Modeling and Analysis of the Dynamic Progress of Chronic Lung Cancer Due to Smoking

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Abstract

Lung cancer is the leading cause of cancer death worldwide irrespective of age, sex and region. Cigarette smoking is the main cause of lung cancer and about 85 percent of the disease are caused by smoking. The smokers not only endangering themselves but also contribute comprehensively in developing lung cancer among the non-smokers who are in interaction with them. So a mathematical model of lung cancer has been developed in terms of non-linear ordinary differential equation to study the disease dynamics and it's relation with smoking as well as interacting non-smokers. Considering the realistic phenomena of lung cancer a SEIRS model with five compartments including susceptible, active smoker, non-smoker victim, infected with lung cancer and recovered has been developed. The boundedness and positivity of the model is investigated and the basic reproduction number and equilibrium point (smoke free and endemic) has been calculated. The stability analysis has also been performed to investigate whether the equilibrium points are locally/globally asymptotically stable or not and finally numerical simulation is performed to show the result's effectiveness graphically. Including interaction between smokers and non-smokers, the model will describe the dynamics of lung cancer completely which will help us to minimize the fatality.

Keywords
lung cancer, smokers, victim of smoking, SEIRS model, numerical analysis

1. Introduction

Lung cancer is the most life threatening cancer disease of the world. Statistics shows that it has been the leading cause of cancer deaths worldwide with approximately 2.09 million new diagnoses each year and around 1.76 million deaths (WHO, 2018). According to GLOBOCAN-2018 lung cancer is estimated to be the second top most prevalent cancer in the world according to the number of new incidences and be at the top of the list according to number of deaths (see Figure 1). South Asian developing country Bangladesh are also facing danger with cancer disease with 13 to 15 lakh cancer patients and about 2 lakh patients are newly diagnosed each year. Lung cancer can start from anywhere in the lungs and can affect any part of the respiratory system. Unlike other normal cells, these cancer cells grow without control and can destroy the healthy lung tissue around them. This growth spread, or metastasize, beyond the lung to the lymph nodes by the process of metastasis into nearby tissue or other parts of the body. Smoking tobacco is by far known as the leading cause of lung cancer and about 80% of the lung cancer deaths are caused by

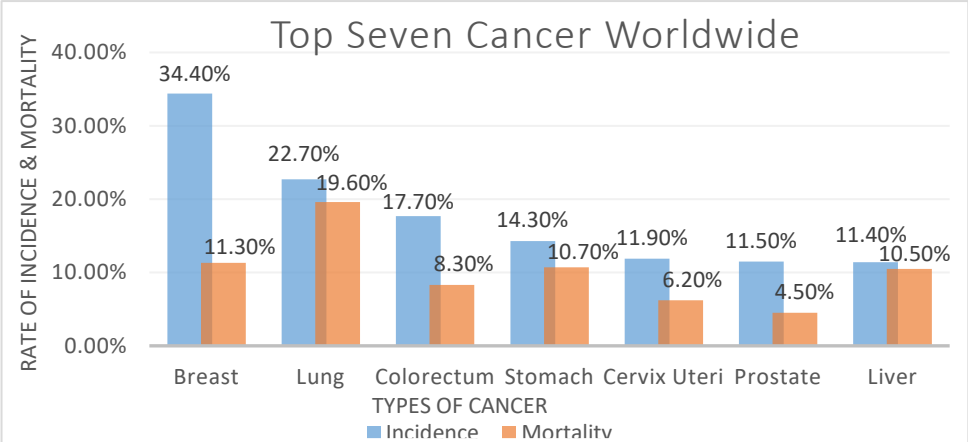

Figure 1: Top Seven Cancer Incidence and Death Rate Worldwide [10].

smoking. The major cancer-causing agents is known as carcinogens and tobacco smoke contains over 60 carcinogens (WHO, 2018). Mathematical modeling plays a significant role for providing quantitative insight into multiple fields. It has already contributed for better understanding of the mechanisms of many non-communicable diseases. It is mainly used to describe the real phenomena which leads to design better prediction, management and control strategies. There is a large body of work to develop mathematical models and optimal control policies of infectious diseases. Biswas (2014) (see also Biswas *et al*., 2014) and Khatun (2020) investigated and analyzed the treatment of most devastating infectious diseases independently in which mathematical modeling was the key tool.
In this paper, we would like to propose a mathematical model to study the dynamics of lung cancer. Our aim is to analyze the model of lung cancer to show the effect of smoking to this fatal diseases, determine the basic reproduction number and study the existence and stability of the disease free and endemic equilibrium points of the model. Finally numerical simulations will be performed.

