Mathematics

http://www.aimspress.com/journal/Math

AIMS Mathematics, 3(4): 584–599
DOI:10.3934/Math.2018.4.584
Received: 12 July 2018
Accepted: 21 November 2018
Published: 30 November 2018

Research article

Smoking dynamics with health education effect

Pengcheng Xiao[1,*], **Zeyu Zhang**[1,2] **and Xianbo Sun**[3,4]

[1] Department of Mathematics, University of Evansville, Evansville, IN 47722, USA
[2] Department of Computer Science and Engineering, The University of Texas at Arlington, Arlington, TX 76019, USA
[3] Department of Applied Mathematics, Western University, London, Ontario, N6A 5B7, Canada
[4] Department of Information and Statistics, Guangxi University of Finance and Economics, Nanning, Guangxi, 750003, China

* **Correspondence:** Email: px3@evansville.edu; Tel: +18124882213.

Abstract: This paper provides a mathematical study for analyzing the dynamics of smoking with health education campaigns involved. The method of next generation matrix is used to derive the basic reproduction number R_0. We prove that the smoking-free equilibrium is both locally and globally asymptotically stable if $R_0 < 1$; and the smoking-present equilibrium is globally asymptotically stable if $R_0 > 1$. By comparing with smoking dynamics without health education involved, we conclude that health education can decrease smoking population. Numerical simulations are used to support our conclusions.

Keywords: smoking dynamics; mathematical modeling; health education; equilibrium; stability; Lyapunov function
Mathematics Subject Classification: 92D25, 34D20

1. Introduction

The smoking behaviors have been considered as a critical problem on both health and social aspects for a long time. It is well-known that smoking can increase the risks of having serious diseases such as cancer and cardiovascular disease. WHO has estimated that tobacco use (smoking and smokeless) is currently responsible for the death of about six million people across the world each year with many of these deaths occurring prematurely. Although often associated with ill-health, disability and death from noncommunicable chronic diseases, tobacco smoking is also associated with an increased risk of death from communicable diseases [1].

To reduce such serious effect, many nations and global health organizations had applied control policies. According to WHO Comprehensive Information Systems, During the most recent decade, the prevalence of tobacco smoking in men fell in 125 (72%) countries, and in women fell in 155 (87%) countries. If these trends continue, only 37 (21%) countries are on track to achieve their targets for men and 88 (49%) are on track for women, and there would be an estimated 1.1 billion current tobacco smokers (95% credible interval 700 million to 1.6 billion) in 2025 [2]. Among many control policies, health education campaigns played an important role. Ian Bier *et al*. [3] studied the relationship between auricular acupuncture, education, and smoking cessation. They concluded that acupuncture and education, alone and in combination, significantly reduce smoking. Damiende Walque [4] collected data from smoking population, and concluded that education does affect smoking decisions: educated individuals are less likely to smoke, and among those who initiated smoking, they are more likely to have stopped. Moreover, Sarah Durkin *et al*. [5] directly studied how mass media campaigns to promote smoking cessation among adults. Their studies showed that mass media campaigns conducted in the context of comprehensive tobacco control programmes can promote quitting and reduce adult smoking prevalence. Mass media campaigns to promote quitting are important investments as part of comprehensive tobacco control programmes to educate about the harms of smoking, set the agenda for discussion, change smoking attitudes and beliefs, increase quitting intentions and quit attempts, and reduce adult smoking prevalence.

Above evidences motivated us to construct a mathematical model to mimic the smoking dynamics with health educational campaigns involved. We think it can be a helpful tool to analyze smoking behaviors and their control.

Back to 90's, smoking dynamics were only been studied by using basic SIR model. In a recent decade, several more sophisticated models about smoking dynamics have been studied. In 2008, Sharomo and Gumel [6] introduced new classes Q_t (temporary quitter) and Q_p (permanent quitter) into the model and presented a more realistic dynamics about smoking population. In 2014, Alkhudhari *et al*. [7] further developed Sharomo and Gumel's model by considering peer pressure effect on the transmission from Q_t (temporary quitter) to S (Smoker). Besides, several researchers like Din *et al*. [8] have studied the effect of introducing the class Z of smoker with illnesses. Similar models about smoking, drinking can also be found in other studies including [9–15].

Above works guide us to derive a smoking model along with health educational campaigns involved. The paper is organized as follows. In Section 2, we present the model with health education effect, and prove the model is well posed. Section 3 focuses on the existence of smoking-free equilibrium and smoking-present equilibrium. Derivations for the reproduction number and both local and global stability properties for equilibria are also included in this section. In Section 4, we provide some numerical simulation results to support our analytic results. Section 5 includes discussions of the results.

2. Model formulation and its properties

2.1. Formulation of the model

In this section we describe our smoking model with health educational campaigns involved. We first divide the whole population into 6 groups:

$P_N(t)$: Normal susceptible population, who do not smoke or smoke occasionally and do not get health education, may become smokers in future.

$P_E(t)$: Educated susceptible population, who get health education and do not smoke or smoke occasionally, have lower chance to develop smoking behaviors.

$S(t)$: Smoking population

$Q_t(t)$: Temporary quitters, who are currently abstaining smoking, but may not succeed.

$Q_p(t)$: Permanent quitters, who permanently quit smoking, never smoke again.

$Z(t)$: Smokers with associated diseases, yield extra death rate.

