

## **Modeling and Simulation Study of Population Subjected to the Smoking Habit**

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**Abstract:** *The habit of smoking affects the health of both the active and passive smokers and their descendents. The health problems are potential enough leading to loss of lives, social and economical problems. Smoking is a bad habit and has become a big problem of the entire world. Despite of the health risks and economic losses, the spread of smoking habit has been increasing and socially accepted worldwide. These facts encourage mathematicians to model and simulate the dynamics of populations subject to the smoking habit. In this work, we presented a non-linear mathematical model and analyzed the effect of educated smokers and patient smokers on the spread of smoking and increase of mortality rate. The model can be used to predict the smoke attributed mortality (SAM) for long periods of times. The stability analysis of the present model is carried out and presented the results. Numerical simulation of the model is conducted results are presented.*

**Keywords:** *Smoking habit, Mathematical modeling, Mortality, Stability analysis, Numerical simulation, Government.*

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### **I. Introduction**

Smoking is a process of burning tobacco and experiencing its smoke through pipes, cigars and other means by in healing into lungs through mouth and then release. Tobacco consists of a variety of harmful particles and when its smoke is in healed then these particles would enter in to human body and do harm [2]. The major cause of smoking tobacco leaves a negative impact on the health of people around the world possibly leading to death [3]. It is estimated that up to half of the smokers die worldwide due to smoking habit alone. The mortality rate due to tobacco consumption has been increasing and is now approximately doubling for every decade. Most of these deaths occur in developed countries. Smoking kills about six million people each year. Of these more than five millions are smokers and over a half million are non-smokers.

The non – smokers referred here are being exposed to a second hand smoke. The smokers are referred as ‘active smokers’ and the non – smokers but exposed to second hand smoking are referred to as ‘passive smokers’. The report of the world health organization on the global tobacco epidemic estimates and says that the mortality number due to smoking is increasing gradually every year [7]. The world health organization forecasts and it is true that if the smokers population continuous to grow with the current rates then from the year 2030 the number of dead persons due to cigarette smoking will be doubled each year [8].

People start smoking of tobacco for fun during initial times. If continued this habit leads to an addiction. The addiction of smoking influences both personal and social life. Tobacco consists of a substance called nicotine that has addictive nature. Once addicted for smoking it is difficult for giving up. The main reasons which prevent giving up smoking include nicotine in the tobacco, psychological conditions and genetics history of the patient, smoking rate, sex, age, education, social and economical states, cigarette price, youth access laws, nicotine replacement therapy, parental smoking, and peer influences [9].

The influence of smoking habit on people has been an important and interesting area of study as this habit potentially affects lives. It has been well proved medically that the smoking addiction causes more than thirty diseases [1]. In addition, the cigarette smoking increases poly unsaturated fatty acid in smoker’s body that causes cancer. If the amount of poly unsaturated fatty acid increases then the cigarette smoker will die within a short span of time. The death rate due to cancer is twice bigger and the risk of lung cancer is forty times more in case of smokers in comparison with non smokers. It is obvious that the risk of death also increases with the rate of cigarettes smoked. Consumption of high amount of tobacco is a major cause for many of the world’s top most killer diseases. These facts motivated us to model, simulate and study the dynamics of smoking population of human beings.

Modeling is a science that requires sufficient knowledge and creativity so as to link a whole group of physical variables and parameters [2]. Modeling helps to predict future values of physical quantities up on providing the observed initial values. The object of present model is to understand the spread of smoking in the human population and to predict is impact on the death rate. The model can also be used in reducing the number and the mortality rate of smokers in the community [5].

It is important to suggest ways to reduce tobacco caused deaths. The efforts to be implemented in tobacco control programs are of great interest to a researcher working in this area. Goals are to be effectively implemented in tobacco control programs and reduce the practice of cigarette smoking. There are many reasons

to begin tobacco consumption or cigarette smoking. Most of the reasons are connected with psychology of human. The other partial reasons include the effect of friends and peers, nicotine conception, and utter failures and great successes in life. Controlling of potential-smokers, new-smokers, educated-smoker and patient-smokers will help in reducing the transmission rate. These controls have enough influence and it shows its affect on the existence of equilibrium point and its stability [2].

