IOSR Journal of Mathematics (IOSR-JM)
e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 18, Issue 1 Ser. I (Jan. – Feb. 2022), PP 28-38
www.iosrjournals.org

Mathematical Analysis of a Smoking Model with Social Factor

Muhammad Said*, Jae Hun Jung, and Il Hyo Jung
Department of Mathematics, Pusan National University, Busan 46241, Republic of Korea

Abstract: *The mathematical modeling of cigarette smoking has an important role in understanding the transmission dynamics and developing effective prevention methods. In this study, a non-linear mathematical model is used to examine the extent of smoking from person to person and in a group of large social networks, as well as the social influence on temporary quit smokers. Firstly, the positivity and boundedness of the model solution are determined by the complete differential process, followed by the computation of the infection-free and endemic equilibrium of the system. The basic reproductive number is calculated by the next-generation matrix method. The local and global stability of the system is also presented. For local asymptotical stability, linearization and Ruth-Hurwitz criterion are used, which show that when* $R_0 < 1$, *the system is locally asymptotically stable, otherwise unstable. Furthermore, for global asymptotical stability, the geometrical approach is used. We evaluate the sensitivity analysis to figure out the rule of most sensitive parameters in the transmission of smoking. Finally, numerical simulation is performed to show the feasibility of our analytical work. The results show that when* $R_0 = 2.25 > 1$, *a significant increase is observed in the population of light and chain smokers.*

Keywords: *Smoking model, social factor, smoking generation number, stability analysis, sensitivity analysis, numerical simulation.*

Date of Submission: 01-01-2022 Date of Acceptance: 12-01-2022

I. Introduction

Cigarette smoking is the single most serious preventable health risk of the modern era, as well as a primary cause of premature mortality worldwide that kills more than five million people each year, with the figure expected to rise [1]. Smoking causes several diseases including stroke, diabetes, premature, chronic obstructive pulmonary diseases, chronic obstructive lung diseases and asthma. Tobacco use also increases the risk of death from cancers of the mouth, lung, stomach, kidney, pancreas, cervix, and liver. In Australia, approximately 14,900 people died from smoking-related diseases in the financial year 2004-2005. Heart disease kills around 4,000 people each year, which is the main cause of heart attack. Lung cancer is related to around 6,057 deaths, COPD is related to 3,870 deaths, and stroke is related to 580 deaths. In 2001, roughly 22 percent of adults in Australia were shown to be smokers. Males are about 24.3% more likely to smoke than females (19.9 %). Around one-third of 17-year-old students smoked, and the 20-29 year old age group had the highest smoking rates [2].

Smoking behavior spreads through both close and distant social links, as groups of related people begin to smoke together, and smokers become progressively socially isolated [3,4]. Young individuals begin smoking for the sake of having pleasure. Some people smoke in order to regulate or lose weight, as smoking suppresses hunger along with the senses of taste and smell. The majority of individuals smoke to unwind and relieve the stress and anxiety of daily life. However, they are not aware of the several health problems prior to adopting the habit of smoking. Therefore, different health care programs and strategies have been implemented in several countries to spread awareness regarding smoking and its adverse effects [5].

Many epidemic models have been presented to study the dynamics of smoking. In 2000, Castillo-Garsow presented the first simple mathematical model to quit smoking [6]. In their model, the total population was divided into three classes of smokers, namely potential smokers (P), smokers (S), and quitters (Q). Sharomi and Gummel proposed a mathematical model by introducing mild and chain classes [7]. Later, Zaman extended the work of Castillo-Garsow and derived a mathematical model, taking into account the occasional smokers, and studied its qualitative behavior [8]. In this paper, we use a mathematical model to investigate two types of smoking transmission in a community. It is important to verify that all solutions of the model with nonnegative initial conditions remain nonnegative for all time; therefore, the model's positivity and boundedness are discussed. Then, the system's different equilibrium points are examined and the threshold parameter R_0 is evaluated by the Driessche and Watmough method. We use Ruth-Hurwitz criteria for the local stability of the

model. The global stability is also investigated by the geometrical approach. We discuss sensitivity analysis to observe how essential each epidemic parameter is in smoking transmission. Finally, for easy understanding, all theoretical results are evaluated using numerical simulation.

II. Model Formulation and Analysis

The total population of smokers is divided into five classes of smokers, namely non-smokers P, light smokers S_l, chain smokers S_c, temporary quit smokers Q_t, and permanent quitter Q_p. The non-smokers begin smoking when they are in contact with a smoker or are influenced by a large group of social smokers in a community ($at\ rate\ \beta_1 and\ \beta_2$). The number of light smokers S_l do not smoke a pack every day, but sooner or later, they become chain smokers ($at\ rate\ \alpha$). The chain smoker is highly addicted and smokes about three packs a day. This smoker is physically addicted to nicotine and convinces himself that he must smoke. The chain smokers reach for a cigarette immediately upon awakening and often smoke in the middle of the night. Moreover, they either quit smoking temporarily or permanently quit smoking ($at\ rate\ \phi\ and\ \xi$). The temporary quitting smokers may become light smokers again ($at\ rate\ \delta$), while others permanently quit smoking ($at\ rate\ \phi$). The schematic diagram of the proposed model is given below in Figure no 1.