1.1 Objectives

The present study focuses on the following objectives aiming

i) to study the biological insights of dynamics transmission of lung cancer
ii) to propose a mathematical model of lung cancer in terms of a system of nonlinear ordinary differential equations (NODEs) considering the assumptions of the disease using conservation and balance principle
iii) to verify the boundedness and positivity of the solution of the model
iv) to calculate the basic reproduction number as well as the smoke free and endemic equilibrium point
v) to analyze the stability of the smoke free and endemic equilibrium point
vi) to ascertain the mathematical model numerically

2. Literature Review

Mathematical model based study of lung cancer was discussed by many mathematicians. A numbers of mathematical model have been proposed by researchers relating the smoking behavior and lung cancer. Acevedo-Estefania *et al.* (2000) proposed a model to describe the dynamics of lung cancer at the population level where smoking and second-hand smoke is the main cause of lung cancer. The model determined that the best way to lower the number of smokers and individuals developing lung cancer is by increasing the number of well-educated population about the effect of smoking. Andest (2013) also formulated a mathematical model that describes nicotine accumulation in lung of a smoker which is the main cause of lung cancer. Wardah *et al.* (2017) presented a mathematical model that discuss of lung cancer as the effect of smoking behavior on both active and passive smoker. Trisilowati (2019) analyze the stability as well as used optimal control strategy to Wardah *et al.* (2017) model and illustrate that the optimal control is effective to control the growth of passive smokers, active smokers and lung cancer patients. There is an extensive body of work which developed models associating with the treatment of cancer using chemotherapy, radiotherapy, targeted agent treatment etc. Beljanski *et al.* (2012) mentioned that multi-drug resistance to these therapeutic treatment is the major cause of failure in clinical therapeutic treatment. Genetically modified oncolytic viruses (OVs) kill tumor cell via completely unique mechanisms compare to other therapeutic treatments. Thus Beljanski *et al.* (2012) claims that treatments with oncolytic viruses (OVs) which is under development will open the possibility to overcome drug resistance and module the immune response to fight against cancer.
Taking the above discussions into account, we propose a model to study the dynamics of lung cancer and it's relation with smoking. Many theoretical as well as mathematical models of lung cancer have been proposed by researchers. But this is a newly proposed mathematical model of lung cancer on the basis of some basic assumptions. We observed from real phenomena that patients recovered from lung cancer are not out of danger at all. They can be affected with lung cancer again. It indicates that people recovered from lung cancer are becoming susceptible again and can develop this fatal disease for multiple time. So our SEIRS model will be more realistic and effective than other SIR or SEIR model discussed above. Our goal is to study the disease dynamics of lung cancer and the relation between smoking and lung cancer both analytically and numerically.

3. Methods

A mathematical model of a system of nonlinear ordinary differential equations (ODEs) will be governed in this study. The basic reproduction number will be determined by next generation matrix. The condition for the existence of equilibrium points (smoke free and endemic equilibrium points) and their stability analysis will be investigated. At last, the mathematical model will be solved numerically by using ode45 solver written in MATLAB

programming. The effective methods to control the disease or to prevent people from being infected with lung cancer are as follows:

i) Before infection, awareness among the people can be raised about not to smoke as well as keep away from smokers. At the same time public awareness can be developed about the effect of air pollution especially radon gas emission.
ii) Early detection of lung cancer is very important for the survival of the patients and so public awareness is exigent in this respect.
iii) After infection, treatment on the basis of stage and infection quality should be taken.

4. Model Formulation

Let $N(t)$ be the total population in a region at any time t which is subdivided into five compartments such as, $S(t)$ (susceptible population that is the vulnerable subpopulation who are not infected with lung cancer, but at a high risk of infection as a result of smoking), population who are active smoker $E_a(t)$, victim of smoking $E_v(t)$, number of individual infected with lung cancer $I(t)$ and number of population recovered from lung cancer $R(t)$.