In addition to biological effects, the addiction of smoking also affects humans in many ways socially and economically. Smoking reduces the quality of the works done. The studies of students will get disturbed. Peace and welfare of the society will get affected negatively [9].

The premature death of a human due to smoking will take down social and economical conditions of his family. Medical and fitness costs will increase and that will push down financial development of the family. In this way the smoking habit will bring more problems into both personal and public life of a human. The smoking also causes spending of more money on health care and disease control practices [3].

In literature the mathematical modeling and simulation studies have been successfully applied to various epidemics and population growths. Also interesting and important conclusions have been drawn. The epidemics include Ebola, harvesting fisheries, breast cancer, malaria, tuberculosis, flu and HIV/AIDS [9 – 18].

In the present study we have developed a mathematical model and analyzed the spread of cigarette smoking and the increase of mortality rate. Further, we have evaluated the conditions at which non smokers will start smoking and the smokers will stop smoking using the basic reproduction number.

## **II. Compartmentalization Of Human Population Based On Smoking Habit**

We have categorized the human population into four compartments based on the intensity of smoking habit. The compartmental description and human flow directions of the model are illustrated in Figure 1. The present model is formulated using five first order non-linear ordinary differential equations. The total human population at any time  $t$  is denoted by  $N(t)$  and is divided into four compartments in the present model. These four compartments are represented by variables. The notations and interpretations of the compartmental variables are as follows:

**Compartment of potential smokers:** The number of potential smokers at any time  $t$  is represented by  $P(t)$ . The people of this compartment are at this moment non smokers but they have chances to become smokers in future. People will enter into this compartment with a natural birth rate  $\lambda$ . Some of the new smokers of the compartment  $Q(t)$  will quit smoking and enter into  $P(t)$  with a rate of  $\beta_1$ . Few of the educated smokers of the compartment  $E(t)$  will quit smoking and enter into  $P(t)$  with a rate of  $\beta_2$ . Some of the patient smokers of the compartment  $R(t)$  will quit smoking and enter into  $P(t)$  with a rate of  $\beta_3$ . People of this compartment  $P(t)$  will die naturally with a rate of  $\mu$  and quit. Also people or potential smokers of this compartment  $P(t)$  start smoking and become new smokers and then will enter into the compartment  $Q(t)$  with a rate of  $\alpha_1$ .

**Compartment of new smokers:** The number of new smokers at any time  $t$  is represented by  $Q(t)$ . The people of this compartment were non smokers earlier but at this moment these have become smokers. People or potential smokers of the compartment  $P(t)$  start smoking and become new smokers and then will enter into this compartment  $Q(t)$  with a rate of  $\alpha_1$ . People of this compartment  $Q(t)$  will die naturally with a rate of  $\mu$  and then they quit. Also people or new smokers of this compartment  $Q(t)$  stop smoking and become potential smokers and then will go back into the compartment  $P(t)$  with a rate of  $\beta_1$ . Further people or new smokers of this compartment  $Q(t)$  continue smoking, become educated smokers after knowing the pros and cons of the habit, and then they will enter into the compartment  $E(t)$  at a rate of  $\alpha_2$ .

**Compartment of educated smokers:** The number of educated smokers at any time  $t$  is represented by  $E(t)$ . The people of this compartment have been smokers since some time and not educated. But by this moment of time these people have been educated and they are completely aware of the pros and cons of smoking. Further people or new smokers of the compartment  $Q(t)$  continue smoking, become educated smokers after knowing the pros and cons of the habit, and then they will enter into this compartment  $E(t)$  at a rate of  $\alpha_2$ . People or the educated smokers of this compartment  $Q(t)$  will die naturally with a rate of  $\mu$  and then they quit. Also people of this compartment  $E(t)$  stop smoking and become potential smokers and then will go back into the compartment  $P(t)$  with a rate of  $\beta_2$ . Further, people of this compartment  $E(t)$  continue smoking, become patient smokers after getting some disease due to smoking, and then they will enter into the patient smoker's compartment  $R(t)$  at a rate of  $\alpha_3$ .

