Chapter 5

Quantifying Cancer Risk from Smoking: A Fractional Mathematical Approach

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5.1 Introduction

The smoking disease, also known as chronic obstructive pulmonary disease (COPD), is a group of progressive lung diseases that cause breathing difficulties [62]. It is primarily caused by long-term exposure to cigarette smoke, although exposure to second hand smoke, air pollution, and certain occupational fumes can also contribute to its development. Common symptoms of COPD include chest tightness, wheezing, shortness of breath, and persistent cough. Over time, the disease progressively worsens, resulting in a reduced ability to carry out physical activities and everyday tasks. Individuals with COPD may also experience frequent respiratory infections, fatigue, unintended weight loss, and swelling in the ankles, feet, or legs [124].

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 [142]. 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 [8]. 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 [67].

Smoking is a major cause of various types of cancer, including lung, oral, throat, bladder, kidney, pancreas, and stomach cancer, among others [104]. 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 [150].

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 [116]. 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 [95].

Quitting smoking significantly decreases the risk of developing cancer. Although it can be challenging, quitting smoking at any age can provide immediate and long-term health benefits. Additionally, early detection through cancer screening programs and regular medical check-ups is crucial for detecting and treating cancer at an early stage, improving the chances of successful treatment and survival [105, 141].

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 second hand 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 modelling [130, 131]. 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 [60].

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 [20]. This can lead to improved patient outcomes and resource allocation. Mathematical modelling 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 for money of various interventions and allocate resources accordingly [65].

The chapter is framed as follow: Section 5.2 includes the fractional smoking cancer model, its diagram, and detailing of the parameters. Section 5.3 covers the well defined definitions and theorems which we have used in this work. Qualitative and stability analysis are discussed in sections 5.4 and 5.5, respectively. Numerical computation and its behavior in form of figures are displayed in section 5.6. Result's valedictory and graphical interpretation are discussed in section 5.7.

5.2 Model formation

The present mathematical model of smoking cancer classified in seven specific compartments: Smoker individuals as 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 cancer individuals as $C(t)$, and Recovered individuals as $R(t)$.

We contemplated some parameters in the formation of model. The disease transmission coefficient from susceptible $P(t)$ to lately infected class $I_L(t)$ is β_1, and from lately infected $I_L(t)$ to chronically infected class $I_C(t)$ is β_2. The total population size is denoted as N. The infected individuals without treatment $I(t)$ to with treatment $T(t)$ transmit rate is defined as δ_1, and smoker individuals $P(t)$ to cancer patient $C(t)$ transmit rate is δ_2. For TB infective patient that transmit from lately infected $I_L(t)$ to infected with treatment class $T(t)$ as δ_3, infected with treatment $T(t)$ to smoking cancer class $C(t)$ as δ_4, and infected with treatment $T(t)$ to recovered class $R(t)$ as δ_5. Death rate due to smoke is marked as μ.

Notation α_1, α_2, and α_3 are assigned for infection grow 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 $I_L(t), I_C(t)$, and $I(t)$ compartments are γ_1, γ_2, and γ_3, respectively.

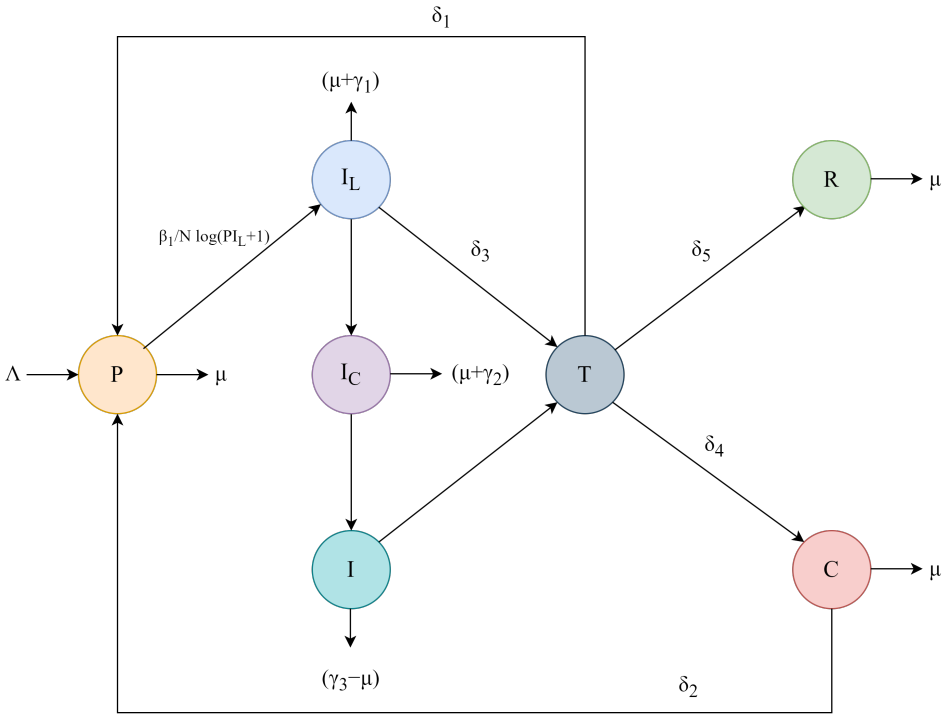

Figure 5.1: Diagram of the smoking cancer model.

By the linking above mentioned parameter, the mathematical model as a system of differential equations is represented as:

$$
\begin{aligned}
\frac{dP(t)}{dt} =& \Lambda - \frac{\beta_1}{N(t)} \log\left(P(t)I_L(t)+1\right) + \delta_1 T(t) + \delta_2 C(t) - \mu P(t),\\
\frac{dI_L(t)}{dt} =& \frac{\beta_1}{N(t)} \log\left(P(t)I_L(t)+1\right) - \left(\alpha_1 + \delta_3 + \gamma_1 + \mu\right) I_L(t) - \beta_2 I_L(t) I_C(t),\\
\frac{dI_C(t)}{dt} =& \beta_2 I_L(t) I_C(t) - \alpha_2 I_C(t) - \left(\gamma_2 + \mu\right) I_C(t) + \alpha_1 I_L(t),\\
\frac{dI(t)}{dt} =& \alpha_2 I_C(t) - \left(\alpha_3 + \gamma_3 - \mu\right) I(t),\\
\frac{dT(t)}{dt} =& \delta_3 I_L(t) - \delta_4 T(t) - \left(\delta_1 + \mu\right) T(t) + \alpha_3 I(t) - \delta_5 T(t),\\
\frac{dC(t)}{dt} =& \delta_4 T(t) - \delta_2 C(t) - \mu C(t),\\
\frac{dR(t)}{dt} =& \delta_5 T(t) - \mu R(t),
\end{aligned}
\tag{5.1}
$$

with the initial conditions $P(0) = P_0$, $I_L(0) = I_{L_0}$, $I_C(0) = I_{C_0}$, $I(0) = I_0$, $T(0) = T_0$, $C(0) = C_0$, $R(0) = R_0$.