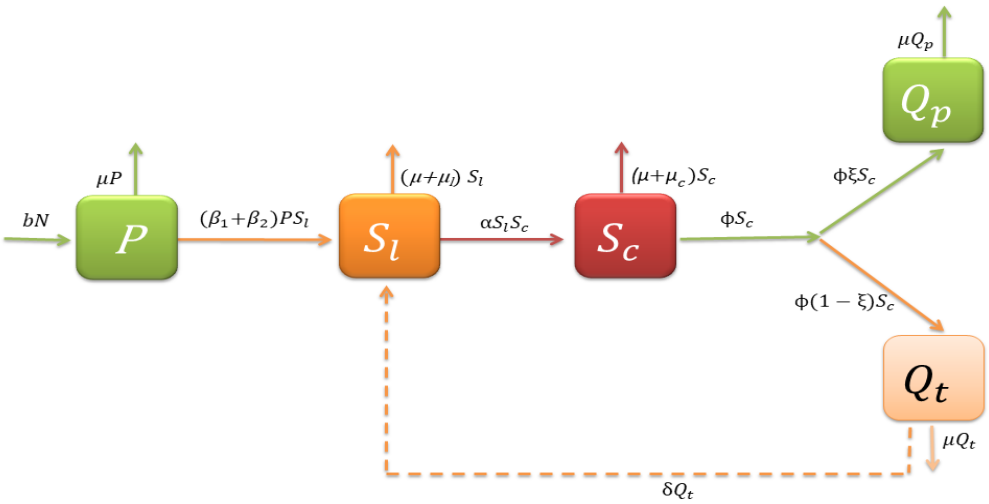


Figure 1. Schematic diagram of the model (1).

We construct the following mathematical model:

$$\begin{aligned}
\frac{dP}{dt} &= bN - (\beta_1 + \beta_2)PS_l - \mu P,\\
\frac{dS_l}{dt} &= (\beta_1 + \beta_2)PS_l + \delta Q_t - \alpha S_l S_c - (\mu + \mu_l)S_l,\\
\frac{dS_c}{dt} &= \alpha S_l S_c - \phi S_c - (\mu + \mu_c)S_c,\\
\frac{dQ_t}{dt} &= \phi(1-\xi)S_c - \delta Q_t - \mu Q_t,\\
\frac{dQ_p}{dt} &= \phi\xi S_c - \mu Q_p.
\end{aligned} \tag{1}$$

with initial conditions

$$P(0) \geq 0, S_l \geq 0, S_c(0) \geq 0, Q_t(0) \geq 0, Q_p(0) \geq 0.$$

The different parameters used in the model are described below in the table.

Parameter	Description	Unit
b	Birth rate	time^{-1}
μ	Natural death rate	time^{-1}
μ_l	Disease death rate of light smokers	time^{-1}
μ_c	Disease death rate of chain smokers	time^{-1}
β_1	Transmission rate from person to person	1
β_2	Transmission rate in group of social network	1
α	Transmission rate from light to chain smokers	1
ϕ	Quit smoking rate	time^{-1}
$(1-\xi)$	Fraction of smokers who temporarily quit smoking at ϕ	time^{-1}
ξ	Permanent quit smoking rate	time^{-1}
δ	Rate at which temporary quitters revert back to smoking	time^{-1}

Table 1. Parameters description and units.

1. Equilibrium analysis:

The smoking free equilibrium of model (1) is given by

$$E_0 = \left(\frac{bN}{\mu}, 0,0,0,0,0\right). \tag{2}$$

The endemic equilibrium E_* of the model (1) is given by

$$E_* = \left(P^*, S_l^*, S_c^*, Q_t^*, Q_p^*\right). \tag{3}$$

Where

$$P^* = \frac{bN}{\mu - (\beta_1+\beta_2)S_l^*}, \qquad S_l^* = \frac{\delta Q_t^*}{(\mu+\mu_c) + \alpha S_c^* - (\beta_1+\beta_2)P^*}, \qquad S_c^* = \frac{(\delta+\mu)Q_t^*}{\phi(1-\xi)},$$

$$Q_t^* = \frac{\phi(1-\xi)S_c^*}{\delta-\mu}, \qquad Q_p^* = \frac{\phi\xi S_c^*}{\mu}.$$

2. Positivity and Boundedness of solution:

Theorem: For all time t ≥ 0, there exists a domain D:

$$D = \left\{\left(P, S_l, S_c, Q_t, Q_p\right) \in R_+^5, 0 < \left(P + S_l + S_c + Q_t + Q_p\right) \leq \frac{bN}{\mu}\right\}.$$

Proof: Let $\left(P, S_l, S_c, Q_t, Q_p\right)$ be any solution with a positive initial condition $P \geq 0, S_l \geq 0, S_c \geq 0, Q_t \geq 0, Q_p \geq 0$. with $\left(P, S_l, S_c, Q_t, Q_p\right) = \left(P + S_l + S_c + Q_t + Q_p\right)$, since $N(t)$ represents the total population. Now differentiating with respect to time t, we get

$$\frac{dN(t)}{dt} = (b-\mu)N(t) - (\mu_l S_l + \mu_c S_c) \leq (b-\mu)N(t). \tag{4}$$

Equation (4) can be expressed as:

$$\left(\frac{dN(t)}{dt}\right) + \frac{1}{N(t)} \leq b - \mu. \tag{5}$$

Solving the above inequality yields:

$$N(t) \leq (b-\mu)(1-e^{-\mu t}) + N^{-1}(0)e^{-\mu t}. \tag{6}$$

Consequently, taking the limits $t \to \infty$, gives $N(t) \leq (b-\mu)$. Thus, D is positively invariant, and all the solutions are bounded in the interval $[0, \infty]$.

