

Mathematical Analysis on Physiological Effect of Chronic Obstructive Pulmonary Cancer Due To Habit of Smoking

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Abstract:

Smoking is a major cause of various types of cancer, including lung, oral, throat, bladder, kidney, pancreas, and stomach cancer, this smoking-related disease mainly affects the lungs' alveoli, or air sacs, and airways. The toxic compounds in tobacco smoke affect the respiratory system and cause damage to the airways, leading to a narrowing of the air passages and mucus production. As a result, the lungs become less efficient at transferring oxygen into the bloodstream, making breathing more difficult. In advanced stages, COPD can also affect the heart, leading to complications such as heart failure. While there is currently no cure for COPD, there are treatments that can help to control the symptoms and delay the illness's progression. In order to effectively manage COPD, changes in lifestyle like giving up smoking, avoiding triggers in the environment, and engaging in regular exercise are essential. Medications like bronchodilators, inhaled corticosteroids, and oxygen therapy are commonly prescribed to improve breathing and reduce inflammation. Mathematical modelling is employed to analyse the effects of smoking lead to lungs problems. Runge- Kutta fourth order method is employed to demonstrate the how the normal cells are converted into diseased cells due to smoking leads to respiratory disease like cancer.

Keywords: Cancer, toxic, lung, respiratory, oxygen, bloodstream.

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I. Introduction:

This smoking-related disease mainly affects the lungs' alveoli, or air sacs, and airways. The toxic compounds in tobacco smoke affect the respiratory system and cause damage to the airways, leading to a narrowing of the air passages and mucus production. As a result, the lungs become less efficient at transferring oxygen into the bloodstream, making breathing more difficult. In advanced stages, COPD can also affect the heart, leading to complications such as heart failure. While there is currently no cure for COPD, there are treatments that can help to control the symptoms and delay the illness's progression. In order to effectively manage COPD, changes in lifestyle like giving up smoking, avoiding triggers in the environment, and engaging in regular exercise are essential. Medications like bronchodilators, inhaled corticosteroids, and oxygen therapy are commonly prescribed to improve breathing and reduce inflammation. Smoking is a major cause of various types of cancer, including lung, oral, throat, bladder, kidney, pancreas, and stomach cancer, among others. It is estimated that smoking is responsible for nearly one-third of all cancer deaths in the United States. When tobacco is smoked, thousands of harmful chemicals are released into the body. These chemicals can cause damage to the DNA in cells, leading to the formation of cancerous cells. Smoking also weakens the immune system, making it harder for the body to fight off cancer cells.

Lung cancer is the most common and deadliest form of cancer associated with smoking. The risk of developing lung cancer is directly related to the duration and intensity of smoking. The longer a person smokes and the more cigarettes they consume, the higher their risk of developing lung cancer. Oral and throat cancers are also strongly linked to smoking. The toxic chemicals from tobacco can cause mutations in the cells of the mouth and throat, leading to the formation of cancerous growths. Smokers are also at a higher risk of developing bladder, kidney, pancreas, and stomach cancer, as these organs come into contact with the harmful chemicals through the bloodstream or urine.

Mathematical modeling is highly significant in various fields and has numerous practical applications. Mathematical models provide a means to simulate and analyze real-world phenomena, allowing scientists to conduct virtual experiments and test hypotheses. This enables researchers to explore complex systems and

generate insights that may be difficult or impossible through direct experimentation. Therefore, mathematical modeling is a great tool as it provides a framework for understanding complex systems, predicting future outcomes, solving problems, making informed decisions, optimizing designs, evaluating policies, conducting scientific research, and fostering education.

