MEKELLE UNIVERSITY

COLLEGE OF NATURAL AND COMPUTATIONAL SCIENCES

DEPARTMENT OF MATHEMATICS

A

Thesis

On

Mathematical modeling of smoking dynamics in society with impact of media information and awareness in the case of Mekelle Town

A thesis Submitted to the Department of Mathematics College of Natural and Computational Science

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By

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Mathematical modeling of smoking dynamics in society with impact of media information and awareness in the case of Mekelle Town, Tigray, Ethiopia

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Declaration

First, I stated that this study was my own, and that I worked under the supervision of Abreha Tesfay (Ph.D.) to achieve the research's results. The requirements for a M.Sc. degree in mathematics for the academic year 2025 have been partially met by this study. In accordance with library regulations, it was placed at the university library to ensure its dependability for patrons. I wanted to make it clear that no other school is considering this work for the award of any academic degree or diploma.

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This study has been submitted for the examination with my approval as university advisor.

Name of the advisor: Abreha Tesfay (Ph.D)

Signature of the advisor with date:

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Dedication

I dedicate this study to my mother, Sindayo Tadeg, my wife, Tsegabrhan G/tsadkan and my lovely kids Dawit Mengstu, Abel Mengstu and Tsion Mengstu and my great friends for their moral and spiritual support.

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Last but not least, I want to express my sincere gratitude to my beautiful mother Sindayo Tadeg, my beautiful wife Tsegabrhan G/tsadkan, as well as my friends and coworkers who have always supported me in all areas of life financially, morally, and materially.

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List of Abbreviation

1. AIDS -Aware Individuals Defeating Sickness
2. COPD -Chronic Obstructive Pulmonary Disorder
3. CSA -Community Statistics Alliance
4. CVD -Cardiac Vitality Defense
5. EDHS -Ethiopian Data for Health and Society
6. EE -Endemic Equilibrium
7. GATS -Global Awareness on Tobacco Survey
8. GYTS -Global Youth Tobacco Study
9. IHD -Ischemic Heart Defense
10. LAS-Local Asymptotical Stability
11. Mat lab-Matrix Laboratory
12. MSIR -Medical Statistics and Information Resource
13. NCD -Non-Communicable Disease
14. $PLHQ_tQ_pS_mM$ –Potential, Light, Heavy, Temporary quit, Permanent quit, Aware population and Media
15. R_s – Smoking Generation Number
16. SEIS –Susceptible Exposed Infected Susceptible
17. SFE -Smoke-Free Equilibrium
18. SIR -Susceptible Infected Recover
19. SIRS -Susceptible Infected Recover Susceptible
20. SIS –Susceptible Infected Susceptible

Abstract

This study came up with a mathematical model to examine the smoking dynamics of the Mekelle Town in Ethiopia where the rate of smoking is a rising issue. The study categorized the population in compartments of potential smokers, light smokers, heavy smokers, temporary and permanent quitters, informed non-smokers and media campaign($PLHQ^tQ^pS_mM$) based on survey data of 400 respondents throughout four sub-cities. The basic reproduction number was determined to be 1.192555 which proved that smoking is being actively propagated in the community. The sensitivity analysis showed that the most important parameters that increase the rate of diffusion of smoking are contact rate (β = 0.5454) and the rate of light into heavy smoking (α_2 = 0.833), and the rate of elimination among heavy smokers (γ= 0.2922) is the most effective to reduce the prevalence. The analysis of the three control measures using the optimal control theory included counseling/education (u_1), public smoking prohibition (u_2), and treatment of the heavy smokers (u_3). The mathematical model proved this combination of all three controls was the most effective and significantly decreased all the populations of smokers, and single interventions or a combination of two interventions were inadequate to overturn the smoking trend. It offers a conclusion that the multi-strategy programs that will work with potential, light, and heavy smokers simultaneously are necessary, with the emphasis on tightening the smoking bans, offering the cessation programs, and continuous local language awareness campaigns, and additional long-term research is needed to prove the effectiveness of the suggested method in the real-life scenario.

Keywords: Mathematical models, dynamics related to smoking, basic reproduction number, optimal control, sensitivity analysis, Mekelle Town, tobacco control, intervention of public health, epidemic model, Ethiopia.

Chapter 1: Introduction

1.1 Definition of Smoking Tobacco

Smoking is a global habit with deep roots. It began with Native American traditions before spreading to Europe and beyond, eventually becoming the multi-billion dollar cigarette and cigar industry we see today. Most people smoke for the nicotine; it's a powerful chemical that can make you feel relaxed and energized at the same time, which is exactly why it's so hard to quit, despite the well-known risks[1].

Figure 1 : Cigarette Butts(Adapted from https://www.britannica.com/topic/smoking-tobacco

Defining smoking as inhaling tobacco does not tell the whole story. Smoking is not about a physical craving for nicotine it is a complex issue. To really understand the harm that smoking does we have to look at the person who smokes their genetics, their stress levels and where they come from. If we do not consider someones environment and daily life a simple definition of smoking is not enough(2).

Tobacco smoking is basically the act of breathing in the smoke from burning tobacco leaves. When people smoke cigarettes they usually breathe the smoke into their lungs. People who smoke pipes or cigars often just taste the flavor in their mouth. Then let it out. Smoking has a long history people in Mesoamerica and South America were using tobacco in their daily lives and rituals as far back as 5000 BC(26). The tobacco plant we know today Nicotiana tabacum is a plant with leaves that can grow up to six feet tall. This plant originally came from places but now it is grown in many fields, around the world. Scientists are still trying to figure out the

history of tobacco they think it came from a mix of three different wild plants. Because tobacco leaves are very special they are the common type of tobacco plant that people grow(3).

1.1.1 Health Effects of Cigarette Smoking

When someone lights a cigarette they are not just breathing in smoke they are starting a fast chemical reaction that happens away. The smoke goes into the airways. Then into the lungs, where it gets into the blood really quickly. This all happens fast and within a few seconds the chemicals from the cigarette are, in the brain and moving through the rest of the body. People usually smoke cigarettes. The same thing happens if someone uses a pipe or a cigar or even smokes herbal blends or marijuana (4).

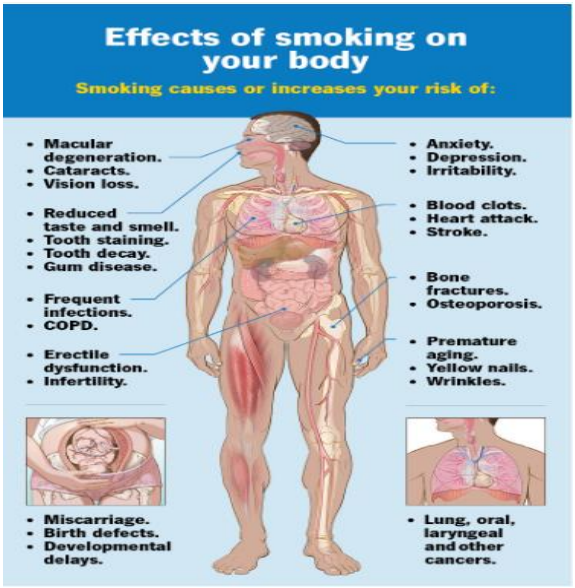


Figure 2: Smoking can damage all parts of our body(Adapted from https://.clevelandclinic.org/health/articles/17488-amoking)

1.1.2 Smoking and its health risk

It's a devastating misconception that smoking only harms the smoker, as the smoke from a single cigarette endangers those nearby who never chose to light up leading to 1.2 million global deaths annually, including a heartbreaking 65,000 children who lose their lives to secondhand smoke-related illnesses simply by breathing contaminated air in their own homes and communities, with nearly half of the world's youngsters exposed. This cycle of harm can begin even before birth, as pregnant women who smoke pass along direct exposure to their developing babies, often resulting in a lifetime of chronic health struggles from the very first breath (6).

A long-term smoker faces a significantly higher risk of developing a wide range of serious and often life-threatening health issues. This includes numerous cancers, such as those affecting the lungs, mouth, throat, pancreas, kidneys, and bladder, as well as leukemia in the bone marrow. The damage isn't limited to cancer; smoking severely compromises the lungs, leading to chronic bronchitis, emphysema, and other forms of chronic obstructive pulmonary disease. It also takes a heavy toll on the cardiovascular system, dramatically increasing the chances of a stroke, heart disease, and poor circulation in the hands and feet a condition that can become so severe it leads to gangrene and even amputation. Furthermore, smokers are more susceptible to developing type 2 diabetes, rheumatoid arthritis, stomach ulcers, and osteoporosis, which raise the risk of debilitating hip fractures. In total, tobacco use is a major contributor to over 20 different types of cancer, along with a host of other devastating and crippling medical conditions (5).

Nicotine is also highly addictive. Over 8 million individuals die from tobacco usage each year. The majority of tobacco-related deaths take place in low- and middle-income nations, which are frequently the focus of intense marketing and corporate influence (27).