Here, the total population by adding all the compartments is defined as $N(t) = P(t) + I_L(t) + I_C(t) + I(t) + T(t) + C(t) + R(t)$.

The proposed smoking cancer model by implementing the ABC fractional derivative [14] is as follows:

$$\begin{aligned}
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}P(t) =&\Lambda - \frac{\beta_1}{N(t)}log(P(t)I_L(t)+1) + \delta_1 T(t) + \delta_2 C(t) - \mu P(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}I_L(t) =&\frac{\beta_1}{N(t)}log(P(t)I_L(t)+1) - \beta_2 I_L(t)I_C(t) - (\alpha_1+\delta_3+\gamma_1+\mu)I_L(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}I_C(t) =&\beta_2 I_L(t)I_C(t) - \alpha_2 I_C(t) - (\gamma_2+\mu)I_C(t) + \alpha_1 I_L(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}I(t) =&\alpha_2 I_C(t) - (\alpha_3+\gamma_3-\mu)I(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}T(t) =&\delta_3 I_L(t) - \delta_4 T(t) - (\delta_1+\mu)T(t) + \alpha_3 I(t) - \delta_5 T(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}C(t) =&\delta_4 T(t) - \delta_2 C(t) - \mu C(t),\\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}R(t) =&\delta_5 T(t) - \mu R(t).
\end{aligned} \tag{5.2}$$

5.3 Preliminaries

Definition 5.3.1. *[14] Let $G \in \mathbb{H}^1(a,b), a > b$, then the ABC fractional derivative is given as*

$${}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}G(t) = \frac{\mathbb{N}(\alpha)}{1-\alpha}\int_a^t E_\alpha\left[-\frac{\alpha(\mathsf{t}-\delta)^\alpha}{1-\alpha}\right]G'(\delta)d\delta. \tag{5.3}$$

Definition 5.3.2. *[14] The ABC fractional integral of order α is defined as:*

$${}^{\mathsf{ABC}}_{0}I^{\alpha}_{\mathsf{t}}G(t) = \frac{1-\alpha}{\mathbb{N}(\alpha)}G(t) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t G(\delta)(t-\delta)^{\alpha-1}d\delta. \tag{5.4}$$

Lemma 5.3.1. *[35] The solution of any Fractional differential equation as*

$${}^{\mathsf{ABC}}_{0}D^{\alpha}_{t}\psi(t) = f(t),\ f(t) \in C\left([0,T)\right),\ 0 < \alpha < 1, \tag{5.5}$$

with $\psi(0) = \psi_0$, *is given by*

$$\psi(t) = \psi_0 + \frac{(1-\alpha)}{\mathbb{N}(\alpha)} f(t) + \frac{\alpha}{\Gamma(\alpha)\mathbb{N}(\alpha)} \int_0^t (t-u)^{\alpha-1} f(u) du. \tag{5.6}$$

5.4 Existence and Uniqueness

The existence and uniqueness of the considered model (5.2) will be covered in this section. The considered model (5.2) can be rewritten as follows:

$$\begin{aligned}
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} P(t) &= g_1(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} I_L(t) &= g_2(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} I_C(t) &= g_3(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} I(t) &= g_4(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} T(t) &= g_5(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} C(t) &= g_6(t, P, I_L, I_C, I, T, C, R), \\
{}^{\mathsf{ABC}}_{0}D^{\alpha}_{t} R(t) &= g_7(t, P, I_L, I_C, I, T, C, R),
\end{aligned} \tag{5.7}$$

where,

$$\begin{aligned}
g_1(t, P, I_L, I_C, I, T, C, R) &= \Lambda - \frac{\beta_1}{N} log(PI_L + 1) + \delta_1 T + \delta_2 C - \mu P, \\
g_2(t, P, I_L, I_C, I, T, C, R) &= \frac{\beta_1}{N} log(PI_L + 1) - \beta_2 I_L I_C - (\alpha_1 + \delta_3 + \gamma_1 + \mu) I_L, \\
g_3(t, P, I_L, I_C, I, T, C, R) &= \beta_2 I_L I_C - \alpha_2 I_C - (\gamma_2 + \mu) I_C + \alpha_1 I_L, \\
g_4(t, P, I_L, I_C, I, T, C, R) &= \alpha_2 I_C - (\alpha_3 + \gamma_3 - \mu) I, \\
g_5(t, P, I_L, I_C, I, T, C, R) &= \delta_3 I_L - \delta_4 T - (\delta_1 + \mu) T + \alpha_3 I - \delta_5 T, \\
g_6(t, P, I_L, I_C, I, T, C, R) &= \delta_4 T - \delta_2 C - \mu C, \\
g_7(t, P, I_L, I_C, I, T, C, R) &= \delta_5 T - \mu R.
\end{aligned} \tag{5.8}$$

For the purpose of simplicity, we refer to the suggested model (5.7) as follows:

$$\begin{aligned} {}_{0}^{\mathsf{ABC}}D_t^{\alpha}\mathbb{G}(t) &= \Delta\left(t, \mathbb{G}(t)\right), \\ \mathbb{G}(0) &= \mathbb{G}_0 \geq 0, \end{aligned} \tag{5.9}$$

where,

$$\begin{aligned} \mathbb{G}(t) &= \left(P(t), I_L(t), I_C(t), I(t), T(t), C(t), R(t)\right)^T, \\ \mathbb{G}_0 &= \left(P_0, I_{L_0}, I_{C_0}, I_0, T_0, C_0, R_0\right)^T, \\ \Delta(t, \mathbb{G}(t)) &= \left(g_1, g_2, g_3, g_4, g_5, g_6, g_7\right)^T. \end{aligned}$$

Applying fractional ABC integral (5.4) on (5.9), we get

$$\mathbb{G}(t) = \mathbb{G}_0 + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\Delta(t, \mathbb{G}(t)) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\Delta(\delta, \mathbb{G}(\delta))d\delta. \tag{5.10}$$

Utilizing $\chi = [0, T]$ as $\Omega = \mathsf{C}(\chi, \mathbb{R}^7)$ under the norm specified as follows:

$$\|\mathbb{G}\| = \sup_{t\in\chi}\left\{\mathbb{G}(t) : \mathbb{G} \in \Omega\right\}.$$

Assume that the function $\Delta\left(t, \mathbb{G}(t)\right)$ satisfies the following two conditions for each $\mathbb{G} \in \Omega$ and $t \in [0, T]$.