3. Basic reproductive number:

The basic reproductive number or smoking generation number is found by the next generation matrix method [9]. Here, let $X = (P, S_l, S_c, Q_t)$.

$$\bar{F} = \begin{bmatrix} (\beta_1 + \beta_2)PS_l \\ (\beta_1 + \beta_2)PS_l + \alpha S_l S_c \\ \alpha S_l S_c \\ 0 \end{bmatrix}, \quad \bar{V} = \begin{bmatrix} bN + \mu P \\ \delta Q_t + (\mu + \mu_l)S_l \\ \phi S_c + (\mu + \mu_c)S_c \\ \phi(1-\xi)S_c + \delta Q_t + \mu Q_t \end{bmatrix}.$$

The Jacobean matrix for $\bar{F}$ and $\bar{V}$ for infectious free equilibrium point is given by

$$J_{\bar{F}} = \begin{bmatrix} 0 & \frac{(\beta_1 + \beta_2)bN}{\mu} & 0 & 0 \\ 0 & \frac{(\beta_1 + \beta_2)bN}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

And

$$J_{\bar{V}} = \begin{bmatrix} \mu & 0 & 0 & 0 \\ 0 & (\mu + \mu_l) & -\delta & 0 \\ 0 & 0 & (\phi + \mu + \mu_c) & 0 \\ 0 & \phi(1-\xi) & 0 & \delta + \mu \end{bmatrix},$$

Where

$$J_{\bar{V}}^{-1} = \begin{bmatrix} \frac{1}{\mu} & 0 & 0 & 0 \\ 0 & \frac{1}{(\mu + \mu_l)} & \frac{-\delta}{(\mu + \mu_l)(\phi + \mu + \mu_c)} & 0 \\ 0 & 0 & \frac{1}{(\phi + \mu + \mu_c)} & 0 \\ 0 & \frac{\phi(\xi - 1)}{(\delta + \mu)(\mu + \mu_l)} & \frac{\delta\phi(\xi - 1)}{(\delta + \mu)(\mu + \mu_l)(\phi + \mu + \mu_c)} & \frac{1}{(\mu + \delta)} \end{bmatrix}.$$

Therefore, we consider the smoking generation number for our proposed model as follows:

$$R_0 = \frac{(\beta_1 + \beta_2)bN}{\mu^2(\mu + \mu_l)(\delta + \mu)(\phi + \mu + \mu_c)}. \tag{7}$$

4. Stability Analysis:

The following theorems are used to discuss the system local and global qualitative analysis at the disease-free and endemic equilibrium.

Theorem 1. If $R_0 < 1$, then model (1) is locally asymptotically stable at disease-free equilibrium point E_0, otherwise unstable.

Proof. To prove the local stability of the model (1) at smoking-free equilibrium, linearizing the proposed model (1) around the disease-free equilibrium E_0, we get the following matrix J_1,

$$J_1 = \begin{bmatrix} -\mu & 0 & 0 & 0 & 0 \\ 0 & -(\mu + \mu_l) & \delta & 0 & 0 \\ 0 & 0 & -(\phi + \mu + \mu_c) & 0 & 0 \\ 0 & \phi(1-\xi) & 0 & -\delta - \mu & 0 \\ 0 & 0 & \phi\xi & 0 & -\mu \end{bmatrix}.$$

The characteristic equation of the above matrix J_1 is given by,

$$\det[J_1 - \lambda I] = 0.$$

As all the eigenvalues have negative real parts, thus, the system (1) at disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$. □

Theorem 2. If $R_0 > 1$, then the model (1) is locally asymptotically stable at disease-free equilibrium point E_*, otherwise unstable.

Proof. To prove the local stability of the model (1) at smoking-free equilibrium, linearizing the proposed model (1) around the disease-free equilibrium E_*, we get the following matrix J_1,

$$J_2 = \begin{bmatrix} -\mu-(\beta_1+\beta_2)S_l & -(\beta_1+\beta_2)P & 0 & 0 & 0 \\ -(\beta_1+\beta_2)S_l & -(\mu+\mu_l)-(\beta_1+\beta_2)P & \delta & 0 & 0 \\ 0 & 0 & -(\phi+\mu+\mu_c) & 0 & 0 \\ 0 & \phi(1-\xi) & 0 & -\delta-\mu & 0 \\ 0 & 0 & \phi\xi & 0 & -\mu \end{bmatrix}.$$

Auxiliary equation of matrix J_2 takes the following form,

$$(\lambda+\mu)(\lambda+\delta+\mu)(\lambda+\phi+\mu+\mu_c)a_1\lambda + a_2 = 0,$$

Where

$$a_1 = -(-\mu-(\beta_1+\beta_2)S_l) - (-(\mu+\mu_l)-(\beta_1+\beta_2)P),$$
$$a_2 = (-\mu-(\beta_1+\beta_2)S_l)(-(\mu+\mu_l)-(\beta_1+\beta_2)P) - (-(\beta_1+\beta_2)P)(-(\beta_1+\beta_2)S_l).$$

By Ruth-Hurwitz criteria [10], the equilibrium state E_* is locally asymptotically stable if $a_1, a_2 > 0$. Hence, for $R_0 > 1$, the model (1) is locally asymptotically stable. □

Lemma 1: If $\dot{x} = F(x)$, $[F: D \to R^n]$ containing an endemic equilibrium of the form ẋ and there is a compact absorbing set, then this system is globally asymptotically stable around that equilibrium if there exist a function $D(x)$ and a Lozinskii measure l such that $lim_{t\to\infty} supsup \frac{1}{t}\int_0^t l(B)dt < 0$. We discuss the function D, B *and* l in the next section.

Theorem 3. If $R_0 > 1$, then the smoking model (1) is globally asymptotically stable at endemic equilibrium E_*.