**Compartment of patient smokers:** The number of patient smokers at any time  $t$  is represented by  $R(t)$ . The people of this compartment have been patient smokers since some time. Further, people or educated smokers of

the compartment  $E(t)$  continue smoking, become patient smokers after getting some disease due to smoking, and then they will enter into the patient smoker's compartment  $R(t)$  at a rate of  $\alpha_3$ . People or the patient smokers of the compartment  $R(t)$  will die naturally at a rate of  $\mu$ . Also people of this patient compartment  $R(t)$  will stop smoking and become potential smokers and then will go back into the compartment  $P(t)$  with a rate of  $\beta_3$ .

The model diagram consisting of the compartments and flow directions of the people is given in Figure 1 below. Natural birth and death rates are also mentioned. All the newly born people are assumed to join only potential smoker's compartment  $P(t)$  while the people of all the compartments will die naturally with the same rate of  $\mu$ . All these and the other facts have been described pictorially in the flow chart.

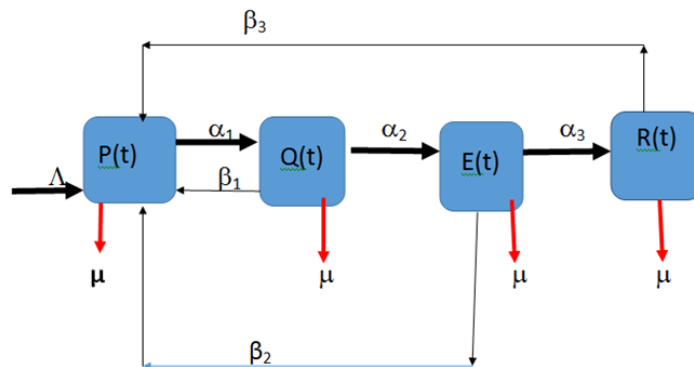


Figure 1: Flow chart showing compartmental division and flow directions of the people with respect to smoking habit

The population dynamics of the compartments shown in the flow chart, Figure 1, can be described by a system of five first order nonlinear ordinary differential equations as

$$dP/dt = \Lambda N - \alpha_1 PQ + \beta_1 Q + \beta_2 E + \beta_3 R - \mu P \tag{1(a)}$$

$$dQ/dt = \alpha_1 PQ - \alpha_2 QE - (\beta_1 + \mu) Q \tag{1(b)}$$

$$dE/dt = \alpha_2 QE - \alpha_3 ER - (\beta_2 + \mu) E \tag{1(c)}$$

$$dR/dt = \alpha_3 ER - (\beta_3 + \mu) R \tag{1(d)}$$

The variables and parameters appearing in the system of model equations (1) are described and interpreted in Table 1 and Table 2 respectively.

Variables	Description
$P(t)$	Human population size in the 'compartment of non smokers' at any time $t$
$Q(t)$	Human population size in the 'compartment of new smokers' at any time $t$
$E(t)$	Human population size in the 'compartment of educated smokers' at any time $t$
$R(t)$	Human population size in the 'compartment of patient smokers' at any time $t$ . The patients considered here are caught by smoking based diseases.
$N(t)$	Total human population size at any time $t$ . Always $N(t) = P(t) + Q(t) + E(t) + R(t)$ true.

Table 1: Notations and descriptions of the variables used in the model

Parameter	Description
$\Lambda$	The natural birth rate of human population
$\alpha_1$	Transmission rate of humans from non smokers or potential smokers compartment to new smokers compartment
$\alpha_2$	Transmission rate of humans from new smokers compartment to educated smokers compartment
$\alpha_3$	Transmission rate of humans from educated smokers compartment to patient smokers compartment
$\beta_1$	Progression rate of humans from new smoker compartment to non smoker compartment. This is also the rate of giving up smoking in the compartment of new smokers.
$\beta_2$	Progression rate of humans from educated-smoker compartment to non-smokers compartment. This is also the rate of giving up smoking in the compartment of educated smokers.
$\beta_3$	Progression rate of humans from patient-smokers compartment to non-smokers compartment. This is also the rate of giving up smoking in the patient-smokers compartment.
$\mu$	Natural death rate of humans. It occurs in all the compartments.

