mu,\ \lambda_2=-\mu$. The roots of the quadratic equation $A\lambda^2+B\lambda+C=0$ are the remaining two eigenvalues.

Which is $\mu\lambda^2+\mu(d+\tau+\gamma+2\mu+\xi)\lambda+\mu(d+\gamma+\mu)(\tau+\mu+\xi)(1-\mathfrak{R}_{smok})=0$

Using Routh Hurwitz criteria, the system of equations including the aforementioned characteristic equation is consequently locally asymptotically stable because all of the matrix's eigenvalues have negative real portions.

**Analysis of the Smoking-Free Equilibrium Point's Global Stability**

We'll look into the system's overall stability using the method Castillo-Chavez and Song developed.

Let re-write the system (1) as follows

$\frac{dX}{dt}=F(X,Z)$,

$\frac{dZ}{dt}=G(X,Z), G(X,0)=0$

Where $X$ represents the nonsmoker population, that is $X=\{S,R\}$ while $Z$ represents smokers' population, that is Z= $\{E,M\}$. The smoking-free equilibrium point of the model is denoted by U= ($X^*$,0). The point U= ($X^*$,0) is globally asymptotically stable equilibrium for the model provided that $\mathfrak{R}_{smok}<1$, which is locally asymptotically stable, and the following conditions must be met:

($\mathbb{H}_1$): For $\frac{dX}{dt}=F(X,0)$, $X^*$ is globally asymptotically stable.

($\mathbb{H}_2$):$G(X,Z)=AZ-\widetilde{G}(X,Z), \widetilde{G}(X,Z)\geq 0$ for $(X,Z)\in\Omega$

If the model (1) meets the given two criteria, then the following theorem holds

**Theorem:** The smoking free equilibrium point U= ($X^*$,0) is globally asymptotically stable provided $R_{smok}<1$ and conditions ($\mathbb{H}_1$) and ($\mathbb{H}_2$) are satisfied.

**Proof:** from the system we can get F (X, Z) and G(X, Z):

$$F(X,Z)=\begin{pmatrix}\pi-\beta SM-\mu S\\ \xi E+\gamma M-(\mu+(1-\rho)\beta M)R\end{pmatrix}$$

$$G(X,Z)=\begin{pmatrix}\beta SM+(1-\rho)\beta MR-(\tau+\mu+\xi)E\\ \xi E-(\gamma+\mu+d)M\end{pmatrix}$$

At $E=M=R=0$

$$\frac{dX}{dt}=F(X,0)=\begin{pmatrix}\pi-\mu S\\ 0\end{pmatrix} \quad (2)$$

From the above system, we see that $X^*=(\frac{\pi}{\mu},0)$ is the globally asymptotic point. This can be verified from the solution, namely

$S(t)=\frac{\pi}{\mu}+(S(0)-\frac{\pi}{\mu})e^{-\mu t}$. As t→∞, the solution $S(\infty)\to\frac{\pi}{\mu}$

Which implies the global convergence of (2) in $\mathfrak{D}$ and this satisfies condition $\mathbb{H}_1$. Now from $\mathbb{H}_2$ we have that $G(X,Z)=\mathcal{H}Z-\widetilde{G}(X,Z), \widetilde{G}(X,Z)\geq 0$ for $(X,Z)\in\mathfrak{D}$

Therefore, $\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z}) = \mathcal{H}\mathrm{Z} - \mathrm{G}(\mathrm{X},\mathrm{Z})$.
Where A is an n x n matrix, Z is a column vector and $\mathrm{G}(\mathrm{X},\mathrm{Z})$ is a column vector formed from the smoker equations.