Then the compartmental model diagram will be,

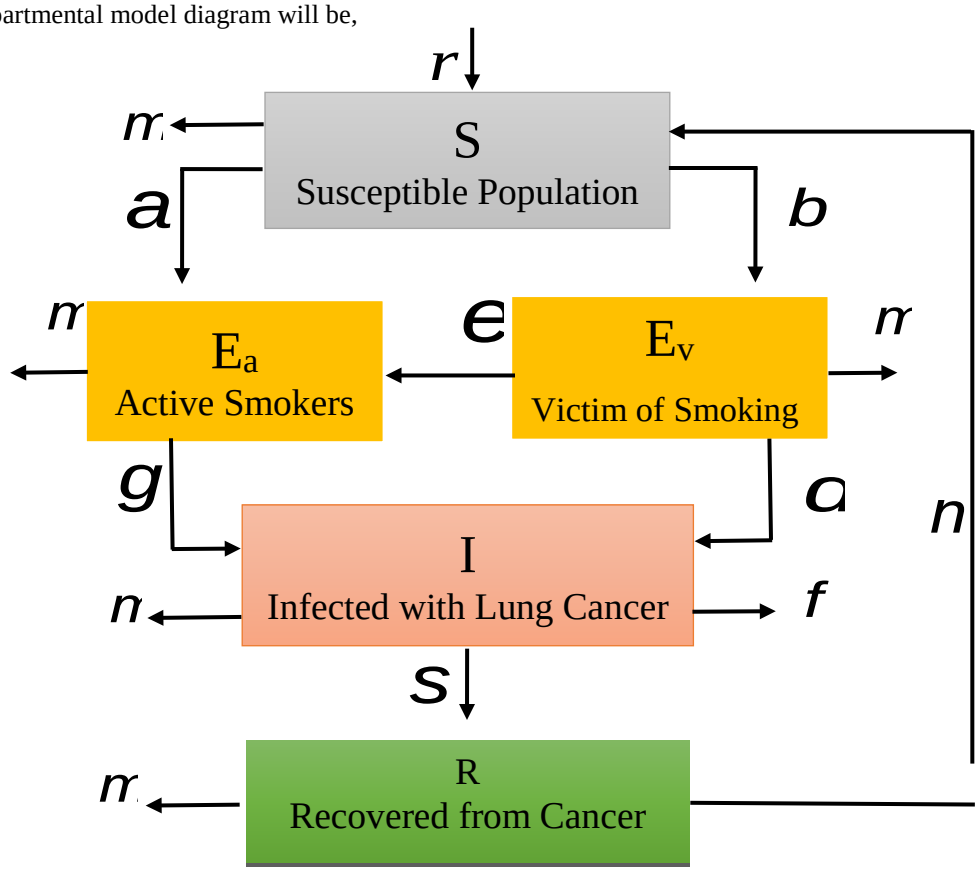

Figure 2: Compartmental model diagram of lung cancer.

Considering the above diagram, the mathematical model can be represented by the following system of non-linear ordinary differential equations.

$$\frac{dS}{dt} = r - (a+b)E_a S - mS + hR$$

$$\frac{dE_a}{dt} = aE_a S + eE_a E_v - gE_a - mE_a$$

$$\frac{d\,\mathrm{E}_v}{dt} = b\,\mathrm{E}_a\,S - e\mathrm{E}_a\mathrm{E}_v - d\mathrm{E}_v - m\mathrm{E}_v \tag{4.1}$$

$$\frac{dI}{dt} = gE_a + dE_v - sI - (m+f)I$$

$$\frac{dR}{dt} = sI - (m+h)R$$

where $S(0) \geq 0, \mathrm{E}_a(0) \geq 0, \mathrm{E}_v(0) \geq 0, I(0) \geq 0, R(0) \geq 0$ and $N = S + E_a + E_v + I + R$

In the above model, r is the natural growth rate of population, m is the natural mortality rate, a and b is rate at which susceptible population become active smoker and victim of smoking respectively. We have considered the direct contact between active smoker and victim non-smoker and e is the migration rate of victim group to active smoker. The constant g and d represents the rate at which active smokers and victim non-smokers become infected with lung cancer. The constant s represents the recovery rate from lung cancer by getting proper treatment and f represents the disease induced death rate. In real life, despite of getting treatment and recovered from lung cancer, people may become susceptible of lung cancer again and the rate is denoted by h.

5. Model Analysis

The model (4.1) has to be analyzed in order to describe the dynamics of lung cancer. The desire of this analysis is to show the effect of smoking in lung cancer and the object of this analysis is to control the adverse situation from locality.

Since it is impossible to find the exact solution of the nonlinear autonomous system (4.1), we have to analyze the qualitative behaviour of the solutions in the neighbourhood of the equilibrium points. First we find the boundedness and positivity of the solutions then find out the equilibrium points followed by analyzing the stability of the equilibrium points and basic reproduction number R_0. The basic reproduction ratio is important because it tells us if a disease will persists or extinct. For the analysis of model (4.1), a closed set has been considered as

$$\Omega = \left\{ \left(S(t), E_a(t), E_v(t), I(t), R(t)\right) \in \mathbb{R}^5_+ \;\middle|\; 0 \leq N \leq \frac{r}{\mu} \right\}$$

with the initial condition $S(0) > 0,\ E_a(0) \geq 0, E_v(0) \geq 0, I(0) \geq 0, R(0) \geq 0$.

5.1 Boundedness of Solutions of the Model

We have to show that the total population is bounded for all $t \geq 0$.

Lemma 1: The region $\Omega = \left\{ \left(S(t), E_a(t), E_v(t), I(t), R(t)\right) \in \mathbb{R}^5_+ \;\middle|\; 0 \leq N \leq \frac{r}{\mu} \right\}$ is a positively invariant set for the model (4.1).

Proof: Since the population size is $N(t)$ so that $N(t) = S(t) + E_a(t) + E_v(t) + I(t) + R(t)$. Now the rate of change of total population is $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE_a}{dt} + \frac{dE_v}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$

$$\Rightarrow \frac{dN}{dt} = r - \mu\left(S + E_a + E_v + I + R\right) - \phi I$$

$$\Rightarrow \frac{dN}{dt} = r - \mu N - \phi I$$

$$\Rightarrow \frac{dN}{dt} + \phi I = r - \mu N$$

In the absence of the disease lung cancer $(\text{i.e. } I = 0)$, we get $\frac{dN}{dt} \leq r - \mu N$

Now solving this we obtain, $N(t) \le \frac{r}{\mu} + \left(N_0 - \frac{r}{\mu} \right) e^{-\mu t}$

From this solution, it is clear that the total population $N(t)$ will approach the threshold $\frac{r}{\mu}$ as $t \to \infty$. This indicates that if the initial total population N_0 is less than $\frac{r}{\mu}$ i.e. if $N_0 \le \frac{r}{\mu}$ then $\lim_{t\to\infty} N(t) = \frac{r}{\mu}$. So, definitely $\frac{r}{\mu}$ is the upper bound of N.

