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Mathematical analysis of a smoking model with anti-smoking campaign rate in Thailand

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ABSTRACT

Smoking is one of the leading causes of preventable mortality worldwide, affecting multiple organ systems and contributing to numerous chronic diseases. It is well established that smoking increases the risk of lung cancer, cardiovascular diseases such as heart attacks and strokes, and chronic respiratory conditions, including chronic obstructive pulmonary disease (COPD). In Thailand, mathematical modeling has not yet been applied to the study of smoking behavior. To better understand smoking behavior and the effects of anti-smoking interventions, this study develops a nonlinear mathematical model of smoking in Thailand that incorporates the influence of anti-smoking campaigns. The population is categorized into non-smokers, active smokers, permanent quitters, and temporary quitters. This simplification makes the model easier to analyze and more practical to use. The model assumes that non-smokers may become smokers through social interactions and that smokers may quit either temporarily or permanently, with temporary quitters susceptible to relapse. The boundedness of the model is proven, and the basic reproductive number (R_0) is derived using the next-generation matrix method. Stability analyses of both the smoking-free and smoking-present equilibrium points using the Routh–Hurwitz criteria show that the system is locally asymptotically stable when $R_0 < 1$, indicating effective control of smoking prevalence under these conditions. Numerical simulations further demonstrate that increasing the rate of anti-smoking campaigns significantly reduces the number of active smokers, highlighting the importance of sustained public health interventions. Overall, this combined analytical and numerical framework offers valuable insights into smoking dynamics and provides a practical tool for designing and evaluating strategies to reduce smoking rates and associated health burdens.

Keywords: Smoking, Mathematical model, Anti-smoking campaign, Stability analysis

INTRODUCTION

Tobacco use remains a significant public health concern in Thailand, with far-reaching consequences for individual health and the national economy [1]. The detrimental effects of smoking are particularly evident in the mortality rates associated with secondhand smoke exposure. Secondhand smoke leads to over 9,400 deaths annually in Thailand, surpassing even the United States' figure of 7,300 deaths [2]. Smoking-related illnesses such as lung cancer, heart disease, and chronic respiratory conditions contribute to substantial healthcare costs, with costs estimated at approximately 0.5% of Thailand's GDP annually [3]. Despite efforts to reduce smoking prevalence, rates among Thai adults aged 15 and above have remained stagnant at 19-21% as of 2024, threatening the country's goal target of reducing smoking by 30% by 2025 [4, 5]. High-risk groups include working-age males (39.8%) and young males aged 10-14 (11.3%) [5], emphasizing the necessity for targeted interventions.

Socioeconomic factors significantly influence and are important for predicting smoking behaviors among Thai adults in mathematical models [6, 7]. Moreover, these factors can help identify target groups for anti-smoking awareness campaigns. Research indicates that households with financial strain are 2.41 times more likely to include smokers, while extended families are 1.53 times more likely to experience smoking compared to nuclear families [8]. Globally, tobacco consumption has decreased by 33% since 2000, according to the World Health Organization (WHO) [9]. However, countries such as Indonesia and Egypt have seen increases in smoking rates [10]. Brazil serves as a successful model of tobacco control through MPOWER strategies like taxation and advertising bans, which have reduced smoking prevalence by 35% [11]. Using ideas from other countries together with information about Thailand's own society and economy gives Thailand a chance to create better policies that fit its special needs.

Several dynamic models of smoking behavior have been developed to explore different intervention

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strategies. In 2019, Verma & Bhadauria [12] introduced a mathematical model dividing the population into three groups (potential smokers, current smokers, and permanent quitters) to analyze how anti-smoking campaigns impact smoking behavior, mainly through education and media. Khyar et al. [13] presented a five-compartment smoking model utilizing optimal control theory to identify effective intervention strategies. In 2022, Said et al. [14] studied a mathematical model to analyze the spread of smoking, considering both individual and social influences. The nonlinear model divides the population into different smoking statuses and examines the transmission dynamics within large social networks and among individuals. Next year, Sofia et al. [15] focused on the model by integrating media-aware populations, demonstrating that sustained awareness campaigns could reduce smoking prevalence.

Building on previous research, we improved the model by reducing the number of population classes from five to four. This simplification makes the model easier to analyze and more practical to use. It focuses on the most critical stages of smoking by removing redundant or overlapping groups without losing important behavior details. As a result, the model is mathematically simpler and more applicable in practice, while still capturing the essential elements needed to designing effective interventions.

This study developed a mathematical model of smoking behavior by investigating the effect of the anti-smoking campaign rate. The local stability of the model's equilibrium points, smoking-free and smoking-present, was proven and demonstrated the conditions under which smoking prevalence can either persist or decline. Furthermore, this paper has been appropriately adapted to the social and cultural context in Thailand.

