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Awareness and Control of Smoking Habit through Education: A MathematicalModel

K. Siva[1], S. Athithan[2*]

[1,2]Department of Mathematics, College of Engineering and Technology,

SRM Institute of Science and Technology, Kattankulathur, Chennai-603 203, Tamilnadu, India

[2*] athithas@srmist.edu.in, [1] shivak@srmist.edu.in

ABSTRACT

In this paper we proposed a mathematical model for analyzing the dynamics of controlling smoking activity under the influence of educational awareness programmers, as well as the determination of individuals to stop smoking. The model exhibits two equilibrium points which are named as smoking free equilibrium and endemic equilibrium. These equilibrium points of the model are analyzed through stability analysis both analytically and numerically. Further, the model is enhanced by introducing stochasticity and analyzed the effect of stochastic model by comparing it with deterministic model. Numerical simulation of both the deterministic and stochastic model is exhibited to validate our analytical findings. Our result shows the better ways for eradicate smoking habit through the parameter effects of the model.

Keywords
Smoking Habit; Local Stability; Global stability; Stochastic model.

1. Introduction

Tuberculosis (TB) is one of the most common causes of death in early modern societies and may also be the captain of these men of death (as alluded to in 1680 by Bunyan). It is also the second common purpose of the planet to die after HIV / AIDS. Around one-third of the world population is infected with My co bacterium tuberculosis (Mtb), the Tuberculosis (TB) bacteria. Generally, with a ready immune system, the bacteria are made inactive. In 2003, 8.8 million people developed active tuberculosis, a rise in incidence of about 1% per annul [1], while 9.6 million people contracted the disease in 2006.

Noncommunicable diseases (NCDs), such as cancer, heart diseases and vascular diseases etc., have probably extreme socioeconomic results and major hurdle in Economic and social improvement of a country. Almost six million humans die from tobacco use annually, every from direct tobacco use and second-hand smoke. NCDs additionally kill at a younger age in low- and middle-income countries, wherever 29% of NCD deaths occur among people below the age of 60 (for complete detail refer to WHO report, 2010 [2]).

The paper concludes tobacco smoking, latent smoking, and biomass indoor air pollution have been implicated as dangerous factors for infection, illness, and death from tuberculosis (TB). Tobacco smoking and air pollution indoors are persistent or exposures grow in areas where TB presents a serious health risk [3].

This paper proposes and analyze mathematical models to consider the dynamics of smoking behavior under the influence of educational programs and also individual's determination to stop smoking. We establish the positivity and boundedness of the solutions in a biologically feasible region. A threshold value responsible for persistence of smoking is obtained and stability analysis on models is performed. We find that determination alone isn't sufficient to kill smoking however it can diminish the prevalence of smoker population [4].

In this paper tobacco use by teens and young adults remains shockingly high in the United States. Today, more than 3.6 million middle and high school students smoke cigarettes. In fact, for every person who dies due to smoking—more than 1,200 each day—at

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least two youth or young adults become regular smokers. Nearly 90% of these replacement smokers try their first cigarette by age 18. Clearly, we have not solved the problem [5].

Due to epidemic aspect of smoking, a lot of researchers concentrate on understanding the dynamics of smoking through mathematical models [[6], [7], [8], [9], [10], [11]].Policymakers play a very important role in having the insight into mathematical models. There are very few research papers published related to smoking dynamics, to the best of our knowledge. In order to predict the smoking behavior dynamics, most of the models Consider compartment modeling method used in infectious disease modelling.

As per WHO's tobacco-free initiative [[12]] the chance of successful quitting rises in presence of smoking cessation measures. In epidemiological models the impact of behavior influencing factors such as educational initiatives, awareness programmes and other social initiatives play an important role in controlling the disease transmission in population and have been a topic of interest in last decade [[13], [14], [15], [16]].

In 2000 Garsow et al. presented a general epidemiological model in the sense of drug use. They identified the dynamics of increasing drug and tobacco use trends [7]. The proposed model was based on substance addiction and also quantified such social factors, such as peer pressure, relapse, therapy and counseling, and examined the complexities of substance use, especially tobacco use. Brauer and Chavez referred to this work as a project problem in the book [17]. The entire population in this work is divided into three compartments: potential smokers, smokers and smokers who permanently quit.

The power of educational programmes, as well as the willingness of individuals to stop smoking. We first suggest a compartmental model in which the entire population is divided into four classes: potential smokers (those who don't smoke yet but may start smoking), smokers, education or counseling and quitters (those who quit smoking and never smoke again). An individual's actions towards using some hazardous product will change if he knows about the fatality caused by that product. This is also valid in the case of the use of tobacco. Therefore, we believe that people in smoker class are stopping smoking because of information and awareness about the fatality caused by smoking.

Using the concept of ordinary differential equation, we analyse our model and re- port detailed results of numerical simulations to support the analytical findings. First, our model is expanded to the concept of stochastic differential equations. We also com- pare the results of deterministic models with stochastic models. The remaining of this article is structured as follows: Section 2 explains the equilibrium model and existence and demonstrates local stability, Global stability of equilibria. The stochastic model is discussed in Section 3. Section 4 presents the results of simulation for both deterministic and stochastic models. Finally, our results are summarized as a conclusion in Section 5.

2. The Model and Analysis

We assume that total population (N) is constant for all time t. We dividing the population into four different compartments: Potential smoker class (P), smoker class(S), Education/Counseling class (E) and Quitters class (Q). Potential smokers are those people who are inclined to smoking; Smokers are the ones who are effectively smoking and Quitters are ones who have stopped smoking. The total population N = P+S+E+Q. Let μ be inflow rate of recruitment and mortality. It also represents natural death rate in each compartment. Let β be rate of transmission of smoking habit, so that $\beta\frac{PS}{N}$ represent the smoking incidence rate.

A fraction $\gamma(1-\rho_1)S$ of these quitters will return to potential smoker class because of low assurance level and remaining $\gamma\rho_1 S$ will continue to quitter's class. ρ_1 $(0 \leq \rho_1 \leq 1)$ is the

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measure of determination. It may be noted that if quitter's determination is 100%, at that point every one of the individuals who quit will move to Q.

