Backward bifurcation arises from the smoking transmission model considering media campaign

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Abstract. In this paper, we present and analyze a smoking cessation model with intervention from media campaigns. In our model, we consider potential smokers choosing not to smoke because of media campaign interventions. From the mathematical analysis, we obtain a threshold value of the model, called the basic reproduction number, which is the threshold condition for the smoking-free and endemic equilibrium stability. Next, it is shown that two endemic equilibrium may exist when the threshold basic reproduction number less than unity, and a unique endemic equilibrium exists if the threshold basic reproduction number exceeds unity. Using the center-manifold theory, we show that a backward bifurcation may occur when the value of the basic reproduction number equal to unity. This result implies that the classical epidemiological requirement of making basic reproduction number less than unity is no longer sufficient, although necessary, for effectively controlling the spread of smoking in a population. The numerical simulation was conducted for several scenarios to support and visualize our analytical results.

1. Introduction
Smoking behavior is the activity of sucking tobacco smoke into the body and blowing it back out. Smoking behavior is not something strange anymore to society because, in every place such as a place to eat, a place around the house, road, or city park, we can meet someone smoking. Smoking has been a crucial issue in many countries for many decades. It was estimated more than one hundred million people died due to smoking. Most of these individuals come from rich countries. Over 80% of the 1.3 billion tobacco users worldwide live in low- and middle-income countries, where the burden of tobacco-related illness and death is heaviest.

Meanwhile, cigarettes are very poisonous. One smoke contains approximately “seven thousand harmful chemicals,” and 200 of them have an impact on damaging cells in the body (Saktyowati, 2008). Damaged body cells can trigger several serious diseases such as lung cancer, coronary heart disease, stroke, and liver disease. WHO reports that tobacco kills more than 8 million people each year. More than 7 million of those deaths are the result of direct tobacco use while around 1.2 million are the result of non-smokers being exposed to second-hand smoke. In Indonesia alone, cigarettes are responsible for 14.7% of the total number of deaths or the equivalent of 225,720 people each year, of which 65% are caused by cardiovascular disease.

There are many reasons behind smoking behavior in adolescents. In general, according to Kurt Lewin, smoking behavior is a function of the environment and the individual. This means that smoking behavior is not only caused by internal factors, but also environmental factors.
Examples of environmental factors include; parents who smoke, friends who smoke, and the convenience of getting cigarettes. Much of the evidence for peer pressure to smoke comes from cross-sectional studies which reveal a relationship between the smoking, and non-smoking, behaviour of friends [3], [4], [5].

Many mathematical models have been introduced for smoking cessation. First introduced by Castillo-Garsow et al, 1997 [6] in their research with 3 divisions of individual groups, namely groups of potential smokers or potential smokers ($P$), individual groups of smokers or smokers ($S$), and quitting individual quitters ($Q$). Eleven years later, Sharomi and Gumel[7] introduced a new group of individuals, namely the group of individuals who quit smoking temporarily or quitters temporary ($Q_t$). Also, pay attention to the groups of individuals who smoke every day and individuals who smoke occasionally. Apart from the two papers above, there are many other papers such as [8], [9], [10], [11], [12].

Here in this article, we modify a smoking spread model introduced in [8]. The modification lies on the fact that intensity of media campaign may trigger a potential smoker individual to be a permanent non-smoker individual. We derive the model in detail for each subpopulation, and construct the positive invariant region for the model. Mathematical analysis regarding the qualitative behaviour of the model analyzed, such as existence and local stability of the equilibria, bifurcation analysis using the center-manifold theory, and sensitivity analysis for the basic-reproduction number.

The layout of this article as follows. We derive the model in section 2, and conduct the mathematical analysis regarding the basic reproduction number and existence of equilibrium in section 3. Existence of backward bifurcation of the model when the basic reproduction number is equal to unity analyzed in section 4 using the center-manifold theorem which introduced by author in [23]. Some numerical experiments conducted in section 5 and followed with some discussion and conclusion in section 6.

2. Model construction

Smoking is a habit that can harm yourself, one of which makes the body of a smoker so easily attacked by disease. Smoking habits can be transmitted to others through intense social interaction with smokers. Based on this assumption, here in this article we construct a mathematical model to describe the dynamic of smoking behaviour among closed human population. The model based on a compartmental model with system of non-linear ordinary differential equation approach.