MATERIALS AND METHODS

1. Model formulation

The total population is divided into four classes of individual, namely non-smokers (S_1), active smokers (I), permanent quitters (Q) and temporary quitters (S_2). We assumed that individuals in the non-smoker class become active smokers when they interact with smokers or social smokers (at rate $1-\phi$ and β). Moreover, they either quit smoking temporarily (at rate $\sigma+\phi$ and $1-\xi$) or permanently quit smoking (at rate $\sigma+\phi$ and ξ). The temporary quitting smokers may become smokers again. In Figure1, it illustrates the diagram of the smoking model consisting of non-smokers, active smokers, permanent quitters, and temporary quitters classes from the total population (N).

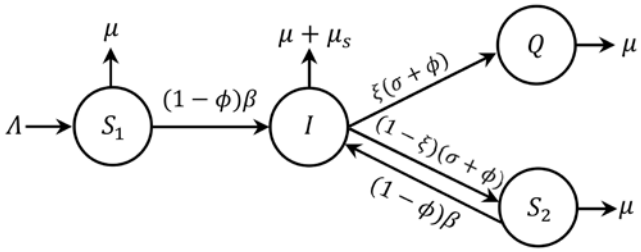

Figure 1 The smoking model.

From Figure 1, the smoking model identifies the coefficients viz Λ to represent the recruitment rate of potential smokers, μ for natural death rate, μ_s to denote disease (smoking) death rate, ϕ for campaign rate, β to represent rate of potential smokers becoming smokers, σ to denote quitting smoking rate, and ξ for proportion of permanent quitting smoking. The model variables and parameters are summarized in Table 1.

Table 1 Description of the model variables and parameters.

	Symbol	Description	Unit
Variable	S_1	Non-smokers	Person
	I	Active smokers	Person
	Q	Permanent quitters	Person
	S_2	Temporary quitters	Person
Parameter	Λ	Recruitment rate of potential smokers	Person per day
	μ	Natural death rate	Per day
	μ_s	Disease (smoking) death rate	Per day
	ϕ	Campaign rate	Per day
	β	Rate of potential smokers becoming smokers	Per person per day
	σ	Quitting smoking rate	Per day
	ξ	Proportion of permanent quitting smoking	-

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The mathematical representation of the smoking behavior has shown as a system of nonlinear ordinary differential equations (1) given as:

$$\begin{aligned}\frac{dS_1}{dt} &= \Lambda - (1-\phi)\beta S_1 I - \mu S_1\\ \frac{dI}{dt} &= (1-\phi)\beta(S_1+S_2)I - (\sigma+\phi)I - (\mu+\mu_s)I\\ \frac{dQ}{dt} &= \xi(\sigma+\phi)I - \mu Q\\ \frac{dS_2}{dt} &= (1-\xi)(\sigma+\phi)I - (1-\phi)\beta S_2 I - \mu S_2\end{aligned} \quad (1)$$

with initial conditions $S_1(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, and $S_2(0) \geq 0$. Here $N(t) = S_1(t) + I(t) + Q(t) + S_2(t)$ is the total population.

We differentiate $N(t)$ with respect to t, then

$$\begin{aligned}\frac{dN}{dt} &= \frac{dS_1}{dt} + \frac{dI}{dt} + \frac{dQ}{dt} + \frac{dS_2}{dt}\\ &= \Lambda - \mu(S_1 + I + Q + S_2) - \mu_s I\\ &= \Lambda - \mu N - \mu_s I\\ &\leq \Lambda - \mu N\end{aligned}$$

$$\frac{dN}{dt} + \mu N \leq \Lambda.$$

By solving the above inequality, we get

$$N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t} - \frac{\Lambda}{\mu}e^{-\mu t}.$$

Taking the limit $t \to \infty$, yields

$$N(t) \leq \frac{\Lambda}{\mu} = \mathcal{N}.$$

2. Mathematical analysis

2.1 Existence of equilibria

From the system of nonlinear ordinary differential equations (1), stability analysis has been carried out the equilibrium points. To determine the equilibrium points, each equation in the system of equation (1) must be equal to zero, that is, $\frac{dS_1}{dt} = 0$, $\frac{dI}{dt} = 0$, $\frac{dQ}{dt} = 0$, and $\frac{dS_2}{dt} = 0$.

The system (1) can be written as:

$$\Lambda - (1-\phi)\beta S_1 I - \mu S_1 = 0 \quad (2)$$

$$(1-\phi)\beta(S_1+S_2)I - (\sigma+\phi)I - (\mu+\mu_s)I = 0 \quad (3)$$

$$\xi(\sigma+\phi)I - \mu Q = 0 \quad (4)$$

$$(1-\xi)(\sigma+\phi)I - (1-\phi)\beta S_2 I - \mu S_2 = 0 \quad (5)$$

Using equations (2) - (5), two equilibrium points are found, namely the smoking-free equilibrium point $E_0 = (S_1^0, I^0, Q^0, S_2^0)$ and the smoking-present equilibrium point $E_1 = (S_1^*, I^*, Q^*, S_2^*)$.