Proof. Let J be the Jacobean matrix and $J^{|2|}$ be the second additive compound matrix contained in first three equations of model (1).

$$J = \begin{bmatrix} -\mu-(\beta_1+\beta_2)S_l & -(\beta_1+\beta_2)P & 0 \\ (\beta_1+\beta_2)S_l & -(\mu+\mu_l)-\alpha S_c+(\beta_1+\beta_2)P & \alpha S_l \\ 0 & \alpha S_c & -\phi-(\mu+\mu_c)+\alpha S_l \end{bmatrix},$$

$$J^{|2|} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix},$$

$$\Rightarrow J^{|2|} = \begin{bmatrix} h_1 & \alpha S_l & 0 \\ \alpha S_c & h_2 & -(\beta_1+\beta_2)P \\ 0 & (\beta_1+\beta_2)S_l & h_3 \end{bmatrix}.$$

Where,

$$h_1 = -\mu-(\beta_1+\beta_2)S_l-(\mu+\mu_l)-\alpha S_c+(\beta_1+\beta_2)P,$$

$$h_2 = -\mu-(\beta_1+\beta_2)S_l-\phi-(\mu+\mu_c)+\alpha S_l,$$

$$h_3 = -(\mu+\mu_l)-\alpha S_c+(\beta_1+\beta_2)P-\phi-(\mu+\mu_c)+\alpha S_l.$$

Let us consider the function $F(X) = (P, S_l, S_c) = diag\{\frac{P}{S_l}, \frac{P}{S_l}, \frac{P}{S_l}\}$, which implies that $F^{-1}(X) = diag\{\frac{S_l}{P}, \frac{S_l}{P}, \frac{S_l}{P}\}$. Taking the time derivative, we have

$$F'(X) = diag\left\{\frac{\dot{P}}{P} - \frac{P\dot{S}_l}{S_l^2}, \frac{\dot{P}}{P} - \frac{P\dot{S}_l}{S_l^2}, \frac{\dot{P}}{P} - \frac{P\dot{S}_l}{S_l^2}\right\}. \quad (8)$$

Now $F'F^{-1} = diag\left\{\frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l}, \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l}, \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l}\right\}$, and $FJ^{|2|}F^{-1} = J^{|2|}$, thus we can take $B = F^{'}F^{-1} + FJ^{|2|}F^{-1}$, which can be written as

$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}$, where $B_{11} = \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \mu - (\beta_1+\beta_2)S_l - (\mu+\mu_l) - \alpha S_c + (\beta_1+\beta_2)P$, $B_{12} = (\alpha S_l \quad 0)$,

$B_{21} = (\alpha S_c \quad 0)^t$, and

$$B_{22} = \begin{pmatrix} -\mu-(\beta_1+\beta_2)S_l-\phi-(\mu+\mu_c)+\alpha S_l, & -(\beta_1+\beta_2)P \\ (\beta_1+\beta_2)S_l & -(\mu+\mu_l)-\alpha S_c+(\beta_1+\beta_2)P-\phi-(\mu+\mu_c)+\alpha S_l \end{pmatrix}$$

Let (x_1, x_2, x_3) be a vector in R^3 and its norm $\|.\|$ defined by

$$\|x_1, x_2, x_3\| = \max\{\|x_1\|, \|x_2\| + \|x_3\|\}.$$

Let l be the Lozinskii measure with respect to the above norms as described in [11]. We have $l(B) \leq \sup\{g_1, g_2\} = \sup\{l(B_{11}) + \|l(B_{12})\|, l(B_{22}) + \|l(B_{21})\|\}$, where $g_i = l(B_{ii}) + \|l(B_{ij})\|$, for $i = 1,2$ *and* $i \neq j$, which implies that

$$g_1 = l(B_{11}) + \|l(B_{12})\|, g_2 = \{l(B_{22}) + \|l(B_{21})\|\},$$

Where

$l(B_{11}) = \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \mu - (\beta_1 + \beta_2)S_l - (\mu + \mu_l) - \alpha S_c + (\beta_1 + \beta_2)P$, $l(B_{12}) = \alpha S_l$,

$$l(B_{22}) = max(\frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \mu - \phi - (\mu + \mu_c) + \alpha S_l, -\frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - (\mu + \mu_l) - \alpha S_c - \phi - (\mu + \mu_c) + \alpha S_l)$$

$\Rightarrow l(B_{22}) = \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \phi - (\mu + \mu_c) + \alpha S_l(-\mu, -(\mu + \mu_l) - \alpha S_c,$ *and* $\|l(B_{21})\| = \alpha$ Therefore, g_1 *and* g_2 becomes,

$$g_1 = \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \mu - (\beta_1 + \beta_2)S_l - (\mu + \mu_l) - \alpha S_c + (\beta_1 + \beta_2)P + \alpha S_l,$$

$$g_2 = \frac{\dot{P}}{P} - \frac{\dot{S}_l}{S_l} - \phi - (\mu + \mu_c) + \alpha S_l. \quad (9)$$

Thus, we can write $g_1 \leq \frac{\dot{P}}{P} - \mu_l - 2\mu,$, and $g_2 \leq \frac{\dot{P}}{P} - \phi - 2\mu - \mu_c$, which implies that $l(B) \leq \left\{\frac{\dot{P}}{P} - 2\mu - \right.$ min$\mu l, -\phi - \mu c.$ Hence, $lB \leq \dot{P}P - 2\mu.$ Now integrating the lozinski measure lB with respect to t in the interval $[0, t]$ and taking $lim_{t\to\infty}$, we get

$$lim_{t\to\infty} supsup \frac{1}{t}\int_0^t l(B)dt \leq -2\mu < 0. \quad (10)$$

Finally, we can write,

$$\hat{y} = lim_{t\to\infty} supsup \frac{1}{t}\int_0^t l(B)ds < 0. \quad (11)$$

Thus, the system containing the first three equations of the model (1) is globally asymptotically stable around its interior equilibrium (P^*, S_l^*, S_c^*). Now considering the last two equations of model (1), we have

$$\frac{dQ_t}{dt} = \phi(1 - \xi)S_c - \delta Q_t - \mu Q_t,$$

$$\frac{dQ_p}{dt} = \phi\xi S_c - \mu Q_p. \quad (12)$$

Taking limits of model (12), we get

$$\frac{dQ_t}{dt} = \phi(1 - \xi)S_c^* - \delta Q_t - \mu Q_t,$$

$$\frac{dQ_p}{dt} = \phi\xi S_c^* - \mu Q_p. \quad (13)$$

Solving system (13) and using the initial conditions $Q_t(0)$, $Q_p(0)$, for large time t, that is $t \to \infty$, $Q_t \to Qt*$ *and* $Qp \to Qp*$, which is sufficient to prove that the endemic equilibrium point $E*$ is globally asymptotically stable.