In view of the above considerations the mathematical model is proposed as follows:

Table 1: Description of parameters

Parameter	Description
α	Rate of potential smokers become smokers themselves
β	Rate of transmission of smoking habit
γ	Rate of education awareness in smoker class
δ	Rate of education awareness
ε	Rate of getting intention towards smoking
ψ	Rate of quitters after getting education awareness
μ	Rate of recruitment and mortality
ρ_1	Fraction of smokers going for education awareness
ρ_2	Fraction of smokers going for education awareness
$\gamma\rho_1 S$	Smokers entering for education awareness
$\gamma(1-\rho_1)S$	Smokers entering to quitters class
$\delta(1-\rho_2)P$	Smokers entering to quitters class

$$\frac{dP}{dt} = \mu N - \frac{\beta PS}{N} - \alpha P + \varepsilon Q - \mu P - \delta P,$$

$$\frac{dS}{dt} = \frac{\beta PS}{N} - \mu S + \alpha P - \gamma S,$$

$$\frac{dE}{dt} = \gamma\rho_1 S + \delta\rho_2 P - \mu E - \psi E,$$

$$\frac{dQ}{dt} = \delta(1-\rho_2)P + \gamma(1-\rho_1)S - \varepsilon Q + \psi E - \mu Q.$$

where

$k_1 = \alpha + \mu + \delta$
$k_2 = \mu + \gamma$
$k_3 = \mu + \psi$

The parameters used in the model 2 are described in Table. 1

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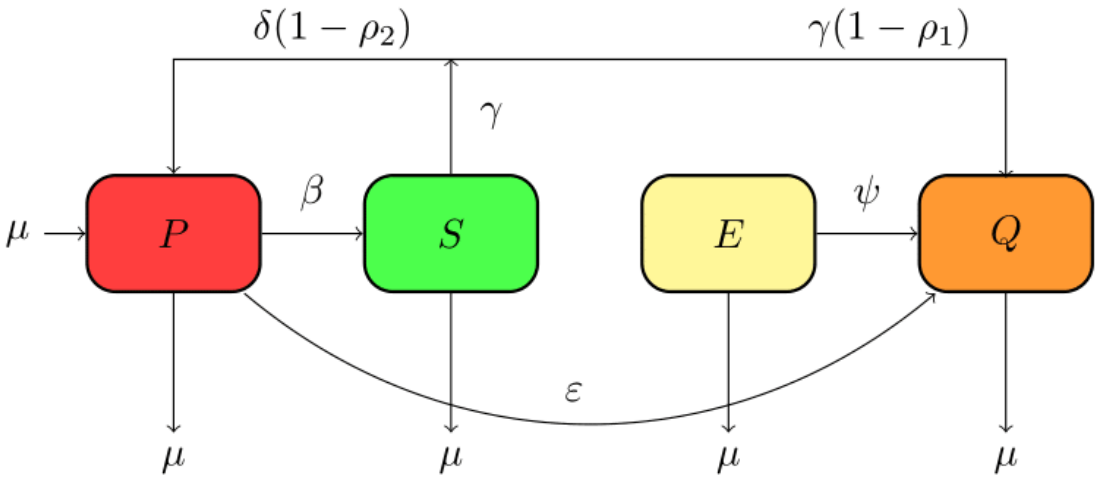

Figure 1: Transfer Diagram of the Model

1.1 Existence of Equilibria

$P(0) \geq 0$, $S(0) \geq 0$, $E(0) \geq 0$ and $Q(0) \geq 0$ as initial conditions. As N is constant, we use following transformation
$p = \frac{P}{N}$, $s = \frac{S}{N}$, $e = \frac{E}{N}$, $q = \frac{Q}{N}$ we get:

$$\begin{aligned}
\frac{dp}{dt} &= \mu - \beta ps - \alpha p + \varepsilon q - \mu p - \delta p, \\
\frac{ds}{dt} &= \beta ps - \mu s + \alpha p - \gamma s, \\
\frac{de}{dt} &= \gamma \rho_1 s + \delta \rho_2 p - \mu e - \psi e, \\
\frac{dq}{dt} &= \delta(1-\rho_2)p + \gamma(1-\rho_1)s - \varepsilon q + \psi e - \mu q
\end{aligned} \tag{1}$$

$P(0) \geq 0$, $S(0) \geq 0$, $E(0) \geq 0$ and $Q(0) \geq 0$ as initial conditions Note that
$p + s + e + q = 1$.
The equilibria for our model are determined by setting right hand side of the model to zero. The system (1) has following two steady states

(1) Smoking-free steady state,

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$$E^0 = (p^0, s^0, e^0, q^0) = (\frac{-\mu\varepsilon + \mu}{\varepsilon + k_1}, 0, 0, \frac{-\mu}{\varepsilon})$$

(2) Smoking-persistent steady state,

$$E^* = (p^*, s^*, e^*, q^*)$$

Where

$$p^* = \frac{k_2 s}{\alpha + \beta s}$$

$$e^* = \frac{\gamma\rho_1 s(\alpha + \beta s) + \delta\rho_2(k_2 s)}{k_3(\alpha + \beta s)} \quad (2)$$

$$q^* = \frac{k_2 s(k_1 + \beta s) - \mu(\alpha + \beta s)}{\varepsilon(\alpha + \beta s)}$$

Where

$$k_1 = \alpha + \mu + \delta$$
$$k_2 = \mu + \gamma$$
$$k_3 = \mu + \psi$$

$R_0 = \frac{\beta p}{\mu+\alpha}$ is the basis reproduction number of system (1)

a non-smoking steady state E^0 occurs at all times, but only $R_0 \geq 1$ has a smoking – persistent steady state E^*.