Table 2: Notations and descriptions of the parameters used in the model

The total population is distributed into four compartments and hence the sum of the populations of these four compartments is always the same as the total populations. So that  $N(t) = P(t) + Q(t) + E(t) + R(t)$

$R(t)$ . All the variables of the foregoing relation and those of all the compartments represent human population sizes. Due to this reason all the compartment variables are non-negative.

**Invariant region of the model variables:** Here we will identify the invariant region of the model variables. Now on differentiating the total population equation  $N(t) = P(t) + Q(t) + E(t) + R(t)$  with respect to time coordinate  $t$  we get  $N'(t) = P'(t) + Q'(t) + E'(t) + R'(t)$ . The foregoing relation of the differential terms, up on using (1), takes the form as  $N'(t) = \lambda N(t) - \mu N(t)$  or it can be rewritten as  $[dN(t)/dt] = (\lambda - \mu)N_0$ . By integrating this equation we obtain its general solution as  $N(t) = N_0 e^{(\lambda - \mu)t}$ . Here  $N(0) = e^c \cdot e^{(\lambda - \mu)0} = N_0$  is the size of initial human population. It can be observed that as  $t \rightarrow \infty$  the human population size  $N(t)$  approaches the value  $N_0 e^{(\lambda - \mu)t}$ . Therefore, the feasible solution of human population for the model is restricted to the region  $\Omega = \{(P, Q, E, R) \in \mathbb{R}_+^4, N(t) = N_0 e^{(\lambda - \mu)t}\}$ .

### III. Stability Analysis of the Model

In this section, (i) we identify two equilibrium points viz., smoking free equilibrium point and endemic equilibrium point (ii) we analyze local stability of smoking free equilibrium point, (iii) we analyze global stability of smoking free equilibrium point and (iii) we construct the formula for reproduction number  $R_0$ .

#### 3.1 Existence of equilibrium Points

The equilibrium points are obtained by setting the right hand sides of system equations (1) to zero. That means  $(dP/dt) = (dQ/dt) = (dE/dt) = (dR/dt) = 0$ . That is, the foregoing condition is a requirement for existence of equilibrium points.

#### 3.2 The smoking free Equilibrium Point

Smoking equilibrium point denoted by  $E_0$  is a steady state solution when there are no cigarette smokers. The human populations of new smoker, educated smoker and patient smoker compartments can be considered as cigarette smokers compartments of human populations. To stabilize this model we consider that  $Q(t) = E(t) = R(t) = 0$ . Since in the present model, if new smokers, educated smokers and patient smokers stop smoking then the potential smokers population will become stable. Then in that case  $P(t)$  alone contains the total human population size.

Thus we get the equilibrium point at steady state as  $E_0 = (P, Q, E, R) = (\lambda/\mu N, 0, 0, 0)$ . The reproduction number of smoking habit is denoted by  $R_0$ . It can be obtained by constructing next generation matrix for the system of equations (1) as

$$F = \begin{pmatrix} \frac{\alpha_1 \lambda N}{\mu} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V^{-1} = \begin{pmatrix} \frac{1}{\beta_1 + \mu} & 0 & 0 \\ 0 & \frac{1}{\beta_2 + \mu} & 0 \\ 0 & 0 & \frac{1}{\beta_3 + \mu} \end{pmatrix} \text{ and also } F V^{-1} = \begin{pmatrix} \frac{\alpha_1 \lambda N}{\mu(\beta_1 + \mu)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Recall that the reproduction number is defined as the dominant eigenvalue of the matrix  $[F V^{-1}]$ . That is  $R_0 = \rho(F V^{-1})$ . In case of the present model we have  $R_0 = \lambda_1 = \{\alpha_1 \lambda N / [\mu(\beta_1 + \mu)]\}$ .

#### 3.3 Local Stability of the equilibrium point $E_0$

The local stability can be found by Jacobean matrix that is, by finding the eigenvalues of Jacobean matrix. If all the eigenvalues are negative or having negative real part then the system will be locally asymptotically stable. That is, cigarette smoking does not spread but deteriorates. Otherwise unstable, i.e. cigarette smoking continues to spread and increases.