The total number of population at time t is given by

$$N(t) = P_N(t) + P_E(t) + S(t) + Q_t(t) + Q_p(t) + Z(t)$$

The following system of ODEs forms our model (Figure 1):

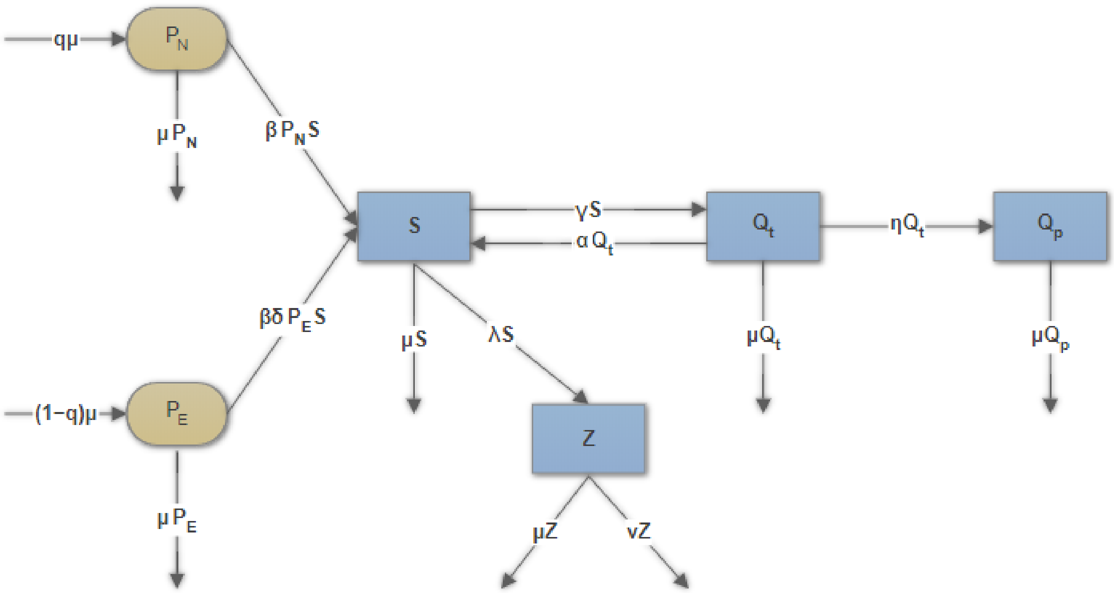

Figure 1. Transfer diagram of the model.

$$\begin{aligned}
\dot{P}_N &= q\mu - \mu P_N - \beta P_N S \\
\dot{P}_E &= (1-q)\mu - \mu P_E - \beta\delta P_E S \\
\dot{S} &= -(\mu + \gamma + \lambda)S + \beta S(P_N + \delta P_E) + \alpha Q_t \\
\dot{Q}_t &= -\mu Q_t - \alpha Q_t - \eta Q_t + \gamma S \\
\dot{Q}_p &= -\mu Q_p + \eta Q_t \\
\dot{Z} &= \lambda S - (\mu + \nu)Z
\end{aligned}$$

First of all, every group share same death rate μ. For simplicity, we assume the new recruitment rate of the system to be same as the death rate μ. The new population recruited into the system is divided into 2 portions - uneducated and educated. The proportion $q(0 < q < 1)$ of new recruitment is uneducated portion, and the proportion $(1-q)$ is educated portion. Since smoking population cannot be isolated, through peer pressure, we have both educated and uneducated susceptible population transferring to smokers with transmission coefficient β. However, educated people have lower chance to become smokers, hence we assume addition immunity coefficient δ to reflect this effect, where $0 < \delta < 1$. Smokers can turn into temporary quitters by getting treatment or self-abstaining, hence we assume γ as the corresponding transmission coefficient. On the other hand, temporary quitters can also relapse, hence we assume α as the corresponding transmission coefficient. There does exist some quitters could abstain smoking permanently. By enough treatment and perseverance, a temporary quitter can become a permanent quitter. For this type of transmission, we assume a transmission coefficient η. We have mentioned in introduction that smoking is highly related to some serious diseases. Therefore, it is reasonable to have a transmission from ordinary smokers to diseased smokers with coefficient λ. In addition, this diseased population yield extra death rate ν.

2.2. Properties of the model

Boundedness is one of important properties of a system, and we shall provide it for our system by following lemma.

Lemma 2.1. *If $P_N(0) > 0$, $P_E(0) > 0$, $S(0) > 0$, $Q_t(0) > 0$, $Q_p(0) > 0$, $Z(0) > 0$, then the solutions $P_N(t) \geq 0$, $P_E(t) \geq 0$, $S(t) \geq 0$, $Q_t(t) \geq 0$, $Q_p(t) \geq 0$, $Z(t) \geq 0$ for all $t > 0$.*

Proof. Suppose above lemma does not hold, then at least one of $P_N(t)$, $P_E(t)$, $S(t)$, $Q_t(t)$, $Q_p(t)$, $Z(t)$ is less than 0 for some t's. We have following 6 cases:

1. There exists a first time t_1 such that $P_N(t_1) = 0$, $P'_N(t_1) < 0$, and $P_E(t) \geq 0$, $S(t) \geq 0$, $Q_t(t) \geq 0$, $Q_p(t) \geq 0$, $Z(t) \geq 0$ for $0 \leq t \leq t_1$. But $P'_N(t_1) = q\mu \geq 0$, so this case is impossible.
2. There exists a first time t_2 such that $P_E(t_2) = 0$, $P'_E(t_2) < 0$, and $P_N(t) \geq 0$, $S(t) \geq 0$, $Q_t(t) \geq 0$, $Q_p(t) \geq 0$, $Z(t) \geq 0$ for $0 \leq t \leq t_2$. But $P'_E(t_2) = (1-q)\mu \geq 0$, so this case is impossible.
3. There exists a first time t_3 such that $S(t_3) = 0$, $S'(t_3) < 0$, and $P_N(t) \geq 0$, $P_E(t) \geq 0$, $Q_t(t) \geq 0$, $Q_p(t) \geq 0$, $Z(t) \geq 0$ for $0 \leq t \leq t_3$. But $S'(t_3) = 0 \geq 0$, so this case is impossible.
4. There exists a first time t_4 such that $Q_t(t_4) = 0$, $Q'_t(t_4) < 0$, and $P_N(t) \geq 0$, $P_E(t) \geq 0$, $S(t) \geq 0$, $Q_p(t) \geq 0$, $Z(t) \geq 0$ for $0 \leq t \leq t_4$. But $Q'_t(t_4) = \gamma S(t_4) \geq 0$, so this case is impossible.
5. There exists a first time t_5 such that $Q_p(t_5) = 0$, $Q'_p(t_5) < 0$, and $P_N(t) \geq 0$, $P_E(t) \geq 0$, $S(t) \geq 0$, $Q_t(t) \geq 0$, $Z(t) \geq 0$ for $0 \leq t \leq t_5$. But $Q'_p(t_5) = \eta Q_t(t_5) \geq 0$, so this case is impossible.
6. There exists a first time t_6 such that $Z(t_6) = 0$, $Z'(t_6) < 0$, and $P_N(t) \geq 0$, $P_E(t) \geq 0$, $S(t) \geq 0$, $Q_t(t) \geq 0$, $Q_p(t) \geq 0$ for $0 \leq t \leq t_6$. But $Z'(t_6) = \lambda S(t_6) \geq 0$, so this case is impossible.