The first partial derivative of $\mathrm{G}(\mathrm{X},\mathrm{Z})$ with respect to $E$, and $M$ computed at the smoker free equilibrium point gives matrix A.

$$\mathcal{H} = \begin{pmatrix} -(\tau+\mu+\xi) & \frac{\beta\Lambda}{\mu} \\ \xi & -(\gamma+\mu+d) \end{pmatrix}$$

$$\mathcal{H}\mathrm{Z} = \begin{pmatrix} -(\tau+\mu+\xi)E + \frac{\beta\Lambda}{\mu}M \\ \xi E - (\gamma+\mu+d)M \end{pmatrix}$$

From the expression $\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z}) = \mathcal{H}\mathrm{Z} - \mathrm{G}(\mathrm{X},\mathrm{Z})$, we have

$$\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z}) = \begin{pmatrix} -(\tau+\mu+\xi)E + \frac{\beta\Lambda}{\mu}M \\ \xi E - (\gamma+\mu+d)M \end{pmatrix} - \begin{pmatrix} \beta SM + (1-\rho)\beta MR - (\tau+\mu+\xi)E \\ \xi E - (\gamma+\mu+d)M \end{pmatrix}$$

$$\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z}) = \begin{pmatrix} -(1-\rho)\beta MR \\ 0 \end{pmatrix}$$

From $\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z})$,we can see that $\tilde{G}_1(\mathrm{X},\mathrm{Z}) \le 0, \tilde{G}_2(\mathrm{X},\mathrm{Z}) = 0$ which leads to $\widetilde{\mathrm{G}}(\mathrm{X},\mathrm{Z}) \le 0$. That means the second condition $(\mathbb{H}_2)$ is not satisfied. So, the system of the equation may not be globally asymptotically stable when $\mathfrak{R}_{\mathrm{smok}} < 1$.

**Smoking Equilibrium Point $E^*_{smok}$**

We compute the smoking equilibrium points in terms of the smokers using the model system equation (1). We solved for the variables and obtained the endemic equilibrium point by setting each equation to zero.

$$E^*_{smok} = \left(\frac{\pi}{\beta\mathrm{M}^* + \mu}, \frac{(\gamma+\mu+d)\mathrm{M}^*}{\xi}, \mathrm{M}^*, \frac{\xi(\gamma+\mu+d)\mathrm{M}^* + \xi\gamma\mathrm{M}^*}{\xi(\mu+(1-\rho)\beta\mathrm{M}^*)}\right)$$

When the values of $\mathrm{S}^*$, $\mathrm{E}^*$, $\mathrm{M}^*$ and $\mathrm{R}^*$ are substituted into the system (1)'s second equation and simplified, we obtain

$$\beta\pi\xi(\mu+(1-\rho)\beta\mathrm{M}^*) + (1-\rho)\beta(\xi(\gamma+\mu+d)\mathrm{M}^* + \xi\gamma\mathrm{M}^*)(\beta\mathrm{M}^*+\mu) - (\tau+\mu+\xi)(\gamma+\mu+d)(\beta\mathrm{M}^*+\mu)(\mu+(1-\rho)\beta\mathrm{M}^*) = 0$$

$$(\beta^2(\rho-1)(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi) - \beta^2(\rho-1)(\xi(\mathrm{d}+\gamma+\mu)+\gamma\xi))\,\mathrm{M}^2 + (-(\beta\mu - \beta\mu(\rho-1))(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi) - \pi\beta^2\xi(\rho-1) - \beta\mu(\rho-1)(\xi(\mathrm{d}+\gamma+\mu)+\gamma\xi))\,\mathrm{M} + (\pi\beta\mu\xi - \mu^2(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi)) = 0$$

Which gives

$$\mathcal{S}_1 M^2 + \mathcal{S}_2 M + \mathcal{S}_3 = 0$$

Where,

$$\mathcal{S}_1 = \beta^2(\rho-1)(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi) - \beta^2(\rho-1)(\xi(\mathrm{d}+\gamma+\mu)+\gamma\xi)$$

$$\mathcal{S}_2 = -(\beta\mu - \beta\mu(\rho-1))(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi) - \pi\beta^2\xi(\rho-1) - \beta\mu(\rho-1)(\xi(\mathrm{d}+\gamma+\mu)+\gamma\xi)$$

$$\mathcal{S}_3 = -\mu^2(\mathrm{d}+\gamma+\mu)(\tau+\mu+\xi)(1-\mathfrak{R}_{smok})$$

The plot of smokers versus the basic reproduction number gives the backward bifurcation diagram below.