The smoking-free equilibrium refers to the situation in which the population has no smokers, then $I = 0$.

From equation (2), we get $S_1 = \Lambda/\mu$. Then the smoking-free equilibrium point of the model is:

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$$

2.2 The basic reproduction number

The basic reproduction number (R_0) represents how many new people, on average, will start smoking due to the influence of one current smoker throughout their entire smoking lifetime. We calculate R_0 by using the next generation matrix method [16].

Let $x = (S_1, I, Q, S_2)^T$, the system (1) can be rewritten in the matrix form,

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathrm{V}(x),$$

where

$$\mathcal{F}(x) = \begin{bmatrix}\Lambda\\ (1-\phi)\beta(S_1+S_2)I\\ \xi(\sigma+\phi)I\\ (1-\xi)(\sigma+\phi)I\end{bmatrix},$$

and

$$\mathrm{V}(x) = \begin{bmatrix}(1-\phi)\beta S_1 I + \mu S_1\\ (\sigma+\phi)I + (\mu+\mu_s)I\\ \mu Q\\ (1-\phi)\beta S_2 I + \mu S_2\end{bmatrix}.$$

The Jacobian matrices of $\mathcal{F}(x)$ and $\mathrm{V}(x)$ are evaluated as follows:

$$F_{E_0} = \begin{bmatrix}0 & 0 & 0 & 0\\ 0 & (1-\phi)\beta\frac{\Lambda}{\mu} & 0 & 0\\ 0 & \xi(\sigma+\phi) & 0 & 0\\ 0 & (1-\xi)(\sigma+\phi) & 0 & 0\end{bmatrix},$$

and

$$V_{E_0} = \begin{bmatrix}\mu & (1-\phi)\beta\frac{\Lambda}{\mu} & 0 & 0\\ 0 & \sigma+\phi+\mu+\mu_s & 0 & 0\\ 0 & 0 & \mu & 0\\ 0 & 0 & 0 & \mu\end{bmatrix}.$$

Therefore, the basic reproduction number is given by the spectral radius (ρ) of the next generation matrix:

$$R_0 = \rho\left(F_{E_0}V_{E_0}^{-1}\right) = \frac{(1-\phi)\beta\Lambda}{(\sigma+\phi+\mu+\mu_s)\mu} = \frac{a_0\Lambda}{a_1\mu}$$

where $a_0 = (1-\phi)\beta$ and $a_1 = \sigma+\phi+\mu+\mu_s$.

Theorem 2.1 The smoking-present equilibrium point of the system (1) is

$$E_1 = \left(\frac{\Lambda}{a_0 I^* + \mu}, \frac{\Lambda(R_0-1)}{a_2 R_0}, \frac{a_3 I^*}{\mu}, \frac{(1-\xi)a_4 I^*}{a_0 I^* + \mu}\right)$$

where $a_0 = (1-\phi)\beta$, $a_2 = \xi(\sigma+\phi)+\mu+\mu_s$, $a_3 = \xi(\sigma+\phi)$, and $a_4 = \sigma+\phi$.

Proof For the smoking-present equilibrium, the population $S_1^* \neq 0, I^* \neq 0, Q^* \neq 0$, and $S_2^* \neq 0$.

From equation (2): $\Lambda - (1-\phi)\beta S_1 I - \mu S_1 = 0$, thus, we find

$$S_1^* = \frac{\Lambda}{(1-\phi)\beta I^* + \mu} = \frac{\Lambda}{a_0 I^* + \mu}.$$

From equation (4): $\xi(\sigma+\phi)I - \mu Q = 0$, which implies that

$$Q^* = \frac{\xi(\sigma+\phi)I^*}{\mu} = \frac{a_3 I^*}{\mu}.$$

From equation (5), we can write

$$S_2^* = \frac{(1-\xi)(\sigma+\phi)I^*}{(1-\phi)\beta I^* + \mu} = \frac{(1-\xi)a_4 I^*}{a_0 I^* + \mu}.$$

From equation (3), we obtain

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$$[(1-\phi)\beta(S_1^*+S_2^*)-(\sigma+\phi+\mu+\mu_s)]I^*=0.$$

Since $I^*\neq 0$, that is,

$$(1-\phi)\beta(S_1^*+S_2^*)-(\sigma+\phi+\mu+\mu_s)=0$$

$$S_1^*+S_2^*=\frac{\sigma+\phi+\mu+\mu_s}{(1-\phi)\beta}$$

$$\frac{\Lambda+(1-\xi)(\sigma+\phi)I^*}{(1-\phi)\beta I^*+\mu}=\frac{\Lambda}{\mu R_0}$$

$$\Lambda\mu R_0+(1-\xi)(\sigma+\phi)\mu R_0 I^*=\Lambda(1-\phi)\beta I^*+\Lambda\mu$$

$$I^*=\frac{\Lambda\mu(1-R_0)}{(1-\xi)(\sigma+\phi)\mu R_0-\Lambda(1-\phi)\beta}$$

$$I^*=\frac{\Lambda\mu(1-R_0)}{(1-\xi)(\sigma+\phi)\mu R_0-(\sigma+\phi+\mu+\mu_s)\mu R_0}.$$