We use the next generation matrix method to measure the basic reproduction number. We get two non-negative matrices F and V evaluated at E^0 using this approach. such that,

$$F = \begin{pmatrix} \beta p & 0 \\ 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \mu + \gamma & 0 \\ -\gamma\rho_1 & \mu + \psi \end{pmatrix}$$

[18] is a good resource for identifying and evaluating matrices F and V. The spectral radius of FV^{-1}, which corresponds to the basic reproduction number, is now

$$R_0 = \frac{\beta p}{\mu + \gamma}$$

Since p, s, e, and q represent a proportion of the population, they must be positive. We note that:

$$\frac{dp}{dt}|_{P=0} = \mu + \varepsilon q \geq 0\,, \quad \frac{ds}{dt}|_{s=0} = \alpha p \geq 0$$

$$\frac{de}{dt}|_{e=0} = \gamma\rho_1 s + \delta\rho_2 p \geq 0\,, \quad \frac{dq}{dt}|_{q=0} = \delta(1 - \rho_2)p + \gamma(1 - \rho_1)s + \psi e \geq 0.$$

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As a result, the solution of the system (1) that starts in the positive octant will stay there indefinitely, ensuring the positivity of p, s, e, and q. As a result, the positively invariant set for the model system (1) is:

$$\Omega = \{(p, s, e, q) \in \mathbb{R}^3_+ \,|0 \leq p + s + e + q \leq 1, \qquad p \geq 0, s \geq 0, e \geq 0, q \geq 0\}$$

1.2 Stability Analysis

Theorem 2.1. (i) Smoking-free steady E^0 is locally asymptotically stable for $R_0 < 1$.

(ii) Smoking-persistent steady state E^* , whenever it exists is locally asymptotically stable.

Proof : The Variational matrix for the system (1) is given by

$$J = \begin{pmatrix} -(\beta s + k_1) & -\beta p & 0 & \varepsilon \\ \beta s + \alpha & \beta p - k_2 & 0 & 0 \\ \delta\rho_2 & \gamma\rho_1 & k_3 & 0 \\ \delta(1-\rho_2) & \gamma(1-\rho_1) & \psi & -(\varepsilon+\mu) \end{pmatrix}$$

Stability analysis of Smoking-free point

The Variational matrix, M^* corresponding to the Endemic Equilibrium point E^0 is given by

$$M^* = \begin{pmatrix} n_{11} & n_{12} & 0 & n_{14} \\ n_{21} & n_{22} & 0 & 0 \\ n_{31} & n_{32} & n_{33} & 0 \\ n_{41} & n_{42} & n_{43} & n_{44} \end{pmatrix}$$

Where

$n_{11} = -k_1,\ n_{12} = -\beta p, \qquad n_{14} = \varepsilon$

$n_{21} = \alpha, \qquad n_{21} = \beta p - k_2$

$n_{31} = \delta\rho_2,\ n_{32} = \gamma\rho_1, \qquad n_{33} = -k_3$

$n_{41} = \delta(1-\rho_2), \qquad n_{42} = \gamma(1-\rho_1), \quad n_{43} = \psi, \quad n_{44} = -(\varepsilon+\mu)$

The bi-quadratic equation

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

Where

$a_1 = -(n_{11} + n_{22} + n_{33} + n_{44})$

$a_2 = n_{11}n_{22} + n_{22}n_{33} + n_{33}n_{44} + n_{11}n_{33} + n_{11}n_{44} + n_{22}n_{44} - n_{12}n_{21} - n_{14}n_{41}$

$a_3 = n_{14}n_{21}n_{42} + n_{14}n_{41}n_{22} - n_{14}n_{31}n_{44} - n_{11}n_{22}n_{33} - n_{11}n_{22}n_{44} - n_{11}n_{33}n_{44} - n_{22}n_{33}n_{44} + n_{12}n_{21}n_{33} + n_{12}n_{21}n_{44}$

$a_4 = n_{11}n_{22}n_{33}n_{44} - n_{12}n_{21}n_{33}n_{44} + n_{14}n_{22}n_{31}n_{43} - n_{14}n_{21}n_{32}n_{43} - n_{14}n_{22}n_{33}n_{41} + n_{14}n_{21}n_{33}n_{42}$

By using Routh-Hurwitz criteria, E^0 will be locally asymptotically stable if the following conditions are satisfied: $a_1 > 0, a_3 > 0,\ \ a_1a_2a_3 - a_3^2 - a_1^2a_4 > 0, a_3 > 0$

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$E^0$is locally asymptotically stable whenever $R_0 < 1$

Stability analysis of EE point

The Variational matrix, M^* corresponding to the Endemic Equilibrium point E^* is given by

$$M^* = \begin{pmatrix} n_{11} & n_{12} & 0 & n_{14} \\ n_{21} & n_{22} & 0 & 0 \\ n_{31} & n_{32} & n_{33} & 0 \\ n_{41} & n_{42} & n_{43} & n_{44} \end{pmatrix}$$

where

$n_{11} = -(\beta s + k_1), \quad n_{12} = -\beta p, \quad n_{14} = \varepsilon$

$n_{21} = \beta s + \alpha, \qquad n_{21} = \beta p - k_2$

$n_{31} = \delta\rho_2, \quad n_{32} = \gamma\rho_1, \quad n_{33} = -k_3$

$n_{41} = \delta(1-\rho_2), \quad n_{42} = \gamma(1-\rho_1), \quad n_{43} = \psi, \quad n_{44} = -(\varepsilon + \mu)$

The bi-quadratic equation

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

Where

$a_1 = -(n_{11} + n_{22} + n_{33} + n_{44})$

$a_2 = n_{11}n_{22} + n_{22}n_{33} + n_{33}n_{44} + n_{11}n_{33} + n_{11}n_{44} + n_{22}n_{44} - n_{12}n_{21} - n_{14}n_{41}$

$a_3 = n_{14}n_{21}n_{42} + n_{14}n_{41}n_{22} - n_{14}n_{31}n_{44} - n_{11}n_{22}n_{33} - n_{11}n_{22}n_{44} - n_{11}n_{33}n_{44} - n_{22}n_{33}n_{44} + n_{12}n_{21}n_{33} + n_{12}n_{21}n_{44}$

$a_4 = n_{11}n_{22}n_{33}n_{44} - n_{12}n_{21}n_{33}n_{44} + n_{14}n_{22}n_{31}n_{43} - n_{14}n_{21}n_{32}n_{43} - n_{14}n_{22}n_{33}n_{41} + n_{14}n_{21}n_{33}n_{42}$

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 2571-2587
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By using Routh-Hurwitz criteria, E^* will be locally asymptotically stable if the following conditions are satisfied: $b_1 > 0, b_3 > 0, \; b_1 b_2 b_3 - b_3^2 - b_1^2 b_4 > 0,$ $b_3 > 0$
E^* is locally asymptotically stable whenever $R_0 > 1$.