That shows the contradiction, therefore the lemma has to be true. □

By summing the equations of our system, we find that

$$\begin{aligned} P'_N + P'_E + S' + Q'_t + Q'_p + Z' &= \mu[1 - (P_N + P_E + S + Q_t + Q_p + Z)] - \nu Z \\ &\leq \mu[1 - (P_N + P_E + S + Q_t + Q_p + Z)] \end{aligned}$$

It follows that $P_N(t) + P_E(t) + S(t) + Q_t(t) + Q_p(t) + Z(t) \leq 1$, so the set

$$\Omega = \{(P_N, P_E, S, Q_t, Q_p, Z) \in \mathbb{R}_+^6 : P_N + P_E + S + Q_t + Q_p + Z \leq 1\}$$

is positively invariant for our system. Hence, the global stability of the system will be only considered within set Ω. Also, the whole population has the scaled upper bound 1 in this model, and the number of each population group can be interpreted as the portion of the whole population.

3. Equilibria and stabilities

3.1. Equilibria and local stabilities

By setting the right-hand side of the model to 0, we get following equations:

$$\begin{aligned}
P_N &= \frac{q\mu}{\mu + \beta S} \\
P_E &= \frac{(1-q)\mu}{\mu + \beta\delta S} \\
S &= \frac{\alpha Q_t}{(\mu + \gamma + \eta) - \beta(P_N + \delta P_E)} \\
Q_t &= \frac{\gamma S}{\mu + \eta + \alpha} \\
Q_p &= \frac{\eta}{\mu} Q_t \\
Z &= \frac{\lambda S}{\mu + \nu}
\end{aligned}$$

We see that the model has a smoking-free equilibrium $E_0 = (P_{N_0}, P_{E_0}, 0, 0, 0, 0)$, where

$$P_{N_0} = q \qquad P_{E_0} = 1 - q$$

The smoking infected compartments are S, Q_t, and Z, giving $m = 3$. Since each function in our model represents a direct transfer of individuals, each function is non negative. And if one population group is empty, then there is no transfer of individuals out of that population group. Also, our model assumes that incidence of smoking infection for uninfected population groups is zero, the smoking free subspace is always invariant, and the smoking free equilibrium is stable in the absence of new infection. This indicates that our model satisfies the five conditions in lemma 1 from van den Driessche and Watmough [16]. Let $X = (S, Q_t, Z, P_N, P_E, Q_p)^T$, then the model can be rewritten as

$$\frac{dX}{dt} = F(X) - V(X)$$

where

$$F(X) = \begin{pmatrix} \beta P_N S + \beta\delta P_E S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \qquad V(X) = \begin{pmatrix} (\mu + \gamma + \lambda)S - \alpha Q_t \\ (\alpha + \mu + \eta)Q_t - \gamma S \\ (\mu + \nu)Z - \lambda S \\ \mu P_N + \beta P_N S - q\mu \\ \mu P_E + \beta\delta P_E S - (1-q)\mu \\ \mu Q_p - \eta Q_t \end{pmatrix}$$

By computing the Jacobian matrices at E_0, we got

$$DF(E_0) = \begin{pmatrix} F_{3\times3} & 0 \\ 0 & 0 \end{pmatrix} \quad DV(E_0) = \begin{pmatrix} V_{3\times3} & 0 \\ J_1 & J_2 \end{pmatrix}$$

where

$$F = \begin{pmatrix} \beta P_{N_0} + \beta\delta P_{E_0} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad V = \begin{pmatrix} \mu+\gamma+\lambda & -\alpha & 0 \\ -\gamma & \alpha+\mu+\eta & 0 \\ -\lambda & 0 & \mu+\nu \end{pmatrix}$$

$$J_1 = \begin{pmatrix} \beta P_{N_0} & 0 & 0 \\ \beta\gamma P_{E_0} & 0 & 0 \\ 0 & -\eta & 0 \end{pmatrix} \quad J_2 = \begin{pmatrix} \mu & 0 & 0 \\ 0 & \mu & 0 \\ 0 & 0 & \mu \end{pmatrix}$$

Further, F is non-negative, V is a non-singular 3-matrix and all eigenvalues of J_2 have positive real part. Thus, the basic reproduction number of the model can be derived by the method of next generation matrix [16]. And we got the basic reproduction number R_0

$$R_0 = \rho(FV^{-1}) = \frac{\beta(P_{N_0} + \delta P_{E_0})(\mu+\eta+\alpha)}{(\mu+\gamma+\lambda)(\mu+\eta+\alpha) - \alpha\gamma}$$

By Theorem 2 from van den Driessche and Watmough [16], the local stability of smoking-free equilibrium E_0 can be summarized as following:

Theorem 3.1. *The smoking-free equilibrium E_0 is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.*

Now we look at smoking-present equilibrium $E^* = (P_N^*, P_E^*, S^*, Q_t^*, Q_p^*, Z^*)$. Similarly, by he right-hand side of the model to 0, we get

$$\begin{aligned} P_N &= \frac{q\mu}{\mu+\beta S} \\ P_E &= \frac{(1-q)\mu}{\mu+\beta\delta S} \\ Q_t &= \frac{\gamma S}{\mu+\eta+\alpha} \\ &S[\beta(P_N + \delta P_E) - (\mu+\gamma+\lambda)] + \alpha Q_t = 0 \end{aligned}$$

By substituting Q_t into last equation, we have

$$\begin{aligned} &S[\beta(P_N + \delta P_E) - (\mu+\gamma+\lambda)] + \frac{\gamma S}{\mu+\eta+\alpha} = 0 \\ \Rightarrow\ &S\left(\beta(P_N + \delta P_E) - (\mu+\gamma+\lambda) + \frac{\gamma}{\mu+\eta+\alpha}\right) = 0 \end{aligned}$$