Over the past years, Steven Plantadosi et al [1] describes the formulation and behavior of a cell cycle model on the previously outlined concepts and the model given a postulational basis, which is not unique, but is simple and appealing, and has a foundation in other work. M. A. J. Chaplain et al [2] presented a theoretical mathematical model for the diffusion of TAF into the surrounding tissue in which an attempt has been made to reflect all of the main events associated with angiogenesis described in the introduction and offer an explanation for the occurrence of anastomosis. Zeljko Bajzer et al [3] examined Winsor's postulate of growth within the confines of the inference that the "power to grow" is quantified by the directly measurable growth fraction, the fraction of proliferating cells in the system. Ashraf Mohamed et al [4] compared real brain tumor-induced deformations observed in serial-time MRI scans of a human subject and 3 canines with surgically transplanted gliomas and Results indicate that the model can reproduce the real deformations with an accuracy that is similar to that of manual placement of landmark points to which the model is compared. Alexandros Roniotis et al [5] studied parameter estimation for treatment term, proliferation rate and initial state. Khairia El-Said El-Nadi et al [6] aimed to measure tumor growth in time and space and the used to understand the behavior of the tumor and the growth models to study the tumor cells under the influence of random disturbances. Eugeny Petrovich Kolpak et. al. [7] developed mathematical model of a growing tumor distinguished from most models proposed for description of neoplasms as individual objects in living organisms – the growth of tumor cells occurs in a functioning organ. Sana Abdulkream Alharbi [8] proposed a new model by considering the main population cells, which had a significant effect on the tumor cell appearance in a tissue. Samuel Ruiz-Arrebola et al [9] analyzed the capabilities of different classical mathematical models to describe the growth of multicellular spheroids simulated with an on-lattice agent-based Monte Carlo model Nuverah Mohsin et al [10] compared three models of tumor volume dynamics and studied the implications of the model assumptions in terms of the effects on rates of change in tumor volume, parameter sensitivity and parameter identifiability.

Formulation:

The present mathematical model of smoking-related cancer is classified into seven specific compartments: smoking individuals as the susceptible class $P(t)$, lately infected individuals as $I_L(t)$, chronically infected individuals without treatment as $I_C(t)$, infected individuals with and without treatment as $T(t)$ and $I(t)$, smoking-related cancer individuals as $C(t)$, and recovered individuals as $R(t)$. We contemplated some parameters in the formation of the model. The disease transmission coefficient from the susceptible $P(t)$ to the lately infected class $I_L(t)$ is β_1 , and from the lately infected $I_L(t)$ to the chronically infected class $I_C(t)$ is β_2 . The total population size is denoted as A . The infected individuals without treatment $I(t)$ to with treatment $T(t)$ transmission rate is defined as δ_1 , and smoker individuals $P(t)$ to cancer patient $C(t)$ transmit rate is δ_2 . For TB-infected patients that transmit from the lately infected $I_L(t)$ to infected with treatment class $T(t)$ is defined as δ_3 , those infected with treatment $T(t)$ that transmit to the smoking-related cancer class $C(t)$ as δ_4 , and those that transmit from the infected with treatment $T(t)$ to recovered class $R(t)$ as δ_5 . The death rate due to smoking is marked as μ . Notations α_1, α_2 and α_3 are assigned for infection growth from $I_L(t)$ to $I_C(t)$ class, from $I_C(t)$ to $I(t)$ class, and from $I(t)$ to $T(t)$ class, respectively. The disease death rate in the $I_L(t)$, $I_C(t)$, and $I(t)$ compartments are γ_1, γ_2 and γ_3 , respectively. By linking the above-mentioned parameters, the mathematical model as a system of differential equations is represented as:

$$\frac{dP}{dt} = A - \frac{\beta_1}{N(t)} \log(P(t)I_L(t) + 1) + \delta_1 T(t) + \delta_1 C(t) - \mu P(t) \quad (1)$$

$$\frac{dI_L}{dt} = \frac{\beta_1}{N(t)} \log(P(t)I_L(t) + 1) - (\alpha_1 + \delta_3 + \gamma_1 + \mu)I_L(t) - \beta_2 I_L(t)I_C(t) \quad (2)$$

$$\frac{dI_C(t)}{dt} = \beta_2 I_L(t)I_C(t) - \alpha_2 I_C(t) - (\gamma_2 + \mu)I_C(t) + \alpha_1 I_L(t) \quad (3)$$

$$\frac{dI(t)}{dt} = \alpha_2 I_C(t) - (\alpha_3 + \gamma_3 - \mu)I(t) \quad (4)$$

$$\frac{dT(t)}{dt} = \delta_3 I_L(t) - \delta_4 T(t) - (\delta_1 + \mu)T(t) + \alpha_3 I(t) - \delta_5 T(t) \quad (5)$$

$$\frac{dC(t)}{dt} = \delta_4 T(t) - \delta_2 C(t) - \mu C(t) \quad (7)$$

$$\frac{dR}{dt} = \delta_5 T(t) - \mu R(t) \quad (8)$$

Mathematical models can be used to determine the probability of developing different types of cancer due to smoking. Researchers can incorporate various factors such as smoking duration, intensity, and exposure to secondhand smoke to estimate individual and population-level cancer risks. We can simulate the natural history and progression of smoking-related cancers with the help of modeling. Researchers can create models that simulate the effects of implementing intervention strategies like tobacco taxes, smoking cessation programs, or smoke free regulations, allowing policy-makers to make evidence based decisions. Models can assist in optimizing treatment strategies for smoking-related cancers. By simulating treatment outcomes and comparing different treatment approaches, researchers can identify the most effective combinations of surgery, chemotherapy, radiation therapy and targeted therapies. This can improve patient outcomes and resource allocation. Mathematical modeling can also be used to evaluate the economic implications of different smoking-related cancer interventions. By considering factors such as healthcare costs, productivity losses, and quality-adjusted life years gained, policymakers can assess the value of various interventions and allocate resources accordingly.