Theorem 2.2. (i) Smoking-free steady state E^0 is globally asymptotically stable for $R_0 \leq 1$ in feasible region Ω.

(ii) Smoking-persistent steady state E^*, whenever it exists, is globally asymptotically stable in feasible region Ω

Proof. (i) consider Lyapunov function as:

$$V(p, s, e, q) = \frac{1}{2}\left((p - p^0) + s + e + (q - q^0)\right)^2 + s$$

The derivative of V along solutions of system (1) is given by

$$\begin{aligned}\dot{V}(p, s, e, q) &= \left((p - p^0) + s + e + (q - q^0)\right)(\dot{p} + \dot{s} + \dot{e} + \dot{q}) + s \\ &= \left(\beta p - (\mu + \gamma)\right)s \\ &\leq -(\mu + \gamma)\left(1 - \frac{\beta p}{\mu + \gamma}\right)s \\ &\leq -(\mu + \gamma)(1 - R_0)s\end{aligned}$$

If $R_0 \leq 1, \dot{V} \leq 0$ We infer that E^0 is globally asymptotically stable for $R_0 \leq 1$ using the Lyapunov LaSalle theorem [19].

In part (ii), we consider the Lyapunov function to determine the global stability of E^*.

$$V(p, s, e, q) = \frac{1}{2}\left((p - p^*) + (s - s^*) + (e - e^*) + (q - q^*)\right)^2 + \frac{1}{2}(p - p^*)^2$$

$$+ \frac{k_1 + \varepsilon}{\beta}(s - s^* - s^* \log\frac{s}{s^*})$$

This is a Ω positive definite function, and its derivative along the solutions of system (1) is:

$$\dot{V}(p, s, e, q) = \left((p - p^*) + (s - s^*) + (e - e^*) + (q - q^*)\right)(\dot{p} + \dot{s} + \dot{e} + \dot{q})$$

$$\begin{aligned}&+(p - p^*)\dot{p} + \frac{k_1 + \varepsilon}{\beta}(s - s^*)\frac{s^*}{s} \\ &= +(p - p^*)(\mu - \beta p s + \varepsilon q - k_1 p) + \frac{k_1 + \varepsilon}{\beta}(s - s^*)(\beta p - k_2)\end{aligned}$$

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$$= (-\beta s - k_1)(p - p^*)^2 + (p - p^*)(s - s^*)(\beta p^* - k_2)$$

$$= -(\beta s + k_1)(p - p^*)^2$$

Clearly, $\dot{V} \leq 0$ if $R_0 > 1$. We infer that E^* is globally asymptotically stable whenever $R_0 > 1$ using the Lyapunov-LaSalle theorem.

3. Stochastic model

Here we expand our deterministic model to stochastic systems, as stochastic models are more capable of capturing random variations of the problem's biological dynamics. The derivation of an SDE model is based on the method developed by Yuan et al. [20]. Let $Y(t) = (Y_1(t), Y_2(t), Y_3(t), Y_4(t))^T$ be a continuous random variable for $(p(t), s(t), e(t), q(t))^T$ and T denotes the transpose of a matrix.

Let $\Delta X = Y(t + \Delta t) - Y(t) = (\Delta Y_1, \Delta Y_2, \Delta Y_3, \Delta Y_4)^T$ denotes the random vector for the change in random variables during time interval Δt. Here, we'll write transition maps that define all possible changes in the SDE model between states. Based on our ODE model system (1), here we see that with in a small-time interval Δt, there are 9 possible changes between states. State changes and their probabilities are discussed in Table 2. In the case, the state change ΔY is denoted by ΔY = (-1,1,0,0); The probability of this change is determined by

$$\text{Prob}\,(\Delta Y_1, \Delta Y_2, \Delta Y_3, \Delta Y_4) = (-1,1,0,0)|(Y_1, Y_2, Y_3, Y_4)$$

$$= P_2 = \beta Y_1 Y_2 + o(\Delta t)$$

by neglecting terms higher than $o(\Delta t)$, the following expectation change $E(\Delta Y)$ and its covariance matrix $V(\Delta Y)$ associated with ΔY, can be identified.
The expectation of ΔY is

$$E(\Delta Y) = \sum_{i=1}^{14} P_i (\Delta y)_i \Delta t = \begin{pmatrix} \mu - \beta Y_1 Y_2 + \varepsilon Y_4 - \alpha Y_1 - \mu Y_1 - \delta Y_1 \\ \beta Y_1 Y_2 + \alpha Y_1 - \mu Y_2 - \gamma Y_2 \\ \gamma \rho_1 Y_2 + \delta \rho_2 Y_1 - \mu Y_3 - \psi Y_3 \\ \delta(1 - \rho_2) Y_1 + \gamma(1 - \rho_1) Y_2 - \varepsilon Y_4 + \psi Y_3 - \mu Y_4 \end{pmatrix} \Delta t$$

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 2571-2587
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$$= f(Y_1, Y_2, Y_3, Y_4)\Delta t.$$