Since $S \neq 0$,

$$\beta(P_N + \delta P_E) - (\mu + \gamma + \lambda) + \frac{\gamma}{\mu + \eta + \alpha} = 0$$
$$\Rightarrow P_N + \delta P_E = \frac{1}{\beta}\left((\mu + \gamma + \lambda) - \frac{\gamma}{\mu + \eta + \alpha}\right)$$

By substituting P_N and P_E, we have

$$Y(S) := \frac{q(\mu + \beta\delta S) + (1 - q)\delta(\mu + \beta S)}{(\mu + \beta S)(\mu + \beta\delta S)} - \frac{(\mu + \gamma + \lambda)(\mu + \eta + \alpha) - \alpha\gamma}{\mu\beta(\mu + \eta + \alpha)} = 0$$

By taking the derivative of $Y(S)$, we have

$$Y'(S) = -\frac{\beta\{\beta^2\delta^2 S^2 + \mu S[2\beta\delta^2(1 - q) + 2\beta\delta q] + \mu^2[\delta^2(1 - q) + q]\}}{(\mu + \beta S)^2(\mu + \beta\delta S)^2} < 0$$

Hence, the function $Y(S)$ is decreasing for $S > 0$. In addition, since $(\mu + \beta\delta S)(\mu + \beta S) > (\mu + \beta\delta S)\beta S$ and $q(\mu + \beta\delta S) + (1 - q)\delta(\mu + \beta S) \leq u + \beta\delta S$, we have

$$Y(S) < \frac{1}{\beta S} - \frac{(\mu + \gamma + \lambda)(\mu + \eta + \alpha) - \alpha\gamma}{\mu\beta(\mu + \eta + \alpha)}$$

Thus,

$$\begin{aligned}
Y(0) &= \frac{(\mu + \gamma + \lambda)(\mu + \eta + \alpha) - \alpha\gamma}{\mu\beta(\mu + \eta + \alpha)}(R_0 - 1)\\
Y(1) &< \frac{1}{\beta} - \frac{(\mu + \gamma + \lambda)(\mu + \eta + \alpha) - \alpha\gamma}{\mu\beta(\mu + \eta + \alpha)}\\
&= -\frac{\lambda(\mu + \eta + \alpha) + \gamma(\mu + \eta)}{\mu\beta(\mu + \eta + \alpha)} < 0
\end{aligned}$$

If $R_0 > 1$, by the monotonicity of $Y(S)$, there exist an unique root in $(0, 1)$. If $R_0 \leq 1$, there is not root in $(0, 1)$. Since the smoking-present equilibrium E^* lives in the set $\Omega = \{(P_N, P_E, S, Q_t, Q_p, Z) \in \mathbb{R}^6_+ : P_N + P_E + S + Q_t + Q_p + Z \leq 1\}$, following theorem can be established:

Theorem 3.2. *The system always has the smoking-free equilibrium E_0. If $R_0 > 1$, the system has an unique smoking-present equilibrium E^*, where*

$$\begin{aligned}
P_N^* &= \frac{q\mu}{\mu + \beta S^*}\\
P_E^* &= \frac{(1 - q)\mu}{\mu + \beta\delta S^*}\\
Q_t^* &= \frac{\gamma S^*}{\mu + \eta + \alpha}
\end{aligned}$$

$$Q_p^* = \frac{\eta}{\mu} Q_t^*$$
$$Z^* = \frac{\lambda S^*}{\mu + \nu}$$

and S^* is the unique root of $Y(S) = 0$.

Theorem 3.3. *The smoking-present equilibrium E^* is locally stable, and there is no hopf bifurcation.*

Proof. Since variables Q_p and Z do not appears in first four equations of the system, the dynamics of the system is same as the following one:

$$\begin{aligned}
\dot{P_N} &= q\mu - \mu P_N - \beta P_N S \\
\dot{P_E} &= (1-q)\mu - \mu P_E - \beta\delta P_E S \\
\dot{S} &= -(\mu + \gamma + \lambda)S + \beta S(P_N + \delta P_E) + \alpha Q_t \\
\dot{Q_t} &= -\mu Q_t - \alpha Q_t - \eta Q_t + \gamma S
\end{aligned}$$

Consider the previous four equations in the original system, we get its Jacobian matrix at the smoking-present equilibrium E^*,

$$J(E^*) = \begin{bmatrix} -\beta S - \mu & 0 & -\beta P_N & 0 \\ 0 & -\mu\delta S - \mu & -\beta\delta P_E & 0 \\ \beta S & \beta\delta S & \beta(\delta P_E + P_N) - \mu - \gamma - \lambda & \alpha \\ 0 & 0 & \gamma & -\mu - \alpha - \eta \end{bmatrix}.$$

$R_0 = 1$ reveals that

$$\beta(\delta P_E + P_N) - \mu - \gamma - \lambda = -\frac{\alpha\gamma}{\alpha + \eta + \mu}.$$

Hence, we have

$$J(E^*) = \begin{bmatrix} -\beta S - \mu & 0 & -\beta P_N & 0 \\ 0 & -\mu\delta S - \mu & -\beta\delta P_E & 0 \\ \beta S & \beta\delta S & -\frac{\alpha\gamma}{\alpha+\eta+\mu} & \alpha \\ 0 & 0 & \gamma & -\mu - \alpha - \eta \end{bmatrix}.$$

Our aim is to prove $J(E^*)$ has no positive or zero-real part eigenvalues. In order to reduce complexity due to multiple parameters, we introduce new variables, which are all positive from the original parameters are positive.

$$a_{11} = \beta S + \mu,\ a_{13} = \beta P_N,\ a_{22} = \mu\delta S + \mu,\ a_{23} = \beta\delta P_E,\ a_{31} = S\beta,\ a_{32} = S\delta\beta,\ a_{44} = \mu + \alpha + \eta.$$

Even the new variables are not independent, we would like to investigate them in a broader ranges.