For this reason, here the smoking behaviour model is formulated based on monitoring the dynamics of the proportion of subpopulations of potential smokers ($P(t)$), proportion of smokers ($S(t)$), proportion of smoker who quit for smoke for temporary ($Q_t(t)$), and a proportion of permanent non smoker individual ($Q_p(t)$). This sub-population include individual who quit permanently from smoking or whom decide to not smoke from the beginning caused by their awareness. Therefore, we have that the total of population at time $t$ is given by

$$N(t) = P(t) + S(t) + Q_t(t) + Q_p(t) = 1.$$  

In realistic, the population changes over time at a constant rate of natural birth rate $\mu$. For a model simplification, we assume that the total population is constant over time. Therefore, we assume that the natural death rate of population also given by $\mu$. Furthermore, here in this article we also consider the intervention by policymakers to control the smoking behaviour by campaign a healthy life style or educate community about the danger of smoking using media campaign. Therefore, we introduce the level of media campaign intensity as a time dependent variable $M(t)$ which has its own dynamics. The model constructed in this article is based on the model introduced by authors in [7] which then modified in [11] and later by author in [9].
The dynamic of each sub-population following the transmission diagram given in Figure 1 and described as follows. The number of susceptible individual increases by exponential growth rate \( \mu \), and also decrease caused by natural death rate \( \mu \). Since we assume that all newborn is a potential smoker, we have that \( \frac{dP}{dt} = \mu N - \mu P \). Furthermore, the sub-population is decreases due to intense social contact between potential smokers and a smokers. If \( \frac{P}{N} \) is a probability of single smoker meet the potential smoker individual, then we have that \( b \frac{P}{N} S \) is total counter between smoker and potential smoker per unit time, where \( b \) describe the average number of meet between \( P \) and \( S \) per unit time. If we assume that \( c \) is the success "infection" of a smoker succeeding in making a potential smoker become a smoker, then we have that \( bc \frac{P}{N} S \) describe a rate of new new smoker per unit tame from potential smoker individual due to intensive contact with a smokers. Since we have \( b, c \) and \( N \) is a constant, then we can simplify \( bc/N \) as \( \beta \). Therefore, we have \( \beta PS \) describe infection term from smoker to a potential smoker. As previously mentioned, here we consider the dynamic of media campaign as an effort by policymaker to control the increase of smoking behaviour. The purpose of this media campaign is to develop human awareness on the danger of smoking behaviour. Therefore, if we assume that \( \delta \) as a probability of media campaign succeed to develop human awareness to the danger of smoking, then we have that \( \delta PM \) is a number of potential smoker who decided to be a permanent non-smoking sub-population per unit time due to media campaign. Therefore, we have that the dynamic of potential smoker is given by

\[
\frac{dP}{dt} = \mu(N - P) - \beta PS - \delta PM.
\]

Next, we model how smokers sub-population evolve respect to time. This sub-population increases due to new infection from potential smokers with rate of \( \beta \), and also from sub-population of smoker who quit temporary due to contact with smoker with rate of \( \alpha \). On the other hand, this sub-population decreases due to natural death rate \( \mu \), quit to smoke because of self-awareness that can be triggered by health conditions, the encouragement of family and closest people or other factors except the influence of the media with the rate of \( \gamma \), and also due to effect of media campaign with the rate of \( \eta \). Therefore, the dynamic of smoker is given by

\[
\frac{dS}{dt} = \beta PS + \alpha Q_t S - \eta SM - \gamma S - \mu S.
\]
The temporal quitter sub-population is increase due to transition from smoker sub-population who stop to smoke caused by self-awareness (γ), or effect of media campaign (η). Note that only $1-\sigma$ portion of $\gamma S$ who become temporal quitter, while $\sigma \gamma S$ become a permanent quitter. This sub-population decreased due to "reinfection" from smoker individual with rate of $\alpha$ and become a permanent quitter with the rate of $\epsilon$. Using $\mu$ as the natural death rate, the dynamic of temporal quitter is given as

$$\frac{dQ_t}{dt} = \eta SM + \gamma (1-\sigma) S - \alpha Q_t S - \epsilon Q_t - \mu Q_t.$$

The permanent quitter increases due to transition from potential smoker who decide to not smoke caused by media campaign with rate of $\delta$, smoker who decide to stop smoking permanently with rate of $\gamma (1-\sigma)$, transition from temporal quitter with rate of $\epsilon$. On the other hand, this sub-population only decreases due to natural death rate, since we assume that once individual decide to stop smoke, it will become non-smoker forever. Therefore, the dynamic of permanent quitter is given by

$$\frac{dQ_p}{dt} = \delta P M + \gamma \sigma S + \epsilon Q_t - \mu Q_p.$$

Furthermore, we assume that media campaign is disseminate among potential smoker and smokers. We assume that the level of media campaign is increase due it constant rate of media campaign rate $A$, as a response of increase number of smoker with rate of $\phi$. On the other hand, we assume that effect of media campaign have a duration for it to succeed to encourage human awareness to the danger of smoking. Therefore, we denote $\phi_0$ as a rate of media campaign fading. Hence, we have :