- $\exists$ constants μ_Δ and ν_Δ such that, $|\Delta(t, \mathbb{G}(t))| \leq \mu_\Delta |\mathbb{G}| + \nu_\Delta$.
- $\exists$ constant $\lambda_\Delta > 0$ such that. $|\Delta(t, \mathbb{G}_1(t)) - \Delta(t, \mathbb{G}_2(t))| \leq \lambda_\Delta |\mathbb{G}_1 - \mathbb{G}_2|$.

Now we define the operators Λ_1 and Λ_2 (with $\Lambda_1 + \Lambda_2 = \Omega$.) such that

$$\begin{aligned} \Lambda_1\mathbb{G} &= \mathbb{G}_0 + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\Delta(t, \mathbb{G}(t)), \\ \Lambda_2\mathbb{G} &= \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\Delta(\delta, \mathbb{G}(\delta))d\delta, \end{aligned}$$

Theorem 5.4.1. *Suppose that the above mentioned the Lipschitz and growth conditions are hold. If the following are true, then there is at least one solution for* (5.10)*:*

1. $\dfrac{(1-\alpha)}{\mathbb{N}(\alpha)}\lambda_{\Delta} < 1.$
2. $\Psi_1 = \left[\dfrac{(1-\alpha)}{\mathbb{N}(\alpha)} + \dfrac{T^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\nu_{\Delta} < 1.$
3. $\Psi_2 = \left[\dfrac{(1-\alpha)}{\mathbb{N}(\alpha)} + \dfrac{T^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\mu_{\Delta} < 1.$

Proof. Assume a closed convex set $B_{\tau} = \{\mathbb{G} \in \Omega : \|\mathbb{G}\| \leq \tau\}$. In order to establish that $\Lambda_1\mathbb{G}_1 + \Lambda_2\mathbb{G}_2 \in B_{\tau}$ for any $\mathbb{G}_1, \mathbb{G}_2 \in B_{\tau}$. Using growth condition, we get

$$\begin{aligned}
\|\Lambda_1\mathbb{G}_1 + \Lambda_2\mathbb{G}_2\| &\leq \sup_{t\in\chi}\left\{|\mathbb{G}_0| + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}|\Delta(t,\mathbb{G}(t))| + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}|\Delta(\delta,\mathbb{G}(\delta))|d\delta\right\} \\
&\leq \left\{|\mathbb{G}_0| + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}(\mu_{\Delta}\|\mathbb{G}\| + \nu_{\Delta}) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}(\mu_{\Delta}\|\mathbb{G}\| + \nu_{\Delta})d\delta\right\} \\
&= |\mathbb{G}_0| + \left\{\frac{(1-\alpha)}{\mathbb{N}(\alpha)} + \frac{T^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right\}\nu_{\Delta} + \left\{\frac{(1-\alpha)}{\mathbb{N}(\alpha)} + \frac{T^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right\}\mu_{\Delta}\tau \\
&= \Psi_1 + \Psi_2\tau \leq \tau.
\end{aligned}$$

This shows that, $\Lambda_1\mathbb{G}_1 + \Lambda_2\mathbb{G}_2 \in B_{\tau}$.

Next to show that Λ_1 is contraction. For any $\mathbb{G}_1, \mathbb{G}_2 \in B_{\tau}$, using Lipschitz condition, we get :

$$\begin{aligned}
\|\Lambda_1\mathbb{G}_1 + \Lambda_2\mathbb{G}_1\| &= \sup_{t\in[0,\mathsf{T}]}\frac{(1-\alpha)}{\mathbb{N}(\alpha)}\left|\Delta(t,\mathbb{G}_1(t)) - \Delta(t,\mathbb{G}_2(t))\right| \\
&\leq \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\lambda_{\Delta}\sup_{t\in[0,\mathsf{T}]}|\mathbb{G}_1 - \mathbb{G}_2| \\
&\leq \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\lambda_{\Delta}\|\mathbb{G}_1 - \mathbb{G}_2\|,
\end{aligned}$$

where, $\dfrac{(1-\alpha)}{\mathbb{N}(\alpha)}\lambda_{\Delta} < 1$. Thus, Λ_1 is a contraction.

Let us now demonstrate the relative compactness of Λ_2. Considering $\mathbb{G} \in B_\tau$ for this

$$\begin{aligned}
\|\Lambda_2\mathbb{G}\| &\leq \sup_{t\in[0,\mathsf{T}]} \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_0^t (t-\delta)^{\alpha-1} |\Delta(\delta, \mathbb{G}(\delta))| d\delta \\
&\leq \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_0^t (t-\delta)^{\alpha-1} \sup_{t\in[0,\mathsf{T}]} [\mu_\Delta |\mathbb{G}| + \nu_\Delta] d\delta \\
&\leq \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_0^t (t-\delta)^{\alpha-1} [\mu_\Delta \|\mathbb{G}\| + \nu_\Delta] d\delta \\
&\leq \frac{T^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} [\mu_\Delta \tau + \nu_\Delta].
\end{aligned}$$

Therefore, on B_τ, Λ_2 has a uniformly bounded. Lastly, We demonstrate the equicontinuous nature of Λ_2. Let t_1, $t_2 \in [0, T]$ such that $t_1 < t_2$. Let $\mathbb{G} \in B_\tau$.
Following that

$$\begin{aligned}
\|\Lambda_2\mathbb{G}(t_1) - \Lambda_2\mathbb{G}(t_2)\| &\leq \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_{t_1}^{t_2} (t_2-\delta)^{\alpha-1} |\Delta(\delta, \mathbb{G}(\delta))| d\delta \\
&\quad + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_0^{t_2} [(t_1-\delta)^{\alpha-1} - (t_2-\delta)^{\alpha-1}] |\Delta(\delta, \mathbb{G}(\delta))| d\delta \\
&\leq \frac{2[\mu_\Delta \tau + \nu_\Delta]}{\mathbb{N}(\alpha)\Gamma(\alpha)} [(t_2-t_1)^\alpha].
\end{aligned}$$