Respiratory Diseases

Smoking damages every part of the lungs, weakens the immune system, and increases susceptibility to infections. Each infection causes further lung damage, creating a cycle that eventually leads to chronic obstructive pulmonary disease (COPD)(7).

The tiny particles in cigarette smoke are perfectly sized to slip deep into the lungs, where they immediately begin irritating and breaking down delicate tissue. This damage turns the lungs into a sticky trap that holds cancer-causing chemicals close, while other toxins enter the bloodstream and are transformed into even more dangerous forms by the body's own enzymes. In a cruel twist, the body's attempt to fight back with inflammatory cells often backfires, ultimately tearing down the very walls of the lungs that make breathing possible (25).

Tobacco smoking and risk of cardiovascular disease

Cardiovascular disease is the cause of death around the world. It includes different types of diseases like heart disease and problems with the rhythm of the heart. Cardiovascular disease also includes diseases that affect the blood vessels in the brain and the rest of the body(7).

The walls of the arteries get clogged because of the things in cigarettes. This makes the walls sticky and then fatty stuff sticks to them. Over time this fatty stuff can block the arteries. Make it hard for blood to flow through them like it should. This can cause a heart attack because the heart is not getting the blood it needs(28).

When people smoke it can make their cholesterol levels go up. Smokers usually have less of the cholesterol and more of the bad cholesterol than people who do not smoke. Smoking also makes the blood thicker and more likely to clot. This is because smoking increases the amount of proteins in the blood. The bad things in cigarettes also stop the blood from carrying much oxygen as it should. This is because they stick to the blood cells and do not let them carry oxygen like they should.All of these things can cause a disease called disease. This disease happens when the arteries get narrower because of building up inside them. This disease is more common, in people who smoke. It can cause a lot of problems because it stops the blood from flowing like it should. As this disease gets worse the blood flow gets worse too(29).

Efficacy of cessation treatments

Since the 1970s people have been studying ways to help people stop smoking. Around the mid-1980s a lot of people started to get really interested in looking at these smoking cessation therapies. Many groups have been going over all the research that has been done on this topic. They have been making recommendations to doctors and the public health community about how to help people stop smoking because many studies have been done and so many people are interested, in this topic now including doctors and the public health community(30).

1.2 Background of the Study

The tobacco use has still been a major global issue of concern in the global health context of which above 8 million deaths are expected to result per year by 2030 even though the rates of tobacco use have decreased in certain high-income countries. Smoking is widely used in the world especially in the developing and middle-income economies where more than 80 percent of smokers are found. Tobacco substances lead to a number of diseases such as cancer and respiratory illnesses and it causes a higher number of deaths than AIDS, tuberculosis and other significant causes together. Exposure to second hand smoke is also dangerous to health. Economic cost of tobacco-related deaths and disease is high particularly in the economies with

low and middle income where tobacco related deaths are about 80% of the total. Poor awareness, social pressure and poor enforcement of anti-tobacco laws are the reasons why tobacco is widely used [31].

To manage and prevent epidemiological disorders such as tobacco use, one has to be knowledgeable on the dynamics of the disease spread and spread processes. This will surely back our disease-eradication strategies as well as our predictions. Since epidemic dynamics are dynamic with time, it is a significant approach to theory to examine the dynamics of infectious disease transmission. Mathematic modeling has been developed with the help of different analyses and numerical simulations, which are literally grounded on the population dynamics, infectious symptoms, and the interaction with the social and physiological factors. Mathematical models may also come in handy to learn more about the distribution of infectious diseases and how they can be managed or controlled [32].

1.3 Statement of the Problem

The world is becoming more aware of the problem that chronic non-communicable diseases are causing. These diseases are now an issue in Africa too. Every year 41 million people die from these diseases and most of these deaths happen in countries that are not very rich(33). One of the reasons for these diseases is that people use tobacco. Tobacco use causes about 8 million deaths every year. It is linked to many cases of lung cancer breathing problems and heart disease(35,36). The cost of tobacco use is high, Over 1.5 trillion dollars every year. If things do not change, one billion people will have died from tobacco-related causes by the end of this century(35,37).

Ethiopia is facing a problem with tobacco use. Every year 16,800 people die from tobacco use in Ethiopia, which is a higher rate than in some neighboring countries(43). There is not enough information about tobacco use in Ethiopia. The country does not have data on how many people use tobacco and there is no detailed analysis of how tobacco use spreads and how to stop it(44,48).

In Mekelle Town, one of the cities in Ethiopia the problem of tobacco use is not well understood. The town has people from different backgrounds and tobacco use is likely affected by social

economic and cultural factors. There is no information about how tobacco use affects the people of Mekelle Town. We do not know how many people start smoking how many quit or how many try to quit. Without this information it is hard to know whether tobacco use is increasing or decreasing in the town. We also do not know, what factors are driving tobacco uses and health officials do not have guidance on how to use their limited resources to stop tobacco use.

In terms while we know that tobacco use is a big health problem we do not have a good understanding of how it affects the people of Mekelle Town. This study aims to fill this gap by collecting data and doing analysis to understand how tobacco use spreads in the town. This will help local health officials design programs to stop tobacco use and save lives.

The goal of this study is to provide the detailed analysis of tobacco use in Mekelle Town. We will collect data to understand key factors that affect tobacco use calculate the basic reproduction number and do sensitivity analysis. This will give us an understanding of how tobacco use spreads, in the town and how to stop it. We will also solve a control problem to find the best way to use limited resources to stop tobacco use. This study will provide health officials with the information they need to design effective programs to stop tobacco use and save lives.

The researcher faces the following problems:

1. We do not have data about smoking in Mekelle Town to make accurate predictions.

2. We do not understand how people move from one smoking category to another such as from smokers to light smokers.

3. We have not evaluated the effectiveness of public health interventions, such as awareness campaigns and smoking bans using methods.

4. We do not know if current smoking trends in Mekelle Town will lead to a decrease in smoking or an increase.

5. We do not have a basis for deciding which intervention strategies to use to reduce smoking prevalence.

Research Questions

This study seeks to answer the following questions:

1. What are the key factors that affect the spread of smoking in Mekelle Town, including how people come into contact with each other and how often people quit smoking?

2. What is the basic reproduction number for smoking in Mekelle Town? Does it mean that smoking will decrease or increase?

3. Are the points where smoking is eliminated or persisted stable under conditions?

4. Which factors are most important in influencing the spread of smoking? Where interventions should be targeted?

5. How effective are different strategies, including counseling and smoking bans in reducing smoking prevalence?

6. What combination of strategies yields the outcome for minimizing potential smokers, light smokers and heavy smokers while maximizing permanent quitters?

1.4 Objectives of the Study

1.4.1 General Objective

To develop a model of smoking dynamics in Mekelle Town that incorporates local data and evaluates optimal control strategies for smoking reduction.

1.4.2 Specific Objectives

1. To collect data from four sub-cities of Mekelle Town to classify the population into smoking categories.

2. To estimate model parameters, including transmission rates and conversion rates between smoking classes.

3. To calculate the reproduction number and determine whether smoking will spread or decrease.

4. To analyze the stability of both smoking- endemic equilibrium points.

5. To conduct sensitivity analysis to identify parameters with the influence on smoking transmission.

6. To perform simulations to visualize smoking dynamics over time.

7. To. Solve an optimal control problem with three intervention strategies to identify the most effective combination.

8. To compare the effectiveness of dual and triple control strategies in minimizing smoking prevalence.

1.5 Significance of the Study

The findings of this study will:

1. Provide public health authorities in Mekelle Town with evidence-based data to predict smoking trends.

2. Identify the threshold conditions that determine whether smoking will persist or decline.

3. Reveal which parameters are most influential in driving smoking transmission.

4. Offer a comparison of different control strategies.

5. Establish a framework that can be adapted for other urban centers in Ethiopia.

6. Contribute to the literature on mathematical modeling of substance use disorders.

1.6 Scope and Limitations

The study focuses on Mekelle Town, four sub-cities with a sample size of 400 respondents. The mathematical model considers six population compartments, including smokers, light smokers and heavy smokers. While the study provides insights, limitations include assumed values, for certain parameters and the cross-sectional nature of data collection.

1.7 Justification

This study finds its reason in the simple fact that Mekelle Town, like many growing cities, needs to understand what is happening with smoking in its own community, and this work may offer a small step in that direction. The mathematical model developed here could one day give local health planners a way to look at smoking not just as a number from a survey, but as something

that moves through neighborhoods and families, helping them see where things might be headed. For the officials and decision makers in Mekelle who carry the responsibility of protecting public health, the findings might offer a local lens, insights drawn from their own people that could quietly shape the programs they design and the choices they make. On the ground, health workers and community leaders who spend their days talking to young people and parents may find in this research something useful to hold onto, a piece of evidence from their own town to strengthen the conversations they are already having. And for the students and researchers who will come after, the ones who will sit where we are sitting now and wonder where to begin, this work could serve as a quiet foundation, something to build on or push against as they take their own small steps forward.

In the end, this study does not claim to change everything, but it may, in its own modest way, help someone in Mekelle see the problem a little more clearly.

1.8 Organization of the Study

This research is divided into six chapters.