Analysis:

The use of the smoking-related cancer model in research can support the UN's sustainable development goals in several ways. Researchers can contribute valuable insights into the causes and mechanisms of cancer development, which can ultimately lead to improved prevention, early detection, and treatment strategies. This can have a significant impact on good health and well-being, by reducing the burden of cancer worldwide and improving overall public health. Additionally, research on the smoking-related cancer model can also contribute to quality education, by providing valuable training and educational opportunities for researchers and healthcare professionals. By sharing knowledge and expertise gained from studying the smoking cancer model, researchers can help build capacity in cancer research and care, ultimately improving the quality of education and healthcare delivery in this field.

II. Result and Discussions:

We demonstrated a fractional-order smoking-related cancer mathematical model with seven compartments under the duration of 60 days. We utilized the ABC fractional operator as it has non-local and non-singular properties. Further, we acquired a well-known Adams-Bashforth Moulton method which provided a fast convergence to the solution. From Figures 1–4, we see that the behavior of the solution of each class having a stable region. Figures 5 and 6 show that when transmission coefficients β_2 and α_1 change, the lately infected individuals transmit to chronically infected individuals. The transmission rate from infected individuals to cancer due to smoking individuals when infective parameter δ_4 differs. By incorporating data on smoking-related cancer and its treatment effectiveness, we can gain insights into how different factors affect the cancer progression and design strategies for prevention, diagnosis, and treatment. It can help to evaluate the impact of different tobacco control policies on cancer incidence and mortality.

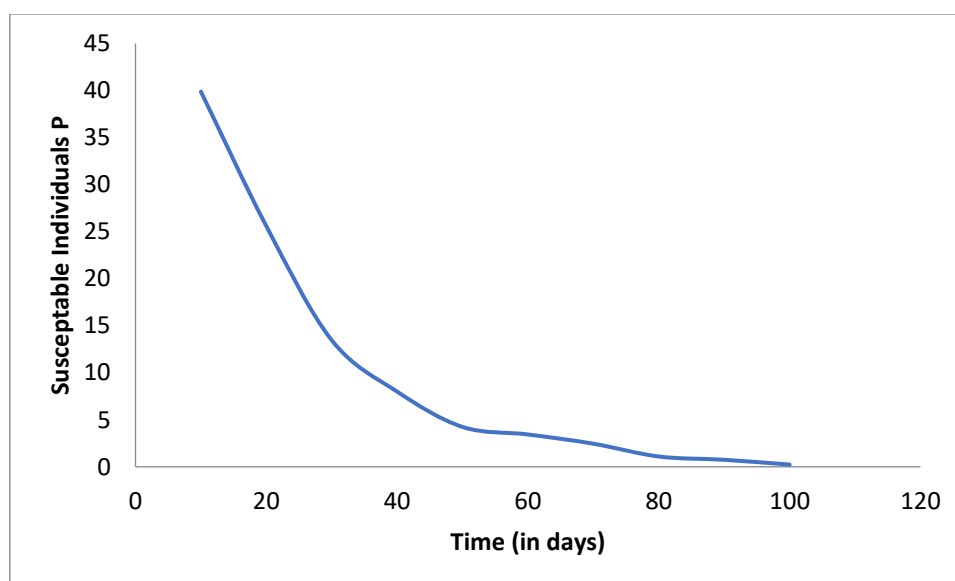


Fig. 1: Behavior of the susceptible class $P(t)$ for certain fractional-order ν vs Time.