□

III. Sensitivity Analysis

The sensitivity analysis of the threshold quantity R_0 has received attention in a wide range of scientific disciplines. The sensitivity analysis is a significant part of the disease model investigation, although it may be time-consuming to compute the complex dynamical systems. We perform sensitivity analysis to determine the relative influence of epidemic parameters in smoking transmission in a community. To conduct this analysis, all epidemic parameters are observed to see how they affect the basic reproductive number. The normalized forward sensitivity index S_ω of R_0 are calculated as follows:

$$S_\omega = \frac{\partial R_0}{\partial \omega} \times \frac{\omega}{R_0}. \quad (14)$$

Important indices are given thus:

$$S_{\beta_1} = \frac{bN\{\mu^2(\mu + \mu_l)(\delta + \mu)(\phi + \mu + \mu_c)\}}{\{\mu^2(\mu + \mu_l)(\delta + \mu)(\phi + \mu + \mu_c)\}^2} \times \frac{\beta_1\{\mu^2(\mu + \mu_l)(\delta + \mu)(\phi + \mu + \mu_c)\}}{R_0} = \frac{1}{\beta_2 bN} > 0,$$

$$S_{\beta_2} = \frac{1}{\beta_1 bN} > 0,$$

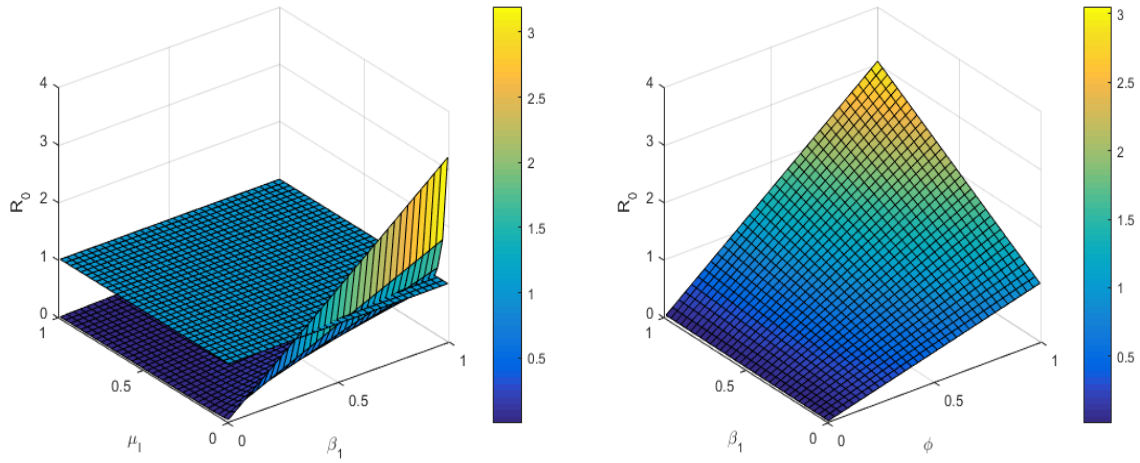
$$S_{\phi} = -\frac{\phi\mu^2(\mu+\mu_l)(\delta+\mu)}{\mu^2(\mu+\mu_l)(\delta+\mu)(\phi+\mu+\mu_c)} < 0,$$

$$S_{\delta} = -\frac{\delta\mu^2(\mu+\mu_l)(\phi+\mu+\mu_c)}{\mu^2(\mu+\mu_l)(\delta+\mu)(\phi+\mu+\mu_c)} < 0,$$

$$S_{\mu} = -\frac{\mu}{\mu^2(\mu+\mu_l)(\delta+\mu)(\phi+\mu+\mu_c)} < 0,$$

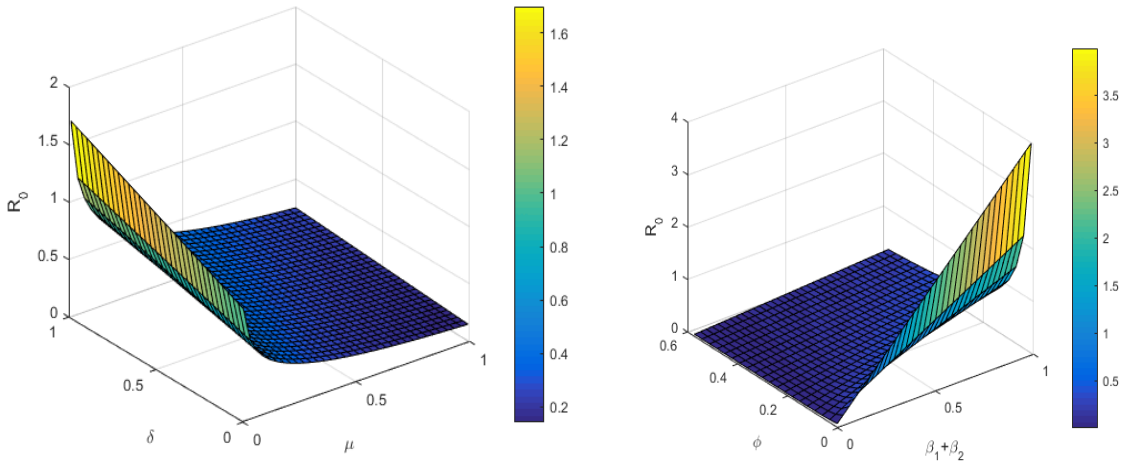
$$S_{\mu_l} = -\frac{\mu_l\mu^2(\delta+\mu)(\phi+\mu+\mu_c)}{\mu^2(\mu+\mu_l)(\delta+\mu)(\phi+\mu+\mu_c)} < 0,$$

$$S_{\mu_c} = -\frac{\mu_c\mu^2(\mu+\mu_l)(\delta+\mu)}{\mu^2(\mu+\mu_l)(\delta+\mu)(\phi+\mu+\mu_c)} < 0.$$