Chapter One introduces the historical and biological context of the study, defines the research problem, outlines the objectives, describes the methodology, justifies the significance of the study, and explains its overall structure.

Chapter Two reviews relevant literature, including both theoretical and empirical studies on the mathematical modeling of tobacco smoking, along with the methods used by previous researchers.

Chapter Three presents the mathematical foundations and the specific methodology applied in this study.

Chapter Four focuses on optimal control and the formulation of the mathematical model.

Chapter Five analyzes the model and discusses the findings.

Finally, **Chapter Six** summarizes the conclusions derived from the study and provides recommendations for future research.

Chapter 2: Literature Review

2.1 Mathematical Model

A mathematical model describes a system using mathematical concepts. The process of creating these models, known as mathematical modeling, is applied across various fields, including social sciences (like economics and psychology), natural sciences (such as physics and biology), and engineering (including computer science)[8].

Mathematical modeling is a cyclical process that involves converting real-world issues into mathematical terms, solving them in a symbolic system, and then testing the answers in the original system [9].

Lesh and Doerr Mathematical modeling is a process that involves using existing conceptual systems and models to develop new models in different contexts. In this framework, a model is considered a product, while modeling refers to the process of creating physical, symbolic, or abstract representations of a situation. This approach enables the adaptation and application of established ideas to address new challenges and scenarios. [10].

Epidemiology studies the distribution and determinants of disease prevalence in humans. Its key functions include [11]:

1. Describing Disease Distribution: Identifying who is affected, how much, where, and when.
2. Identifying Causes and Risk Factors: Understanding why diseases vary among populations.
3. Building and Testing Theories: Developing theoretical frameworks related to diseases.
4. Planning and Evaluating Programs: Implementing and assessing detection, control, and prevention strategies.

2.2 History of Mathematical Modeling in Infectious Disease

Mathematical models are crucial for analyzing the spread and control of infectious diseases. They assist in evaluating epidemiologic explanations and predicting how changes affect disease dynamics. Understanding population dynamics and the impact of control programs is essential for effective disease management. [12].

The first epidemiological model was created by **Daniel Bernoulli** in 1760, using mathematical methods to assess the effectiveness of universal smallpox vaccination. Deterministic epidemiology modeling began to develop in the 20th century [13].

In 1906 **Hame**r formulated and analyzed a discrete-time model in his attempt to understand the recurrence of measles epidemics [6]. And followed by **Ross** was interested in the incidence and control of malaria, so he developed differential equation models for malaria as a host-vector disease in 1911 [14].

Starting in 1926 **Kermack and McKendrick** published papers on epidemic models and obtained the epidemic threshold result that the density of susceptible must exceed a critical value for an epidemic outbreak to occur [15]. Starting in the middle of the 20^{th} century, mathematical epidemiology showed rapid growth. The book on mathematical modeling of epidemiological systems that was published by **Bailey** in 1975 is said to be an important landmark which, in part, led to the recognition of the importance of modeling in public health decision-making [16].

2.3 Review on Mathematical Modeling in Smoking Tobacco

Smoking remains one of the world's most devastating health crises, claiming nearly seven million lives each year. The vast majority over six million people die from lighting up themselves, while close to a million more are innocent bystanders, breathing in smoke that was never theirs. The damage reaches nearly every part of the body: it fuels cancers, attacks the heart and blood vessels, eats away at the stomach, and slowly destroys the lungs' ability to breathe. It even leaves its mark in stained teeth, a small but visible reminder of the destruction happening within [35]. About 80% of the world's 1.1 billion smokers reside in low- to middle-income countries, where the burden of smoking-related illness and death is most severe. Premature deaths among smokers decrease family income, increase healthcare costs, and hinder economic development. Economic losses to society are projected to reach two trillion dollar(52).

Castillo-Garsow and G. Jordan-Salivia originally proposed a mathematical model of smoking behavior, whereas **Rodriguez-Herrera** [53] developed a mathematical model for quitting smoking. This model divides the overall population into three subclasses: potential smokers, smokers, and ex-smokers. Later, **O Sharomi and A Gumel** [54] established the smoking behavior model by categorizing ex-smokers as either temporarily or permanently quitting smoking. **Zaman** [55] developed and analyzed a smoking behavior model that included the occasional smoker compartment in the basic model. In [54], **Z. Alkhudhari, S. Al-Sheikh, and S. Al-Tuwairqi** used the model created and studied in [58] to investigate the impact of peer pressure on temporary quitters.

Z. Alkhudhari, S. Al-Sheikh, and S. Al Tuwairqi proposed a new model in 2014 [56] that divided smokers into two subclasses: occasional smokers and heavy smokers, and examined the impact of these two subclasses on the occurrence and stability of equilibrium points. They also investigated the influence of occasional smokers on potential smokers, followed by the effect of heavy smokers on potential smokers in 2015 [56]. Furthermore, **S Matintu (2017**) [57] studied the smoking behavior model by categorizing ex-smokers as temporary quitters or permanent quitters, as well as smokers into two subclasses: light smokers and heavy smokers.

In this model, **S Matintu** added the component of mortality rates by assuming that all classes had the same mortality rates. However, these assumptions are incorrect because the death rates of potential smokers, light smokers, heavy smokers, smokers who temporarily quit smoking, and smokers who permanently quit smoking are all significantly different, according to health-care data.

In this study, they used the model [57] to create a mathematical model of smoking behavior modifications. They include the rate at which heavy smokers become light smokers, heavy smokers become smokers who temporarily and permanently quit smoking, light smokers become smokers who temporarily and permanently quit smoking, and smokers who temporarily quit smoking become light smokers. They eliminated the rate of change from the class of smokers who quit smoking momentarily and then become heavy smokers using model [57]. Changes are made to the model of the propagation of changes in smoking habit based on the authors' observations of various smokers.

In a recent paper [**Zeb, A., Bibi, F., & Zaman, G. (2015), Square-root Dynamics of a Giving Up Smoking Model, Appl. Math. Model, 37 (2013) 5326-5334]**, the researchers developed a new smoking cessation model using three strategies—education campaigns, anti-smoking gum, and nicotine-blocking medications—to reduce smoking in communities. Using optimal control theory and mathematical analysis, they determined the most effective combination of these tools to convert smokers into quitters. Their findings, validated through MATLAB simulations, provide a data-driven blueprint for communities working toward becoming smoke-free[20].

Labzai, A.Balatif, O., & Rachik,M. (2018). This study developed a mathematical model to analyze smoking behavior by dividing the population into five groups: potential smokers, light smokers, heavy smokers, temporary quitters, and permanent quitters. The goal was to find the most effective way to reduce light smokers, heavy smokers, and those who temporarily quit. Researchers tested three interventions awareness campaigns through media and education, medical treatment, and psychological support with follow-up care. Using discrete-time mathematical analysis and MATLAB simulations, they confirmed that combining these strategies optimally can successfully drive down smoking rates across all categories[19].

Hussain,T., Awan, A.U., Abro, K. A., Ozair, M., & Manzoor, M. (2021). The qualitative study of a smoking model with parametric conditions for diseases controlled under the influence of smoking is investigated through rigorous mathematical study. The mathematical modeling of an epidemiological smoking model having six compartments is traced out. Mathematical expressions for smoke-free and smoke-present equilibrium points have been developed. The strength of the Lyapunov functional theory has been exploited to show that the smoke-free equilibrium point is globally asymptotically stable whenever the basic reproduction number $R_s<1$. The competency of graphical and theoretic processes is utilized to observe the global behavior of unique smoke equilibrium points. The sensitivity analysis of the model is performed through the basic reproduction number and diseased classes effectively to design reliable, robust and stable control strategies[21].

Khyar,O., Danane, J., & Allali, K. (2021). They had studied in mathematically and numerically dynamics of giving up smoking behavior, they suggested smoking model consists of a system of five differential equations representing the potential smokers, the occasional smokers, the chain smokers, the temporarily quit smokers, and the permanently quit smokers. They had established

the well-posedness result of their smoking problem by proving the existence, positivity, and boundedness of the smoking problem solution. Both the equilibrium local and the global stability results are fulfilled. To study the role of control measures on their smoking problem dynamics, two different controls are introduced to the model. The role of the first control is to reduce the contact between nonsmokers and smokers, while the objective of the second is to prevent occasional smokers from becoming chain smokers. The existence and the characterization of the two optimal controls were discussed by using the Pontryagin's minimum principle of optimality system is solved numerically using the classical forward and backward difference numerical scheme. It was established that the numerical simulations concerning the stability of the smoking-free and the smoking-present equilibria were in good agreement with the local and global theoretical results. Moreover, the numerical results confirm the important role of control measures in reducing the number of occasional and chain smokers which may save a significant number of lives in the smoking population[23].