$$\frac{dM}{dt} = A + \phi S - \phi_0 M.$$

Based on description above, the model for the transmission of smoking behaviour incorporating media campaign and self-awareness is given by the following system of nonlinear differential equations :

$$\begin{align*}
\frac{dP}{dt} &= \mu N - \mu P - \beta PS - \delta P M, \\
\frac{dS}{dt} &= \beta PS + \alpha SQ_t - (\mu + \gamma) S - \eta SM, \\
\frac{dQ_t}{dt} &= \eta SM + \gamma (1-\sigma) S - \alpha SQ_t - \mu Q_t - \epsilon Q_t, \\
\frac{dQ_p}{dt} &= \delta P M + \gamma \sigma S + \epsilon Q_t - \mu Q_p, \\
\frac{dM}{dt} &= A + \phi S - \phi_0 M, \\
\end{align*}$$

which supplemented with a non-negative initial condition

$$P(0) \geq 0, S(0) \geq 0, Q_t(0) \geq 0, Q_p(0) \geq 0, M(0) \geq 0. \quad (2)$$

Note that all parameters in system (1) are non-negative which the description is given in Table 1.
Table 1. Description and values of parameters of the smoking behaviour model

| Par. | Description | Value | Reference |
|------|-------------|-------|-----------|
| $\mu$ | Natural newborn / death rate (per year) | $\frac{1}{65}$ | Assumption |
| $\beta$ | Contact rate between $S$ and $P$ | 0.46 | Vinay et al., 2015 [9] |
| $\alpha$ | Contact rate between $S$ and $Q_t$ | 0.34 | Vinay et al., 2015 [9] |
| $\delta$ | Effect of media campaign to $P$ | 0.042 | Assumption |
| $\eta$ | Effect of media campaign to $S$ | 0.042 | Vinay et al., 2015 [9] |
| $\gamma$ | Rate of quitting smoking | 0.03 | Vinay et al., 2015 [9] |
| $\sigma$ | Proportion of quitting smoking permanently | 0.4 | Vinay et al., 2015 [9] |
| $\epsilon$ | Rate of transition from temporal to permanent quitter | 0.23 | Vinay et al., 2015 [9] |
| $A$ | Constant increase of media intensity | 0.046 | Assumption |
| $\phi$ | Increasing rate of media intensity due to number of smoker | 0.3 | Vinay et al., 2015 [9] |
| $\phi_0$ | Fading rate of media intensity | 0.1 | Assumption |

3. Model analysis

3.1. Non-dimensionalization of the smoking model

From Sec. 1, we notice that total of human population is constant. Therefore, we have that $Q_p = 1 - P - S - Q_t$. Next, let us consider $\beta^* = \frac{\beta}{\mu}$, $\alpha^* = \frac{\alpha}{\mu}$, $\delta^* = \frac{\delta}{\mu}$, $\gamma^* = \frac{\gamma}{\mu}$, $\eta^* = \frac{\eta}{\mu}$, $\epsilon^* = \frac{\epsilon}{\mu}$, $\phi^* = \frac{\phi}{\mu}$, $\phi_0^* = \frac{\phi_0}{\mu}$, $A^* = \frac{A}{\mu}$, $\tau = \mu t$. Substituting these into model (1), we have a non-dimensional version of our smoking model given by:

$$
\begin{align*}
\frac{dP}{d\tau} &= 1 - P - \beta^* PS - \delta^* PM \\
\frac{dS}{d\tau} &= \beta^* PS + \alpha^* SQ_t - (1 + \gamma^*) S - \eta^* SM \\
\frac{dQ_t}{d\tau} &= \eta^* SM - \alpha^* SQ_t + \gamma^* (1 - \sigma) S - Q_t - \epsilon^* Q_t, \\
\frac{dM}{d\tau} &= A^* + \phi^* S - \phi_0^* M
\end{align*}
$$

(3)

For convenience of writing, parameters $\beta^*$ will be written as $\beta$ and apply to other parameters as well.

3.2. Basic properties of the model

It is important to make sure our non dimensional model is well posed mathematically and biologically. To guarantee this, we have the following theorem.