Then, we have

$$J(E^*) = \begin{bmatrix} -a_{11} & 0 & -a_{13} & 0 \\ 0 & -a_{22} & -a_{23} & 0 \\ a_{31} & a_{32} & -\frac{\alpha\gamma}{a_{44}} & \alpha \\ 0 & 0 & \gamma & -a_{44} \end{bmatrix}.$$

$J(E^*) - xI = 0$ gives the eigen-polynomial,

$$\begin{aligned} Ep(x) &= a_{44}\, x^4 + \left(a_{44}{}^2 + (a_{11} + a_{22})\, a_{44} + \alpha\,\gamma\right) x^3 \\ &+ \left((a_{11} + a_{22})\, a_{44}{}^2 + (a_{11}\, a_{22} + a_{13}\, a_{31} + a_{23}\, a_{32})\, a_{44} + \gamma\,\alpha\ (a_{11} + a_{22})\right) x^2 \\ &+ \left((a_{11}\, a_{22} + a_{13}\, a_{31} + a_{23}\, a_{32})\, a_{44}{}^2 + (a_{11}\, a_{23}\, a_{32} + a_{13}\, a_{22}\, a_{31})\, a_{44} + \gamma\, a_{11}\, a_{22}\,\alpha\right) x \\ &+ a_{44}{}^2\,(a_{11}\, a_{23}\, a_{32} + a_{13}\, a_{22}\, a_{31}). \end{aligned}$$

All coefficients of $Ep(x)$ are positive, therefore $Ep(x)$ has no non-negative eigenvalues.Suppose $Ep(x)$ has a pair of complex eigenvalues $x^* = a \pm bi$. Let $REp(x^*)$ and $IPp(x^*)$ denote the real part and imaginal part of $Ep(x^*)$. The resultant between $REp(x^*)$ and $IPp(x^*)$ respect to b is a polynomial in a with positive coefficients, which has no non-negative roots. Therefore, $J(E^*)$ could not have complex eigenvalues with positive or zero real part. Hence, the eigenvalues of $J(E^*)$ are negative or complex with negative real part, therefore, the smoking-present equilibria E^* is local stable and could not present hopf-bifurcation.

□

3.2. Global stability of equilibria

Theorem 3.4. *If $R_0 \leq 1$, the smoking-free equilibrium E_0 is globally asymptotically stable.*

Proof. Since variables Q_p and Z do not appears in first four equations of the system, the dynamics of the system is same as the following one:

$$\begin{aligned} \dot{P_N} &= q\mu - \mu P_N - \beta P_N S \\ \dot{P_E} &= (1-q)\mu - \mu P_E - \beta\delta P_E S \\ \dot{S} &= -(\mu + \gamma + \lambda)S + \beta S\,(P_N + \delta P_E) + \alpha Q_t \\ \dot{Q_t} &= -\mu Q_t - \alpha Q_t - \eta Q_t + \gamma S \end{aligned}$$

By proving the global stability of smoking-free equilibrium $\bar{E}_0(P_{N_0}, P_{E_0}, 0, 0)$ of above system, we prove the original one.
For the smoking-free equilibrium $\bar{E}_0$, following equations hold:

$$\begin{aligned} q\mu - \mu P_N &= 0 \\ (1-q)\mu - \mu P_E &= 0 \end{aligned}$$

Hence, we can rewrite above system as

$$\dot{P_N} = P_N\left[q\mu\left(\frac{1}{P_N} - \frac{1}{P_{N_0}}\right) - \beta S\right]$$

$$
\begin{aligned}
\dot{P_E} &= P_E\left[(1-q)\mu\left(\frac{1}{P_E}-\frac{1}{P_{E_0}}\right)-\beta\delta S\right]\\
\dot{S} &= \beta S\ [(P_{N_0}+\delta P_{E_0})+(P_N-P_{N_0})+\delta(P_E-P_{E_0})]+\alpha Q_t-(\mu+\gamma+\lambda)S\\
\dot{Q_t} &= \gamma S\ -(\mu+\eta+\alpha)Q_t
\end{aligned}
$$

Define the Lyapunov function:

$$
V_1=\left(P_N-P_{N_0}-P_{N_0}ln\frac{P_N}{P_{N_0}}\right)+\left(P_E-P_{E_0}-P_{E_0}ln\frac{P_E}{P_{E_0}}\right)+S\ +\frac{\alpha}{\mu+\eta+\alpha}Q_t
$$

By taking the derivative, we have

$$
\begin{aligned}
V_1' &= (P_N-P_{N_0})\frac{P_N'}{P_N}+(P_E-P_{E_0})\frac{P_E'}{P_E}+S'+\frac{\alpha}{\mu+\eta+\alpha}Q_t'\\
&= (P_N-P_{N_0})\left[q\mu\left(\frac{1}{P_N}-\frac{1}{P_{N_0}}\right)-\beta S\right]\\
&+(P_E-P_{E_0})\left[(1-q)\mu\left(\frac{1}{P_E}-\frac{1}{P_{E_0}}\right)-\beta\delta S\right]\\
&+\beta S\ [(P_{N_0}+\delta P_{E_0})+(P_N-P_{N_0})+\delta(P_E-P_{E_0})]+\alpha Q_t-(\mu+\gamma+\lambda)S\\
&+\frac{\alpha}{\mu+\eta+\alpha}[\gamma S\ -(\mu+\eta+\alpha)Q_t]\\
&= \frac{(\mu+\gamma+\lambda)(\mu+\eta+\alpha)-\alpha\gamma}{\mu+\eta+\alpha}(R_0-1)S\ +F(P_N,P_E)
\end{aligned}
$$

,where

$$
\begin{aligned}
F(P_N,P_E) &= q\mu(P_N-P_{N_0})\left(\frac{1}{P_N}-\frac{1}{P_{N_0}}\right)+(1-q)\mu(P_E-P_{E_0})\left(\frac{1}{P_E}-\frac{1}{P_{E_0}}\right)\\
&= q\mu\left(2-\frac{P_N}{P_{N_0}}-\frac{P_{N_0}}{P_N}\right)+(1-q)\mu\left(2-\frac{P_E}{P_{E_0}}-\frac{P_{E_0}}{P_E}\right)
\end{aligned}
$$

Let $x=\frac{P_N}{P_{N_0}}$ and $y=\frac{P_E}{P_{E_0}}$, then

$$
\begin{aligned}
F(P_N,P_E) &= q\mu\left(2-x-\frac{1}{x}\right)+(1-q)\mu\left(2-y-\frac{1}{y}\right)\\
&= q\mu\left(\frac{(-1)(x-1)^2}{x}\right)+(1-q)\mu\left(\frac{(-1)(y-1)^2}{y}\right)
\end{aligned}
$$