**Figure 3. Backward Bifurcation**

**Local Stability of the Smoking Equilibrium Point**

Using the Centre manifold theory, we investigate whether the model system (1) could exhibit forward or backward bifurcation. We can identify the local stability of the system at the endemic equilibrium point by looking at the possibility of the occurrence of backward or forward bifurcation. The fundamental reproduction number is now.

$$\Re_{smok} = \frac{\pi\beta\xi}{\mu(\tau+\mu+\xi)(d+\gamma+\mu)}$$

We let $\beta=\beta^*$ be the bifurcation parameter and consider the value of $\Re_{smok}$ at 1, then, we make β the subject of the formula to obtain

let $\Re_{smok}$ = 1 and define $\beta=\beta^*$ as the bifurcation parameter before making β the formula of the subject.

$$\beta^* = \frac{\mu(\tau+\mu+\xi)(d+\gamma+\mu)}{\xi\pi}$$

Let rename the variables as $S=x_1$,$E=x_2$,$M=x_3$, and R=$x_4$.

Also let use the vector X= $(x_1, x_2, x_3, x_4)^T$

formulated as $\frac{dX}{dt}$ = F(x),

Where F = $(f_1, f_2, f_3, f_4)^T$

Now the smoking-free equilibrium becomes $X_0 = (x_1 = \frac{\pi}{\mu}, x_2 = 0, x_3 = 0, x_4 = 0)$

Substituting these values of $S=x_1$,$E=x_2$,$M=x_3$and R=$x_4$ into our model system of equation we get

$$\frac{dx_1}{dt} = \pi - \beta x_3 x_1 - \mu x_1 ---------- f_1$$
$$\frac{dx_2}{dt} = \beta x_3 x_1 + (1-\rho)\beta x_3 x_4 - (\tau+\mu+\xi)x_2 ---------- f_2$$
$$\frac{dx_3}{dt} = \xi x_2 - (\gamma+\mu+d)x_3 ---------- f_3$$
$$\frac{dx_4}{dt} = \tau x_2 + \gamma x_3 - (\mu + (1-\rho)\beta x_3)x_4 ---------- f_4$$

The Jacobian at the smoking-free equilibrium point is

$$J(E^0_{smok}) = \begin{pmatrix} -\mu & 0 & -\frac{\beta\pi}{\mu} & 0 \\ 0 & -(\tau+\mu+\xi) & \frac{\beta\pi}{\mu} & 0 \\ 0 & \xi & -(\gamma+\mu+d) & 0 \\ 0 & \tau & \gamma & -\mu \end{pmatrix}$$

With characteristic equation, $(\lambda+\mu)^2(A\lambda^2+B\lambda+C)=0$

Where

$A=\mu$

$B=\mu(d+2\mu+\tau+\gamma+\xi)$

$C=\mu(\tau+\mu+\xi)(\gamma+\mu+d)-\xi\beta\pi$

Which means

$(\lambda+\mu)=0$

$(\lambda+\mu)=0$

$(A\lambda^2+B\lambda+C)=0$

or

$[\mu\lambda^2+\mu(d+2\mu+\tau+\gamma+\xi)\lambda+\mu(\tau+\mu+\xi)(\gamma+\mu+d)-\xi\beta\pi]=0$

Solving for λ we get

$\lambda_1=-\mu$

$\lambda_2=-\mu$

$-\mu^2(d+\gamma+\mu)(\tau+\mu+\xi)(1-1)$

The other two eigenvalues are the roots of the quadratic equation:

$[\mu\lambda^2+\mu(d+2\mu+\tau+\gamma+\xi)\lambda+\mu(\tau+\mu+\xi)(\gamma+\mu+d)-\xi\beta\pi]=0$

Which can be simplified as

$\mu\lambda^2+\mu(d+2\mu+\tau+\gamma+\xi)\lambda+\mu(\tau+\mu+\xi)(\gamma+\mu+d)(1-\Re_{smok})=0$

When we substitute the values of $\beta$, we have:

$Or\ \big(\lambda+(d+2\mu+\alpha+\gamma+\xi)\big)\lambda=0$

Which gives

$\lambda_3=-(d+2\mu+\alpha+\gamma+\xi)$ and $\lambda_4=0$

Now $\lambda_4$ is a simple eigenvalue, so that allow us to use the center manifold theorem. So, we proceed with the computation as follows: $W=\ (w_1,w_2\ ,w_3\ ,w_4)^T$

$$\begin{pmatrix} -\mu & 0 & -\frac{\beta\pi}{\mu} & 0 \\ 0 & -(\tau+\mu+\xi) & \frac{\beta\pi}{\mu} & 0 \\ 0 & \xi & -(\gamma+\mu+d) & 0 \\ 0 & \tau & \gamma & -\mu \end{pmatrix}\begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \end{pmatrix}=\begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

The system of equation formed from the matrix is:

$$-\mu w_1-\frac{\beta\pi}{\mu}w_3=0$$

$$-(\tau+\mu+\xi)w_2+\frac{\beta\pi}{\mu}w_3=0$$

$$\xi w_2-(\gamma+\mu+d)w_3=0$$

$$\tau w_2+\gamma w_3-\mu w_4=0$$

Which has solutions:

$$w_1=-\frac{\beta\pi}{\mu^2}w_3, w_2=\frac{\beta\pi w_3}{\mu(\tau+\mu+\xi)}, w_3=w_3>0, w_4=\frac{\tau\beta\pi w_3+\gamma\mu(\tau+\mu+\xi)w_3}{\mu^2(\tau+\mu+\xi)}$$

The transpose of the matrix gives:

$$\begin{pmatrix} -\mu & 0 & 0 & 0 \\ 0 & -(\tau+\mu+\xi) & \xi & \tau \\ -\frac{\beta\pi}{\mu} & \frac{\beta\pi}{\mu} & -(\gamma+\mu+d) & \gamma \\ 0 & 0 & 0 & -\mu \end{pmatrix}$$

With regard to the zero eigenvalue at $\beta = \beta^*$, the left eigenvectors of J($E_0$) are given by V $=(v_1, v_2, v_3, v_4)^T$

$$\begin{pmatrix} -\mu & 0 & 0 & 0 \\ 0 & -(\tau+\mu+\xi) & \xi & \tau \\ -\frac{\beta\pi}{\mu} & \frac{\beta\pi}{\mu} & -(\gamma+\mu+d) & \gamma \\ 0 & 0 & 0 & -\mu \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Write the above into a system of equations, we have:

$$-\mu v_1 = 0$$
$$-(\tau+\mu+\xi)v_2 + \xi v_3 + \tau v_4 = 0$$
$$-\frac{\beta\pi}{\mu}v_1 + \frac{\beta\pi}{\mu}v_2 - (\gamma+\mu+d)v_3 + \gamma v_4 = 0$$
$$-\mu v_4 = 0$$

Now solve for $v_1$,$v_2$,$v_3$,$v_4$ we get:

$$V = \left(0, \frac{\xi v_3}{(\tau+\mu+\xi)}, v_3\ 0\right)^T$$

We proceed to compute the value of $\mathcal{A}$ using the formula

$$\mathcal{A} = \sum_{\mathcal{K},i,j=1}^{n} \mathcal{V}_{\mathcal{K}}\, \mathcal{W}_i \mathcal{W}_j \frac{\partial^2 f_{\mathcal{K}}}{\partial x_i\, \partial x_j}(E_0)$$

Now we compute the second partial derivatives of the $f_i's$ with respect to the $x_i$'s and got $\frac{\partial^2 f_2}{\partial x_1\, \partial x_3} = \frac{\partial^2 f_2}{\partial x_3\, \partial x_1} = \beta$, $\frac{\partial^2 f_2}{\partial x_3\, \partial x_4} = \frac{\partial^2 f_2}{\partial x_4\, \partial x_3} = (1-\rho)\beta$ while the rest of the partial derivatives are zero.