**Theorem 1.** Let $(P, S, Q_t, M)$ be the solution of the non-dimensional smoking model in (3) with initial condition given in (2). The closed set

$$
\Omega = \left\{ (P, S, Q_t, M) \in \mathbb{R}_+^4 : 0 \leq P + S + Q_t \leq 1, 0 \leq M \leq \frac{A + \phi}{\phi_0} \right\}
$$

is positively invariant and attracting for the smoking model in (3).
Proof. Using the non-negative initial condition (2), we have:

\[
\begin{align*}
\frac{dP}{dt} &\bigg|_{(P=0,S\geq0,Q_t\geq0,M\geq0)} = 1 > 0, \\
\frac{dS}{dt} &\bigg|_{(P\geq0,S=0,Q_t\geq0,M\geq0)} = 0 \geq 0, \\
\frac{dQ_t}{dt} &\bigg|_{(P\geq0,S\geq0,Q_t=0,M\geq0)} = \eta SM + \gamma(1-\sigma)S, \\
\frac{dM}{dt} &\bigg|_{(P\geq0,S\geq0,Q_t\geq0,M=0)} = A + \phi S \geq 0.
\end{align*}
\]

The above rates are all non-negative over their boundary planes of the non-negative cone \( \mathbb{R}_+^4 \). Therefore, we have that the direction of the vector fields is tended inward from their boundary. Consequently, we have that starting from the non-negative initial conditions, all solutions of system (3) remain positive for all time \( t > 0 \). Furthermore, since \( P + S + Q_t + Q_p = 1 \), and \( P, S, Q_t \) always positive, we have \( 0 \leq P + S + Q_t \leq 1 \). Next, since

\[
\frac{dM}{dt} = A + \phi S - \phi_0 M \leq A + \phi - \phi_0 M,
\]

therefore,

\[
M(t) \leq \frac{A + \phi}{\phi_0}.
\]

Furthermore, we have \( 0 \leq M \leq \frac{A + \phi}{\phi_0} \). Thus, \( \Omega \) is positively invariant and attracting system (3).

This theorem verifies that every solution of our non-dimensional smoking model in (3) with initial condition (2) will remains in \( \Omega \) for all time \( t > 0 \). Furthermore, our solution is always unique in \( \Omega \), therefore system (3) is mathematically and biologically well posed.

### 3.3. Smoking-free equilibrium and the basic reproduction number

System (3) has a smoking-free equilibrium which given by:

\[
\mathcal{E}_0 = (P_0, S_0, Q_{t0}, M_0) = \left(\frac{\phi_0}{\delta A + \phi_0}, 0, 0, \frac{A}{\phi_0}\right)
\]

Since \( Q_p = 1 - P - S - Q_t \), we have that \( Q_{p0} = \frac{\delta A}{\delta A + \phi_0} \). Therefore, the ratio of potential smoker and permanent non-smoker in \( \mathcal{E}_0 \) is

\[
{U} = \frac{P_0}{Q_{p0}} = \frac{\phi_0}{\delta A}.
\]

**Remark 1.** We notice that the ratio of potential smokers and the permanent quitter in the smoking-free equilibrium is given by the ratio between the fading rate of media campaign and multiplication between the effectiveness of media and constant growth of media campaign. Increasing the ability of media campaigns to exert persuasive influence on the dangers of smoking, making advertisements more memorable, and increasing the number of routine campaigns can be used as an option to increase the number of people who stop smoking permanently.
The basic reproduction number, denoted by $R_0$ on our smoking model, describe the average number of new smokers caused by one primary smoker in it period of infection on a completely potential smokers population. Using the next generation matrix approach [13], the basic reproduction number of the non-dimensional smoking model in (3) is

$$R_0 = \frac{\beta \phi_0^2}{(A\delta + \phi_0)(A\eta + \gamma \phi_0 + \phi_0)}.$$  

For further example about application of the next-generation method to calculating the basic reproduction number in some epidemiological model, readers can see [14, 15, 16, 17, 18, 19, 20, 21, 22]. From equation in (7), it can be seen that $\alpha, \phi, \epsilon$ and $\sigma$ do not effect the size of $R_0$. Further discussion of this fact will be discussed in the next section.

3.4. Local stability of smoking-free equilibrium

**Theorem 2.** The smoking-free equilibrium, $E_0$, of smoking model in (3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

**Proof.** Linearizing smoking model (3) in $E_0$ results in

$$J(E_0) = \begin{bmatrix}
-\frac{A\delta}{\phi_0} - 1 & -\frac{\phi_0 \beta}{A\delta + \phi_0} & 0 & -\frac{\delta \phi_0}{A\delta + \phi_0} \\
0 & -\frac{A\eta}{\phi_0} + \frac{\phi_0 \beta}{A\delta + \phi_0} - 1 - \gamma & 0 & 0 \\
0 & \frac{A\eta}{\phi_0} + \gamma (1 - \sigma) & -\epsilon - 1 & 0 \\
0 & \phi & 0 & -\phi_0 \\
\end{bmatrix}.$$  

The eigenvalues are $\lambda_1 = -1 - \frac{A\delta}{\phi_0}$, $\lambda_2 = -1 - \epsilon$, $\lambda_3 = -\phi_0$, and $\lambda_4 = \frac{(R_0 - 1)(A\eta + \phi_0 + \gamma \phi_0)}{\phi_0}$. If $R_0 < 1$, then we have $\lambda_4 < 0$. Consequently, the smoking-free equilibrium is locally asymptotically stable. If $R_0 > 1$, $\lambda_4$ will be positive, and $E_0$ become unstable. 