On the other hand, if $N_0 > \frac{r}{\mu}$, then $N(t)$ will decrease to $\frac{r}{\mu}$ as $t \to \infty$. This means that if $N_0 > \frac{r}{\mu}$, then the solutions $\left(S(t), E_a(t), E_v(t), I(t), R(t)\right)$ enters the region Ω or approaches it asymptotically.

Thus we conclude that the region Ω is positively invariant under the flow induced by the model (4.1). Therefore, the model is both mathematically and epidemiologically well-posed in the region Ω. It is therefore sufficient to study the dynamics of the model (4.1) in Ω. Hence the lemma is proved.

5.2 Positivity of solution of the Model

Since the model (4.1) describes the human population, it is necessary to prove that all the state variables $S(t), \mathrm{E}_a(t), \mathrm{E}_v(t), I(t), R(t)$ are non-negative i.e. the solutions of the model (4.1) with positive initial conditions, $S(0) \ge 0, \mathrm{E}_a(0) \ge 0, \mathrm{E}_v(0) \ge 0, I(0) \ge 0, R(0) \ge 0$, are non-negative $\forall t \ge 0$.

Lemma 2: If $S(0) > 0$, $E_a(0) \ge 0$, $E_v(0) \ge 0$, $I(0) \ge 0$, and $R(0) \ge 0$, then the solutions $S(t)$, $E_a(t), E_v(t), I(t), R(t)$ of the model (4.1) are all non-negative for all $t \ge 0$.

Proof: The initial conditions for the model 4.1 is, $S(0) \ge 0, \mathrm{E}_a(0) \ge 0, \mathrm{E}_v(0) \ge 0, I(0) \ge 0, R(0) \ge 0$

The first equation of the model (4.1) is denoting the rate of change of susceptible population with time.

$$i.e.\ \frac{dS}{dt} = r - (a+b)\mathrm{E}_a S - mS + hR \tag{5.1}$$

For positivity, (5.1) can be written as, $\frac{dS}{dt} + mS \ge r$ (5.2)

$$\therefore S \ge \frac{r}{m} + ce^{-m} \tag{5.3}$$

Applying initial condition, (at $t = 0, S(0) \ge 0$), we get from (4.4), $c = S(0) - \frac{r}{m}$

By putting the value of c in (5.3) we get,

$$S(t) \ge \frac{r}{m} + \left(S(0) - \frac{r}{m} \right) e^{-m} \tag{5.4}$$

So, at $t \to \infty$, $S(t) \ge \frac{r}{m}$, which is also greater that 0.

So the first solution $S(t)$ of the model (4.1) is positive for all $t \ge 0$

Therefore, all the solutions $S(t), \mathrm{E}_a(t), \mathrm{E}_v(t), I(t), R(t)$ of the dynamic model (4.1) with positive initial conditions, $S(0) \ge 0$, $\mathrm{E}_a(0) \ge 0$, $\mathrm{E}_v(0) \ge 0$, $I(0) \ge 0$, $R(0) \ge 0$ are non-negative for all $t \ge 0$.

5.3 Smoke-Free Equilibrium Point

The diseases free equilibrium point of a system is the point with no infections or diseases. Let us consider the smoke free equilibrium point of the model (4.1) is w_0. In case of smoke free equilibrium point for the model (4.1), all the state variables, $\mathrm{E}_a, \mathrm{E}_v, I, R$ are zero, except the susceptible compartment S.

So, $\frac{dS}{dt} = E_a = E_v = I = R = 0$

Hence we get, $r - mS = 0$

$$\therefore S = \frac{r}{m}$$

So the smoke free equilibrium for the model (4.1) is, $w_0 = (S, E_a, E_v, I, R) = (\frac{r}{m}, 0, 0, 0, 0)$

5.4 Endemic Equilibrium Point

Let the endemic equilibrium point for the model (4.1) is $w_1(S^*, E_a^*, E_v^*, I^*, R^*)$ that can be obtained by considering,

$$\frac{dS^*}{dt} = \frac{E_a^*}{dt} = \frac{E_v^*}{dt} = \frac{I^*}{dt} = \frac{R^*}{dt} = 0$$

i.e. $\frac{dS}{dt} = r - (a+b)E_a^* S^* - mS^* + hR^* = 0$ (5.5)

$$\frac{dE_a}{dt} = aE_a^* S^* + eE_a^* E_v^* - gE_a^* - mE_a^* = 0 \quad (5.6)$$

$$\frac{dE_v}{dt} = bE_a^* S^* - eE_a^* E_v^* - dE_v^* - mE_v^* = 0 \quad (5.7)$$