Table: 2 Possible changes of states and their probabilities

Possible stage change	Probability of state changes
$(\Delta y)_1 = (1,0,0,0)^T$	$P_1 = \mu\Delta t + o(\Delta t)$
$(\Delta y)_2 = (-1,1,0,0)^T$	$P_2 = \beta Y_1 Y_2 \Delta t + o(\Delta t)$
$(\Delta y)_3 = (1,0,0,-1)^T$	$P_3 = \varepsilon Y_4 \Delta t + o(\Delta t)$
$(\Delta y)_4 = (-1,1,0,0)^T$	$P_4 = \alpha Y_1 \Delta t + o(\Delta t)$
$(\Delta y)_5 = (-1,0,0,0)^T$	$P_5 = \mu Y_1 \Delta t + o(\Delta t)$
$(\Delta y)_6 = (-1,0,0,1)^T$	$P_6 = \delta Y_1 \Delta t + o(\Delta t)$
$(\Delta y)_7 = (0,-1,0,0)^T$	$P_7 = \mu Y_2 \Delta t + o(\Delta t)$
$(\Delta y)_8 = (0,-1,0,1)^T$	$P_8 = \gamma Y_2 \Delta t + o(\Delta t)$
$(\Delta y)_9 = (0,0,1,-1)^T$	$P_9 = \gamma \rho_1 Y_2 \Delta t + o(\Delta t)$
$(\Delta y)_{10} = (0,0,1,-1)^T$	$P_{10} = \delta \rho_2 Y_1 \Delta t + o(\Delta t)$
$(\Delta y)_{11} = (0,0,-1,0)^T$	$P_{11} = \mu Y_3 \Delta t + o(\Delta t)$
$(\Delta y)_{12} = (0,0,-1,1)^T$	$P_{12} = \psi Y_3 \Delta t + o(\Delta t)$
$(\Delta y)_{13} = (0,0,0,-1)^T$	$P_{13} = \mu Y_4 \Delta t + o(\Delta t)$
$(\Delta y)_{14} = (0,0,0,0)^T$	$P_{14} = (1 - \sum_{i=1}^{13} P_i) + o(\Delta t)$

It should be noted that the expectation vector as well as the function f have the same form as in the ODE method (1).

Since the covariance matrix $V(\Delta Y) = E\big((\Delta Y)(\Delta Y)^T\big) - E(\Delta Y)((\Delta Y)^T)$ and $E\big((\Delta Y)(\Delta)^T\big) = f(Y)(f(Y)^T)\Delta t$, it can be approximated with diffusion matrix Ω times Δt by neglecting the term of $(\Delta t)^2$ such that $\mathrm{V}(\Delta\mathrm{Y}) \approx \mathrm{E}\Big((\Delta\mathrm{Y})(\Delta\mathrm{Y})^{\mathrm{T}}\Big)$. That is,

$$\mathrm{E}\left((\Delta\mathrm{Y})(\Delta\mathrm{Y})^{\mathrm{T}}\right) = \sum_{\mathrm{i}=1}^{13} \mathrm{P_i}(\Delta\mathrm{Y})_{\mathrm{i}}(\Delta\mathrm{Y})_{\mathrm{i}}^{\mathrm{T}}\Delta\mathrm{t} = \begin{pmatrix} \mathrm{V}_{11} & \mathrm{V}_{12} & 0 & \mathrm{V}_{14} \\ \mathrm{V}_{21} & \mathrm{V}_{22} & 0 & \mathrm{V}_{24} \\ 0 & 0 & \mathrm{V}_{33} & \mathrm{V}_{34} \\ \mathrm{V}_{41} & \mathrm{V}_{42} & \mathrm{V}_{43} & \mathrm{V}_{44} \end{pmatrix}.\Delta\mathrm{t} = \Omega$$

where each component of the 4×4 diffusion matrix is symmetric, positive-definite, and can be obtained by

$$\mathrm{V}_{11} = \mathrm{P}_1 + \mathrm{P}_2 + \mathrm{P}_3 + \mathrm{P}_4 + \mathrm{P}_5 + \mathrm{P}_6 = \mu + \beta\mathrm{Y}_1\mathrm{Y}_2 + \varepsilon\mathrm{Y}_4 + \alpha\mathrm{Y}_1 + \mu\mathrm{Y}_1 + \delta\mathrm{Y}_1$$

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 2571-2587
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$$V_{22} = P_2 + P_4 + P_7 + P_8 = \beta Y_1 Y_2 + \alpha Y_1 + \mu Y_2 + \gamma Y_2$$
$$V_{33} = P_9 + P_{10} + P_{11} + P_{12} = \gamma\rho_1 Y_2 + \delta\rho_2 Y_1 + \mu Y_3 + \psi Y_3$$
$$V_{44} = P_3 + P_6 + P_8 + P_9 + P_{10} = \delta(1-\rho_2)Y_1 + \gamma(1-\rho_1)Y_2 + \varepsilon Y_4 + \psi Y_3 + \mu Y_4$$
$$V_{12} = V_{21} = -P_2 = -\beta Y_1 Y_2$$
$$V_{14} = V_{41} = -P_3 = -\varepsilon Y_4$$
$$V_{12} = V_{21} = -P_4 = -\alpha Y_1$$
$$V_{14} = V_{41} = -P_6 = -\delta Y_1$$
$$V_{24} = V_{42} = -P_8 = \gamma Y_2$$
$$V_{34} = V_{43} = -P_9 = -\gamma\rho_1 Y_2$$
$$V_{34} = V_{43} = -P_{10} = -\delta\rho_2 Y_1$$
$$V_{34} = V_{43} = -P_{12} = -\psi Y_3$$

A matrix D square root of the symmetric, positive-definite diffusion matrix Ω such that $K = \Omega^{1/2}$. Use an equivalent matrix K, such that $\Omega = KK^T$, where K has the dimension of a 4×8 matrix.

$$K = \begin{pmatrix} M_{11} & M_{12} & M_{13} & 0 & 0 & 0 & 0 & 0 \\ 0 & M_{22} & 0 & M_{24} & M_{25} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & M_{36} & M_{37} & 0 \\ 0 & 0 & M_{43} & 0 & M_{45} & M_{46} & 0 & M_{48} \end{pmatrix}$$

$$M_{11} = \sqrt{P_1 + P_5}, \quad M_{12} = -\sqrt{P_2 + P_4}, \quad M_{13} = \sqrt{P_3 + P_6}$$
$$M_{22} = \sqrt{P_2 + P_4}, \quad M_{24} = -\sqrt{P_7}, \quad M_{25} = -\sqrt{P_8}$$
$$M_{36} = \sqrt{P_9 + P_{10} + P_{12}}, \quad M_{37} = -\sqrt{P_{11}}$$
$$M_{43} = \sqrt{P_3 + P_6}, \quad M_{45} = \sqrt{P_8}, \quad M_{46} = \sqrt{P_9 + P_{10} + P_{12}}, \quad M_{48} = -\sqrt{P_{12}}$$