Sofia, I. R., Bandekar, S. R., & Ghosh, M. (2023). In this work, a nonlinear model of smoking dynamics was framed and analyzed with the inclusion of media awareness and information. A detailed mathematical analysis was carried out and presented in detail covering the positivity and roundedness of solutions, stability analysis, and basic reproduction number. Numerical simulations were performed and the analytical results were supported graphically. To solidify the study, the work was expanded by performing the sensitivity analysis of basic reproduction numbers concerning several parameters to signify the key parameters that can impact the former. The variations in different populations with changes in rates related to media awareness and depletion are showcased in detail. The overall study emphasizes the importance of media awareness in the achievement of smoking cessation. A drastic decrease in the population of potential smokers and subsequently a substantial increase in the aware population are observed with an increasing rate of spread of awareness. The study is further extended to an optimal control model which further proves the significance of counseling, treatment methods, and other approaches in contributing towards smoking cessation. Through optimal control, the authors stressed on the importance of counseling and therapies for potential smokers, where there is a plausible section who might be dealing with mental health-related issues to not dwell on this addiction [22].

Ullah,A., Sakidin, H., Shah, K., Hamed, Y., & Abdeljawad, T. (2024). In this work, the author enhanced the NERA model by introducing two additional compartments: The prisoner's class and the hospitalized class (smokers under treatment). The inclusion of these classes is deemed realistic, and their absence posed challenges in mitigating and recovering the prevalence of marijuana smokers within the population. The mathematical formulation employed in this study utilized a system of first-order non-linear ordinary differential equations. They addressed various aspects, including the basic reproduction number, invariant region, and sensitivity analysis, each serving distinct purposes. The invariant region was solved to validate the modified model. The basic reproduction number was computed for the initial rate of marijuana smoking transmission, and sensitivity analysis identified the most crucial parameters influencing marijuana transmission. To curtail marijuana smoking, two strategies were derived based on the most sensitive (targeted) parameters. Through numerical simulation, the results indicated that Strategy 1 proves to be more efficacious than Strategy 2 in controlling marijuana smoking. Moreover, the modified model exhibited more rapid convergence compared to the previous model, leading to the conclusion that the modified model holds greater significance. They suggested potential future research directions, including the exploration of novel techniques such as “optimal control problems” and “threshold conditions”, to optimize the control of marijuana smoking with minimized costs and time investment[24].

Chapter 3: Methodology

3.1 Introduction

Compartments

Imagine we are trying to understand how smoking moves through a community. One helpful way is to picture the population sorted into different rooms based on their smoking status. Some people have never smoked, some are current smokers, and some have quit. These rooms are what mathematicians call **compartments**, and a model that tracks how people flow between them is called a compartmental model(72).

In mathematical terms, if we let N(t) represent the total population at time t, and we divide them into n compartments labeled $X_1, X_2, \ldots X_n$ then the sum of people in all compartments equals the total population:

$$N(t) = \sum_{i=1}^{n} X_i(t)$$

What makes the model useful is that it watches how these numbers change over time. The rate at which people enter or leave each compartment is described using differential equations. For any compartment X_i, the change over time is simply what comes in minus what goes out:

$$\frac{dX_i}{dt} = \sum (in\ flow\ in\ to X_i) - \sum (out\ flow X_i)$$

To make this concrete, consider a basic smoking model with just three compartments: P for potential smokers (those who have never smoked), S for current smokers and Q for those who have quit. The movement between them might look like this:

$$\frac{dP}{dt} = -\beta PS$$

$$\frac{dS}{dt} = \beta PS - \gamma s$$

$$\frac{dQ}{dt} = \gamma s$$

Here, β represents the rate at which people start smoking, perhaps through social influence, and γ is the rate at which smokers quit. These equations simply tell a story: people leave the potential group to become smokers, and smokers eventually leave to join the quitters(72).

Deterministic Models

Think of a deterministic model like a recipe you follow exactly every time. If you use the same ingredients in the same amounts and follow the same steps, your dish will turn out identical each time. There is no room for luck, chance, or surprise. A deterministic model works the same way: given the same starting point and the same rules, it will always produce the exact same result(72).

In mathematical language, imagine we have a system described by a set of ordinary differential equations:

$$\frac{dX}{dt} = f(X, t, \theta)$$

Here, X represents the state of the system at any given time, like how many people are in each compartment. The symbol tt stands for time, and θ represents the parameters, things like how fast people start smoking or how quickly they quit. If we know where we started, written as $X(0) = X_0$ then the path the system takes into the future is completely fixed. There is only one possible outcome [72].

What sets deterministic models apart from their cousins, stochastic models, is that they do not try to capture randomness or luck. They assume the population is large enough that individual surprises average out, and the overall behavior is smooth and predictable. It is like looking at a river from far away: you do not see every splash and ripple, just the steady current carrying the water forward [73].

In this chapter we create a math model to understand how tobacco smoking spreads in Mekelle Town.

This model will help us predict how smoking will spread and find ways to control it in Mekelle Town. We use a type of math model called a model because we are studying a large population.

In this model we divide the population into groups, each representing a stage of the smoking epidemic. The groups are: people who can potentially smoke, light smokers, heavy smokers, people who quit smoking temporarily people who quit smoking permanently people who are aware of the risks and the media.

We use a model called Potential smokers, Light smokers, Heavy smokers, Quit temporary smokers, Quit permanent smokers, the aware population, and Media ($PLHQ^tQ^pS_mM$) to study the spread of smoking. This model is helpful in explaining how tobacco smoking spreads in Mekelle Town using a ($PLHQ^tQ^pS_mM$) model.

The model helps us understand tobacco smoking in Mekelle Town and the spread of tobacco smoking.

The capital and special zone of Ethiopia's Tigray Region is Mekelle, also known as Mekelle (Tigrigna: መቐለ). In the past, Mekelle served as the capital of Enderta awraja in Tigray. At an elevation of 2,254 meters (7,395 feet) above sea level, it is situated around 480 miles (780 kilometers) north of Addis Ababa, the capital of Ethiopia [67].
Mekelle, which is separated into seven sub-cities, is regarded as a Special Zone administratively. It serves as the political, cultural, and economic center of northern Ethiopia. With 61,000 residents in 1984, 97,000 in 1994 (of whom 96.5% spoke Tigrinya), and 170,000 in 2006 (i.e., 4% of Tigray's total population), Mekelle has had tremendous population growth since 1991. Mekelle is the second-biggest city in Ethiopia after Addis Ababa [66].The current metro area population of Mekele in 2024 is 612,000, a 4.26% increase from 2023. The metro area population of Mekele in 2023 was 587,000, a 3.89% increase from 2022. The majority of Mekelle's population is employed by the government and makes their living through small businesses and commerce. New textile, cement, and engineering factories that catered to both

domestic and international markets were located in Mekelle in 2007. The Arid Agricultural College existed prior to 1991 and gave rise to Mekelle University [67].

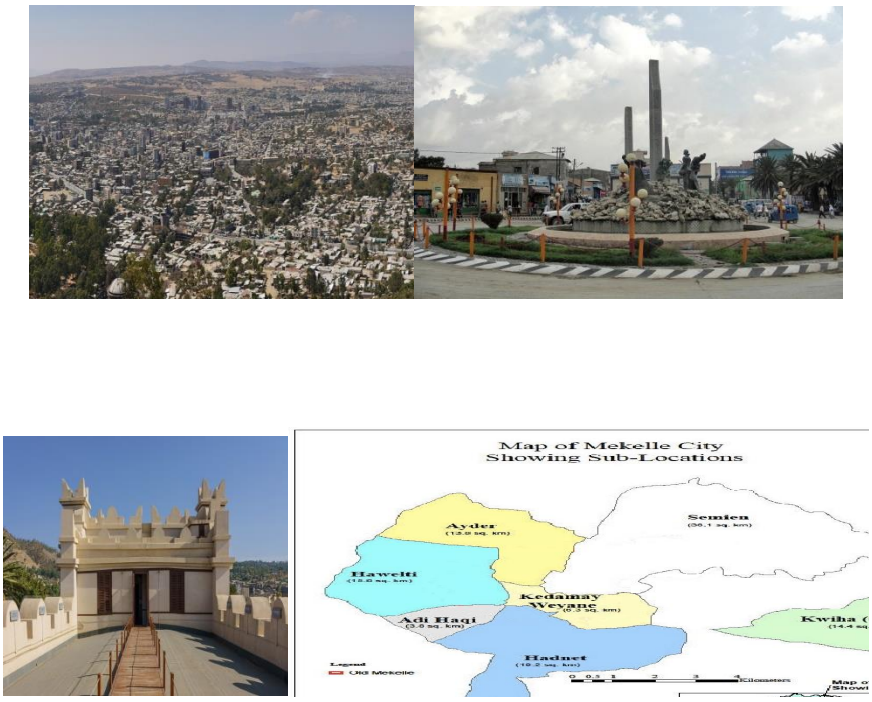


Figure 3: The Administration Map of the Mekelle town

3.2 Mathematical Preliminaries

3.2.1 Equilibrium Point/Steady State

Consider a system of differential equation given by:

$$\frac{dx}{dt} = f(x), \qquad x \in R^n \tag{3.2.1}$$

A point x^* that satisfies $f(x^*) = 0$ is called the equilibrium point or fixed point of the equation system [50].