It is obvious that $F(P_N,P_E)\le 0$ for $x,y>0$. In particular, $F(P_N,P_E)=0$ if and only if $P_N=P_{N_0}$ and $P_E=P_{E_0}$. Hence, if $R_0\le 1$, $V_1'<0$ for $P_N\ne P_{N_0}$, $P_E\ne P_{E_0}$ and $S\ne 0$. Therefore, by Lyapunov stability criterion, the smoking-free equilibrium $\bar{E}_0$ is globally asymptotically stable, and so is E_0. □

Theorem 3.5. *If $R_0>1$, the smoking-present equilibrium E^* is globally asymptotically stable.*

Proof. Similarly, we prove the stability of original smoking-present equilibrium E^* by proving the stability of $\bar{E}^*(P_N^*, P_E^*, S^*, Q_t^*)$.
For $\bar{E}^*$, following equations hold:

$$\begin{aligned} q\mu - \mu P_N - \beta P_N S &= 0 \\ (1-q)\mu - \mu P_E - \beta\delta P_E S &= 0 \\ -(\mu+\gamma+\lambda)S + \beta S(P_N + \delta P_E) + \alpha Q_t &= 0 \\ \gamma S - Q_t(\mu+\eta+\alpha) &= 0 \end{aligned}$$

Let $a = \frac{P_N}{P_N^*}$, $b = \frac{P_E}{P_E^*}$, $c = \frac{S}{S^*}$, and $d = \frac{Q_t}{Q_t^*}$, we have

$$\begin{aligned} a' &= a\left[\frac{q\mu}{P_N^*}\left(\frac{1}{a}-1\right) - \beta S^*(c-1)\right] \\ b' &= b\left[\frac{(1-q)\mu}{P_E^*}\left(\frac{1}{b}-1\right) - \beta\delta S^*(c-1)\right] \\ c' &= c\left[\beta P_N^*(a-1) + \beta\delta P_E^*(b-1) + \frac{\alpha Q_t^*}{S^*}\left(\frac{d}{c}-1\right)\right] \\ d' &= d\left[\frac{\gamma S^*}{Q_t^*}\left(\frac{c}{d}-1\right)\right] \end{aligned}$$

Define the Lyapunov function:

$$V_2 = P_N^*(a-1-lna) + P_E^*(b-1-lnb) + S^*(c-1-lnc) + \frac{\alpha}{\mu+\eta+\alpha}Q_t^*(d-1-lnd)$$

By taking the derivative, we have

$$\begin{aligned} V_2' &= P_N^*\left(\frac{a-1}{a}\right)a' + P_E^*\left(\frac{b-1}{b}\right)b' + S^*\left(\frac{c-1}{c}\right)c' + \frac{\alpha}{\mu+\eta+\alpha}Q_t^*\left(\frac{d-1}{d}\right)d' \\ &= (a-1)\left[q\mu\left(\frac{1}{a}-1\right) - \beta P_N^* S^*(c-1)\right] \\ &+ (b-1)\left[(1-q)\mu\left(\frac{1}{b}-1\right) - \beta\delta P_E^* S^*(c-1)\right] \\ &+ (c-1)\left[\beta P_N^* S^*(a-1) + \beta\delta P_E^* S^*(b-1) + \alpha Q_t^*\left(\frac{d}{c}-1\right)\right] \\ &+ \frac{\alpha\gamma S^*}{\mu+\eta+\alpha}(d-1)\left(\frac{c}{d}-1\right) \\ &= q\mu(a-1)\left(\frac{1}{a}-1\right) - \beta P_N^* S^*(a-1)(c-1) + (1-q)\mu(b-1)\left(\frac{1}{b}-1\right) - \beta\delta P_E^* S^*(b-1)(c-1) \\ &+ \beta P_N^* S^*(c-1)(a-1) + \beta\delta P_E^* S^*(c-1)(b-1) + \alpha Q_t^*(c-1)\left(\frac{d}{c}-1\right) + \frac{\alpha\gamma S^*}{\mu+\eta+\alpha}(d-1)\left(\frac{c}{d}-1\right) \\ &= q\mu\left(-\frac{(a-1)^2}{a}\right) + (1-q)\mu\left(-\frac{(b-1)^2}{b}\right) + \alpha Q_t^*\left(d-c-\frac{d}{c}+1\right)\frac{\alpha\gamma S^*}{\mu+\eta+\alpha}\left(c-d-\frac{c}{d}+1\right) \end{aligned}$$

$$= F(a,b) + G(c,d)$$

where

$$\begin{aligned} F(a,b) &= q\mu\left(-\frac{(a-1)^2}{a}\right) + (1-q)\mu\left(-\frac{(b-1)^2}{b}\right) \\ G(c,d) &= \frac{\alpha\gamma S^*}{\mu+\eta+\alpha}\left(2-\frac{c}{d}-\frac{d}{c}\right) \\ &= \frac{\alpha\gamma S^*}{\mu+\eta+\alpha}\left(-\frac{(c-d)^2}{cd}\right) \end{aligned}$$

It is easy to see that $F(a,b) \le 0$ for $a,b > 0$. In particular, $F(a,b) = 0$ if and only if $P_N = P_N^*$ and $P_E = P_E^*$. Also, $G(c,d) \le 0$ for $c,d > 0$. In particular, $G(c,d) = 0$ if and only if $\frac{S}{S^*} = \frac{Q_t}{Q_t^*}$. Hence, $V_2' < 0$ for $P_N \ne P_N^*$, $P_E \ne P_E^*$, $S \ne S^*$, and $Q_t \ne Q_t^*$. Therefore, by byLyapunov stability criterion, the smoking-free equilibrium $\bar{E}^*$ is globally asymptotically stable,and so is E^*. □

4. Numerical simulation

In this section, we provide some numerical results to support our analytic results from above. For the choices of parameters, some are chosen from medical researches, and others are estimated. The values for normal mortality μ and additional disease death rate ν are provided by McEvoy, John W., *et al*. [9]. Other parameters are estimated. All the parameter values show in Table 1.
The model is simulated for following different initial values such that $P_N(0) + P_E(0) + S(0) + Q_t(0) + Q_p(0) + Z(0) = 1$:

1. $P_N(0) = 0.8$, $P_E(0) = 0.1$, $S(0) = 0.1$, $Q_t(0) = 0$ $Q_p(0) = 0$, $Z(0) = 0$.
2. $P_N(0) = 0.1$, $P_E(0) = 0.1$, $S(0) = 0.8$, $Q_t(0) = 0$ $Q_p(0) = 0$, $Z(0) = 0$.
3. $P_N(0) = 0.2$, $P_E(0) = 0.2$, $S(0) = 0.2$, $Q_t(0) = 0.2$ $Q_p(0) = 0.2$, $Z(0) = 0$.
4. $P_N(0) = 0.1$, $P_E(0) = 0.1$, $S(0) = 0.5$, $Q_t(0) = 0$ $Q_p(0) = 0$, $Z(0) = 0.3$.