Therefore,

$$I^*=\frac{\Lambda(R_0-1)}{[\xi(\sigma+\phi)+\mu+\mu_s)]R_0}=\frac{\Lambda(R_0-1)}{a_2R_0}.$$

Hence, the smoking-present equilibrium point of the model is:

$$E_1=\left(\frac{\Lambda}{a_0I^*+\mu},\frac{\Lambda(R_0-1)}{a_2R_0},\frac{a_3I^*}{\mu},\frac{(1-\xi)a_4I^*}{a_0I^*+\mu}\right),$$

where $a_0=(1-\phi)\beta$, $a_2=\xi(\sigma+\phi)+\mu+\mu_s$, $a_3=\xi(\sigma+\phi)$, and $a_4=\sigma+\phi$. □

2.3 Stability analysis

According to the system (1), the Jacobian matrix (J) is found:

$$J=\begin{bmatrix}-a_0I-\mu & -a_0S_1 & 0 & 0\\ a_0I & a_0(S_1+S_2)-a_1 & 0 & a_0I\\ 0 & \xi(\sigma+\phi) & -\mu & 0\\ 0 & (1-\xi)(\sigma+\phi)-a_0S_2 & 0 & -a_0I-\mu\end{bmatrix} \quad (6)$$

where $a_0=(1-\phi)\beta$ and $a_1=\sigma+\phi+\mu+\mu_s$.

At the smoking-free equilibrium point, the local stability is determined by the eigenvalues (λ) from:

$$\det(J_{E_0}-\lambda I)=0 \quad (7)$$

where J_{E_0} is the Jacobian matrix at the smoking-free equilibrium point and I is the identity matrix. If all the eigenvalues of the characteristic equation have negative real parts, E_0 is stable.

At the smoking-present equilibrium point, the local stability is determined by the eigenvalues from:

$$\det(J_{E_1}-\lambda I)=0 \quad (8)$$

where J_{E_1} is the Jacobian matrix at the smoking-present equilibrium point. If all the eigenvalues of the characteristic equation have negative real parts, E_1 is stable.

Theorem 2.2 If $R_0<1$, the smoking-free equilibrium point E_0 is local asymptotically stable while E_0 is unstable if $R_0>1$

Proof At the smoking-free equilibrium point, substituting $S_1=\Lambda/\mu$, $I=0$, and $S_2=0$ into equation (6), it obtains:

$$J_{E_0}=\begin{bmatrix}-\mu & -a_0\frac{\Lambda}{\mu} & 0 & 0\\ 0 & a_0\frac{\Lambda}{\mu}-a_1 & 0 & 0\\ 0 & \xi(\sigma+\phi) & -\mu & 0\\ 0 & (1-\xi)(\sigma+\phi) & 0 & -\mu\end{bmatrix},$$

where $a_0=(1-\phi)\beta$ and $a_1=\sigma+\phi+\mu+\mu_s$.

The eigenvalues are found by solving the characteristic equation (7).

Therefore, the eigenvalues of J_{E_0} are given as follows:

$\lambda_1=\lambda_2=\lambda_3=-\mu$, and $\lambda_4=a_1(R_0-1)$.

It is obvious that $\lambda_4<0$ if $R_0<1$. Thus, all eigenvalues of equation (7) are negative real parts. Therefore, the smoking-free equilibrium point E_0 is local asymptotically stable.

If $R_0>1$, equation (7) has a positive eigenvalue. Therefore, E_0 is unstable. □

Theorem 2.3 If $R_0>1$, the smoking-present equilibrium point E_1 is local asymptotically stable.

Proof At the smoking-present equilibrium point, substituting $S_1^*=\frac{\Lambda}{a_0I^*+\mu}$, $I^*=\frac{\Lambda(R_0-1)}{a_2R_0}$, and $S_2^*=\frac{(1-\xi)a_4I^*}{a_0I^*+\mu}$ into equation (6), it obtain:

$$J_{E_1}=\begin{bmatrix}-\frac{\mu a_1(R_0-1)}{a_2}-\mu & -\frac{a_1a_2R_0}{a_1(R_0-1)+a_2} & 0 & 0\\ \frac{\mu a_1(R_0-1)}{a_2} & 0 & 0 & \frac{\mu a_1(R_0-1)}{a_2}\\ 0 & a_3 & -\mu & 0\\ 0 & \frac{(a_4-a_3)a_2}{a_1(R_0-1)+a_2} & 0 & -\frac{\mu a_1(R_0-1)}{a_2}-\mu\end{bmatrix},$$

where $a_1=\sigma+\phi+\mu+\mu_s$, $a_2=\xi(\sigma+\phi)+\mu+\mu_s$, $a_3=\xi(\sigma+\phi)$, and $a_4=\sigma+\phi$.