3.2.2 Stability of the Equilibrium Points/Steady States

Theorem3.1: Consider the autonomous system $\frac{dx}{dt} = f(x)$ where f is continuously differentiable (C') in a neighborhood of an equilibrium point x* (i.e., f (x*) = 0). Let J = D f(x*) denote the Jacobian matrix of f evaluated at x* [48].

1. If all eigenvalues of J have negative real parts, then x* is locally asymptotically stable (LAS).
2. If at least one eigenvalue of J has a positive real part, then x* is unstable.
3. If all eigenvalues have non-positive real parts but at least one has zero real part, the stability cannot be determined from linearization alone and requires further investigation of nonlinear terms.

The eigenvalues are obtained as the roots of the characteristic equation of the Jacobian matrix evaluated at the equilibrium point.

For differential equations of this form, there are two approaches to determine the stability of the fixed points: Graphical stability of analysis and Linearization stability analysis. For the study, the study will consider the Linearization stability.

Linearization of Stability Analysis

Linearization is about finding a straight line that is very close to a curve at a certain point. When we study how things change over time like in systems linearization helps us understand if something is stable at a certain point. This is really useful, for systems that have a lot of movements like nonlinear differential equations or discrete dynamical systems and we want to know what happens at a specific Equilibrium point [49].

Suppose that p is a point such that $f(p) = 0$, i.e., p is a fixed point for the differential equation $x'(t) = f\big(x(t)\big)$. The linear part f at p denoted $Df(p)$, is a matrix of partial derivatives at p.

For $x \in R^N$ and $f(x) \in R^N$, we can write; $$f(x) = \begin{pmatrix} f_1(x) \\ f_2(x) \\ f_3(x) \\ . \\ . \\ . \\ f_n(x) \end{pmatrix}$$

The function f_i is called the component function of f.

Define

$$Df(p) = \begin{pmatrix} \frac{\partial f_1}{\partial x_1}(p) & \frac{\partial f_1}{\partial x_2}(p) & \frac{\partial f_1}{\partial x_3}(p)\dots & \frac{\partial f_1}{\partial x_n}(p) \\ \frac{\partial f_2}{\partial x_1}(p) & \frac{\partial f_2}{\partial x_2}(p) & \frac{\partial f_2}{\partial x_3}(p)\dots & \frac{\partial f_2}{\partial x_n}(p) \\ \frac{\partial f_3}{\partial x_1}(p) & \frac{\partial f_3}{\partial x_2}(p) & \frac{\partial f_3}{\partial x_3}(p)\dots & \frac{\partial f_3}{\partial x_n}(p) \\ \vdots & \vdots & \vdots & \vdots \\ \frac{\partial f_n}{\partial x_1}(p) & \frac{\partial f_n}{\partial x_2}(p) & \frac{\partial f_n}{\partial x_3}(p)\dots & \frac{\partial f_n}{\partial x_n}(p) \end{pmatrix} \qquad (3.2.2)$$

This is called the Jacobean matrix [54].

There are several approaches to the study of the stability of the flow of nonlinear systems, but for the purpose of this study, the study will consider the Routh - Hurwitz stability criterion.

Routh-Hurwitz Criterion

Routh-Hurwitz stability criterion is a method that can be used to establish the stability of a system without solving its characteristic equation [50]. Consider the characteristic equation

$$\lambda^{n} + a_1 \lambda^{n-1} + a_2 \lambda^{n-2}\lambda + \dots + a_{n-1}\lambda + a_n = 0 \qquad (3.2.3)$$

The necessary condition for stability is satisfied if all the coefficients $a_i > 0$ where i =1, 2, . . . , n. This is called the Hurwitz matrix. It is composed as follows;

The main diagonal of the matrix contains elements a_1, a_2, a_3,. . . , a_n. The first column contains numbers with odd indices a_1, a_3, a_5, ... In each row, the index of each following number (counting from left to right) is 1 less than the index of its predecessor, all other coefficients a_i with indices greater than n or less than 0 are replaced by zeros. The result is a matrix shown below:

$$\Delta_1 = (a_1), \quad \Delta_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}, \quad \Delta_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{pmatrix},$$

$$\Delta_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & 0 & 0\dots & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0\dots & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 0\dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \qquad (3.2.4)$$

Where$a_n = 0$, if j > n. All of the roots of the polynomial $p(\lambda)$ are negative or have negative real part if only if the determinants of all Hurwitz matrices are positive.

$$\det\Delta_j > 0,\ j = 1, 2...n$$

For the most common systems of the 2nd, 3rd and 4th order, we obtain the following stability criteria:

For a 2^{nd} order system, the condition of the stability is given by

$$det\Delta_1 = a_1 > 0 \ and \ det\Delta_2 = \begin{vmatrix} a_1 & 1 \\ 0 & a_2 \end{vmatrix} = a_1 a_2 > 0$$

Hence $a_1 > 0$, $a_2 > 0$.

For a 3 rd order system, the stability criterion is defined by the inequalities

$$det\Delta_1 = \ a_1 > 0, \ det\Delta_2 = a_1 a_2 > 0, \ det\Delta_3 = \begin{vmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{vmatrix} = a_3 \begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix} = a_3(a_1 a_2 - a_3) > 0$$

Hence, $a_1 > 0, a_2 > 0, a_3 > 0, a_1 a_2 - a_3 > 0$

For a 4th order system, the stability criterion is defined by

$$det\Delta_1 = a_1 > 0,\ det\Delta_2 = a_1 a_2 > 0,\ a_3(a_1 a_2 - a_3) > 0,\ det\Delta_4 = \begin{vmatrix} a_1 & 1 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 \\ 0 & a_4 & a_3 & a_2 \\ 0 & 0 & 0 & a_4 \end{vmatrix}$$

$a_4(a_1 a_2 a_3 - {a_2}^2 a_4 - {a_3}^3) > 0$

Hence, $a_i > 0$ (i =1, ..., 4), $a_1 a_2 - a_3 > 0$, $a_1 a_2 a_3 - {a_1}^2 a4 - {a_3}^2 > 0$

For polynomial (3.2.3) of degree n = 2, 3, 4,… the Routh − Hurwitz criteria are summarized below;

For n = 2 : $a_1 > 0$, and, $a_2 > 0$

$For\ n = 3$: $a_1>0, a_2>0, a_3>0$, and, $a_1 a_2 - a_3 > 0$ (3.2.5)

For n = 4: $a_i > 0$, where i = 1, 2, 3, 4 and, $a_1a_2a_3 - a_2{}^2a_4 - a_3{}^2 > 0$

For $n = a_5\Delta_4$, where $\Delta_4 = a_1a_2a_3 - a_2{}^2a_4 - a_3{}^2 > 0$

For n = 6 $a_6[2a_1{}^2a_2a_5a_6 + a_1{}^2a_4{}^2a_5 + a_1a_2a_3a_4a_5 + 2a_1a_4a_5{}^2 + a_2a_3a_5{}^2 - (a_1{}^3a_6{}^2 + a_1a_2{}^2a_5{}^2 + a_1a_2a_3{}^2a_6 + 3a_1a_3a_5a_6)] > 0$

3.2.3 Basic Reproduction Number (R_S)

The basic reproduction number R_s Kermack and McKendrick is defined as the average number of Secondary infections that occur when one infective individual is introduced into a susceptible population with no immunity to the disease, in the absence of interventions [15]. Therefore, when R_s < 1, the SFE is locally asymptotically stable and the EE is unstable, that is the disease dies out. When R_s > 1, it follows that an infected individual will cause more than one additional infection on average and thus smoking will spread and the EE will be stable, whereas the SFE will be unstable. When R_s = 1, then smoking becomes endemic, meaning smoking remains in the population at a constant rate. This means that the threshold quantity for eradicating smoking is to reduce the value of R_s to a value less than one.

An alternative method proposed by Diekmann, Heester Beek, and Metz and elaborated by Van den Driessche and Watmough gives a way of determining R_s for an ODE compartmental model by using the next-generation matrix [50, 51].

R_s is determined by the dominant eigenvalue of the Jacobean matrix at the SFE. Now calculate R_s using the Next Generation Matrix Approach comprising two matrices F and V. The elements in matrix F constitute the new infections transfer from susceptible, while that of matrix V constitutes the transfer of infections from one compartment to another.

The difference $F_i(x) - V_i(x)$ gives the rate of change of x_i. Then the form of the next-generation matrix from the partial derivatives of F_i and is V_i is

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right], \qquad V = \left(\frac{\partial V_i(x_0)}{\partial x_j}\right)$$

Where, j = 1, 2, 3... n and x_0 is the initial condition of the epidemic, that is free equilibrium point (SFE). The basic reproduction ratio R_s is given by the dominant eigenvalue (spectral radius) of the matrix FV^{-1}. That is,

$$R_s = \rho(FV^{-1}) \qquad (3.2.6)$$

3.3 Model Formulation

To understand how smoking affects a community this thesis uses an idea called a compartmental deterministic model. We take the people in the community. Put them into different groups based on their smoking habits. Some people have never smoked some smoke all the time. Some have quit smoking. Then we see how people move from one group to another over time using math to describe how this happens.