After some calculation we got the expression for $\mathcal{A}$

$$\mathcal{A} = \frac{2\beta\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\Big((1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) - \beta\pi(\tau+\mu+\xi)\Big)$$

$$\mathcal{A} = \frac{2\beta\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\mathfrak{J}$$

Where $\mathfrak{J} = (1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) - \beta\pi(\tau+\mu+\xi)$

Now we compute the value of $\mathcal{B}$ using the formula:

$$\mathcal{B} = \sum_{\mathcal{K},i=1}^{n} \mathcal{V}_{\mathcal{K}}\, \mathcal{W}_i \frac{\partial^2 f_{\mathcal{K}}}{\partial x_i\, \partial\beta}(E_0, \beta)$$

Now let compute the values of the second partial derivatives of the $f_i'$s with respect to the $x_i'$s and β's to get:

$$\frac{\partial^2 f_2}{\partial x_3\, \partial\beta} = \beta x_1 = \frac{\beta\pi}{\mu}$$

The rest of the partial derivatives are zero.

Substitute the values of $\frac{\partial^2 f_2}{\partial x_3\, \partial\beta}$, $\mathcal{V}_2$ and $\mathcal{W}_3$ into the expression for $\mathcal{B}$, we got:

$$\mathcal{B} = \frac{\xi\beta\pi}{\mu(\tau+\mu+\xi)}\mathcal{V}_3\mathcal{W}_3 > 0$$

We now use Castillo-Chavez and Song stated below:

**Theorem 6:** consider the following general system of ordinary differential equation with parameter φ

$$\frac{dx}{dt} = f(x, \varphi), f: \mathrm{R}^{\mathrm{n}} \mathrm{x}\ \mathrm{R} \text{ and } f \in C^2(\mathrm{R}^{\mathrm{n}} \mathrm{x}\ \mathrm{R})$$

Where 0 is an equilibrium point of the system is($that\ is\ f(0, \varphi) \equiv 0$ for all φ) and assume:

1. A=$D_x f(0,0) = (\frac{\partial f_i}{\partial x_j}(0,0))$ is the linearization matrix of the system around the equilibrium point 0 with φ evaluated at 0.
2. All other eigenvalues of A have negative real components except for the simple eigenvalue zero.
3. Matrix A has right eigenvector W and a left eigenvector V corresponding to the zero eigenvalue.

Let $f_{\mathcal{K}}$ be the $k^{th}$ component of $f$ and

$$\mathcal{A} = \sum_{\mathcal{K},i,j=1}^{\mathrm{n}} \mathcal{V}_{\mathcal{K}}\, \mathcal{W}_i \mathcal{W}_j \frac{\partial^2 f_{\mathcal{K}}}{\partial x_i\, \partial x_j}(E_0), \qquad \mathcal{B} = \sum_{\mathcal{K},i=1}^{\mathrm{n}} \mathcal{V}_{\mathcal{K}}\, \mathcal{W}_i \frac{\partial^2 f_{\mathcal{K}}}{\partial x_i\, \partial \mathrm{a}}(E_0, \beta)$$

Then the local dynamics of the system around the x=0 are totally determined by $\mathcal{A}$ and $\mathcal{B}$. Particularly,

1. $\mathcal{A} > 0, \mathcal{B} > 0$, when $\varphi < 0$ with $||\varphi|| \ll 1$, (0,0) is locally asymptotically stable hence there exists a positive unstable equilibrium. When $0 < \varphi \ll 1$, (0,0) is unstable then there exists a negative and locally asymptotically stable equilibrium.
2. $\mathcal{A} < 0, \mathcal{B} < 0$, when $\varphi < 0$ with $||\varphi|| \ll 1$, (0,0) is unstable, when $0 < \varphi \ll 1$, (0,0) is locally unstable and there exists a positive unstable equilibrium.
3. $\mathcal{A} > 0, \mathcal{B} < 0$, when $\varphi < 0$ with $||\varphi|| \ll 1$, (0,0) is unstable, and there exists locally asymptotically stable equilibrium; when $0 < \varphi \ll 1$, (0,0) is stable and positive unstable equilibrium appears.
4. $\mathcal{A} < 0, \mathcal{B} > 0$, when φ changes from negative to positive, x=0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes locally asymptotically stable.

Now from our values of $\mathcal{A}$ and $\mathcal{B}$, The value of $\mathcal{B}$ is positive so whether the bifurcation will be forward bifurcation or backward at β=β* depends on the values of $\mathcal{A}$ and in turn depend on the values of $\mathfrak{J}$. If the value of $\mathfrak{J}$is negative we will have forward bifurcation while if $\mathfrak{J}$ is positive we will have backward bifurcation.