Then, the Ito stochastic differential model has the following form:

$$d\big(Y(t)\big) = f(Y_1, Y_2, Y_3, Y_4)dt + K.\,dw(t)$$

with initial condition $Y(0) = \big(Y_1(0), Y_2(0), Y_3(0), Y_4(0)\big)^T$ and a Wiener process, $W(t) = \big(W_1(t), W_2(t), W_3(t), W_4(t), W_5(t), W_6(t), W_7(t), W_8(t)\big)^T$ We get the stochastic differential equation model as follows:

$$dp = [\mu - \beta ps - \alpha p + \varepsilon q - \mu p - \delta p]dt + \sqrt{\mu + \mu p}\, dW_1 - \sqrt{\beta ps + \alpha p}\, dW_2$$

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$$+\sqrt{\varepsilon q + \delta p}\, dW_3$$

$$ds = [\beta ps - \mu s + \alpha p - \gamma s]dt + \sqrt{\beta ps + \alpha p}\, dW_2 - \sqrt{\mu s}\, dW_4 - \sqrt{\gamma s}\, dW_5$$

$$de = [\gamma \rho_1 s + \delta \rho_2 p - \mu e - \psi e]dt + \sqrt{\gamma \rho_1 s + \delta \rho_2 p + \psi e}\, dW_6 - \sqrt{\mu e}\, dW_7$$

$$dq = [\delta(1-\rho_2)p + \gamma(1-\rho_1)s - \varepsilon q + \psi e - \mu q]dt + \sqrt{\varepsilon q + \delta p}\, dW_3 + \sqrt{\gamma s}\, dW_5$$

$$+\sqrt{\gamma \rho_1 s + \delta \rho_2 p + \psi e}\, dW_6 - \sqrt{\mu q}\, dW_8 \qquad \text{----------} \qquad (3)$$

4. Numerical simulation

Here, we simulate both deterministic and stochastic models for the following set of parameters:

$\alpha = 0.4,\ \beta = 0.0093,\ \mu = 0.167,\ \gamma = 0.5,\ \delta = 0.2,\ \psi = 0.2,\ \varepsilon = 0.6,$ $\rho_1 = 0.2,\ \rho_2 = 0.03$

The system (1) is simulated for various set of parameters satisfying the condition of local and globally asymptotic stability of equilibrium E^*. The simulation results for both deterministic and stochastic models are shown in Fig 2. The stochastic model (SDE model) is simulated by Euler–Maruyama method, and mean of the 100 runs is plotted in Fig 2. Here, the results of stochastic model seem better than the deterministic model as the curve corresponding to Smoking lies below the one that corresponds to the deterministic model.

$\alpha = 0.2,\ \beta = 0.009,\ \mu = 0.167,\ \gamma = 0.4,\ \delta = 0.5,\ \rho_1 = 0.7,\ \rho_2 = 0.7,$ $\psi = 0.5,\ \varepsilon = 0.2, p = 0.9,\ s = 0.9,\ q = 0.3,\ e = 0.5$

The system (1) is simulated for different set of parameters satisfying the condition of local and globally asymptotic stability of equilibriumE^* (see Fig.3).

Figs 4 - 7 demonstrate the impact of various parameters on the equilibrium level of Smokers and quitters.

Annals of R.S.C.B., ISSN:1583-6258, Vol. 25, Issue 6, 2021, Pages. 2571-2587
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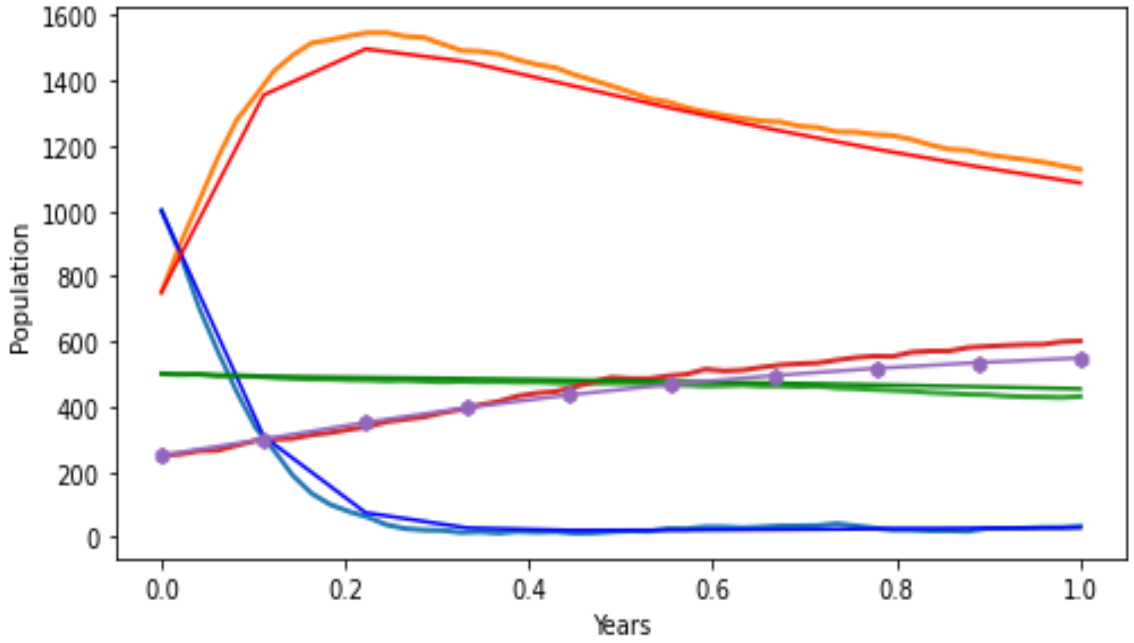