We need to make important assumptions to do this. First we assume that the number of people in each group changes slowly over time like water flowing from one container to another. This lets us use math to understand how people move between groups. Second we assume that the whole process follows rules meaning that if we know how many people start smoking or quit we can predict what will happen to the community without worrying about luck or chance. This is what makes the model work.

These assumptions make sense when we are talking about a community, like the people of Mekelle Town. When there are thousands of people individual stories are not as important. The overall pattern becomes clear. So while we cannot say if one person will start smoking tomorrow we can say how many people in the community will likely start smoking.. That is what this study is trying to find out.

This study uses a compartmental model, called ($PLHQ^tQ^pS_mM$), which divides the community into seven groups based on their smoking habits and how they influence each other. The first three groups are, about how much people smoke:

- The **Potential smoker (P)** compartment contains healthy individuals who have not yet started smoking but are susceptible to starting;
- The **Light smoker (L)** compartment consists of individuals who have recently taken up smoking; and the
- **Heavy smoker (H)** compartment represents those who smoke regularly and heavily.

The model also tracks cessation through two compartments:

- **Temporary Quit (Q^t)** and

- **Permanent Quit (Q^p)** .

  Finally, the model accounts for external influences via the
- **Aware non-smokers (S_m)** , who are conscious of the risks, and
- The **Media (M)** compartment, which represents the dynamic influence of anti-smoking campaigns.

The researcher looked at how people start and stop smoking in a community by watching what people do. The researcher found six kinds of people: people who might start smoking people who smoke a little people who smoke a lot and cannot stop people who stop smoking for a while people who stop smoking for good and people who know the dangers of smoking and never start. The researcher also looked at -smoking messages on television, social media and from health workers but not as a group of people rather as a way to help people stop smoking.

The researcher saw that the people around us matter and people who might start smoking are more likely to start if they are around people who smoke a lot.. If people learn about the dangers of smoking from television and social media they are less likely to start smoking. For people who already smoke it is always possible to stop: people who smoke a little might stop or smoke more and even people who smoke a lot for a time can stop, either for a while or for good. When someone decides to stop smoking for a while the researcher thinks that they really want to stop smoking for good and will not go back to smoking.

The anti-smoking messages on television, social media and from health workers are very important. How well they work depends on how strong they are and how long they last. By looking at how all these things work we can see how to help more people stop smoking. The details of the researchers work are, in Tables 1 and 2. There is a picture of the researcher's idea in section 3.3.3.

Model Assumption

1. The likelihood of an individual's exposure to smoking is independent of demographic factors such as age, sex, social status, or race, as well as local climatic conditions.
2. The population size remains constant over time, as the birth rate is balanced by an equal natural death rate. All newborns are assumed to be susceptible to smoking.
3. An individual's initial exposure to smoking occurs solely through contact with active smokers, which leads to smoking uptake at a non-heavy (light) level.
4. The dynamics of smoking spread are modeled within a closed population; therefore, migration (emigration or immigration) is not considered.
5. Susceptible individuals initiate smoking primarily through social interaction with smokers. However, a portion of the population is assumed to have a natural immunity to smoking temptation and will not take up the habit, regardless of exposure.

3.3.1 Variables and Parameters

Table1: Description of Variables

Variables	Definitions
$P(t)$	The number of potential smokers
$L(t)$	The number of light smokers
$H(t)$	The number of heavy smokers
$Q^t(t)$	The number of temporary smokers
$Q^p(t)$	The number of permanent smokers
$S_m(t)$	The number of aware people
$M(t)$	The media compartment

Table 2: Description of Parameters

Parameter	Definition	Parameter	Definition
π	Recruitment / birth rate	β	Rate: **P** → **L** (initiation)
α_1	Rate: **L** → $\mathbf{Q^p}$ (permanent quit)	α_2	Rate: **L** → **H** (progression)
α_3	Rate: **L** → $\mathbf{Q^t}$ (temporary quit)	σ	Probability: **H** → $\mathbf{Q^t}$
γ	Rate: **H** → quitting ($\mathbf{Q^t}$/$\mathbf{Q^p}$)	η	Rate: $\mathbf{Q^t}$ → $\mathbf{Q^p}$
ω	Relapse rate: $\mathbf{Q^t}$ → **L**	μ	Natural death rate
d_1	Death rate: light smokers (**L**)	d_2	Death rate: heavy smokers (**H**)
d_3	Death rate: temporary quitters ($\mathbf{Q^t}$)	ϵ_0	Rate: **P** → aware ($\mathbf{S_m}$) via media
ϵ_1	Rate: aware ($\mathbf{S_m}$) → **P** (relapse)	$\emptyset$	Media campaign intensity
φ	Media campaign decay rate	$\mathbf{R_s}$	Smoking generation number
μ_m	Media effectiveness decay	$1-\delta$	Probability: **H** → $\mathbf{Q^p}$

3.3.2 Flow Chart

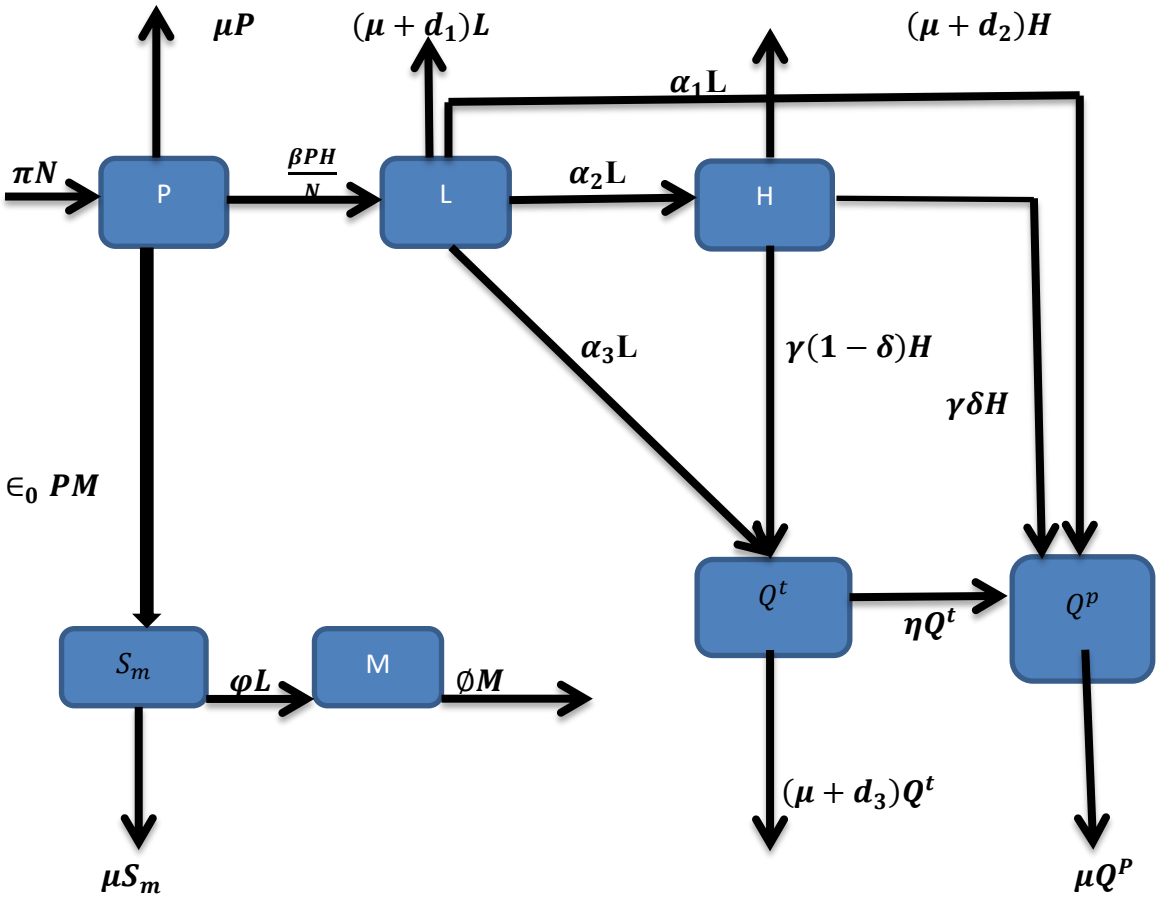


Figure 4: Tobacco transmission Diagram

3.3.3 Model Equation

The human population is categorized into seven classes such that at time t > 0, there are P is Potential smokers, L is light smokers, H is Heavy smokers, Q^t is Temporarily quit smokers, Q^p is Permanently quit smokers , S_m is aware of people and with additional the media compartment (M). Thus, the size of the human population is given as

$$N = P + L + H + Q^t + Q^P + S_M + M$$

Based on the assumption, the researcher is looking at a population that's already reached its maximum size think of it as a community that's completely filled up, where the total number of people (which the researcher will call N) represents this upper limit. In this population, people

are born and die naturally at steady rates: Let's say π represents the birth rate and μ represents the death rate. Here's how it works: new individuals enter the "potential smokers" group at a rate of π N (basically, the birth rate times the total population). Meanwhile, this same group shrinks for two reasons people either dies naturally (which happen at rate μP) or they get introduced to smoking and move out of this category.