Thus, $\|\Lambda_2\mathbb{G}(t_1) - \Lambda_2\mathbb{G}(t_2)\| \to 0$ as $t_2 \to t_1$. The operator Λ_2 is equicontinuous because it is relatively compact, according to the Arzela-Ascoli theorem. Hence, there is at least one solution for (5.10). The considered model has at least one solution since the proposed model is equivalent to (5.10). □

Theorem 5.4.2. *Assuming that the Lipschitz condition is satisfied, then the unique solution of equation* (5.10) *can be found if*

$$\left\{ \frac{(1-\alpha)}{\mathbb{N}(\alpha)} + \frac{T^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \right\} \lambda_\Delta < 1.$$

Proof. Considering $t \in [0, T]$ and for any $\mathbb{G}$, $\mathbb{G}^* \in \Omega$, we have

$$\begin{aligned}\|\Lambda\mathbb{G}(t) - \Lambda\mathbb{G}^*(t)\| &\leq \max_{t\in[0,\mathsf{T}]} \frac{(1-\alpha)}{\mathbb{N}(\alpha)} \left|\Delta(t, \mathbb{G}(t)) - \Delta(t, \mathbb{G}^*(t))\right| \\ &\quad + \max_{t\in[0,\mathsf{T}]} \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)} \int_0^t (t-\delta)^{\alpha-1} \left|\Delta(t, \mathbb{G}(t)) - \Delta(t, \mathbb{G}^*(t))\right| d\delta \\ &\leq \left\{\frac{(1-\alpha)}{\mathbb{N}(\alpha)} + \frac{T^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right\} \lambda_\Delta \left\|\mathbb{G} - \mathbb{G}^*\right\|.\end{aligned}$$

The operator Λ is contraction as per the hypothesis $\left\{\frac{(1-\alpha)}{\mathbb{N}(\alpha)} + \frac{T^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right\} \lambda_\Delta < 1$. The solution to (5.10) is unique according to the Banach contraction theorem. As a result, the solution for the suggested model (5.2) is unique. □

5.5 Stability analysis

5.5.1 Local stability

We discuss the equilibrium points of presented model (5.1). Here, we consider the two stage of the population.

1. Disease-free equilibrium point E^0:

   At this stage, there is no contamination due to smoking habit in class. Hence, we set every infection class to zero. Therefore, Disease-free equilibrium point E^0 can be defined as:

   $$E^0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

2. Endemic equilibrium point E^{end}:

   To obtain the value of endemic stage E^{end}, we will equate system of equation (5.1) with zero. By solving the system of linear equations, we achieve the endemic equilibrium

point E^{end} as:

$$E^{end} = \left(P^{end}, I_L^{end}, I_C^{end}, I^{end}, T^{end}, C^{end}, R^{end}\right),$$

where,

$$P^{end} = \frac{2\beta_1 (1 - PI_L)}{\mu N (1 + PI_L)} + \frac{R (\delta_1\delta_2 + \delta_1\mu + \delta_2)}{\delta_5 (\delta_2 + \mu)},$$

$$I_L^{end} = \frac{2\beta_1 (PI_C (\mu + \alpha_2 + \gamma_2) - \alpha_1 - \beta_2 I_C)}{N (PI_C (\mu + \alpha_2 + \gamma_2) + \alpha_2 + \beta_2 I_C) (\mu + \alpha_1 + \gamma_1 + \delta_3 + \beta_2 I_C)},$$

$$I_C^{end} = \frac{1}{\beta_2} \left(\frac{2\beta_1 (PI_C (\mu + \alpha_2 + \gamma_2) - \alpha_1 - \beta_2 I_C)}{NI_L (PI_C (\mu + \alpha_2 + \gamma_2) + \alpha_2 + \beta_2 I_C)} - (\mu + \alpha_1 + \gamma_1 + \delta_3) \right),$$

$$I^{end} = \frac{\alpha_1\alpha_2 I_L + \alpha_2\beta_2 I_L I_C}{(\mu + \alpha_2 + \gamma_2) (-\mu + \alpha_3 + \gamma_3)},$$

$$T^{end} = \frac{I_L (\alpha_1\alpha_2\alpha_3 + \alpha_2\alpha_3\beta_2 I_C + \delta_3)}{(2\delta_4 + \delta_5 - \mu) (\mu + \alpha_2 + \gamma_2) (-\mu + \alpha_3 + \gamma_3)},$$

$$C^{end} = \frac{I_L (\alpha_1\alpha_2\alpha_3 + \alpha_2\alpha_3\beta_2 I_C + \delta_3)}{(\mu + \delta_2) (2\delta_4 + \delta_5 - \mu) (\mu + \alpha_2 + \gamma_2) (-\mu + \alpha_3 + \gamma_3)},$$

$$R^{end} = \frac{I_L (\delta_3\delta_5 + \alpha_1\alpha_2\alpha_3\delta_5 + \alpha_2\alpha_3\beta_2\delta_5 I_C)}{\mu(2\delta_4 + \delta_5 - \mu) (\mu + \alpha_2 + \gamma_2) (-\mu + \alpha_3 + \gamma_3)}.$$

Reproduction number R_0

To calculate R_0 we employ the next generation matrix method. Let consider the following sub-system by involving the infective compartments as below:

$$\begin{aligned}
\frac{dI_L(t)}{dt} &= \frac{\beta_1}{N(t)} \log\left(P(t)I_L(t)+1\right) - \beta_2 I_L(t) I_C(t) - \left(\alpha_1+\delta_3+\gamma_1+\mu\right) I_L(t),\\
\frac{dI_C(t)}{dt} &= \beta_2 I_L(t) I_C(t) - \alpha_2 I_C(t) - \left(\gamma_2+\mu\right) I_C(t) + \alpha_1 I_L(t),\\
\frac{dI(t)}{dt} &= \alpha_2 I_C(t) - \left(\alpha_3+\gamma_3-\mu\right) I(t),\\
\frac{dC(t)}{dt} &= \delta_4 T(t) - \delta_2 C(t) - \mu C(t).
\end{aligned} \tag{5.11}$$