The eigenvalues are found by solving the characteristic equation:

$$\det(J_{E_1}-\lambda I)=0$$

From the computation, the eigenvalues were $\lambda_1=-\mu$ and the other three eigenvalues are the roots of the following equation:

$$\lambda^3+A\lambda^2+B\lambda+C=0,$$

where

$$A=\frac{2\mu[a_1(R_0-1)+a_2]}{a_2},$$

$$B=\frac{\mu a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_1(R_0-1)+a_2}+\mu^2[a_1(R_0-1)+a_2]^2,$$

and

$$C=\frac{\mu^2a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_2}.$$

When $R_0>1$, then $A>0$,

$$B=\frac{\mu a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_1(R_0-1)+a_2}+\mu^2[a_1(R_0-1)+a_2]^2$$

$$>\frac{\mu a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_1(R_0-1)+a_2}$$

$$=\frac{\mu a_1(R_0-1)[a_3-a_4+(a_4+\mu+\mu_s)R_0]}{a_1(R_0-1)+a_2}$$

$$=\frac{\mu a_1(R_0-1)[a_3+a_4(R_0-1)+(\mu+\mu_s)R_0]}{a_1(R_0-1)+a_2}>0,$$

$$C=\frac{\mu^2a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_2}>0,$$

and

$$AB=\frac{2\mu^2a_1(R_0-1)(a_3-a_4+a_1R_0)}{a_2}+\frac{2\mu^2}{a_2}[a_1(R_0-1)+a_2]^3$$

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$$> \frac{\mu^2 a_1 (R_0 - 1)(a_3 - a_4 + a_1 R_0)}{a_2} = C$$

By the Routh-Hurwitz criterion [17], all eigenvalues of equation (9) are negative real parts. Therefore, the smoking-present equilibrium point E_1 is local asymptotically stable. □

2.4 Sensitivity analysis

A sensitivity analysis of the basic reproduction number was conducted to assess to determine the robustness of the model's stability conclusions and to identify which parameters are the most effective targets for intervention.

The normalized forward sensitivity index ($\Upsilon_p^{R_0}$) is employed by using the formula

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0}$$

where p is any parameter of the model. Then

$$\Upsilon_\Lambda^{R_0} = \frac{\partial R_0}{\partial \Lambda} \cdot \frac{\Lambda}{R_0}$$
$$= \frac{(1-\phi)\beta}{(\sigma+\phi+\mu+\mu_s)\mu} \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\Lambda}{(1-\phi)\beta\Lambda} = 1$$
$$\Upsilon_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \cdot \frac{\mu}{R_0}$$
$$= -\frac{(1-\phi)\beta\Lambda}{(\sigma+\phi+\mu+\mu_s)\mu}\left[\frac{1}{\mu} + \frac{1}{\sigma+\phi+\mu+\mu_s}\right] \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu^2}{(1-\phi)\beta\Lambda}$$
$$= -\frac{\sigma+\phi+2\mu+\mu_s}{\sigma+\phi+\mu+\mu_s}$$
$$\Upsilon_{\mu_s}^{R_0} = \frac{\partial R_0}{\partial \mu_s} \cdot \frac{\mu_s}{R_0}$$
$$= -\frac{(1-\phi)\beta\Lambda}{(\sigma+\phi+\mu+\mu_s)^2\mu} \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\mu_s}{(1-\phi)\beta\Lambda}$$
$$= -\frac{\mu_s}{\sigma+\phi+\mu+\mu_s}$$
$$\Upsilon_\phi^{R_0} = \frac{\partial R_0}{\partial \phi} \cdot \frac{\phi}{R_0}$$
$$= -\frac{(\sigma+\phi+\mu+\mu_s)\mu\beta\Lambda + (1-\phi)\beta\Lambda\mu}{(\sigma+\phi+\mu+\mu_s)^2\mu^2} \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\phi}{(1-\phi)\beta\Lambda}$$
$$= -\frac{(\sigma+\mu+\mu_s+1)\phi}{(1-\phi)(\sigma+\phi+\mu+\mu_s)}$$
$$\Upsilon_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0}$$
$$= \frac{(1-\phi)\Lambda}{(\sigma+\phi+\mu+\mu_s)\mu} \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\beta}{(1-\phi)\beta\Lambda} = 1$$
$$\Upsilon_\sigma^{R_0} = \frac{\partial R_0}{\partial \sigma} \cdot \frac{\sigma}{R_0}$$
$$= -\frac{(1-\phi)\beta\Lambda}{(\sigma+\phi+\mu+\mu_s)^2\mu} \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\sigma}{(1-\phi)\beta\Lambda}$$
$$= -\frac{\sigma}{\sigma+\phi+\mu+\mu_s}$$
$$\Upsilon_\xi^{R_0} = \frac{\partial R_0}{\partial \xi} \cdot \frac{\xi}{R_0} = 0 \cdot \frac{(\sigma+\phi+\mu+\mu_s)\mu\xi}{(1-\phi)\beta\Lambda} = 0.$$

The sign of the normalized forward sensitivity index indicates the direction of influence (positive means R_0 increases when p increases), while the magnitude indicates the strength of the influence.