At the same time, individuals leave the potential smoker group at a specific rate let's call it $\in_0 P$ to join the 'aware' population. In practical terms, what this means is that the change in the number of potential smokers happens when they actually start smoking. It happens through interactions between potential smokers and people who already smoke both the occasional (light) smokers and the more addicted (heavy) ones. Essentially, the number of these encounters isn't random; it's directly linked to how many potential smokers and how many heavy smokers exist at any given moment. The more of each we have, the more opportunities there are for contact.

Hence the rate of change in potential smokers is

$$\frac{dp}{dt} = \pi N - \frac{\beta PH}{N} - \in_0 PM - \mu P$$

Where, βPH is the rate of exposure.

The people who stop being 'potential smokers' are the exact same people who start becoming 'exposed' to smoking. It's a straight swap one out, one in. This means the exposed group keeps growing, and it happens because they're getting new members straight from the potential smoker pool as those individuals encounter smoking for the first time ($\frac{\beta PH}{N}$). This group shrinks for a few different reasons. First, there's the natural death rate people pass away at a rate of μL. Then there's a specific death rate tied to smoking itself, which we called $d_1 L$. On top of that, people leave the exposed category by moving into other smoking groups: some become permanent quit

smokers (that's$\alpha_1 L$), some become heavy smokers ($\alpha_2 L$), and others become temporary quitters ($\alpha_3 L$). So when we put it all together deaths and people moving out to different smoker types that's what determines how the exposed population changes over time.

$$\frac{dL}{dt} = \frac{\beta PH}{N} - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)L$$

The heavy smoker class H is reduced by natural deaths (μH) and the incidence death rate(d_2) as well as those who move to the temporarily quiet smoker($\gamma\sigma$) and to the permanent quiet smoker class γ(1-σ). The rate of change of smoking individuals will be

$$\frac{dH}{dt} = \alpha_2 L - \big(\mu + d_2 + \gamma\sigma + \gamma(1-\sigma)\big)H$$

$$\frac{dH}{dt} = \alpha_2 L - (\mu + d_2 + \gamma)H$$

The temporarily quiet smoker class Q^t is reduced by both natural death rate (μQ^t) and incidence death rate (d_3) as well as by the proportion those who move to permanently quiet smoker (ηQ^t) and increased by the rate α_3 comes from light smokers and by the rate $\gamma\sigma$ comes from the heavy smoker class.

The rate of change of temporarily quiet smoker individuals will be given by

$$\frac{dQ^t}{dt} = \alpha_3 L + \gamma\sigma H - (\eta + \mu + d_3)Q^t$$

The permanent quiet smoker class increases by the rate α_1 comes from the light smoker, the probability rate $\gamma(1-\sigma)$ and by the rate η comes from the temporarily quiet smoker and then decreased by the natural death rateμQ^p.

The rate of permanent quiet smoker individuals will be given by

$$\frac{dQ^p}{dt} = \alpha_1 L + \gamma(1-\sigma)H + \eta Q^t - \mu Q^p$$

The individuals in the potential smoker by knowing the ill effects of smoking moves to the awareness media compartment without being addicted to smoking habit and the aware population reduced by natural death rate μS_m.

$$\frac{dS_m}{dt} = \epsilon_0 PM - (\varphi L + \mu S_m)$$

Similarly the rate of the media compartment is

$$\frac{dM}{dt} = \varphi L - \emptyset M$$

This leads to the following formulations of the **PLHQ^tQ^p**$S_m M$ model from the description, assumptions, and compartmental diagram;

$$\left.\begin{aligned}
\frac{dP}{dt} &= \pi N - \frac{\beta PH}{N} - \frac{\epsilon_0 PM}{N} - \mu P \\
\frac{dL}{dt} &= \frac{\beta PH}{N} - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)L \\
\frac{dH}{dt} &= \alpha_2 L - (\mu + d_2 + \gamma)H \\
\frac{dQ^t}{dt} &= \alpha_3 L + \gamma\sigma H - (\eta + \mu + d_3)Q^t \\
\frac{dQ^p}{dt} &= \alpha_1 L + \gamma(1-\sigma)H + \eta Q^t - \mu Q^p \\
\frac{dS_m}{dt} &= \frac{\epsilon_0 PM}{N} - (\varphi L + \mu S_m) \\
\frac{dM}{dt} &= \varphi L - \emptyset M
\end{aligned}\right\} \qquad (3.3.1)$$

The non-linear system of differential equations formulated above has initial conditions

$P(0) = P_0, L(0) = L_0, H(0) = H_0, Q^t(0) = {Q^t}_0, Q^p(0) = {Q^p}_0, S_m(0) = S_{m_0}, M(0) = M_0$

Hence $N(t) = P(t) + L(t) + H(t) + Q^t(t) + Q^p(t) + S_m(t) + M(t)$ and set P, L, H, Q^t, Q^p, S_m, Mas proportion of the population (N),

$$\frac{P(t)}{N} + \frac{L(t)}{N} + \frac{H(t)}{N} + \frac{Q^t(t)}{N} + \frac{Q^p(t)}{N} + \frac{S_m}{N} = 1$$

Let

$$\frac{P(t)}{N} = p(t), \frac{L(t)}{N} = l(t), \frac{H(t)}{N} = h(t), \frac{Q^t(t)}{N} = q^t(t), \frac{Q^p(t)}{N} = q^t(t), \frac{S_m(t)}{N} = s_m(t), \frac{M(t)}{N} = m(t)$$

(3.3.2)

$$\Rightarrow p(t) + l(t) + h(t) + q^t(t) + q^p(t) + s_m(t) + m(t) = 1$$

Divide equations (3.3.1) by N and then substitute equation (3.3.2) into equations (3.3.1) respectively to obtain

$$\left.\begin{aligned}
\frac{dp}{dt} &= \pi - \beta ph - \epsilon_0\, pm - \mu p \\
\frac{dl}{dt} &= \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l \\
\frac{dh}{dt} &= \alpha_2 l - (\mu + d_2 + \gamma)h \\
\frac{dq^t}{dt} &= \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t \\
\frac{dq^p}{dt} &= \alpha_1 l + \gamma(1 - \sigma)h + \eta q^t - \mu q^p \\
\frac{ds_m}{dt} &= \epsilon_0 pm - (\varphi s_m + \mu s_m) \\
\frac{dm}{dt} &= \varphi l - \emptyset m
\end{aligned}\right\} \qquad (3.3.3)$$

With initial conditions

P(0)= $p_0 \geq 0$, L(0)= $l_0 \geq 0$, H(0)= $h_0 \geq 0$, (0)= $p_0 \geq 0$, $Q^t(0) = {q^t}_0 \geq 0$, $Q^p(0) = {q^p}_0 \geq 0$

$$S_m(0) = s_{m_0} \geq 0, M(0) = m_0$$

The variable q^p (t) does not appear in the first six equations of the model and can always

be determined from the relation q^p(t)=1−p(t)− l(t)−h(t)− $q^t(t) - s_m(t) - m(t)$

Then the above seven ODEs Equations (3.3.3) are reduced to six ODEs as follows:

$$\left.\begin{aligned} \frac{dp}{dt} &= \pi - \beta ph - \epsilon_0\, pm - \mu p \\ \frac{dl}{dt} &= \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l \\ \frac{dh}{dt} &= \alpha_2 l - (\mu + d_2 + \gamma)h \\ \frac{dq^t}{dt} &= \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t \\ \frac{ds_m}{dt} &= \epsilon_0 pm - (\varphi l + \mu s_m) \\ \frac{dm}{dt} &= \varphi l - \emptyset m \end{aligned}\right\} \quad (3.3.4)$$

3.3.4 Feasible and Non-Negative Solutions

To make sure the model (3.3.1) is realistic, the answers need to make sense biologically. We call the set of answers that work the feasible region. Also, the answers need to be positive, because the number of smokers, people who quit, etc., can't be less than zero in real life.

Invariant Region

The following theorem can obtain this region.

Theorem 3.2.

The solutions of the system (3.3.1) are feasible for all t > 0 if they enter the invariant region

$\Phi = \left\{(P,\ L,\ H,\ Q^t, Q^p, S_m, M) \in \mathcal{R}^7{}_+ : P + L + H + Q^t + Q^p + S_m + M = N \leq \frac{\pi}{\mu}\right\}$.

Proof:

Let $\Phi = (P,\ L,\ H,\ Q^t, Q^p, S_m, M) \in \mathcal{R}^7{}_+$ be any solution of the system (3.3.1) with non-negative initial conditions.

From the model equations, the total population of individuals is given by

N = $P + L + H + Q^t + Q^p + S_m + M$.