(a) We have forward bifurcation if:

$$(1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau + \mu + \xi)\big) - \beta\pi(\tau + \mu + \xi) < 0 \text{ or } (1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau + \mu + \xi)\big) < \beta\pi(\tau + \mu + \xi))$$

(b) We have backward bifurcation if:

$$(1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau + \mu + \xi)\big) - \beta\pi(\tau + \mu + \xi) > 0 \text{ or } (1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau + \mu + \xi)\big) > \beta\pi(\tau + \mu + \xi))$$

**Global Stability Analysis of the Endemic Equilibrium Point**

**Theorem:** If $R_{smok}$ >1, the endemic equilibrium $E^0_{smok}$ of the model is globally asymptotically stable.

**Proof:** by Lyapunov's direct method and LaSale's Invariant principle, we prove the above theorem by defining a Lyapunov's function (Tilahun et al., 2017).

$$\mathrm{L}(S^*, E^*, I^*, \mathrm{R}^*) = (S - S^* - \ln \frac{\mathrm{S}^*}{S}) + (E - E^* - E^* \ln \frac{E^*}{E}) + (M - M^* - M^* \ln \frac{M^*}{M}) + (\mathrm{R} - \mathrm{R}^* - \mathrm{R}^* \ln \frac{\mathrm{R}^*}{\mathrm{R}})$$

Differentiating L with respect to t, produced

$$\frac{dL}{dt} = \frac{(S-S^*)}{S}\frac{dS}{dt} + \frac{(E-E^*)}{E}\frac{dE}{dt} + \frac{(M-M^*)}{M}\frac{dM}{dt} + \frac{(R-R^*)}{R}\frac{dR}{dt}$$

Substitute the values of $\frac{dS}{dt}, \frac{dE}{dt}, \frac{dM}{dt}$ and $\frac{dR}{dt}$ into $\frac{dL}{dt}$ and then simplify to get

$$\frac{dL}{dt} = [\pi + S^*\beta M + S^*\mu + E^*(\tau+\mu+\xi) + M^*(\gamma+\mu+d) + R^*(\mu+(1-\rho)\beta M)]$$
$$-\left[\mu S + \frac{S^*}{S}\pi + \mu E + \frac{E^*}{E}\beta SM + \frac{E^*}{E}(1-\rho)\beta MR + \mu M + dM + \frac{M^*}{M}\xi E + \mu R + \frac{R^*}{R}\tau E + \frac{R^*}{R}\gamma M\right]$$

Which is of the form

$$\frac{dL}{dt} = H_1 - H_2$$

Where

$$H_1 = \pi + S^*\beta M + S^*\mu + E^*(\tau+\mu+\xi) + M^*(\gamma+\mu+d) + R^*(\mu+(1-\rho)\beta M)$$
$$H_2 = \mu S + \frac{S^*}{S}\pi + \mu E + \frac{E^*}{E}\beta SM + \frac{E^*}{E}(1-\rho)\beta MR + \mu M + dM + \frac{M^*}{M}\xi E + \mu R + \frac{R^*}{R}\tau E + \frac{R^*}{R}\gamma M$$

$\frac{dL}{dt} \leq 0$ if $H_1$ is less then $H_2$

$\frac{dL}{dt} = 0$ if and only if $S = S^*, E = E^*, M = M^*, R = R^*$

Therefore, the largest invariant impact invariant set in $\{(S^*, E^*, M^*, R^*) \in \Omega: \frac{dL}{dt} = 0\}$ is the singleton set $E^*_{smok}$, where $E^*_{smok}$ is the smoking equilibrium of the system (1). Therefore, by Lasalle's Invariant principle, it implies that $E^*_{smok}$ is globally asymptotically stable in $\Omega$ if $Q_1$ is less than $Q_2$.