Now we find the eigenvalues for the matrix FV^{-1}, to evaluate R_0. where matrix F and V for the system (5.11) can be defined as

$$F = \begin{bmatrix}
-\beta_2 I_C + \frac{\beta_1 P}{N(1+PI_L)} & -\beta_2 I_L & 0 & 0\\
\beta_2 I_C & \beta_2 I_L & 0 & 0\\
0 & 0 & 0 & 0\\
0 & 0 & 0 & 0
\end{bmatrix},$$

$$V = \begin{bmatrix}
\mu+\alpha_1+\gamma_1+\delta_3 & 0 & 0 & 0\\
-\alpha_1 & \mu+\alpha_2+\gamma_2 & 0 & 0\\
0 & -\alpha_2 & -\mu+\alpha_3+\gamma_3 & 0\\
0 & 0 & 0 & \mu+\delta_2
\end{bmatrix}.$$

Next, we find the inverse of matrix V and with the simple matrix multiplication,

which leads to

$$FV^{-1} = \begin{bmatrix} -I_L\beta_2 + \frac{\beta_1 P}{N(1+PI_L)} - \frac{\beta_2 I_L}{(\mu+\alpha_1+\gamma_1+\delta_3)} & -\frac{\beta_2 I_L}{\alpha_1} & 0 & 0 \\ \beta_2 I_C + \frac{\beta_2 I_L}{(\mu+\alpha_1+\gamma_1+\delta_3)} & -\frac{\beta_2 I_L}{\alpha_1} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{5.12}$$

Further, the dominant eigenvalues can be find by $|FV^{-1} - \lambda I| = 0$. And basic reproduction number R_0 can be defined as

$$R_0 = \frac{m_1 + m_2 + m_3}{2\alpha_1 c_1 N}, \tag{5.13}$$

where,

$$m_1 = \alpha_1\beta_1 c_1,$$
$$m_2 = N\beta_2\left(\alpha_1 c_1 I_C + \alpha_1 I_L + c_1 I_L\right),$$
$$m_3 = \sqrt{\alpha_1^2\beta_1^2 c_1^2 + 2\alpha_1 c_1 N\left(\alpha_1 c_1 I_C + \beta_1\beta_2 I_L\left(\alpha_1 - c_1\right) + N^2\left(\alpha_1 c_1 I_C + \beta_2^2 I_L(\alpha_1 + c_1)^2\right)\right)}.$$

Theorem 5.5.1. *Disease-free stage of smoking cancer model* (5.1) *is locally asymptotically stable if basic reproduction number* $R_0 < 0$.

Proof. Available in [125]. □

Theorem 5.5.2. *Endemic stage of smoking cancer model* (5.1) *is locally asymptotically stable if endemic Lyaponove function* $L < 0$ *and basic reproduction number* $R_0 > 0$.

Proof. Available in [125]. □

5.5.2 Global stability

Using the concept of nonlinear functional analysis, we address the Ulam-Hyers (U-H) stability of the suggested fractional model (5.2) in this section.

Definition 5.5.1. *If the following property holds true and* $\exists\ \omega > 0$*, then the proposed system* (5.2) *is U-H stable. For any* $\overline{\mathbb{G}} \in \Omega$ *and* $\epsilon > 0$*. If*

$$\left|{}_0^{\mathsf{ABC}}D_t^{\alpha}\overline{\mathbb{G}}(t) - \Delta\left(t, \overline{\mathbb{G}}(t)\right)\right| \leq \epsilon, \tag{5.14}$$

then $\exists\ \mathbb{G} \in \Omega$ *satisfying the system* (5.2) *with the initial condition* $\mathbb{G}(0) = \overline{\mathbb{G}}(0) = \overline{\mathbb{G}}_0$*, such that* $\left\|\overline{\mathbb{G}} - \mathbb{G}\right\| \leq \omega\epsilon$.
where,

$$\begin{aligned}
\overline{\mathbb{G}}(t) &= \left(\overline{P}(t), \overline{I}_L(t), \overline{I}_C(t), \overline{I}(t), \overline{T}(t), \overline{C}(t), \overline{R}(t),\right)^T, \\
\mathbb{G}_0 &= \left(\overline{P}_0(t), \overline{I}_{L_0}(t), \overline{I}_{C_0}(t), \overline{I}_0(t), \overline{T}_0(t), \overline{C}_0(t), \overline{R}_0(t),\right)^T, \\
\Delta(t, \overline{\mathbb{G}}(t)) &= \left(\overline{g}_1, \overline{g}_2, \overline{g}_3, \overline{g}_4, \overline{g}_5, \overline{g}_6, \overline{g}_7\right)^T, \\
\epsilon &= max(\epsilon_j)^T; j = 1, 2, 3, 4, 5, \\
\omega &= max(\omega_j)^T; j = 1, 2, 3, 4, 5.
\end{aligned}$$

Remark 5.5.1. *Consider a small perturbation* $\rho \in \mathsf{C}[0, \mathsf{T}]$ *such that* $\rho(0) = 0$ *along with the following property :* $|\rho(t)| \leq \overline{\epsilon}$*, for* $t \in [0, \mathsf{T}]$ *and* $\overline{\epsilon} > 0$.

Lemma 5.5.1. *The solution* $\overline{\mathbb{G}}_\rho(t)$ *of the perturbed system*

$${}_0^{\mathsf{ABC}}D_t^{\alpha}\overline{\mathbb{G}}(t) = \Delta\left(t, \overline{\mathbb{G}}(t)\right) + \rho(t), \quad \overline{\mathbb{G}}(0) = \overline{\mathbb{G}}_0, \tag{5.15}$$

satisfies the relation: $\left|\overline{\mathbb{G}}_\rho(t) - \overline{\mathbb{G}}(t)\right| \leq \left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\overline{\epsilon}$,

where, $\rho(t) = (\rho_1(t), \rho_2(t), \rho_3(t), \rho_4(t), \rho_5(t))^T$.