**Theorem 1:** If  $R_0 < 1$  then the smoking free equilibrium point  $E_0$  will be locally asymptotically stable. If  $R_0 > 1$  then the smoking free equilibrium point  $E_0$  will be unstable. Also If  $R_0 = 1$  then the smoking free equilibrium point  $E_0$  will be stable, i.e., the spread is constant and continues as it is.

**Proof of Theorem 1:** To prove this theorem we first construct Jacobean matrix as

$$J(P, Q, E, R) = \begin{pmatrix} -\alpha_1 Q - \mu & -\alpha_1 P + \beta_1 & \beta_2 & \beta_3 \\ \alpha_1 Q & \alpha_1 P - \beta_1 - \alpha_2 E - \mu & -\alpha_2 Q & 0 \\ 0 & \alpha_2 E & \alpha_2 Q - \alpha_3 R - (\beta_2 + \mu) & -\alpha_3 E \\ 0 & 0 & \alpha_3 R & \alpha_3 E - (\mu + \delta) \end{pmatrix}$$

This Jacobean matrix, at equilibrium point  $E_0$  takes the form as

$$J(E_0) = \begin{pmatrix} -\mu & -N^{**} + \beta_1 & \beta_2 & \beta_3 \\ 0 & N^{**} - (\beta_1 + \mu) & 0 & 0 \\ 0 & 0 & -(\beta_2 + \mu) & 0 \\ 0 & 0 & 0 & -(\mu + \delta) \end{pmatrix}$$

Here we have used new variable  $N^{**} = [\alpha_1 AN/\mu]$ . For finding eigenvalues of  $J(E_0)$ , first subtract  $\lambda$  times identity matrix from the Jacobean matrix  $J(E_0)$ , equate the determinant of the resultant matrix to zero and then solve it for  $\lambda$ . That is, the one to be solved is  $\det[J(E_0) - \lambda I] = 0$  for  $\lambda$ . After following the procedure, the four distinct values of  $\lambda$  are obtained as

$$\lambda_1 = -\mu, \quad \lambda_3 = -(\beta_2 + \mu), \quad \lambda_4 = -(\mu + \delta), \quad \lambda_2 = N^{**} - (\beta_1 + \mu) = R_0 - 1$$

It is straight forward to observe that all the three eigenvalues viz.,  $\lambda_1, \lambda_3$  and  $\lambda_4$  are negative. Now we will show that the other eigenvalue  $\lambda_2$  is negative. Note that the system will be stable if  $R_0 < 1$ , unstable if  $R_0 > 1$  and difficult to decided its nature if  $R_0 = 1$ . Now stability of the model requires that  $R_0 < 1$  and this fact leads to  $\lambda_2 = R_0 - 1$  is less than zero. Therefore  $\lambda_2$  is also a negative value. Since all the four eigenvalues are real and negative, we conclude that the system is stable at the smoking equilibrium point  $E_0$ .

### 3.4 Endemic equilibrium point

The point  $E_0^* = (P^*, Q^*, E^*, R^*)$  represents endemic equilibrium point of the system (1). After solving equations of the system at steady state we get  $P^* = \frac{[\lambda N + (\beta_1/\alpha_1)][(\beta_2 + \mu) + (\beta_3 + \mu)] - (\beta_2/\alpha_2)(\beta_1 + \mu) + (\beta_3/\alpha_3)(\beta_3 + \mu)}{(\alpha_1/\alpha_2)(\beta_3 + 2\mu)}$

$$E^* = \frac{[(1)/[(\alpha_1/\alpha_2)(\beta_3 + \mu)]] [\lambda N + (\beta_1/\alpha_1)][(\beta_2 + \mu) + (\beta_3 + \mu)] - (\beta_2/\alpha_2)(\beta_1 + \mu) + (\beta_3/\alpha_3)(\beta_3 + \mu)}{(\alpha_1/\alpha_2)(\beta_3 + 2\mu)}$$

$$Q^* = [(1/\alpha_2)(\beta_2 + \mu)] + [(1/\alpha_2)(\beta_3 + \mu)] \text{ and } R^* = [(1/\alpha_2)(\beta_3 + \mu)]$$

The endemic equilibrium point  $E^*$  is locally asymptotically stable provided that the reproduction number at equilibrium point is greater than one unit, that is  $R_0 > 1$ . And smoking is spread and mortality rate due to cigarette smoking is also increase.