RESULTS AND DISCUSSION

To facilitate a more general and scalable analysis of the numerical results, we now introduce a dimensionless formulation of the system (1) by the new variables: $\bar{S_1} = S_1/\mathcal{N}$, $\bar{I} = I/\mathcal{N}$, $\bar{Q} = Q/\mathcal{N}$, and $\bar{S_2} = S_2/\mathcal{N}$ (see Appendix for more details), then the system (1) becomes

$$\frac{d\bar{S_1}}{dt} = \mu - (1-\phi)\beta\bar{S_1}\bar{I}\mathcal{N} - \mu\bar{S_1}$$
$$\frac{d\bar{I}}{dt} = (1-\phi)\beta(\bar{S_1} + \bar{S_2})\bar{I}\mathcal{N} - (\sigma+\phi)\bar{I} - (\mu+\mu_s)\bar{I}$$
$$\frac{d\bar{Q}}{dt} = \xi(\sigma+\phi)\bar{I} - \mu\bar{Q}$$
$$\frac{d\bar{S_2}}{dt} = (1-\xi)(\sigma+\phi)\bar{I} - (1-\phi)\beta\bar{S_2}\bar{I}\mathcal{N} - \mu\bar{S_2}$$

To illustrate the insights gained from the above mathematical model of smoking, we employed numerical simulations. These simulations allow for the exploration of model behavior under various conditions, through adjustment of the parameters stated in Table 2.

Table 2 Parameters used in the investigation.

Symbol	Description	Value	Source
Λ	Recruitment rate of potential smokers	20	Estimate
μ	Natural death rate	0.0091/365	[18]
μ_s	Disease (smoking) death rate	0.0072/365	[19, 20]
ϕ	Campaign rate	0 - 0.04	Estimate
β	Rate of potential smokers becoming smokers	$0.05/\mathcal{N}$	[15, 21]
σ	Quitting smoking rate	0.011	[20]
ξ	Proportion of permanent quitting smoking	0.023	[22]

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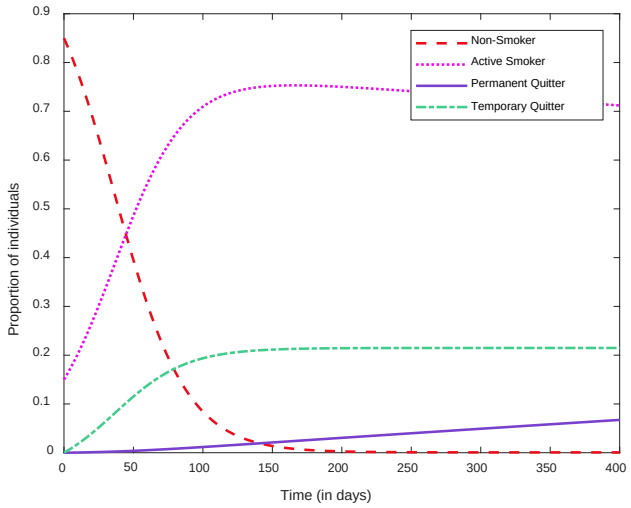

Figure 2 Variation of individuals in each class over 400 days when the campaign rate (ϕ) was 0, with the initial conditions of $(\bar{S_1}, \bar{I}, \bar{Q}, \bar{S_2}) = (0.85, 0.15, 0, 0)$.

Figure 2 shows the simulated changes in population sizes for non-smokers who are at risk of becoming smokers ($\bar{S_1}$), active smokers ($\bar{I}$), ex-smokers who permanently quit smoking ($\bar{Q}$), and ex-smokers who temporarily quit smoking ($\bar{S_2}$) over a year period. The simulation uses hypothetical parameter values to illustrate the dynamics of smoking behavior. Initially, the population consists of 85% non-smokers and 15% active smokers. As time progresses, the active smoker population increases, while the non-smoker population declines. The model also shows the proportions of temporary and permanent quitters changing over time. The temporary quitters initially increase as some smokers attempt to quit, but this number decreases as some of those temporary quitters relapse back to smoking, while others successfully transition to permanent quitters. The permanent quitter population consistently increases, showing the accumulation of individuals who have successfully quit smoking over the long term. The time required for the smoking system to settle into its smoking-present equilibrium point is characterized by different rates of convergence among its classes. While the non-smokers and temporary quitters reach their long-term proportions within approximately 200 days, creating a state of practical stability, the system's absolute convergence takes much longer. This extended time is necessary because the active smoker proportion does not reach the smoking-present equilibrium point within the 400-day, but continues a very slow decline as individuals exit the class through quitting. This slow, continuous flow out of active smokers simultaneously drives the accumulation of permanent quitters, making permanent quitters the slowest-moving class. Since both active smokers is still subtly changing and permanent quitters is still visibly increasing at 400 days, the total time required for the entire system to achieve its strict mathematical steady-state is characterized by an extremely gradual approach, with the final pace governed by the natural death rate and the slow permanent quitter accumulation.