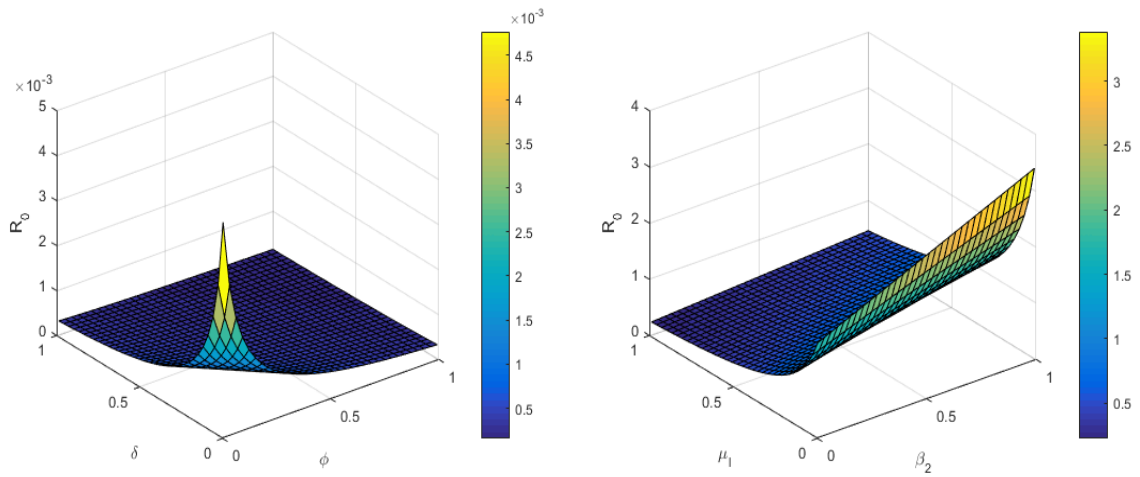
(a) R_0 *vs* β_1 *and* μ_l. (b) R_0 *vs* β_1 *and* ϕ.

Figure 2. The plots show the sensitivity analysis of R_0 in terms of the sensitive parameters β_1, μ_l *and* ϕ.



(a) $R_0\ vs\ \mu\ and\ \delta.$ (b) $R_0\ vs\ \beta_1+\beta_2\ and\ \phi.$

Figure 3. The plots show the sensitivity analysis of R_0 in terms of the sensitive parameters $\mu, \delta, \beta_1+\beta_2\ and\ \phi.$



(a) $R_0\ vs\ \phi\ and\ \delta.$ (b) $R_0\ vs\ \beta_2\ and\ \mu_l.$

Figure 4. The plots show the sensitivity analysis of R_0 in terms of the sensitive parameters , $\phi, \delta, \beta_2\ and\ \mu_l.$

The simulation of the critical parameters versus R_0 is observed in Figures 2-4. The smoking generation number depends on the transmission rates $\beta_1\ and\ \beta_2$, quitting rates ϕ and ξ, reversion rate δ, and death rates μ, μ_l, μ_c as shown in figures 2-5. Figure 2 shows the sensitivity of R_0 against $\beta_1, \phi, and\ \mu_l$. From figure 2(a), it can be seen that when the value of β_1 increases, R_0 also increases, whereas the natural death has no impact on the variation of R_0. Figure 2(b) shows the impact of quit rate and transmission rate on R_0. It shows that when the quit rate increases the R_0 decreases. while the increase in the transmission rate the value R_0 increases from 1 to 3, as shown in figure 2.

Figure 3 shows the sensitivity of R_0 against $\delta, \beta_1+\beta_2, and\ \phi$. It is evident from the figure that the transmission rate and influence of smokers on temporary quitters may increase the value of R_0 whereas the natural death rate and quitting rate have no impact on R_0. Figure 4 shows the sensitivity of R_0 versus $\beta_2, and\ \mu_l$. It is clear from the figure that when the transmission rate β_2 and reversion rate δ increases, then R_0 also increases, whereas the value of $\phi\ and\ \mu_l$ has no impact on R_0. It is obvious from the results of the sensitivity analysis that the transmission rate β_1(person to person), β_2 (transmission in a large group of social networks) and revert rate δ have a very high impact on the smoking generation number R_0. i.e., the value of R_0 increases from 1 and reaches 3. Similarly, the quit rate ϕ and death rates μ, μ_l, μ_c have no impact on the behavior of R_0.