$$\frac{dI}{dt} = gE_a^* + dE_v^* - sI^* - (m+f)I^* = 0 \quad (5.8)$$

$$\frac{dR}{dt} = sI^* - (m+h)R^* = 0 \quad (5.9)$$

By solving equation (5.5-5.9) we get the endemic equilibrium point is $w_1 = (S^*, E_a^*, E_v^*, I^*, R^*)$

where, $S^* = \frac{r + \eta R^*}{k_5 E_a^* + \mu}$ (5.10)

$$E_a^* = \frac{k_3 k_4 - \alpha k_4 S^*}{\varepsilon(k_5 S^* - k_3)} \quad (5.11)$$

$$E_v^* = \frac{k_3 - \alpha S^*}{\varepsilon} \quad (5.12)$$

$$I^* = \frac{(k_3 - \alpha S^*)(\gamma k_4 + \delta k_5 S^* - \delta k_3)}{\varepsilon k_2 (k_5 S^* - k_3)} \quad (5.13)$$

$$R^* = \frac{\sigma(k_3 - \alpha S^*)(\gamma k_4 + \delta k_5 S^* - \delta k_3)}{k_1 k_2 \varepsilon (S^* k_5 - k_3)} \quad (5.14)$$

Also, $k_1 = \mu + \eta, k_2 = \sigma + \mu + \phi, k_3 = \gamma + \mu, k_4 = \delta + \mu, k_5 = \alpha + \beta$

By using (5.11) and (5.14) in (5.10) we get, $AS^{*2} + BS^* + C = 0$

$$\Rightarrow S^* = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A} \quad (5.15)$$

which exists with the following condition, $(i)\ B^2 - 4AC = 0$ and $B < 0$

or $(ii)\ C < 0$

or $(iii)\ C > 0$ and $B < 0$

or $(iv)\ C = 0$ and $B < 0$

where, $A = k_1 k_2 k_5 \mu\varepsilon - k_1 k_2 k_4 k_5 \alpha + \eta\sigma\delta\alpha k_5$

$$B = k_1k_2k_3k_4k_5 - k_1k_2k_3\mu\varepsilon - r\varepsilon k_1k_2k_5 - \eta\sigma\delta k_3k_5 - \eta\sigma\alpha\delta k_3 + \eta\sigma\alpha\gamma k_4 \quad (5.16)$$
$$C = r\varepsilon k_1k_2k_3 + \eta\sigma\delta k_3^2 - \eta\sigma\gamma k_3k_4$$

5.5 Basic Reproduction Number

The basic reproduction number is defined as the secondary infections produced by one primary infection in a wholly susceptible population. It is a key epidemiological quantity, because it determines the size and duration of epidemics. Here the F_i is the gains to infectious compartments, V_i is the losses from infectious compartments.

$$F_i = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \end{bmatrix} = \begin{bmatrix} \alpha E_a S \\ \beta E_a S \\ 0 \end{bmatrix} \text{ and } V_i = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \end{bmatrix} = \begin{bmatrix} (\gamma + \mu - \varepsilon E_v) E_a \\ (\varepsilon E_a + \delta + \mu) E_v \\ (\sigma + \mu + \phi) I - (\gamma E_a + \delta E_v) \end{bmatrix} \quad (5.17)$$

At the disease free equilibrium point $F_{dfe}\left(\frac{r}{\mu}, 0, 0, 0, 0\right)$, we have

$$F = \begin{bmatrix} \frac{\alpha r}{\mu} & 0 & 0 \\ \frac{\beta r}{\mu} & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (5.18)$$

$$V = \begin{bmatrix} \gamma + \mu & 0 & 0 \\ 0 & \delta + \mu & 0 \\ -\gamma & -\delta & \sigma + \mu + \phi \end{bmatrix} = \begin{bmatrix} k_3 & 0 & 0 \\ 0 & k_4 & 0 \\ -\gamma & -\delta & k_2 \end{bmatrix} \quad (5.19)$$

where $\sigma + \mu + \phi = k_2$, $\gamma + \mu = k_3$ and $\delta + \mu = k_4$

Now the characteristic equation is given by setting $\det(G - \lambda I) = 0$, where $G = FV^{-1}$.

$$\therefore G - \lambda I = \begin{bmatrix} \frac{\alpha r}{\mu k_3} - \lambda & 0 & 0 \\ \frac{\beta r}{\mu k_3} & -\lambda & 0 \\ 0 & 0 & -\lambda \end{bmatrix} = 0$$

$$\Rightarrow \left(\frac{\alpha r}{\mu k_7} - \lambda\right)(-\lambda)(-\lambda) = 0$$

$$\therefore \lambda = \frac{\alpha r}{\mu k_3}, 0, 0 \quad (5.20)$$

Equation (5.13) follows that the basic reproduction number which is given by the largest eigen value for the model (4.1) is $R_0 = \frac{\alpha r}{\mu k_3}$