Proof. Applying fractional integral (5.4) on (5.15), we get

$$\begin{aligned}\overline{\mathbb{G}}_\rho(t) = \overline{\mathbb{G}}_0 + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\Delta\left(t, \overline{\mathbb{G}}(t)\right) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\Delta\left(\delta, \overline{\mathbb{G}}(\delta)\right)d\delta \\ + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\rho(t) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\rho(\delta)d\delta.\end{aligned} \tag{5.16}$$

Also,

$$\overline{\mathbb{G}}(t) = \overline{\mathbb{G}}_0 + \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\Delta\left(t, \overline{\mathbb{G}}(t)\right) + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\Delta\left(\delta, \overline{\mathbb{G}}(\delta)\right)d\delta. \tag{5.17}$$

Using Remark 5.5.1,

$$\left|\overline{\mathbb{G}}_\rho(t) - \overline{\mathbb{G}}(t)\right| \leq \frac{(1-\alpha)}{\mathbb{N}(\alpha)}\left|\rho(t)\right| + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\left|\rho(\delta)\right|d\delta,$$

$$\leq \left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\bar{\epsilon}.$$

This complete the proof. □

Theorem 5.5.3. *The proposed fractional system* (5.2) *is U-H stable if* $\left\|\overline{\mathbb{G}}(t) - \mathbb{G}(t)\right\| \leq \omega\ \bar{\epsilon}$.

Proof. Since $\mathbb{G}$ is a unique solution of the system (5.15) and $\overline{\mathbb{G}}$ is the solution of (5.14), then

$$\begin{aligned}\left|\overline{\mathbb{G}}(t) - \mathbb{G}(t)\right| &\leq \left|\overline{\mathbb{G}}_\rho(t) - \overline{\mathbb{G}}(t)\right| + \left|\overline{\mathbb{G}}_\rho(t) - \mathbb{G}(t)\right| \\ &\leq 2\left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\bar{\epsilon} + \frac{1-\alpha}{\mathbb{N}(\alpha)}\left|\Delta(t, \overline{\mathbb{G}}(t)) - \Delta(t, \mathbb{G}(t))\right| \\ &\quad + \frac{\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\int_0^t (t-\delta)^{\alpha-1}\left|\Delta(\delta, \overline{\mathbb{G}}(\delta)) - \Delta(\delta, \mathbb{G}(\delta))\right|d\delta \\ &\leq 2\left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\bar{\epsilon} + \left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^\alpha}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\lambda_\Delta\left|\overline{\mathbb{G}} - \mathbb{G}\right|.\end{aligned}$$

which implies that

$$\left|\overline{\mathbb{G}}(t) - \mathbb{G}(t)\right| \leq \frac{2\xi\overline{\epsilon}}{1-\eta},$$

where

$$\xi = 2\left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right],$$

and,

$$\eta = \left[\frac{(1-\alpha)\Gamma(\alpha) + \mathsf{T}^{\alpha}}{\mathbb{N}(\alpha)\Gamma(\alpha)}\right]\lambda_{\Delta}.$$

For

$$\omega = \frac{2\xi}{1-\eta},$$

then

$$\left|\overline{\mathbb{G}}(t) - \mathbb{G}(t)\right| \leq \omega\overline{\epsilon}.$$

Hence, considered fractional system (5.2) is U-H stable. □

5.6 Numerical results and Discussion

In this work, we obtain the solution of fractional-order smoking cancer model (5.2) using Adams-Bashforth-Moulton method [33]. We take some specific values of necessary parameters $\Lambda = 0.002, \beta_1 = 0.00012, \beta_2 = 0.002, \delta_1 = 0.0032, \delta_2 = 0.00233, \delta_3 = 0.00462, \delta_4 = 0.00023, \delta_5 = 0.000123, \mu = 0.05, \alpha_1 = 0.001, \alpha_2 = 0.0045, \alpha_3 = 0.000123, \gamma_1 = 0.007, \gamma_2 = 0.00023, \gamma_3 = 0.00034$ with initial values $P_0 = 40, I_{L_0} = 20, I_{C_0} = 15, I_0 = 10, T_0 = 10, C_0 = 5, R_0 = 0$. Figures 5.2 to 5.8 are represent the behavior of the each compartment when we utilized Atangana-Baleanu fractional derivative in Caputo sense with some fractional orders between 0.9 to 1. We plot the 3D behavior of the transmission rate from $I_L(t)$ class to $I_C(t)$ class when β_2 and α_1 are between 0 to 0.004 in figures 5.9 and 5.10, respectively. When infective parameter δ_4 is between 0.0001 to 0.0004, rate of transmission from $I(t)$ to $C(t)$

class is demonstrated as figure 5.11. When another infective parameter δ_5 is between 0 to 0.0004, rate of transmission form $I(t)$ to $R(t)$ class is obtained as figure 5.12.

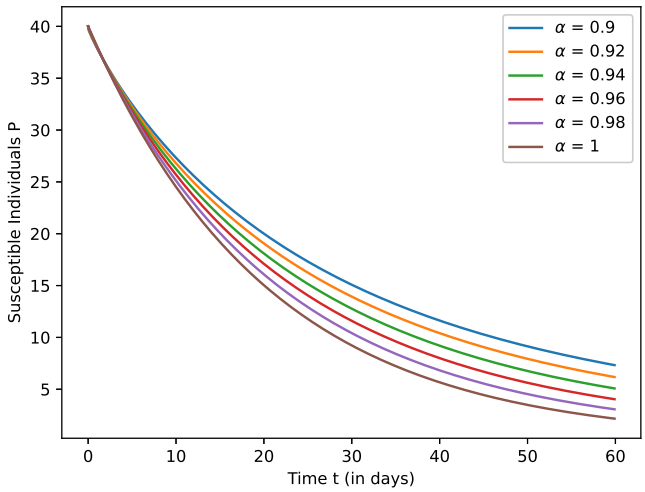

Figure 5.2: Behavior of the susceptible class $P(t)$ for certain fractional-order between 0.9 to 1.

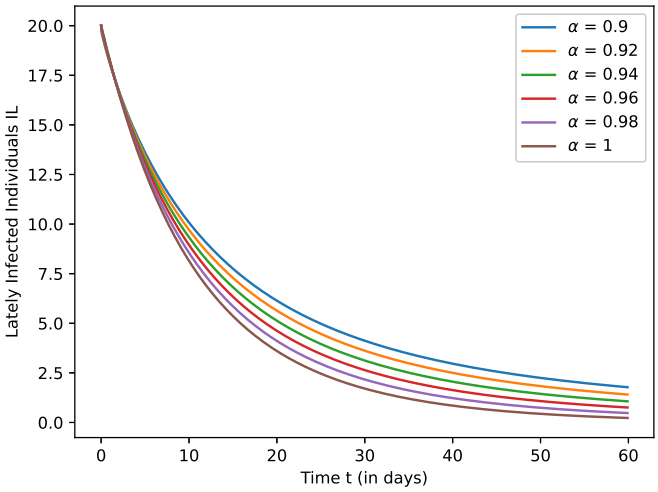

Figure 5.3: Behavior of the lately infected class $I_L(t)$ for certain fractional-order between 0.9 to 1.