**Remark 2.** The local stability of the smoking-free equilibrium depends on the size of the basic reproduction number. Smaller this endemic threshold, a bigger chance to reach a condition of smoking-free in the community. Reducing social contact with smokers, increasing the intensity of the media campaign, is some option to achieve this purpose. However, not all parameters in system (3) appear in the basic reproduction number. Effectiveness of contact rate of the smoker to temporal quitter individual, the effectiveness of media campaign, response of policymaker to increase media campaign intensity as a response to the increasing number of smokers, and fading rate of media campaign do not affect the size of basic reproduction number.

3.5. Smoking-endemic equilibrium

The smoking-endemic equilibrium of he non-dimensional smoking model in (3) is given by

$$E^* = (P, S, Q_t, M) = (P^*, S^*, Q_t^*, M^*),$$

where

$$P^* = \frac{\phi_0}{\beta \phi_0 S^* + \delta \phi S^* + \delta A + \phi_0},$$

$$Q_t^* = \frac{S^* (\eta \phi S^* + \gamma \phi_0 (1 - \sigma) + \eta A)}{\phi_0 (\alpha S^* + \epsilon + 1)},$$

$$M^* = \frac{A + \phi S^*}{\phi_0}.$$ 

and $S^*$ satisfies the equation

$$a_0 S^2 + b_0 S + c_0 = 0,$$

with

$$a_0 = (\alpha (\gamma \sigma + 1) \phi_0 + \eta \phi (\epsilon + 1)) (\beta \phi_0 + \delta \phi) > 0,$$

$$b_0 = \alpha \beta \phi_0^2 (R_c - 1),$$

$$c_0 = (A \delta + \phi_0) (A \eta + \phi_0 (\gamma + 1)) (\epsilon + 1) (1 - R_0),$$

where

$$R_0 = \frac{\beta \phi_0^2}{(A \delta + \phi_0) (A \eta + \gamma \phi_0 + \phi_0)},$$

$$R_c = \frac{1}{\alpha \beta \phi_0^2} \left(\beta \epsilon (1 + \gamma) + (\sigma \alpha + \beta) \gamma + \beta + \alpha\right) \phi_0^2 + 2 A \delta \eta \phi (\epsilon + 1) + \left(\left((1 + \gamma) (\epsilon + 1) \phi + A \alpha (\gamma \sigma + 1)\right) \delta + \eta (\epsilon + 1) (A \beta + \phi)\right) \phi_0.$$

Note that $a_0 > 0$, $c_0 < 0 \iff R_0 > 1$. and $b_0 > 0 \iff R_c > 1$. It follows that:

(i) There is a unique smoking endemic equilibrium if $R_0 > 1$

(ii) There is a unique smoking endemic equilibrium if $R_0 = 1$ and $R_c < 1$ or $b_0^2 - 4a_0c_0 = 0$.

(iii) There are two smoking endemic equilibrium if

$$R_0 < 1, R_c < 1 \text{ and } b_0^2 - 4a_0c_0 > 0.$$ 

(iv) Otherwise, there are no smoking endemic equilibrium point.

Results on this section summarized in the following theorem.

**Theorem 3.** If $R_0 < 1$, the $E_0$ is locally asymptotically stable. On the other hand, there exist a unique smoking-endemic equilibrium if conditions (10) hold, or two smoking-endemic equilibrium if conditions (11) hold. If $R_0 > 1$, then $E_0$ become unstable, and it exist a unique smoking-endemic equilibrium.

In the next section, we will continue our analysis on the stability of the smoking-endemic equilibrium. We will show that the existence of smoking-endemic equilibrium when $R_0 < 1$ comes from a backward bifurcation.

### 4. Bifurcation analysis

From result on Theorem 2 and 3 it appears that $R_0 = 1$ is a point where change of stability of $E_0$ and appearance of new smoking-endemic equilibrium occur. Therefore, we will analyze a type of bifurcation when $R_0 = 1$. First, let use $\beta$ as the bifurcation parameter at $R_0 = 1$ which equivalent to:

$$\beta^* = \frac{(\delta A + \phi_0)(\eta A + \gamma \phi_0 + \phi_0)}{\phi_0^2},$$

so that the $E_0$ is locally asymptotically stable if $\beta < \beta^*$, and unstable otherwise.
We investigate the behaviour of the bifurcation by using the method introduced by author in [23]. The method is based on Center manifold theory, and summarized in Theorem 4. In this theorem, we have to calculate two crucial component, let say it \( A \) and \( B \). These coefficient will decide the direction of the bifurcation when \( R_0 = 1 \). In particular, if \( A < 0 \) and \( B > 0 \), then forward bifurcation appear at \( R_0 = 1 \). On the other hand, when \( A > 0 \) and \( B > 0 \), then backward bifurcation occur. On our non-dimensional smoking model in (3), we have the following theorem.