5.6 Stability at Smoke Free and endemic Equilibrium Point

Theorem 1: The smoke free equilibrium point is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$

Proof: In order to perform the stability analysis at smoke free equilibrium point w_0, we have the Jacobian matrix of the model (4.1) at smoke-free equilibrium point $w_0 = (\frac{r}{m}, 0, 0, 0, 0)$ is,

$$J(w_0)=\begin{pmatrix} -m-(a+b)\frac{r}{m} & 0 & 0 & h \\ 0 & a\frac{r}{m}-g-m & 0 & 0 & 0 \\ 0 & b\frac{r}{m} & -d-m & 0 & 0 \\ 0 & g & d & -(s+m+f) & 0 \\ 0 & 0 & 0 & s & -(m+h) \end{pmatrix}$$

Let λ be the eigen value and I be the identity matrix, then the characteristic equation is,

$$\left|J(w_0)-\lambda I\right|=\begin{vmatrix} -m-\lambda & -(a+b)\frac{r}{m} & 0 & 0 & h \\ 0 & a\frac{r}{m}-g-m-\lambda & 0 & 0 & 0 \\ 0 & b\frac{r}{m} & -d-m-\lambda & 0 & 0 \\ 0 & g & d & -(s+m+f)-\lambda & 0 \\ 0 & 0 & 0 & s & -(m+h)-\lambda \end{vmatrix}=0 \quad (5.21)$$

Solving this, we get

$$(m+\lambda)\left(a\frac{r}{m}+g+m\right)(\lambda+d+m)(\lambda+s+m+f)(\lambda+m+h)=0 \quad (5.22)$$

The eigen values $\lambda_1=-\mu$, $\lambda_3=-d-m, \lambda_4=-(s+m+f)$ and $\lambda_5=-(m+h)$ are negative roots of the characteristic polynomial. Again $\lambda_2=a\frac{r}{m}-g-m=k_3(R_0-1)$ which will be negative when $R_0<1$. Therefore, the smoke free equilibrium point $w_0=(\frac{r}{m},0,0,0,0)$ is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

We analyse the stability of the endemic equilibrium point w_1. So we have the Theorem 2 to determine the stability of the endemic equilibrium point w_1.

Theorem 2: The endemic equilibrium point is locally asymptotically stable if $R_0>1$ and unstable if $R_0<1$.

5.7 Numerical Results

We perform numerical simulations of our model proposed in (4.1) by the ODE45-solver using MATLAB programming. All the values of the parameters used in Table 1 was obtained from different organizations such as the CDC (Center for Disease Control), American Lung Cancer Society, WCRF (world cancer research foundation), WHO (world health organization) Global cancer observatory (GLOBOCAN) and other nonprofit and government agencies.

Table 1: Descriptions of parameters and their values

Descriptions	Parameters	Values
growth rate of population	r	0.05 d^{-1}
natural mortality rate	m	0.01 d^{-1}
active smoker recruitment rate from susceptible population	a	0.4
rate of population become victim of smoking	b	0.5

rate of population migrate from victim group to smokers	e	0.35
smoker's lung cancer incidence rate	g	0.2
lung cancer incidence rate from victim group	d	0.05
lung cancer recovery rate	s	0.2
cancer incident mortality rate	f	0.6
migration rate from recovered to susceptible compartments	n	0.5

We first run the numerical simulation to observe the behavior of all the state variables of the model by using the parameter values showed in the Table 1.

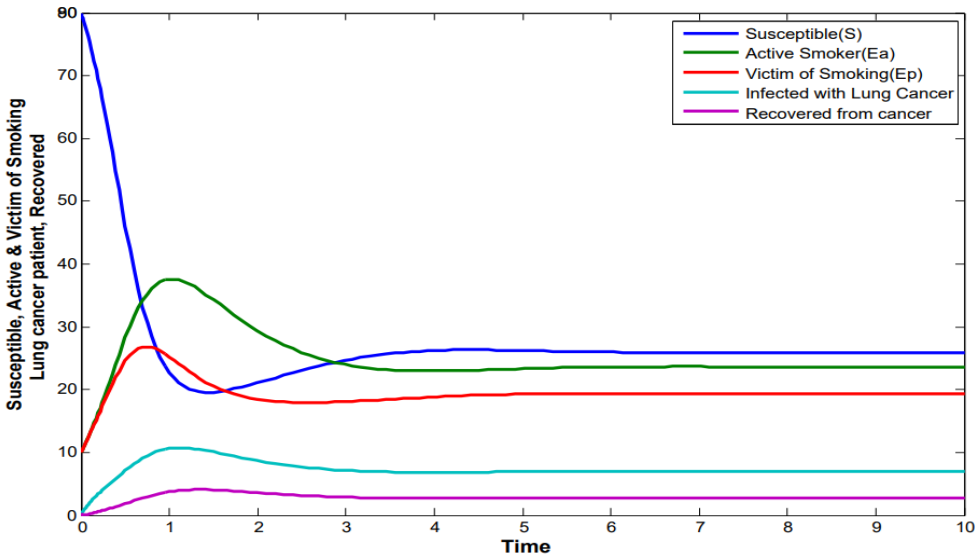

Figure 3: Susceptible population, active smokers and victim of smoking population, population infected with lung cancer and recovered from lung cancer.