### 3.5 Global stability of $E_0$

Here in what follows we analyze global stability of the smoking free equilibrium point  $E_0$ . Here note that the region

$\Omega = \{(P, Q, E, R) \in R_+^4 : P + Q + E + R \leq N_0 e^{(\lambda - \mu)t}\}$  is invariant for the model. Also it can be observed that if  $P > [AN/\mu]$  then  $(dP/dt) < 0$ . Therefore we consider the region of the model as positively invariant and thus  $\Omega = \{(P, Q, E, R) : P > [AN/\mu]\}$ .

**Theorem 2:** The smoking free equilibrium point  $E_0$  is globally stable provided that the reproduction number is less than one unit i.e.  $R_0 < 1$ .

**Proof of Theorem 2:** From Theorem 1, we know that if  $R_0 < 1$  then the model does not possess any positive endemic equilibrium point. Also  $E_0$  is the only the equilibrium for the present model. For proving the present theorem we first construct Lyapunov function for the model as  $L = \ln Q + \ln E + \ln R$ . Now we differentiate Lyapunov function  $L$  with respect to time coordinate and substitute for  $Q, E$  and  $R$  from (1). After some simplifications we obtain  $L' = \alpha_1 P - \alpha_2(E - Q) - \alpha_3(R - E) - [(\beta_1 + \mu) + (\beta_2 + \delta + 2\mu)]$ . At the equilibrium point  $E_0$  the expression for the function  $L'$  reduces and takes the form as  $L' = \alpha_1(AN/\mu) - [(\beta_1 + \mu) + (\beta_2 + \mu) + (\delta + \mu)]$  or it has an equivalent expression as  $L' = [\alpha_1 AN/\mu(\beta_1 + \mu)] - \{1 + \{[(\beta_2 + \mu) + (\delta + \mu)]/(\beta_1 + \mu)\}\}$ . The expression for  $L'$  can also be expressed in terms of the reproduction number  $R_0$  as  $L' = R_0 - [1 + \{1 + \{[(\beta_2 + \mu) + (\delta + \mu)]/(\beta_1 + \mu)\}\}]$ . From the foregoing expression it can be observed that if  $R_0 < 1$  then  $L' \leq 0$ . Holding of this condition is the requirement for  $E_0$  to be globally asymptotically stable. Hence, the statement of the theorem is proved.

## IV. Numerical Simulations

In this section, we illustrate some numerical solutions of the system for different values of the parameters, and show that these solutions are in agreement with the qualitative behavior of the solutions, cigarette smoking and its effect by using different parameters with constant variables of with different initial values such that  $N = P + Q + E + R$ , with  $P(0) = 33800, Q(0) = 7500, E(0) = 16700, R(0) = 3500$ , and  $N(0) = 61500$ .



Thus when we use the parameters  $\lambda = 0.015$ ,  $\mu = 0.004$ ,  $\alpha_1 = 0.000029$ ,  $\alpha_2 = 0.000047$ ,  $\alpha_3 = 0.00005$ ,  $\beta_1 = 0.004$ ,  $\beta_2 = 0.0012$ ,  $\beta_3 = 0.0029$ , and for  $R_0 = 836.0156 > 1$ , Figure 2(a) shows that the number of non smokers decrease rapidly. Because the number of new smokers and the number of patient smokers increase at the same time. But the number of Educated smokers are decrease, because the number of individuals that are transmit from new smokers to educated smokers  $\alpha_2$  are less than that of the sum of the number of natural death rate  $\mu$ , the number of individuals that are transmit from Educated smokers to patient individuals  $\alpha_3$  and the number of individuals that are transmit from Educated smokers to potential smokers  $\beta_2$ . Also in the case of these reasons the number of dead individuals that are caused by cigarette smoking are very fast to increase. Therefore cigarette smoking is quickly spread and also mortality rate is increase, because  $R_0 > 1$ . So these is not good and we must be change by take different techniques to minimize  $R_0$  by minimizing the parameters  $\alpha_1$  and also to maximize  $\beta_1$  to be the model is stable.