**Effect of Re-Infection on the Bifurcation Coefficient**

In this section we look at the effect of the re-cycling the smokers on the bifurcation coefficients $\mathcal{A}$. Now from

$$\mathcal{A} = \frac{2\beta\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\Big((1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) - \beta\pi(\tau+\mu+\xi)\Big)$$

We set $\rho = 1$, then $\mathcal{A}$ becomes

$$\mathcal{A} = -\frac{2\beta^2\pi\xi(\tau+\mu+\xi)}{\mu^2(\tau+\mu+\xi)^2}\mathcal{V}_3\mathcal{W}_3^2 < 0$$

Since the values of $\mathcal{A}$ is negative, the bifurcation will be forward according to center manifold theorem.

Let $(1-\rho)\beta = \mathfrak{T}$ be the re-cycling rate of smokers in the population, then

$$\frac{\partial\rho}{\partial\mathfrak{T}} = -\frac{1}{\beta}$$
$$\frac{\partial\mathcal{A}}{\partial\rho} = -\frac{2\beta\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big)$$
$$\frac{\partial\mathcal{A}}{\partial\mathfrak{T}} = \frac{\partial\mathcal{A}}{\partial\rho} \times \frac{\partial\rho}{\partial\mathfrak{T}} = -\frac{2\beta\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) \times -\frac{1}{\beta}$$
$$\frac{\partial\mathcal{A}}{\partial\mathfrak{T}} = \frac{2\xi\mathcal{V}_3\mathcal{W}_3^2}{\mu^2(\tau+\mu+\xi)^2}\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big)$$

As a result, the bifurcation coefficient $\mathcal{A}$ increases as the smokers' re-cycling rate does. As a result, increasing the rate at which smokers are recycled into the population will raise the bifurcation coefficient, which will lead to backward bifurcation. To end smoking in society, it is necessary to lessen the recycling of smokers into the population.

For forward bifurcation, the value of $\mathfrak{J} < 0$

That is

$$(1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) - \beta\pi(\tau+\mu+\xi) < 0$$
$$(1-\rho)\big(\tau\beta\pi + \gamma\mu(\tau+\mu+\xi)\big) < \beta\pi(\tau+\mu+\xi)$$

$$\rho > 1 - \frac{\beta\pi(\tau + \mu + \xi)}{\left(\tau\beta\pi + \gamma\mu(\tau + \mu + \xi)\right)}$$

$$\rho > 0.42$$

Therefore, the value of the rate of re-cycling smokers that will guarantee forward bifurcation of the model will be from 0% to 58% of the contact rate β. but from 59% to 100% of the contact rate, the model will exhibit backward bifurcation.

$$(1 - \rho)\beta = (1 - 0.42)0.50 = 0.29$$

**Numerical Simulation**

| Parameter | Parameters' Values | Source |
|---|---|---|
| T | 0.650 | Castillo-Garsow, C., Jordan-Salivia, G., & Rodriguez-Herrera, A. (1997). |
| B | 0.500 | Assumed |
| Π | 0.0157 | Assumed |
| Ξ | 0.650 | Sharomi, O., & Gumel, A. B. (2008). |
| D | 0.070 | Sharomi, O., & Gumel, A. B. (2008). |
| M | 0.01301235 | Zaman, G. (2011) |
| P | 0.05 (0 − 1) | Assumed |

**The Effect of Re-cycling Factor($\rho$) on the Bifurcation Diagram of the Model**

**Figure 4a**

**Figure 4b**

**Figure 4c**

**Figure 4d**

Figure 4e

Figure 4f

**Figure 4. The effect of re-cycling smokers back into the population**

**Effect of the Model Parameters on Smokers' Population**

Figure 5a

Figure 5b

Figure 5c

Figure 5d

Figure 5e

Figure 5f

Figure 5. The effect of varying the parameters of the model on the population of the smokers