**Theorem 4.** If \( R_0 < 1 \) and 
\[
A := -2 \left( (1 + \gamma) \phi_0^2 + ((1 + \gamma) \delta + \eta) A + \phi \delta \right) \phi_0 + A^2 \delta \eta \left( (1 + \gamma) \phi_0 + A \eta \right), \\
+2 \frac{\alpha (1 - \sigma) \phi_0 + A \eta}{\epsilon + 1} \phi - \frac{2 \phi_0 \eta}{\phi} > 0, 
\]
then smoking model in (3) exhibits a backward bifurcation at \( R_0 = 1 \). On the other hand, if \( A < 0 \), then system will exhibit a forward bifurcation at \( R_0 = 1 \).

**Proof.** First of all, observe that the eigenvalues of the following Jacobian matrix of system (3) which evaluated at \( \epsilon_0 \) and \( \beta = \beta^* \)
\[
J(\epsilon_0, \beta^*) = 
\begin{bmatrix}
-A\delta + \phi_0 & \frac{(-1-\gamma)\phi_0 - A\eta}{\phi_0} & 0 & -\frac{\delta \phi_0}{A\delta + \phi_0} \\
0 & 0 & 0 & 0 \\
0 & \frac{A\eta}{\phi_0} - \gamma (\sigma - 1) & -\epsilon - 1 & 0 \\
0 & \frac{-\epsilon + 1}{\phi} & 0 & -\phi_0
\end{bmatrix}
\]
is given by 
\[
\lambda_1 = -\frac{A\delta + \phi_0}{\phi_0}, \quad \lambda_2 = 0, \quad \lambda_3 = -(\epsilon + 1), \quad \lambda_4 = -\phi_0.
\]
Obviously, \( \lambda_2 \) is a simple zero eigenvalues and all others eigenvalues of \( A \) have negative real parts. Hence, the method proposed in [23] can be implemented in this model. Denote by \( \vec{w} = (w_1, w_2, w_3, w_4)^T \) and \( \vec{v} = (v_1, v_2, v_3, v_4) \), as the nonnegative right and left eigenvector corresponding to the zero eigenvalue, respectively, such that \( \vec{v} \cdot \vec{w} = 1 \). From direct calculation, we obtain :
\[
w_1 = -\phi \left( (1 + \gamma) \phi_0^2 + ((1 + \gamma) A + \phi \delta) \phi_0 + A^2 \delta \eta \right) w_4 \\
(A\delta + \phi_0)^2 \phi \\
w_2 = \frac{\phi_0}{\phi} w_4, \\
w_3 = \frac{(A\eta + \gamma (1 - \sigma) \phi_0) w_4}{(\epsilon + 1) \phi}, \\
w_4 > 0.
\]

Next, we calculate \( A \) and \( B \) as explained in [23], using the following formula
\[
A = \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\epsilon_0, \beta^*), \quad B = \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \psi}(\epsilon_0, \beta^*).
\]
Since \( v_1 = v_3 = v_4 = 0 \), we only need the partial derivatives of \( f_2 \) to find the values of \( A \) and \( B \). It follows that

\[
\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \frac{(\delta A + \phi_0) (\eta A + \gamma \phi_0 + \phi_0)}{\phi_0^2},
\]
\[
\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = \alpha,
\]
\[
\frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\eta.
\]

and

\[
\frac{\partial^2 f_2}{\partial x_2 \partial \beta} = \frac{\phi_0}{A\delta + \phi_0}.
\]

Hence, we can calculate \( A \) and \( B \) which give us the result as follows.

\[
B = \sum_{k,i=1}^{4} v_kw_i \frac{\partial^2 f_2}{\partial x_i \partial \beta}(E_0, \beta^*) = \frac{\phi_0}{\phi} \frac{\phi_0}{A\delta + \phi_0} > 0,
\] (15)

and

\[
A = \sum_{k,i,j=1}^{4} v_kw_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(E_0, \beta^*)
\]

\[
= v_2 \left( 2w_1w_2 \frac{(\delta A + \phi_0) (\eta A + \gamma \phi_0 + \phi_0)}{\phi_0^2} + 2w_2w_3\alpha - 2w_2w_4\eta \right)
\]

\[
= -2 \left( (1 + \gamma) \phi_0^2 + ((1 + \gamma) \delta + \eta) A + \phi \phi_0 + A^2\delta \eta \right) \left( (1 + \gamma) \phi_0 + A\eta \right)
\]

\[
+ 2 \frac{\alpha (\gamma (1 - \sigma) \phi_0 + A\eta) \phi_0}{(\epsilon + 1) \phi_0}. \] (16)

It can be seen from equation (15) that \( B \) always positive. Therefore system (3) exhibits backward or forward bifurcation according to the sign of \( A \). If \( A > 0 \), then we have system (3) exhibit a backward bifurcation at \( R_0 = 1 \), and exhibit forward bifurcation if \( A < 0 \).