For $R_0 < 1$, Figure 2 shows that the smoking-free equilibrium E_0 is globally asymptotically stable. For $R_0 > 1$, Figure 3 shows that the smoking-present equilibrium E^* is globally asymptotically stable.

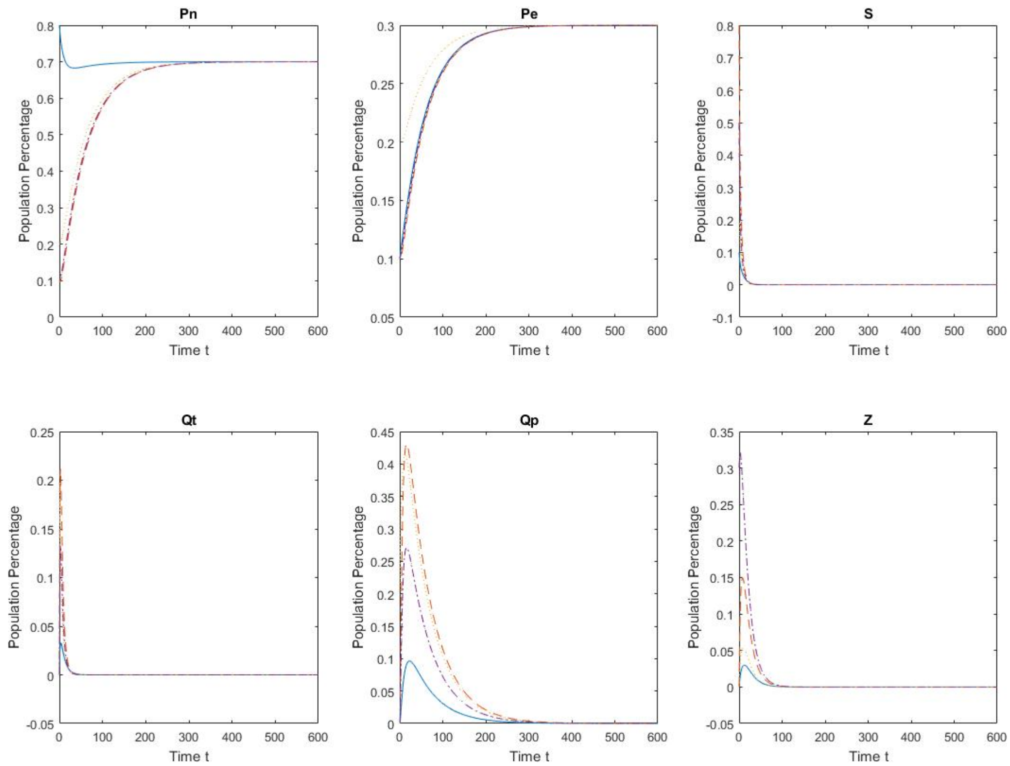

Figure 2. $R_0 < 1$, E_0 is globally asymptotically stable.

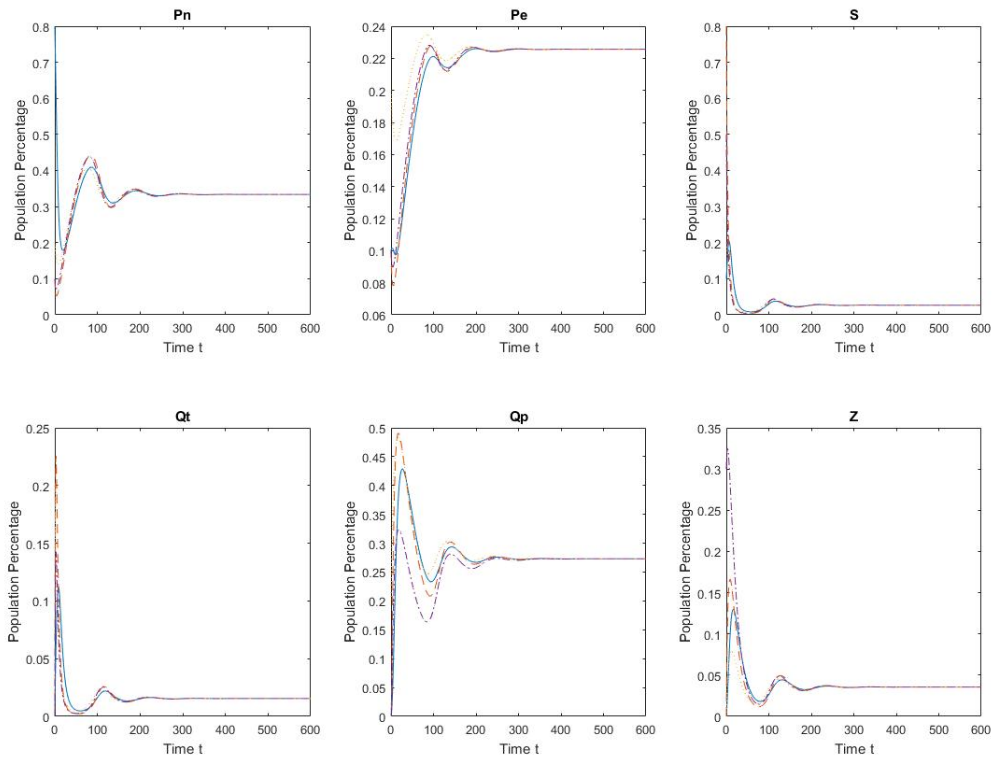

Figure 3. $R_0 > 1$, E^* is globally asymptotically stable.