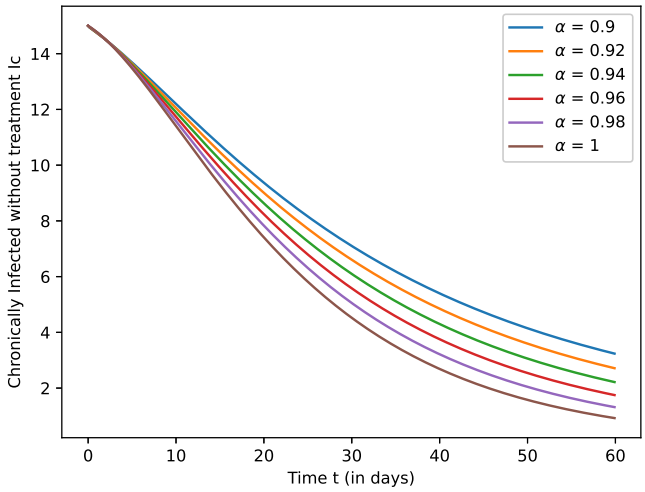

Figure 5.4: Behavior of the chronically infected without treatment class $I_C(t)$ for certain fractional-order between 0.9 to 1.

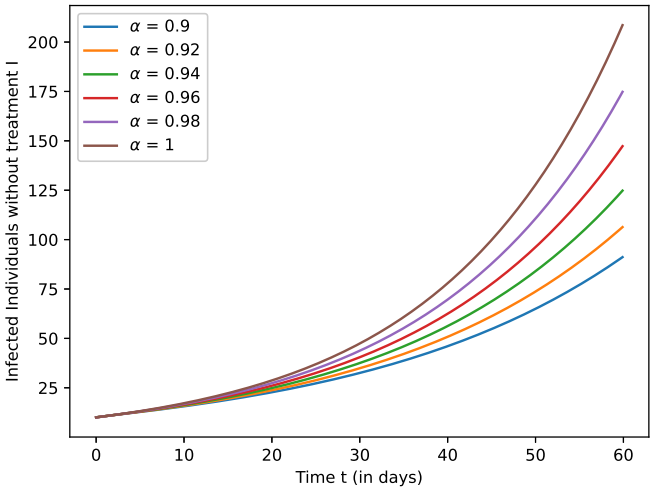

Figure 5.5: Behavior of the infected without treatment class $I(t)$ for certain fractional-order between 0.9 to 1.

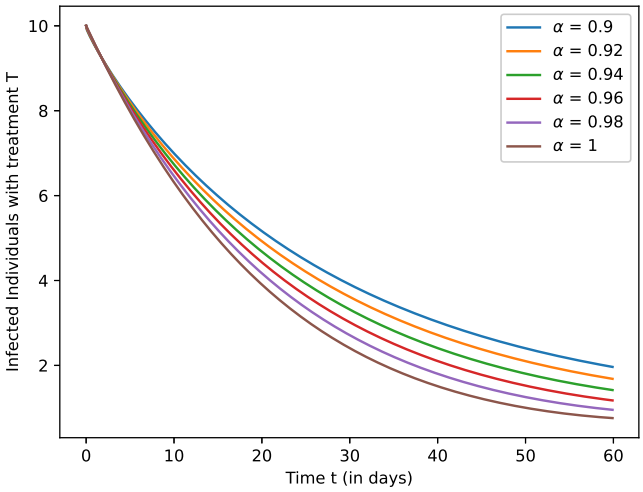

Figure 5.6: Behavior of the infected with treatment class $T(t)$ for certain fractional-order between 0.9 to 1.

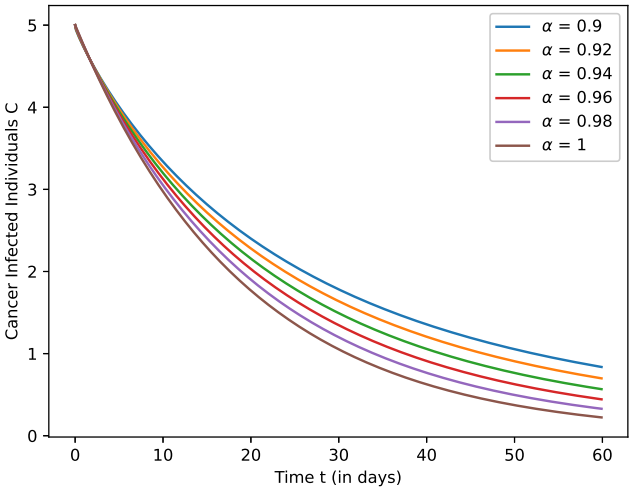

Figure 5.7: Behavior of the cancer infected class $C(t)$ for certain fractional-order between 0.9 to 1..

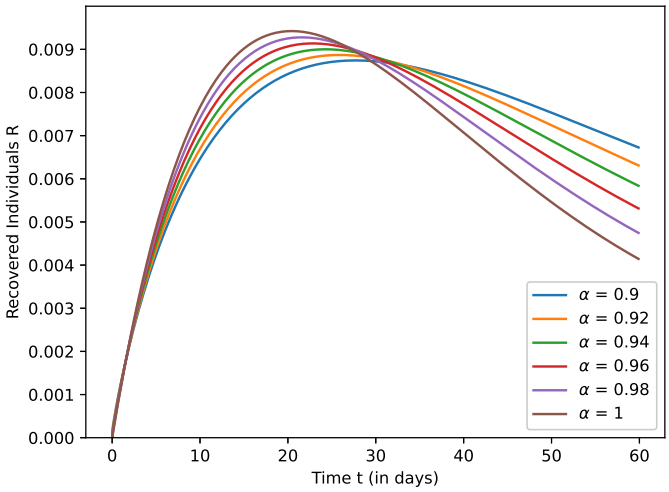

Figure 5.8: Behavior of the recovered class $R(t)$ for certain fractional-order between 0.9 to 1.