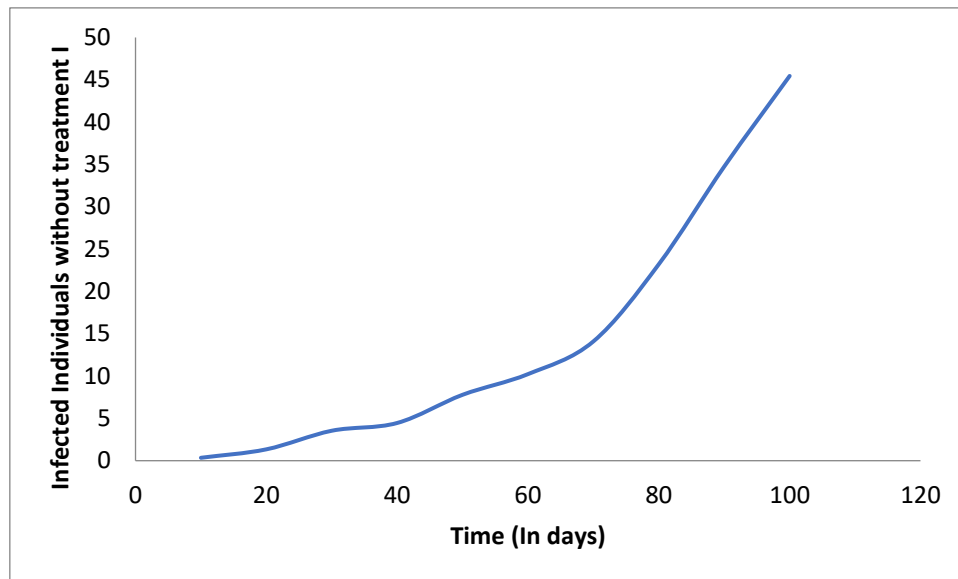


Fig.2: Behaviour of infected individuals without treatment v/s Time

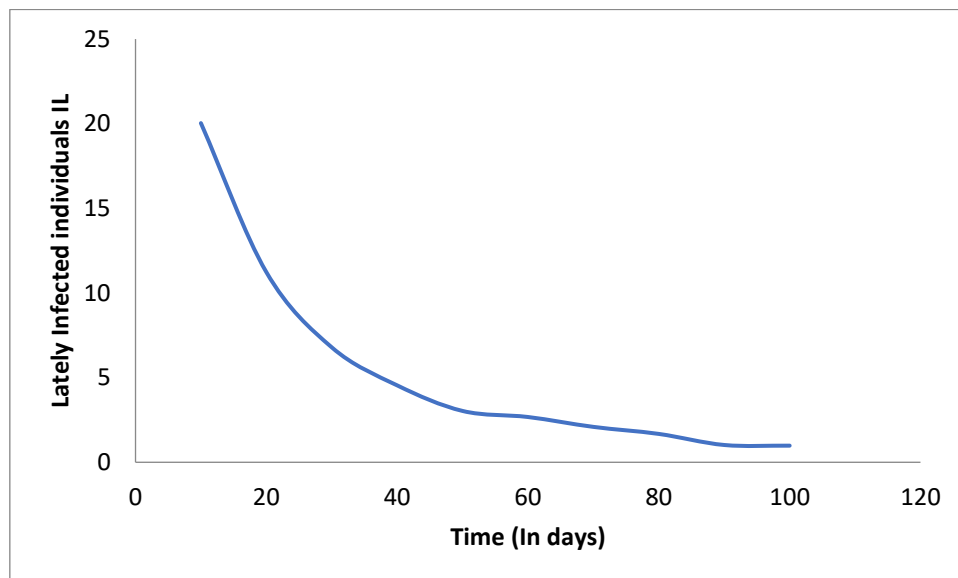


Fig.3: Behaviour of lately infected individuals I_L v/s Time

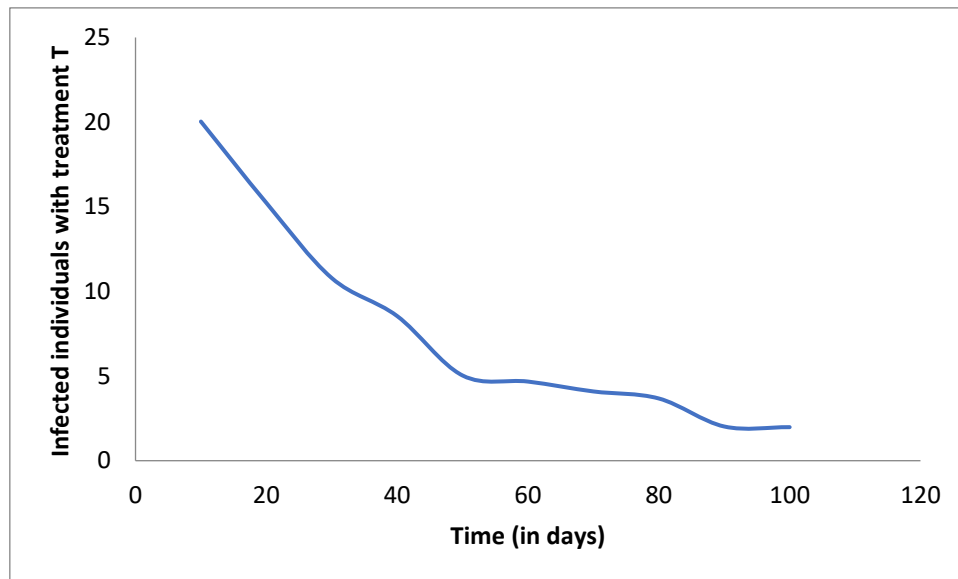


Fig.4: Behaviour of infected individuals with treatment T v/s Time