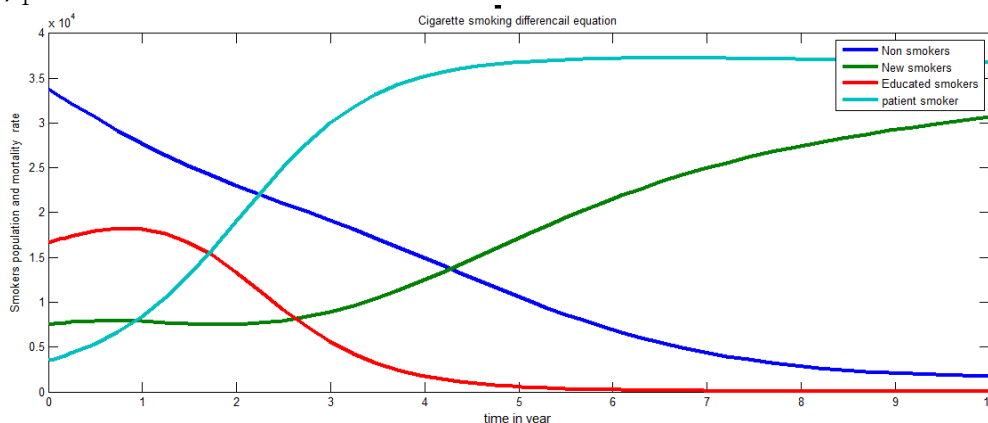


Figure 2: Population dynamics when  $\alpha_1 = 0.0000099$ ,  $\beta_1 = 0.04$  and  $R_0 = 836.0156$

For the same variables by changing only its parameters we have different plots. Thus when we use the following parameters  $\lambda = 0.015$ ,  $\mu = 0.004$ ,  $\alpha_1 = 0.0000099$ ,  $\alpha_2 = 0.0000096$ ,  $\alpha_3 = 0.000017$ ,  $\beta_1 = 0.04$ ,  $\beta_2 = 0.05$ ,  $\beta_3 = 0.03$ , for  $R_0 = 51.89 > 1$ , in Figure 2(b) shows that the number of new smokers and the number of patient smokers are increase, when the number of new smokers increase, the number of non smokers decrease and vice versa. The number of patient individuals that is caused by cigarette smoking are slightly increase, but the number of educated smokers are slightly decrease. As a result the number of dead individuals also increase slightly. So this is also not good and not stable, that is  $R_0 > 1$ , therefore it must be take different techniques also more and more to minimize  $R_0$  by minimizing the parameter  $\alpha_1$  and also to maximize  $\beta_1$  to be the model is stable.

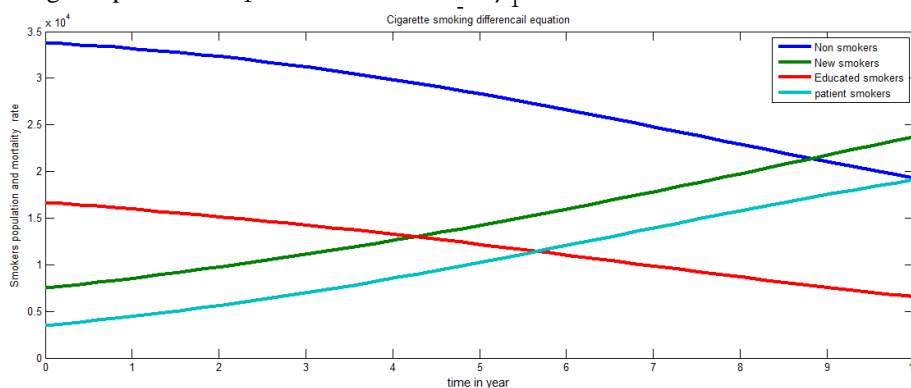


Figure 3: Population dynamics when  $\alpha_1 = 0.00000029$ ,  $\beta_1 = 0.04$  and  $R_0 = 51.89$

For the same variables by changing only its parameters we have different plots. Thus when we use the following parameters  $\lambda = 0.0008$ ,  $\mu = 0.02$ ,  $\alpha_1 = 0.00000029$ ,  $\alpha_2 = 0.00000047$ ,  $\alpha_3 = 0.00000005$ ,  $\beta_1 = 0.8$ ,  $\beta_2 = 0.7$ ,  $\beta_3 = 0.6$ , for  $R_0 = 0.00087 < 1$ , in Figure 2(c) shows that the number of new smokers, the number of patient smokers and the number of educated smokers are decrease quickly, and so the number of potential smokers are increase quickly until the number of total populations, thus the number of patient smokers and the number of Educated smokers becoming to zero. Therefore the

number of dead individuals decrease slightly at some instant and be coming constant ,because the number of dead individuals can't be to zero that is already dead once.