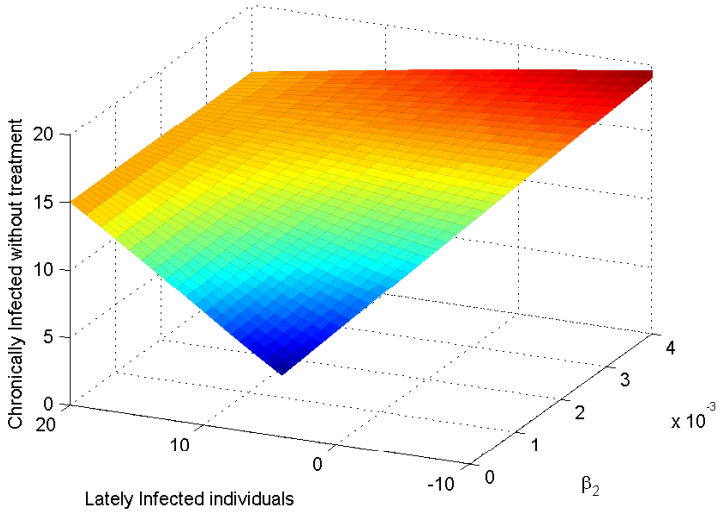

Figure 5.9: Rate of transmission from lately infected to chronically infected without treatment individuals when transmission coefficient β_2 is between 0 to 0.004.

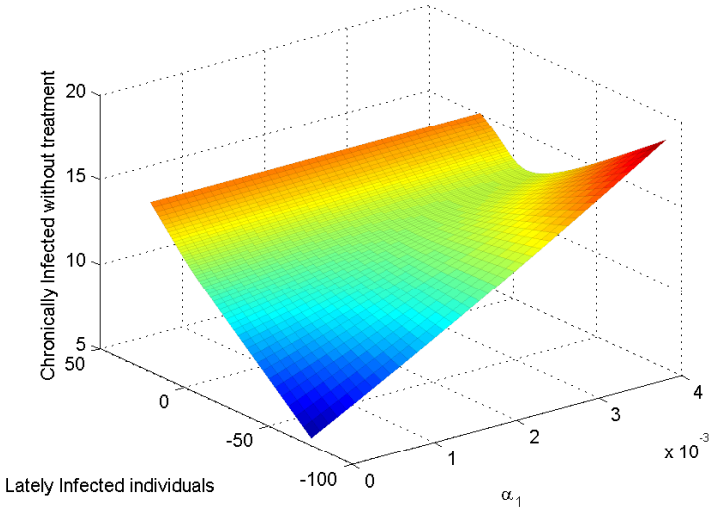

Figure 5.10: When any individuals transferring form I_L to I_C compartment with the rate of α_1 between 0 and 0.004.

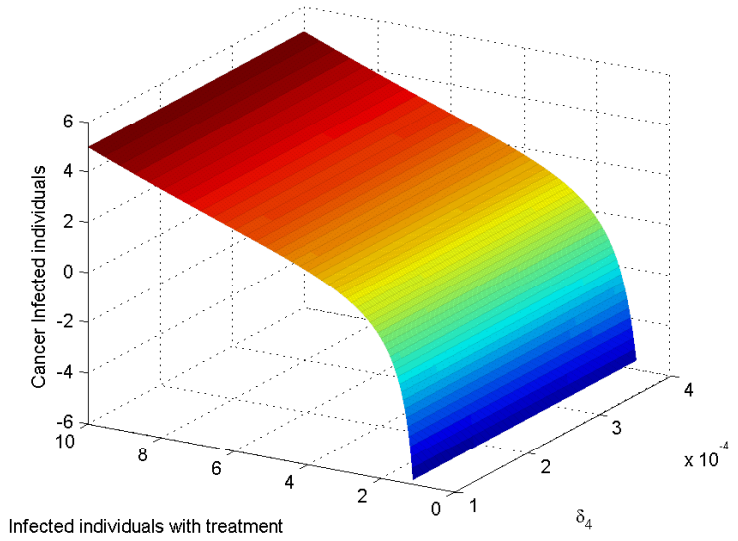

Figure 5.11: Rate of transmission from infected individuals to cancer infected individuals when infective parameter δ_4 is between 0.0001 and 0.0004.

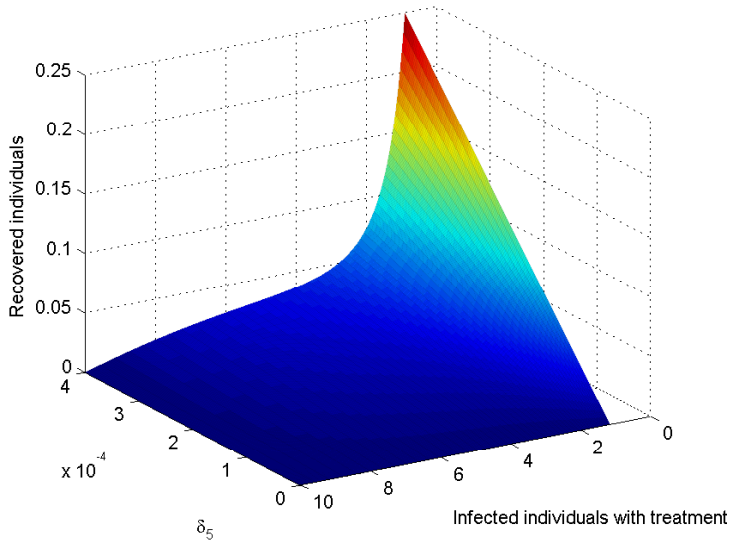

Figure 5.12: Rate of transmission from infected individuals to Recovered individuals when infective parameter δ_5 is between 0 and 0.0004.

5.7 Conclusion

We demonstrated a fractional-order smoking cancer mathematical model with seven compartment under the duration of 60 days. We utilized ABC fractional operator as it has non-local and non-singular property. Further, we acquired a well-known Adams-Bashforth-Moulton method which provides the fast convergence to the solution. The existence and uniqueness of the system is proved with the help of Banach's fix point theorem. By calculating the equilibrium points and basic reproduction number, we validated the local stability and with the use of Ulam-Hyers stability, we admitted the global stability of suggested model. From figures 5.2 to 5.8, we can say that the behavior of the solution of each class having stable region. Figures 5.9 and 5.10 show that when transmission coefficient β_2 and α_1

change, the lately infected individuals transmit to chronically infected individuals. Figure 5.11 is the display of transmission rate from infected individual to cancer due to smoking individuals when infective parameter δ_4 differ. Figure 5.12 is the plot of transmission rate

from infected to recovered individuals when infective parameter δ_5 change. By incorporating data on smoking cancer and its treatment effectiveness, researchers 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.

The use of the smoking 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 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.