The result in this section, regarding the bifurcation analysis of system (3) indicates how the basic reproduction number in (7) do not always become sufficient requirement to eradicate the smoking habit in the field. The reason is simply because when the backward bifurcation occur, there is still a possibility that the smoking habit may appear even the basic reproduction number is already less than unity.

5. Numerical Analysis

In this section, we conduct numerical simulation for the sensitivity analysis of the basic reproduction number in (7), and also the autonomous simulation of system (3).

5.1. Sensitivity analysis

Elasticity and sensitivity analyzes can be performed on static quantities such as \( R_0 \). The static quantity \( R_0 \) depends on the parameter values of the differential equation model.

First, we do an elasticity analysis to determine the percentage change of \( R_0 \) against the \( p \) parameter changes. To perform this experiment, we use parameters value in Table 1 which
Table 2. The value of non-dimensionalized parameters in model (3)

| Parameter | Value |
|-----------|-------|
| $\beta^*$ | 29.90 |
| $\eta^*$  | 2.730 |
| $\alpha^*$| 22.10 |
| $\sigma$  | 0.4  |
| $\gamma^*$| 1.95l|
| $\epsilon^*$| 14.95|
| $\phi$    | 19.5 |
| $\phi^*$  | 0.65 |
| $A^*$     | 3    |
| $\delta^*$| 2.730|

Table 3. Results of $R_0$ Elasticity Analysis Respect to All Parameters in Model (3)

| $R_0$ | $\varepsilon_A^{\beta}$ | $\varepsilon_A^\gamma$ | $\varepsilon_A^\eta$ | $\varepsilon_A^\alpha$ | $\varepsilon_A^\delta$ | $\varepsilon_A$ |
|-------|--------------------------|--------------------------|-----------------------|-------------------------|------------------------|---------------|
| $R_0 < 1$ | -0.85681                 | -0.55752                 | -0.29929              | -0.46318                | 0.85681                | 0 0 0 1       |
| $R_0 > 1$  | -0.56248                 | -0.38650                 | -0.17598              | -0.54469                | 0.56248                | 0 0 0 1       |

in non-dimensional version is given in Table 2. Therefore, we get the results of $R_0$ elasticity analysis in Table 3.

The positive/negative signs of elasticity index in Table 3 has its own interpretation. Positive sign means that increasing the parameter will increase $R_0$, and vice versa. The following is given an example when $R_0 < 1$. For a negative result that is the value $\varepsilon_A^{\beta} = -0.85681$ this means that every 1% increase in the $\beta$ parameter will result in a decrease of 0.85681% on $R_0$. On the other hand, since the value of $\varepsilon_A^\alpha$ is equal to one, then this means that each 1% increase in the $\alpha$ parameter will result in an increase of 1% on $R_0$. Meanwhile, for the elasticity result with a value of 0, namely $\varepsilon_A^\alpha = 0$ means that any change in the value of $\alpha$ does not affect the value $R_0$.

It can be seen that the rate of social contact between the proportion of smokers and potential smokers ($\beta$) and the number of routine campaigns ($A$) respectively have the most influential positive and negative results. That means that $beta$ and $A$ have the most significant effect on the change of $R_0$. Therefore, it is important to analyze the relation between these parameters to $R_0$. Hence, we plot the sensitivity contour map of $R_0$ respect to $A$ and $\beta$ as shown in Figure 2. It can be seen that increasing $\beta$ will increase $R_0$, while increasing $A$ will reduce $R_0$. However, Figure 2 does not provide practical information to determine the exact combination between $\beta$ and $A$ such that $R_0 < 1$. Therefore, we continue the sensitivity analysis by depicting the $R_0 = 1$ curve as shown in Figure 3.