IV. Results and Discussion

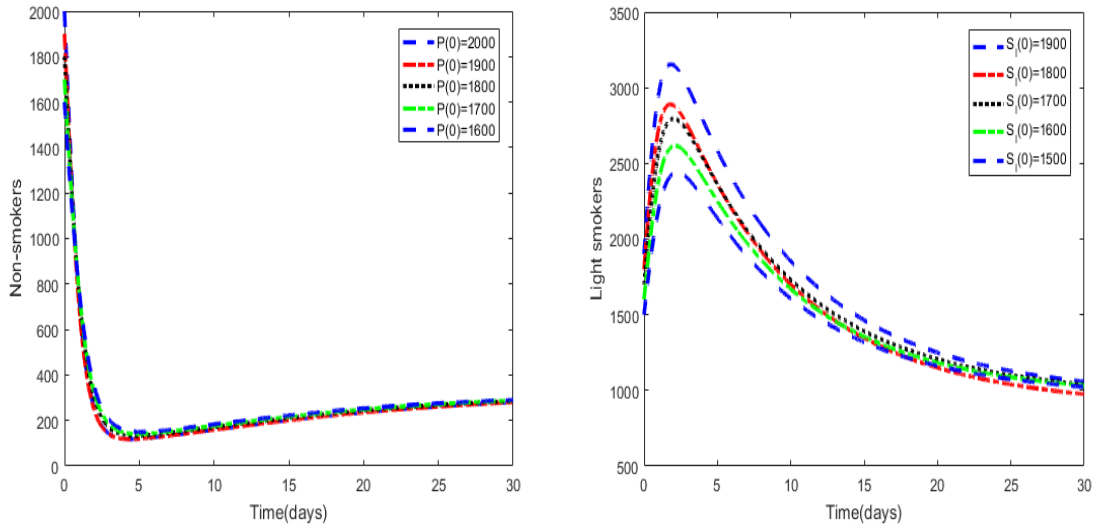
In this section, we numerically solved the proposed epidemic model by applying the Runge-Kutta technique of order 4th [9]. To better understand the dynamic behavior of the epidemic model, we employed numerical simulation to confirm our analytical conclusions. Therefore, we assumed some parameter values, some of which were derived from the data published given in Table 2. The numerical values of the parameter are taken to be more biologically feasible. We consider the crude birth rate of the population in Australia as $b = 12.1\ (over\ 1000\ population)$ =0.0121, and the natural death rate is the average life expectancy of 80 years, therefore, we choose $\mu = \frac{1}{80} = 0.0125$. Table 2 lists all of the parameter values, and Table 3 lists the initial values for state variables.

Parameter	Description	Value	Source
b	Birth rate	0.0121	estimated
μ	Natural death rate	0.0125	estimated
μ_l	Disease death rate of light smokers	0.00021	[9]
μ_c	Disease death rate of chain smokers	0.00037	[9]
β_1	Transmission rate from person to person	0.0380	[9]
β_2	Transmission rate in a group of social smokers	0.05	[12]
α	Transmission rate from light to chain smokers	0.45	[9]
ϕ	Quit smoking rate	0.06	[9]
ξ	Permanently quit smoking rate	0.023	[13]
δ	Rate at which temporary quitters revert back to smoking	0.025	[14]

Table 2. Parameter description and values.

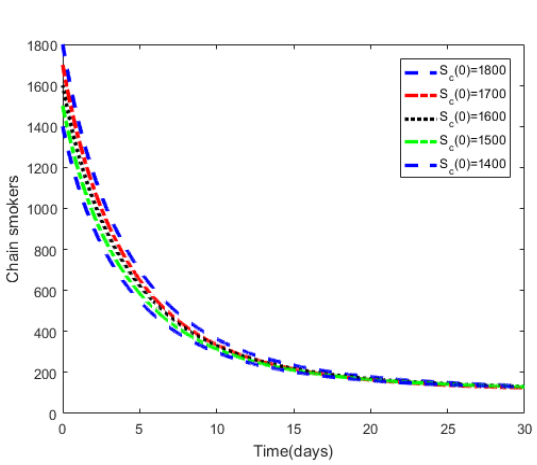
State variables	Description	Initial values
$P(0)$	Non smokers	2000 – 1500
$S_l(0)$	Light smokers	1900 – 1400
$S_c(0)$	Chain smokers	1800 – 1300
$Q_t(0)$	Temporary quit smokers	1700 – 1200
$Q_p(0)$	Permanent quit smokers	1600 – 1100

Table 3. State variables with initial values.

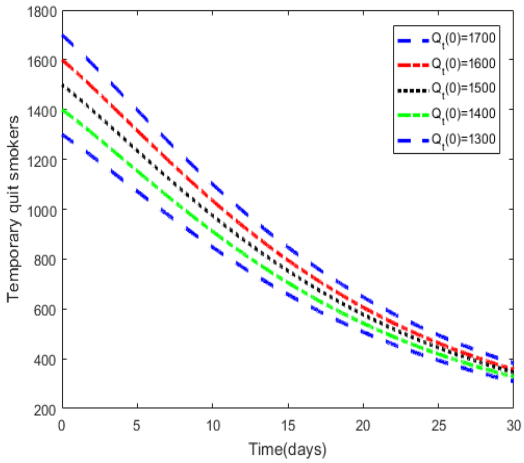


(a) Non-smokers population. (b) Light smokers population.

Figure 5. Population dynamics of the non-smokers and light smokers with respect to time.



(b) Chain smokers population.



(b) Temporary quit smokers population.

Figure 6. Population dynamics of the Chain and Temporary quit smokers with respect to time.

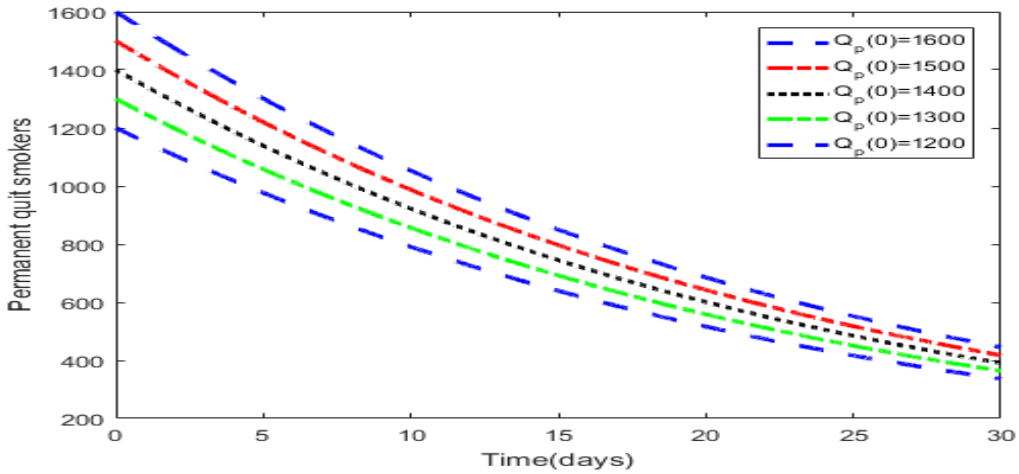


Figure 7: Population dynamics of the permanent quit smokers with respect to time.