Table 1. Table of parameter values.

Parameter	Meaning	Value	Source
q	non-educated portion of new recruitment rate	0.7	Estimated for test case
μ	natural death rate and new recruitment rate	0.017	McEvoy, John W., et al. "Mortality rates in smokers and nonsmokers in the presence or absence of coronary artery calcification." *JACC: Cardiovascular Imaging* 5.10 (2012): 1037-1045.
β	transmission coefficient for potential smokers (both non-educated and educated) transfer to smokers (S)	0.2/0.7	Estimated for test cases
δ	immuity coefficient for educated population (P_E) to lower the transfer to smokers (S)	0.25	Smoking & Tobacco Use.17 Centers for Disease Control and Prevention, *Centers for Disease Control and Prevention*, 3 Feb. 2017
γ	transmission coefficient for smokers (S) transfer to temporary quitters (Q_t)	0.554	Morbidity and Mortality Weekly Report (MMWR).17 Centers for Disease Control and Prevention, *Centers for Disease Control and Prevention*, 14 Aug. 2017
α	transmission coefficient for temporary quitters (Q_t) transfer to smokers (S)	0.48	Morbidity and Mortality Weekly Report (MMWR).17 Centers for Disease Control and Prevention, *Centers for Disease Control and Prevention*, 14 Aug. 2017
η	transmission coefficient for temporary quitters (Q_t) transfer to permanent quitters (Q_p)	0.074	Morbidity and Mortality Weekly Report (MMWR).17 Centers for Disease Control and Prevention, *Centers for Disease Control and Prevention*, 14 Aug. 2017
λ	transmission coefficient for smokers (S) transfer to smokers with diseases (Z)	0.4233	Smoking & Tobacco Use.17 Centers for Disease Control and Prevention, *Centers for Disease Control and Prevention*, 3 Feb. 2017
ν	extra death rate for smokers with diseases (Z)	0.043	McEvoy, John W., et al. "Mortality rates in smokers and nonsmokers in the presence or absence of coronary artery calcification." *JACC: Cardiovascular Imaging* 5.10 (2012): 1037-1045.

5. Discussion

In this paper, we consider the health education effect on the smoking dynamic model. We have derived the reproduction number (R_0) and obtained the following results: when $R_0 < 1$, smoking-free equilibrium is both locally and globally asymptotically stable. As the educated susceptible population increases, the permanent quitter population also increases. When $R_0 > 1$, we proved the smoking-present equilibrium is globally asymptotically stable by constructing Lyapunov function. When the ratio of educated susceptible group increases, the permanent quitter group experiences a time frame of oscillation then becomes stable. The results imply that increasing the health education population not only increases the permanent quitter, but also reduce the difficulty of non-smoking work of the area.

It will be very interesting to consider the time delay in this model, and it will be more realistic and give us more insights into the smoking dynamics, but some complex dynamic behaviors may occur([15, 17]).

Acknowledgments

The authors would like to thank the generous support from the mathematics department at University of Evansville.

Conflict of interest

Authors declare no conflicts of interest in this paper.

References

1. World Health Organization, *WHO global report on trends in prevalence of tobacco smoking 2015*, World Health Organization, 2015.
2. V. Bilano, S. Gilmour, T. Moffiet, et al. *Global trends and projections for tobacco use, 1990–2025: an analysis of smoking indicators from the WHO Comprehensive Information Systems for Tobacco Control*, The Lancet, **385** (2015), 966–976.
3. I. D. Bier, J. Wilson, P. Studt, et al. *Auricular acupuncture, education, and smoking cessation: a randomized, sham-controlled trial*, Am. J. Public Health, **92** (2002), 1642–1647.
4. D. de Walque, *Does education affect smoking behaviors?: Evidence using the Vietnam draft as an instrument for college education*, J. Health Econ., **26** (2007), 877–895.
5. S. Durkin, E. Brennan and M. Wakefield, *Mass media campaigns to promote smoking cessation among adults: an integrative review*, Tob. control, **21** (2012), 127–138.
6. O. Sharomi and A. B. Gumel, *Curtailing smoking dynamics: a mathematical modeling approach*, *Appl. Math. Comput.*, **195** (2008), 475–499.
7. Z. Alkhudhari, S. Al-Sheikh and S. Al-Tuwairqi, *Global dynamics of a mathematical model on smoking*, International Scholarly Research Notices, **2014** (2014), 1–7.
8. D. Qamar, O. Muhammad, H. Takasar, et al. *Qualitative behavior of a smoking model*, Adv. Differ. Equ-NY, **2016** (2016), 96.

9. J. W. McEvoy, M. J. Blaha, J. J. Rivera, et al. *Mortality rates in smokers and nonsmokers in the presence or absence of coronary artery calcification*, JACC-CARDIOVASC IMAG., **5** (2012), 1037–1045.

10. G. Zaman, *Qualitative behavior of giving up smoking models*, B. Malays. Math. Sci. So., **34** (2009), 403–415.

11. H. Xiang, N. N. Song and H. F. Huo, *Modeling effects of public health educational campaigns on drinking dynamics*, J. Biol. Dynam., **10** (2016), 164–178.

12. H. Xiang, C. C. Zhu and H. F. Huo, *Modelling the effect of immigration on drinking behaviour*, J. Biol. Dynam., **11** (2017), 275–298.

13. H. F. Huo and C. C. Zhu, *Influence of relapse in a giving up smoking model*, Abstr. Appl. Anal., **2013** (2013), 1–12.

14. H. F. Huo and N. N. Song, *Global stability for a binge drinking model with two-stage*, Discrete Dyn. Nat. Soc., **2012** (2012), 1–15.

15. H. F. Huo, Y. L. Chen and H. Xiang, *Stability of a binge drinking model with delay*, J. Biol. Dynam., **11** (2017), 210–225.

16. P. Van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci., **180** (2002), 29–48.

17. H. T. Zhao and M. C. Zhao, *Global Hopf bifurcation analysis of an susceptible-infective-removed epidemic model incorporating media coverage with time delay*, J. Biol. Dynam., **11** (2017), 8–24.