Figure 2: Variation of all compartments of the model showing the effect of Stochastic and deterministic

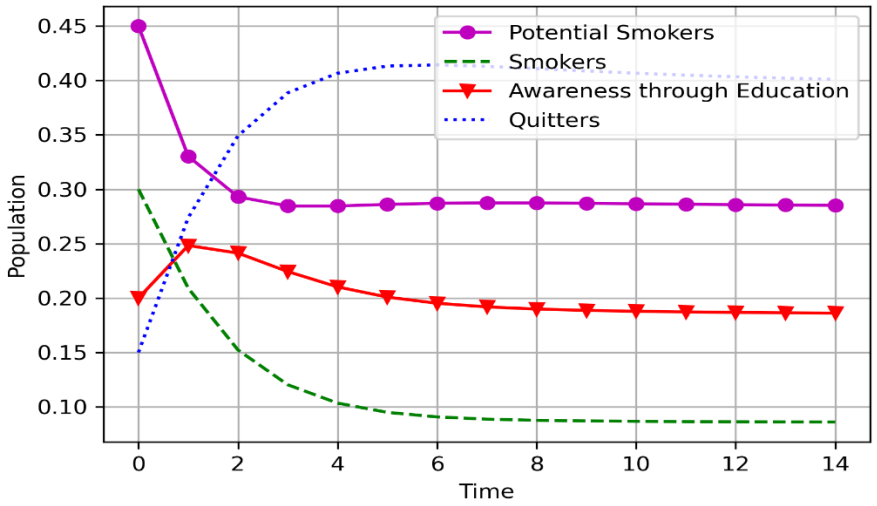

Figure 3: Variation of all compartments of the model showing the stability

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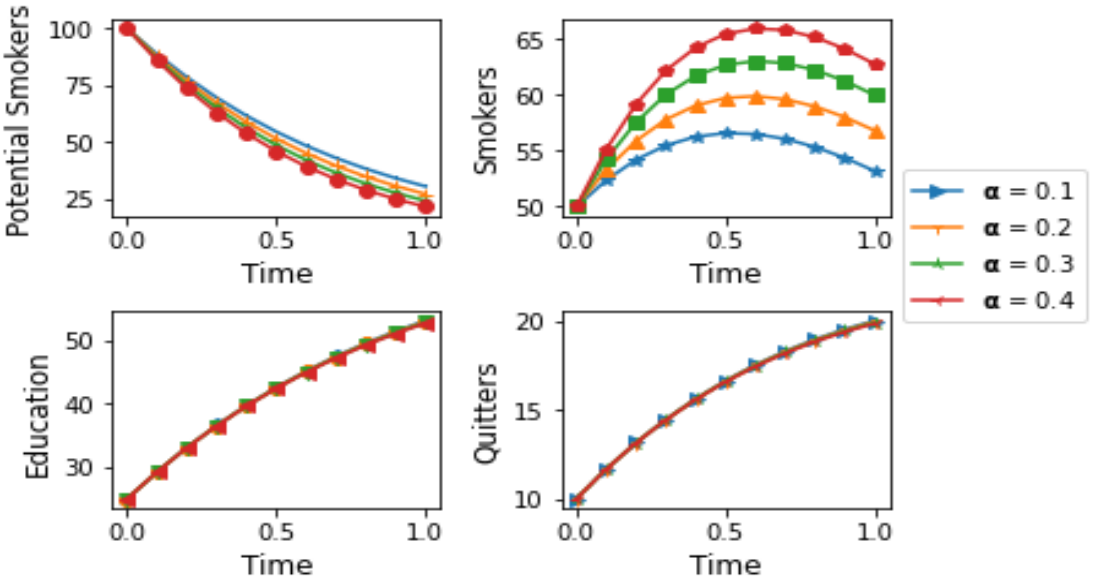