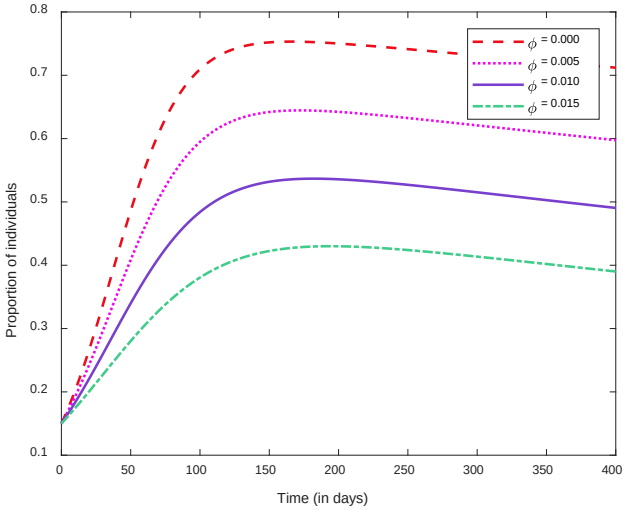

Figure 3 Variation of active smokers ($\bar{I}$) over 400 days with various campaign rates (ϕ) and the initial conditions of $(\bar{S_1}, \bar{I}, \bar{Q}, \bar{S_2}) = (0.85, 0.15, 0, 0)$.

Figure 3 presents the effect of the anti-smoking campaign rate (ϕ) on the proportion of active smokers in the smoking model. The simulation shows that as the campaign rate increases from $\phi = 0.000$ to $\phi = 0.015$, the proportion of individuals who are smokers stabilizes at a lower level over time. This indicates that a higher campaign rate improves the effectiveness of smoking cessation efforts, resulting in a smaller proportion of the population remaining smokers. In addition, since the value of basic reproduction number R_0 is influenced by various factors including the anti-smoking campaign rate, the comparison of R_0 with various campaign rates is shown in Table 3.

To quantify the relative impact of each parameter, we performed the normalized forward sensitivity analysis on R_0. The sensitivity indices, presented in Table 4, reveal the exact magnitude and direction of influence for each parameter.

Table 3 Comparison of basic reproduction number with various campaign rates.

Campaign rate (ϕ)	0.000	0.010	0.020	0.030	0.040
Basic reproduction number (R_0)	4.527	2.352	1.578	1.182	0.940

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Table 4 Sensitivity Indices of the basic reproduction number.

Parameter	Sensitivity Index	Value	Qualitative Impact
Λ	+1	+1	Highly sensitive
μ	$-\frac{\sigma+\phi+2\mu+\mu_s}{\sigma+\phi+\mu+\mu_s}$	-1.002	Highly sensitive
μ_s	$-\frac{\mu_s}{\sigma+\phi+\mu+\mu_s}$	-0.002	Insensitive
ϕ	$-\frac{(\sigma+\mu+\mu_s+1)\phi}{(1-\phi)(\sigma+\phi+\mu+\mu_s)}$	0 at $\phi=0$ and -0.794 at $\phi=0.04$	Insensitive at $\phi=0$. Becomes highly effective immediately above 0.
β	+1	+1	Highly sensitive
σ	$-\frac{\sigma}{\sigma+\phi+\mu+\mu_s}$	-0.996	Highly sensitive
ξ	0	0	Insensitive

The key insight from the baseline scenario (at $\phi = 0$) is that the parameters Λ, μ, β,and σ all have an absolute index value very close to 1, confirming that the model's stability is critically dependent on these fundamental rates in the absence of a public health campaign. In addition, ϕ is insensitive at the boundary of $\phi = 0$ but quickly becomes highly sensitive when any intervention is implemented.

The findings from this nonlinear mathematical model of smoking dynamics in Thailand carry significant policy implications for public health strategies. By demonstrating that the basic reproductive number $R_0 < 1$ ensures local asymptotic stability of the smoking-free equilibrium, and that amplifying anti-smoking campaign rates substantially diminishes active smoker populations in simulations, policymakers can prioritize sustained, high-intensity interventions. These include nationwide media drives, school-based education, and community outreach, to drive $R_0 < 1$. This approach not only curbs preventable mortality from lung cancer, cardiovascular diseases, and COPD but also yields economic dividends through reduced healthcare expenditures and enhanced workforce productivity.