Figure 3 represents the behavior of all the state variable of the model (4.1). We assumed that initially there was no infected or recovered population and less number of smokers as well as less number of victim population. So initially the susceptible group is so large and with time it decreased to a stable position (blue line). Again the number of smokers (green line) as well as victim population (red line) increased with time and move to a stable position. So the number of infected population (sky blue line) and recovered population (purple line) increased.

Again we run the simulation for different recruitment rate of active smoker $a = 0.2, 0.5, 0.9$ observed that different recruitment rate of active smoker results in different number of passive smokers and also change in the number of lung cancer patients.

Figure 4 represented the change in the number of active smoker population with the change of different active smoker recruitment rate a. We observed that higher recruitment rate results in higher number of active smokers which can be predicted easily. We observed that when the value of a is 0.2 then the number of smokers is minimum and it is maximum with the maximum value of a.
Again from Figure 5 we observe that higher number of active smoker which results in higher number of victim population group who are non-smoker also produced more lung cancer infected patient. When a is 0.2 that is less number of smoker results in less number of lung cancer infected patient. But when a increases to 0.9 then this higher number of smokers produced large number of cancer patients. Thus it can easily be predicted that large number of cancer patient will results in more lung cancer induced death which will increase the fatality of the disease and make the situation worse.

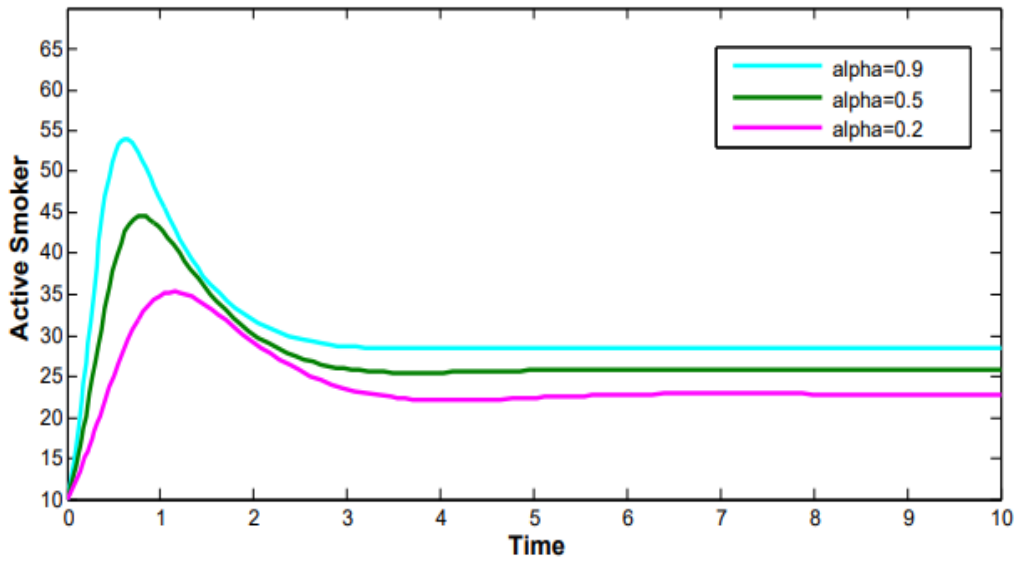

Figure 4: Change in the number of active smokers with the change of active smoker recruitment rate a .

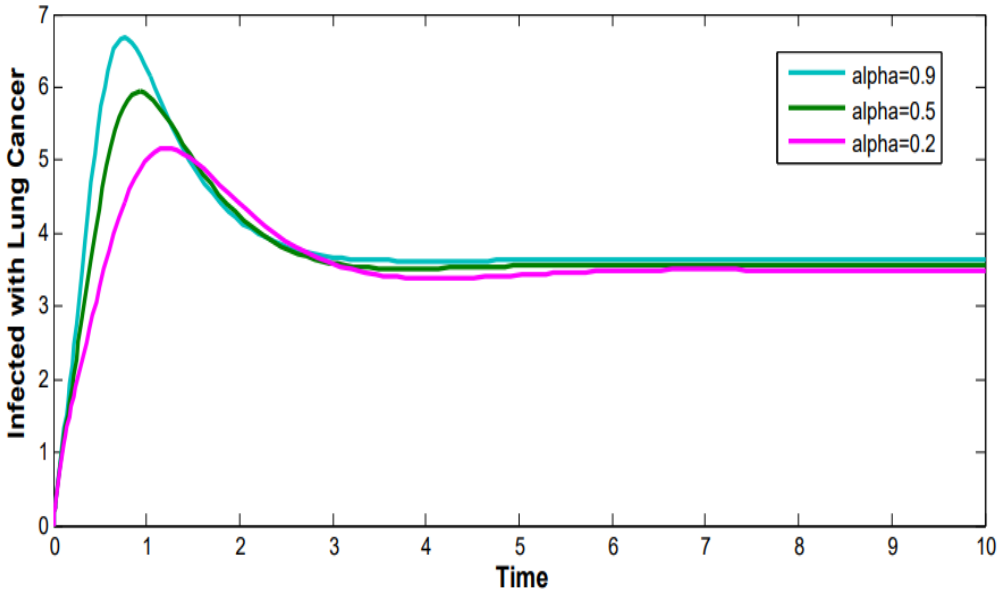

Figure 5: Change in the number of lung cancer infected population with the change of a .