Figure 4: Effect of α on the variation of all compartments of the model

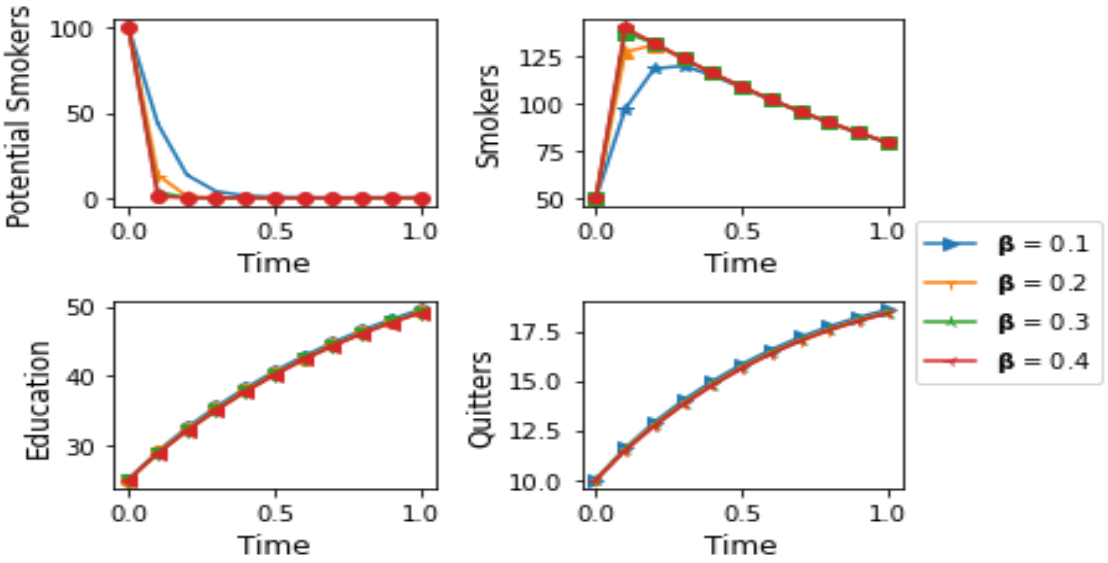

Figure 5: Effect of β on the variation of all compartments of the model

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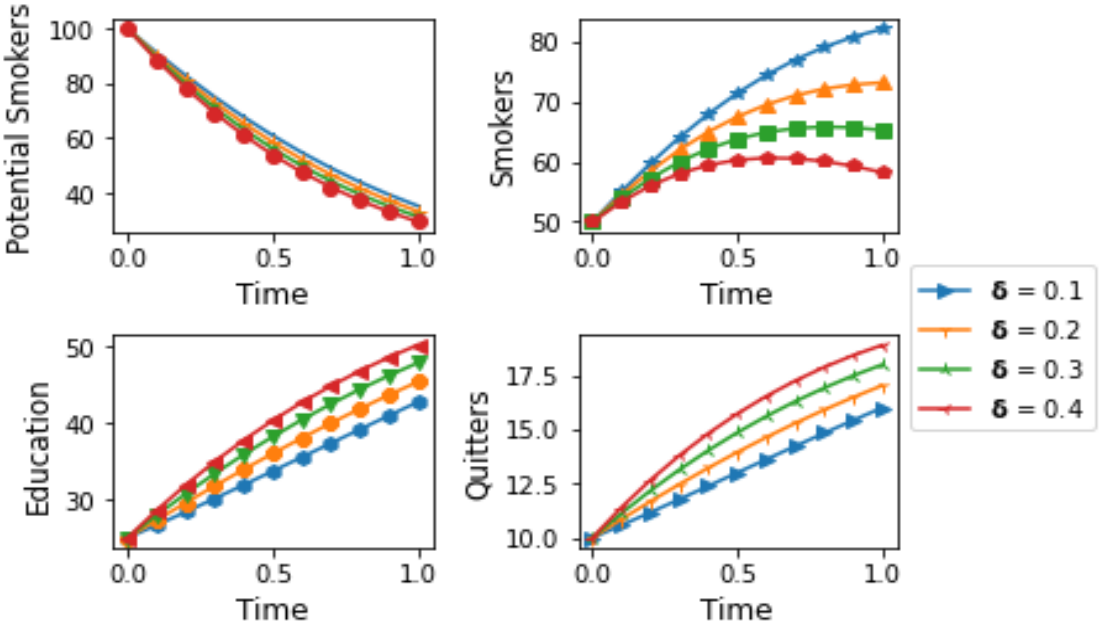

Figure 6: Effect of δ on the variation of all compartments of the model

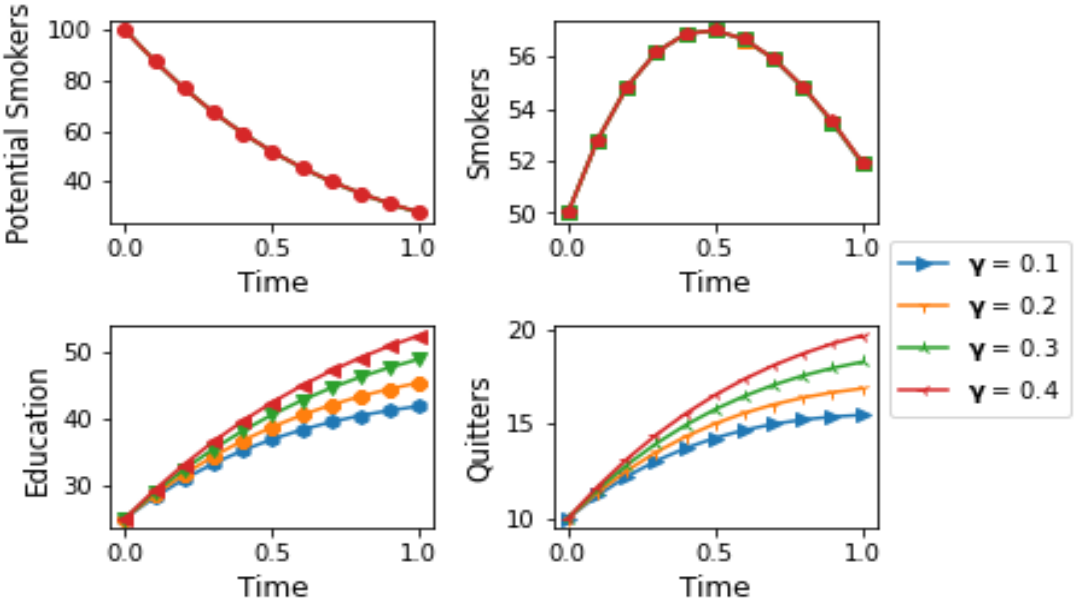

Figure 7: Effect of γ on the variation of all compartments of the model

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5. Result Discussion and Conclusion

In this paper, a deterministic mathematical model of smoking behavior in a population was proposed and analyzed. We calculate the equilibrium of the proposed model and analyses in detail the local stability and global stability of endemic equilibria. In addition, we expanded the proposed deterministic model to a stochastic model and compared the effects of its simulation with deterministic model. The stochastic model results showed that (2) eradicates smoking in comparison to the deterministic model.

We assumed that smokers would quit if they are willing to do so. Relapse is less likely when you have a higher level of resolve. It also considers how potential smoking will be influenced by education. If people are taught about the risks of smoking-related illnesses, they will avoid smoking in the future and thereby fall into the removed class. We remember that the basic reproduction number $R_0 = \beta p/\mu + \gamma$ and we need to bring down R_0 below one in order to eradicate smoking. This can be done by growing educational opportunities and, as a result, education programs.

As the value of α (Rate of potential smokers become smokers themselves) increases in all compartments at the time stable point, the value differs (see Fig. 4). Fig. 5 depicts if β value (Rate of transmission of smoking habit) increase or decrease there is no major different in all compartment. Fig. 6,7 depicts the parameter δ, γ (Rate of education awareness,) value increasing time the education is increased and the quitters is decreased.

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