CONCLUSIONS

In this study, we developed and analyzed a compartmental model to understand the dynamics of smoking behavior within a population. The population of individuals was divided into four classes (non-smokers, active smokers, permanent quitters, and temporary quitters) without accounting for demographic factors such as age groups, gender, or socioeconomic status. The theoretical analysis focused on the local stability of the model's equilibrium points. We demonstrated the conditions under which smoking prevalence can either persist or decline.

Furthermore, numerical simulations were conducted to illustrate the dynamics of smoking behavior in each class. We also determined the effect of the campaign rate while excluding other tobacco control policies, such as taxation, smoking bans in public places, or access restrictions. This showed that a higher campaign rate resulted in a more significant decline in smoking prevalence. These finding aligns with Thailand's smoking trends, where prevalence among individuals aged 15 and older declined to 16.5% in 2024 from 17.4% in 2021 [21]. The decline highlights the effectiveness of tobacco control measures, even as e-cigarette use continues to rise.

In conclusion, the stability analysis and numerical experimentation offer an understanding of the modeled smoking dynamics. This framework can be used as a useful tool to design and evaluate strategies for reducing smoking prevalence.

Appendix

To derive a dimensionless formulation of the system (1) by substituting $\bar{S_1} = S_1/\mathcal{N}$, $\bar{I} = I/\mathcal{N}$, $\bar{Q} = Q/\mathcal{N}$, and $\bar{S_2} = S_2/\mathcal{N}$ into each equation of the system (1), then the equation $\frac{dS_1}{dt}$ becomes

$$\frac{dS_1}{dt} = \frac{d(\bar{S_1}\mathcal{N})}{dt} = \Lambda - (1-\phi)\beta\bar{S_1}\mathcal{N}\bar{I}\mathcal{N} - \mu\bar{S_1}\mathcal{N}$$

$$\mathcal{N}\frac{d\bar{S_1}}{dt} = \Lambda - (1-\phi)\beta\bar{S_1}\mathcal{N}\bar{I}\mathcal{N} - \mu\bar{S_1}\mathcal{N}$$

$$\frac{d\bar{S_1}}{dt} = \frac{\Lambda}{\mathcal{N}} - (1-\phi)\beta\bar{S_1}\bar{I}\mathcal{N} - \mu\bar{S_1}$$

$$\frac{d\bar{S_1}}{dt} = \mu - (1-\phi)\beta\bar{S_1}\bar{I}\mathcal{N} - \mu\bar{S_1}$$

Next, the equation $\frac{dI}{dt}$ becomes

$$\frac{dI}{dt} = \frac{d(\bar{I}\mathcal{N})}{dt} = (1-\phi)\beta(\bar{S_1}\mathcal{N} + \bar{S_2}\mathcal{N})\bar{I}\mathcal{N} - (\sigma+\phi)\bar{I}\mathcal{N} - (\mu+\mu_s)\bar{I}\mathcal{N}$$

$$\mathcal{N}\frac{d\bar{I}}{dt} = (1-\phi)\beta(\bar{S_1}\mathcal{N} + \bar{S_2}\mathcal{N})\bar{I}\mathcal{N} - (\sigma+\phi)\bar{I}\mathcal{N} - (\mu+\mu_s)\bar{I}\mathcal{N}$$

$$\frac{d\bar{I}}{dt} = (1-\phi)\beta(\bar{S_1} + \bar{S_2})\bar{I}\mathcal{N} - (\sigma+\phi)\bar{I} - (\mu+\mu_s)\bar{I}$$

Then, the equation $\frac{dQ}{dt}$ becomes

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$$\frac{dQ}{dt} = \frac{d(\bar{Q}\mathcal{N})}{dt} = \xi(\sigma+\phi)\bar{I}\mathcal{N} - \mu\bar{Q}\mathcal{N}$$

$$\mathcal{N}\frac{d\bar{Q}}{dt} = \xi(\sigma+\phi)\bar{I}\mathcal{N} - \mu\bar{Q}\mathcal{N}$$

$$\frac{d\bar{Q}}{dt} = \xi(\sigma+\phi)\bar{I} - \mu\bar{Q}$$

And the equation $\frac{dS_2}{dt}$ becomes

$$\frac{dS_2}{dt} = \frac{d(\bar{S_2}\mathcal{N})}{dt} = (1-\xi)(\sigma+\phi)\bar{I}\mathcal{N} - (1-\phi)\beta\bar{S_2}\mathcal{N}\bar{I}\mathcal{N} - \mu\bar{S_2}\mathcal{N}$$

$$\mathcal{N}\frac{d\bar{S_2}}{dt} = (1-\xi)(\sigma+\phi)\bar{I}\mathcal{N} - (1-\phi)\beta\bar{S_2}\mathcal{N}\bar{I}\mathcal{N} - \mu\bar{S_2}\mathcal{N}$$

$$\frac{d\bar{S_2}}{dt} = (1-\xi)(\sigma+\phi)\bar{I} - (1-\phi)\beta\bar{S_2}\bar{I}\mathcal{N} - \mu\bar{S_2}$$

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