Again from Figure 6 we observe that increased migration rate from victim group to smoker group results in increased number of lung cancer patients. The smokers not only endangering themselves but also make the in contact victim group sufferer. They also directly or indirectly promote those victim populations to become smoker. If those victim group are not well educated or not properly aware of the effect of smoking there is a big chance of large migration rate from victim to smoker. Figure 6 shows this phenomena that higher number of migration rate e results in higher number lung cancer infected population. When e is 0.2 that is less migration rate results in less number of lung cancer infected patient. But when e increases to 0.9 then it produce large number of cancer patients.

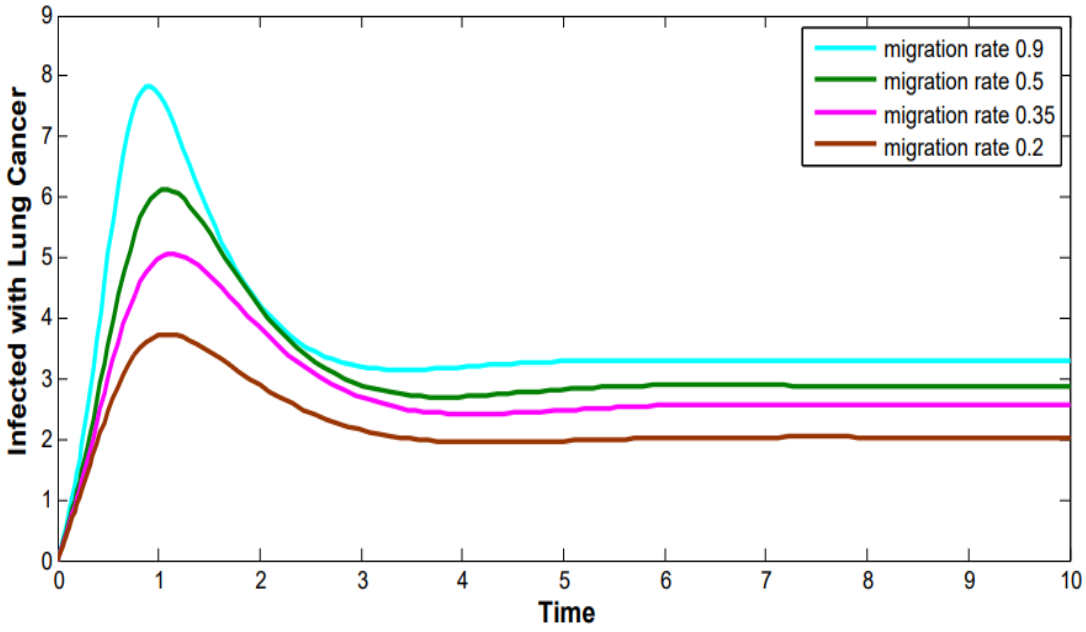

Figure 6: Change in the number of lung cancer infected population with the change of e.

All the four Figures 3-6 clearly indicate that whenever the active smoker recruitment rate increased that is whenever the number of active smoker increased then the number of non-smoker victim population will automatically increase and at the same time both smoker and non-smoker victim group will produce more lung cancer incidence and lung cancer induced mortality. Also the uneducated or unaware victim population group are at a risk of becoming smokers which increase the fatality more. As the smoker are playing the key role in producing lung cancer patient so we have to minimize the number of population of this group and at the same time we should keep the non-smoker population away from the contact of smoker and make them aware of the dangerous effect of smoking to save them from this life threatening diseases.

6. Conclusion

Lung cancer is the most life threatening cancer diseases in the worldwide. It affects millions of patients all over the world and a large number of them results in cancer causing death. Lung cancer occurs throughout the world irrespective of age, sex, region and race. In this paper we observed the dynamics of lung cancer for the changing values of different parameters whether the effectiveness of lung cancer increases or decreases. We observed from the numerical simulations that the increasing number of active smoker results in larger number of victim population group and also increasing number of lung cancer affected patient. This clearly indicated that the fatality of this life taking disease can be minimized by controlling the number of active smokers and at the same time making the victim group safe from the smokers by minimizing the direct contact of smokers and non-smokers when smoking is concern. It is expected that the proposed model can be of help for the clinicians and experimentalists who are associated with the research of lung cancer. It may be helpful for the health researchers to make plans and take decision regarding the prevention and/or cure of the lung cancer. It may also help to identify the causes of lung cancer and control them accordingly and thus can contribute to the public health worldwide.

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