**Discussion and Conclusions**

In this paper, we developed a SEMRE deterministic model for smoking dynamic. The results of the model analysis show that the model is well-posed mathematically and epidemiologically, and that its solutions are positive and bounded. The stability of equilibrium $\mathcal{R}_{smok}$ points was examined, and the fundamental reproduction number was determined. When the$\mathcal{R}_{smok}$< 1 was established, Lyapunov's theory showed that the smokers-free equilibrium point was globally asymptotically stable. Bifurcation analysis of the model was demonstrated using Centre manifold theory, and the model displays backward bifurcation at $\mathcal{R}_{smok}$ = 1. This indicates that when $\mathcal{R}_{smok}$ < 1, both a stable endemic equilibrium and a stable smokers-free equilibrium exist concurrently. This phenomenon of the backward bifurcation indicates that merely reducing the $\mathcal{R}_{smok}$ below 1 will not eradicate smoking in the population. We need to reduce$\mathcal{R}_{smok}$ below a critical value $\mathcal{R}_{smok}^{c}$ which lies between 0 and 1 so that 0 < $\mathcal{R}_{smok}^{c}$ < $\mathcal{R}_{smok}$ < 1 before smokers can be eradicated in the society as shown in Figure 3.

Second, we examine the effect of the re-cycling rate on bifurcation phenomena. It was established that in the absence of re-cycling of smokers back to exposed class, the model shows a forward bifurcation. The rate of re-cycling of smokers back into the population is proportional to the bifurcation phenomena. That is as the rate of re-cycling of smokers decreases, the bifurcation shifts toward the right side of the plane. As the value of the scaling factor increases from 0% to 41%, the bifurcation curve tends to move forward. As soon as the value of the scaling factor reaches 42%, the bifurcation becomes a forward bifurcation. Note that increasing scaling factors reduces the re-cycling rate. When the scaling factor is 0%, the re-cycling rate will be (1-0) β, that 100% of the contact rate. Which means the re-cycling rate will be equal to the contact rate. When the scaling factor is 41%, the re-cycling rate will be (1-41) β = (1-0.41)0.50 = 0.295. That is 295% of the recovered population are being re-cycled back into the exposed population. When the scaling factor is 42%, the re-cycling rate will be (1-42) β = (1-0.42)0.50 = 0.29. That is 29% of the recovered people are being re-cycled back into the exposed population. At this point, the model exhibits the phenomena of forward bifurcation which is maintain for the rest of the values of the scaling factor up to 1. And if the scaling factor is 1, it means the re-cycled rate is (1-1) β = 0. This means there is no re-cycling of the recovered smokers back into the exposed compartment.

Third, we examine the effect of the varying parameters of the model on the population of smokers. We started with the re-cycling scaling factor. When the scaling factor of the recycling parameter is increased from 0 to 1, the population of smokers reduces. This is the same thing as reducing the re-cycling rate from 100% to 0%. Hence, reducing the re-cycling rate helps to reduce the population of smokers. This is shown in Figure (5a). We then examined the contact rate. In Figure (5b), we increase the contact rate from 0.1 to 1.0 and the result shows that the population of smokers increase. We increase the rate at which the exposed class joined the smoking class, and the result shows an increase in the population of the smokers as depicted in Figure (5c). Figure (5d) shows the results of increasing the rate at which smokers quit smoking. This gives a decrease in the population of smokers. We increase the rate at which exposed individuals get recovered without joining the smokers' compartment and the result shows that increasing the rate of recovering of the exposed class without joining the smokers' class from 1 to 10 reduces the population of smokers. This result is shown in Figure (5e). We examine the recruitment rate by increasing its value from 0.1 to 1.0 and the result shows that increasing the recruitment rate increases the population of smokers. It also shows that as the value of the recruitment rate is increased, the endemic equilibrium point also increases. We observed that when the recruitment rate is 0.1, the endemic equilibrium stabilizes at 1 and when it is increased to 0.5 the endemic equilibrium moves upward and stabilizes at 6. When we increase it to 1.0 the endemic equilibrium stabilizes at 12.

Fourth, sensitivity analysis of the parameters of the basic reproduction number was

carried out. Those parameters with positive sensitivity are the recruitment rate π, the contact rate β and the rate at which exposed individuals become smokers ξ. These parameters will increase the basic reproduction numbers when their values are increased. Hence, their values need to be reduced to reduce the basic reproduction number for the eradication of smoking in the population. The parameters with negative sensitivity are the rate at which exposed individuals are recovered without indulged in the smoking τ, natural death rate μ, smoking induced death rate d and recovering rate of smokers γ. Their values when increased will reduce the basic reproduction number.

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