As shown in Figure 3 we can divide the area into four regions. The blue region represents a condition when a stable SFE always occurs for all values of $A$ whenever the average meeting between smokers and potentially smoking individuals are rare and or decreases the persuasive ability of $S$ to invite $P$ to smoke, which is characterized by $\beta < \beta_1$. Meanwhile, the beige region indicates when stable SEE always occurs for all values of $A$, whenever $\beta > \beta_2$. Biologically, this means that if the average encounter between a smoker and an individual is potentially
Figure 2. Sensitivity Contour Map of $R_0$ respect to $A$ and $\beta$

smoking and or the persuasive ability of $S$ to invite $P$ to smoke reaches a value, then the rate of social contact between $S$ and $P$ cannot make the condition free of smoking behavior. Pink and purple regions indicate situations where there is an SFE or SEE that relies on a combination of parameter $\beta$ and $A$.

5.2. Autonomous simulation
In this section, we perform some examples of simulation regarding the behaviour of our model in system (1).

(i) Existence of bistability phenomena. In this paper, it is possible to have a condition that even though $R_0 < 1$, there exist a stable SFE and two SEE where one of them is stable, and the other is unstable (See Figure 1). In this case, a bistability phenomena occurs when backward bifurcation appears. In many mathematical models [24, 25], backward bifurcation is very crucial for devising the control strategies. When backward bifurcation appears, the initial condition of population could determine the final state of population. In our case, bistability phenomena occur when $R_1 < R_0 < 1$, when $R_0 = R_1 = R$ is a condition which fulfill $b_0^2 - 4a_0c_0 = 0$, where $a_0, b_0, c_0$ is taken from polynomial in (9). Figure 5 show several solutions on the number of smokers based on system (3) with various initial condition. It can be seen that in a same set of parameter value, system (3) tends to two different final state, i.e the smoking-free equilibrium and the smoking-endemic equilibrium.

(ii) Effect of Constant Increase of Media Intensity. In this case, we do autonomous simulation with variation of $A$. The result given in Figure 6. The increasing frequency of warnings about the dangers of smoking (which is characterized by an increase of $A$) automatically makes campaigns about the dangers of smoking even more intense. The campaign has become increasingly intense, making the proportion of smokers ($S$) more aware of the dangers of smoking until deciding to quit smoking and become the proportion of individuals quitting temporarily ($Q_t$). Meanwhile, an increasingly intense campaign has raised awareness of the dangers of smoking among the proportion of individuals of potential smoker ($P$) and choosing not to smoke. In conclusion, the increasing frequency of warnings about the dangers of smoking, the less likely it is that the community’s smoking habit is so that the habit can disappear from the population.
Figure 4. Backward Bifurcation Diagram of System (3)

Figure 5. Asymptotic behavior on the number of smokers with different set values of initial condition: \( P(0) = 0.4, S(0) = [0.001, 0.02], Q_t(0) = 0.01, M(0) = 0.4 \)

Figure 6. The dynamic of system (3) for different values of constant media campaign \( A \).

(iii) Effect of Contact Rate between \( S \) and \( P \). The more active the government policy on providing non-smoking areas (as indicated by a reduced of \( \beta \)), the less likely the proportion of potential smoking (\( P \)) individuals will become smokers. We can see from Figure (7) that reducing value of \( \beta \) could maintain number of potential smoker in a high proportion, while
Figure 7. The dynamic of system (3) for different values of infection rate $\beta$.

number of active smokers, and temporal quitter reduced significantly.

6. Discussion and concluding remarks

In this article, we modify a smoking transmission model by author in [8] by adding the effect of the media campaign to trigger potential smokers to be a permanent ”quitter,” or in this case, equivalent with a permanent immune population. The modified model is rigorously analyzed to gain insight into its many dynamical features, such as the existence and local stability of the equilibrium points, bifurcation analysis, and sensitivity analysis.

Mathematical analysis reveals that the smoking model in system (1) could be simplified into a non-dimensional version in (3). This non-dimensionalized model has a locally stable smoking-free equilibrium whenever the associate basic reproduction number $R_0$ in (7) less than unity. Theorem 2 verify that $R_0$ is the key threshold of eradicating the number of smokers. Furthermore, the non-dimensionalized smoking model (3) undergoes backward bifurcation at $R_0 = 1$ whenever $R_0 < 1$ and $A > 0$ which stated in Theorem 3. From the expression of $A$ in Theorem 3, it can be seen that the transition rate from temporal to permanent quitter (\(\epsilon\)), contact rate between a temporal quitter and smokers (\(\alpha\)) and response of the government to increase media campaign due to the number of smokers in the field (\(\phi\)) may trigger the occurrence of backward bifurcation phenomena.

Using the appropriate parameter values, local sensitivity analysis conducted to determine the most influential parameter in determining the value of the basic reproduction number. From our analysis, we reveal that $\beta$ is the most influential parameter in $R_0$. Hence, reducing $\beta$, for example, providing a more closed-smoking area, could be a promising intervention to control the spread of smokers.
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