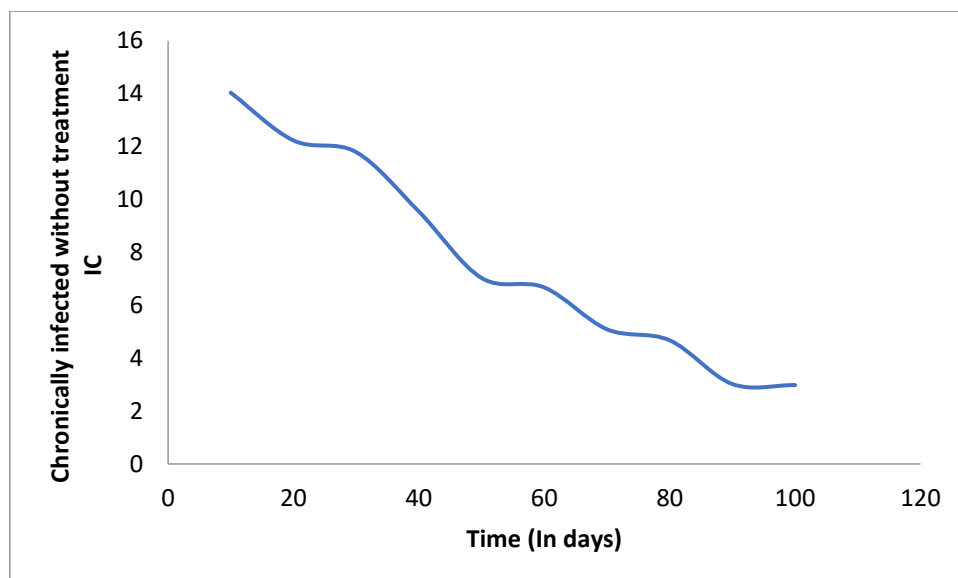


Fig.5: Behaviour of infected individuals without treatment I_C v/s Time

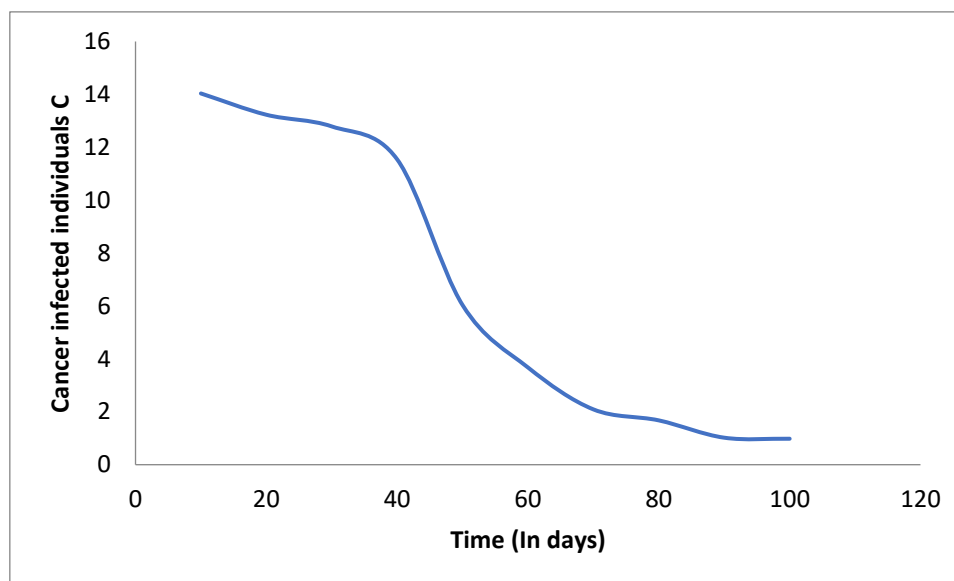


Fig. 6: Behaviour of Cancer infected individuals C v/s Time

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