The numerical simulation presented in Figure 5-7 represents the time dynamics of the model (1) at the endemic equilibrium point when $R_0 = 2.25 > 1$. Figure 5(a), shows that the number of non-smokers decreases with respect to time. Figure 5(b) demonstrates the progression of light smokers over time. The number of light smokers increases and tends to fall into its equilibrium position after a few weeks. Figures 6-7 show the dynamics of chain smokers, temporary quit smokers, and permanent quit smokers, concluding that the number of these populations will decrease and reach their equilibrium position, which ensures the stability of the proposed model. The biological interpretation of these results shows that if $R_0 < 1$, a stable population will be observed, and if R_0 exceeds one, then light and chain smokers exist in the population.

V. Conclusion

In this paper, we proposed an epidemic model with two different types of smoking transmission: person-to-person transmission and smoking among large groups of people. The system equilibrium points and positivity and boundedness are discussed. The basic reproduction number R_0, as an essential parameter in transmission dynamics, is critical to prevent major epidemics and disease control. We calculated the basic reproductive number R_0 by employing the next-generation matrix method. Furthermore, the stability of the model is analyzed by utilizing the Ruth-Hurwitz criterion, which showed that when $R_0 < 1$, the system is locally asymptotically stable and unstable otherwise. However, the global asymptotical stability is analyzed by the geometrical approach. Furthermore, sensitivity analysis is performed based on some important parameters, and the results indicate that the interaction between nonsmokers and groups of smokers, as opposed to a single smoker, has a greater impact on R_0. Finally, numerical simulations are conducted to support our analytical work.

References

[1]. World Health Organization Report on the Global Tobacco Epidemic, http ://whqlibdoc.who.int/publicatios/2009/9789241563918 eng full.pdf. 2009.
[2]. Australian Institute of Health and Welfare. 2004 National Drug Strategy Household Survey: detailed findings. Drug strategy series no.16, AIHW cat. no. PHE 66. Canberra: AIHW, 2005.
[3]. Michael Pearson, Lynn Michell Smoke Rings: social network analysis of friendship groups, smoking and drug-taking, Drugs: Education, Prevention and Policy, 2000, 7:1, 21-37.
[4]. Christakis NA, Fowler JH: The collective dynamics of smoking in a large social network. N Engl J Med 2008, 358:22492258
[5]. C.Castiho, Optimal control of an epidemic through educational campaigns, Electronic Journal of Differential Equations, 2006, no. 125: 1-11.
[6]. C. Castillo-Garsow, G. Jordan-Salivia, and A. Rodriguez Herrera, Mathematical models for the dynamics of tobacco use, recovery, and relapse, Technical Report Series BU-1505-M, Cornell University, Ithaca, NY, USA, 1997.
[7]. O. Sharomi, A.B. Gumel, Curtailing smoking dynamics: A mathematical modeling approach, Applied Mathematics and Computation 195 (2008) 475499.
[8]. G. Zaman, Qualitative behavior of giving up smoking model, Bull. Malays. Math. Sci. Soc. 34(2), 2011, 403-415.
[9]. Van Den Driessche P, Watmough J. Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission. Math Biosc 2002;180:29–48.
[10]. Mahardika R, Sumanto YD. Routh-hurwitz criterion and bifurcation method for stability analysis of tuberculosis transmission model. Phys Conf Ser 217(1); 2019: 012056.
[11]. Martin Jr., R.H. Logarithmic norms and projections applied to linear differential systems. J. Math. Anal. Appl., 1974, 45:432–454.
[12]. An Victoir et al, Smoking status moderates the contribution of social-cognitive and environmental determinants to adolescents' smoking intentions. Health Education Research Theory and practice, Vol.21 no.5, 2006:674–687.
[13]. G. Zaman, Y, H Kong, Il H, Jung, Dynamics of smoking model with smoking death rate Applicationes Mathematicae. 2017, 44,2:281-295.
[14]. Zainab A., Sarah A., Salma A., Stability analysis of a giving up smoking model. International Journal of Applied Mathematical Research, 3 (2) (2014) 168-177.
[15]. J.S.A. Linda, An Introduction to Mathematical Biology, Pearson Education Ltd. USA, 2007.
[16]. D.L. Lukes, Differential Equations: Classical to Controlled, Mathematics in Science and Engineering, Academic Press, New York, 1982.
[17]. J.K. Hale, Ordinary Differential Equations, John Wiley and Sons, New Yark, 1969.
[18]. R. Anguelov, J.M.S. Lubuma, Contriutions to the mathematics of the nonstandard finite difference method and applications, Num. Meth. Part. Diff. Equ., 17 (2001) 518-543.
[19]. R.E. Mickens, M.E. Alexander, B.D. Corbett, A.B.Gumel, A positivity-preserving Mickens-type discretization of an epidemic model, J. Diff. Equ. Appl.,9 (2003) 1037-1051.
[20]. National Cancer Information Center, Death statistics based on vital registration, Retrieved February 17, 2006, from http://www.re.kr/,(2002).

Muhammad Said. "Mathematical Analysis of a Smoking Model with Social Factor." *IOSR Journal of Mathematics (IOSR-JM)*, 18(1), (2022): pp. 28-38.