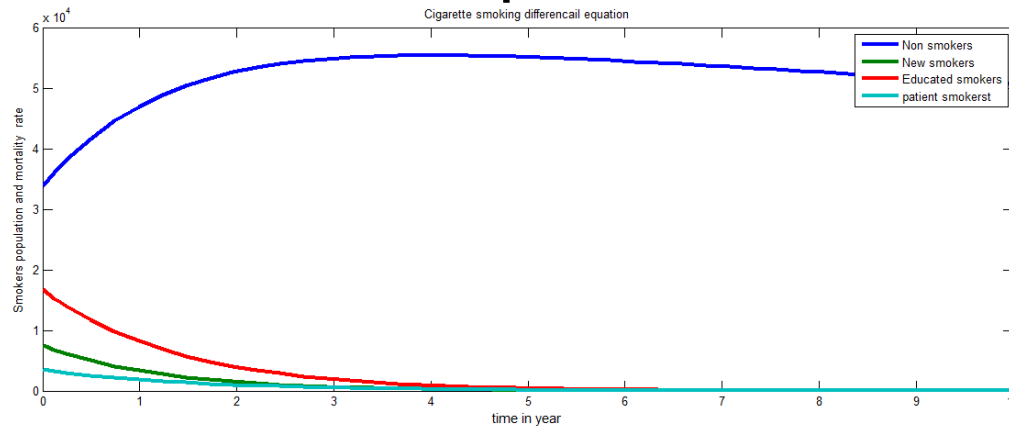


Figure 4: Population dynamics when  $\alpha_1 = 0.0000029$ ,  $\beta_1 = 0.8$  and  $R_0 = 0.00087$

### V. Conclusions

In these paper, a non linear mathematical model was formulated, which describes the overall smoking population and mortality rate when the population is assumed to remain constant. We used the stability analysis theory for non-linear system to analyze the mathematical models and to study both the local and global behavior of smoking dynamics. Locally asymptotic stability for the smoking free equilibrium  $E_0$  can be obtained, if  $\mathfrak{R}_0 = \{(\alpha_1 AN)/[\mu(\beta_1 + \mu)]\}$  and  $\mu(\beta_1 + \mu) > \alpha_1 AN$

Generally , if the transmission rate of potential smoker to new smoker  $\alpha_1$ , the transmission rate of new smoker to Educated smoker  $\alpha_2$ , and the transmission rate of Educated smoker to patients that are caused by cigarette smoking  $\alpha_3$  are increase, that is the number of smokers are increase, and also if the transmission rate of new smoker to potential smokers  $\beta_1$ , the transmission rate of Educated smoker to potential smokers  $\beta_2$ , the transmission rate of patient smokers to potential smokers  $\beta_3$  decrease, then the number of non-smokers decrease and the number of dead individuals increase. But , if the transmission rate of potential smoker to new smoker  $\alpha_1$ , the transmission rate of new smoker to Educated smoker  $\alpha_2$ , and the transmission rate of Educated smoker to patients that are caused by cigarette smoking  $\alpha_3$  are decrease that is, the number of smokers are decrease, and also if the transmission rate of new smoker to potential smokers  $\beta_1$ , the transmission rate of Educated smoker to potential smokers  $\beta_2$ , the transmission rate of patient smokers to potential smokers  $\beta_3$  increase, then the number of non smokers increase and the number of dead individuals decrease. But when we see stability, the reproductive number is less than one (or  $R_0 < 1$ ) if the rate of transmission from potential smokers to new smokers  $\alpha_1$  is decrease and the number of giving up smoking from new smokers  $\beta_1$  is increase.

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