Therefore, adding the first 7 differential equations, results in a first-order linear differential equation of the form

$$\frac{dN}{dt} = \pi - \mu N$$

(3.3.5)

The integrating factor (IF) $=e^{\mu t}$

Multiplying both sides of the equation with $e^{\mu t}$ gives

$$e^{\mu t}\ \frac{dN}{dt} + e^{\mu t}\mu N = e^{\mu t}\ \pi$$

$$\frac{d}{dt}(Ne^{\mu t}) = \pi e^{\mu t}$$

(3.3.6)

Integrating equation (3.3.6) on both sides,

$$N(t)e^{\mu t} = \frac{\pi}{\mu}e^{\mu t} + c$$

,Where c is constant.

Applying the initial conditions at t=0, N(0) = N0,then

$$\text{C}= N_0 - \frac{\pi}{\mu}$$

The solution of the linear differential equation then becomes

$$N(t)e^{\mu t} = \frac{\pi}{\mu}e^{\mu t} + N_0 - \frac{\pi}{\mu}$$

$$N(t) = \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right)e^{-\mu t}$$

So that as

$t \to \infty, N(t) \leq \frac{\pi}{\mu}$ (3.3.7)

Therefore, as t $\to \infty$ in (3.3.7) the population of Mekelle town N approaches K = $\frac{\pi}{\mu}$

(that is N $\to$ K = $\frac{\pi}{\mu}$). The parameter K = $\frac{\pi}{\mu}$ is usually called the carrying capacity.

$\Phi = (P,\ L,\ H,\quad Q^t,\ Q^p, S_m, M) \in \mathcal{R}^7{}_+ : P + L + \ H + \ Q^t + Q^p + S_m, + M = N \leq \frac{\pi}{\mu}$,

p$\geq 0, (L, H,\ Q^t, Q^p, S_m, M,) \geq 0$

Therefore, the region Φ is positively-invariant.

Positivity of Solutions

It is now proved that all the variables in the model equations (3.3.4) are non-negative for $\forall t > 0$.

Theorem 3.2.

Let the initial data be $(p, l, h, q^t, s_m, m,)(0) \geq 0 \in \Phi$

Proof

From equation (3.3.4)

$$\frac{dp}{dt} = \pi - \beta ph - \epsilon_0 pm - \mu p$$

$$\frac{dp}{dt} \geq -\mu p$$

$$\frac{dp}{p} \geq -\mu dt$$

Integrating by separation of variables gives

$$\int \frac{dp}{p} \geq \int -\mu dt$$

$$lnp \geq -\mu t + \mathrm{C}$$

Where C is a constant

$$\mathrm{P}(t) \geq e^{-\mu t} + C$$

$$\mathrm{P}(t) \geq e^{-\mu t} . e^{C}$$

$$\mathrm{P}(t) \geq Ae^{-\mu t}$$

Where A= e^{C}

Let $\mathrm{P}(0) = P_0$ at t = 0, $\mathrm{P}(0) = P_0 \geq A$

Therefore $\mathrm{P}(t) \geq P_0\, e^{-\mu t} \geq 0$ Since $\mu > 0$

(3.3.8)

From equation (3.3.4)

$$\frac{dl}{dt} = \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l$$

$$\frac{dl}{dt} \geq -(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l$$

Integrating by separation of variables gives

$$\frac{dl}{l} \geq -(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)dt$$

That is $\int \frac{dl}{l} \geq \int -(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)dt$

$lnl \geq -(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t + \mathrm{c}$

Where C is a constant

$$l(t) \geq e^{-(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t + C}$$

$$l(t) \geq e^{-(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t}.e^{C}$$

$$l(t) \geq Ae^{-(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t}$$

Where A= e^{C}

Let $L(0) = l_0$ at t= 0, $L(0) = l_0 \geq A$

Therefore

$l(t) \geq l_0 e^{-(\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t} \geq 0\ since\ (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)t\ > 0$ (3.3.9)

From equation (3.3.4)

$\frac{dh}{dt} = \alpha_2 l - (\mu + d_2 + \gamma)h$

$\frac{dh}{dt} \geq -(\mu + d_2 + \gamma)h$

Integrating by separation of variables gives

$\frac{dh}{h} \geq -\big(\mu + d_2 + \gamma\sigma + \gamma(1-\sigma)\big)dt$

$\int \frac{dh}{h} \geq \int -\big(\mu + d_2 + \gamma\sigma + \gamma(1-\sigma)\big)dt$

$$lnh \geq -(\mu + d_2 + \gamma)t + C$$

where C is constant.

$$h(t) \geq e^{-(\mu+d_2+\gamma)t+C} = e^{-(\mu+d_2+\gamma)t}.e^{C}$$

$$h(t) \geq Ae^{-\big(\mu+d_2+\gamma\sigma+\gamma(1-\sigma)\big)t} > 0$$

Where A= e^{C}

Let $H(0) = h_0\ at\ t = 0, H(0) = h_0 \geq A$

$$h(t) \geq h_0 e^{-(\mu+d_2+\gamma)t} > 0 \qquad (3.3.10)$$

Since $(\mu + d_2 + \gamma) > 0$

From equation (3.3.4)

$$\frac{dq^t}{dt} = \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t$$

$$\frac{dq^t}{dt} \geq -(\eta + \mu + d_3)q^t$$

$$\frac{dq^t}{q^t} \geq -(\eta + \mu + d_3)dt$$

Integrating by separation of variables gives

$$\int \frac{dq^t}{q^t} \geq \int -(\eta + \mu + d_3)dt$$

$$lnq^t \geq -(\eta + \mu + d_3)t + C$$

where C is a constant.

$$q^t(t) \geq e^{-(\eta+\mu+d_3)t+C} = e^{-(\eta+\mu+d_3)t}e^C$$

$$q^t(t) \geq Ae^{-(\eta+\mu+d_3)t}, \text{ where A}= e^C$$

Let $Q^t(0) = {q_0}^t,\ at\ t = 0, Q^t(0) = {q_0}^t \geq A$

$$q^t(t) \geq {q_0}^t e^{-(\eta+\mu+d_3)t} > 0$$

(3.3.11)

Since $\eta + \mu + d_3 > 0$

From equation (3.3.4)

$$\frac{ds_m}{dt} = \epsilon_0 pm - (\varphi l + \mu s_m)$$

Then, $\frac{ds_m}{dt} \geq -(\varphi s_m + \mu s_m)$

$$\frac{ds_m}{s_m} \geq -\mu dt$$

Integrating by separation of variables gives

$$\int \frac{ds_m}{s_m} \geq \int -\mu dt$$

$$ln s_m(t) \geq -\mu t + c$$

$$s_m(t) \geq e^{-\mu t+c}$$

$$s_m(t) \geq e^{-\mu t}.e^c$$

$$s_m(t) \geq Ae^{-\mu t}$$

Where $A = e^c$ and c is constant.

Let $S_m(0) = s_{m_0}$ at t= 0, $S_m(0) = s_{m_0} \geq A$

$$s_m(t) \geq s_{m_0}e^{-\mu t} > 0. \qquad (3.4.12)$$

Since μ >0

From equation (3.3.4)

$$\frac{dm}{dt} = \varphi l - \emptyset m$$

$$\frac{dm}{dt} \geq -\emptyset m$$

Integrating by separation of variables gives

$$\frac{dm}{m} \geq -\emptyset dt$$

$$\int \frac{dm}{m} \geq \int -\emptyset dt$$

$$lnm(t) \geq -\emptyset t + c$$

where c is constant.

Then $m(t) \geq e^{-\emptyset t+c} = e^{-\emptyset t}.e^{c}$

$$m(t) \geq Ae^{-\emptyset t}$$

Where $A = e^{c}$

Let $M(0) = m_0\ at\ t = 0, M(0) = m_0 \geq A$

$$m(t) \geq m_0 e^{-\emptyset t} > 0 \qquad (3.4.13)$$

Since $\emptyset > 0$.

Hence, all variables are positive for all time t > 0. That is the rate of contact, the rate of exposed, the recovery rate, the birth and death rate are all non-negative.

3.4 Equilibrium Point and Stability

To understand whether the model is stable, meaning whether it eventually settles into a steady pattern rather than spinning out of control or fluctuating endlessly, we first need to find its equilibrium points. Think of an equilibrium point as a resting state, a moment where everything balances out and nothing changes anymore. In mathematical terms, we find these points by taking the main equations from section (3.3.4) and setting the right-hand side to zero. Solving those equations gives us the values where the system is perfectly balanced, like a seesaw with equal weight on both sides. The equilibrium point of the system described by equation (3.3.4), using the conditions from (3.2.1), is given as follows.

$$\frac{dp}{dt} = \frac{dl}{dt} = \frac{dh}{dt} = \frac{dq^t}{dt} = \frac{ds_m}{dt} = \frac{dm}{dt} = 0$$

Hence, equations (3.3.4) will be

$$\left.\begin{aligned} \pi - \beta p h - \epsilon_0\, p m - \mu p &= 0 \\ \beta p h - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1) l &= 0 \\ \alpha_2 l - (\mu + d_2 + \gamma) h &= 0 \\ \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3) q^t &= 0 \\ \epsilon_0 p m - (\varphi l + \mu s_m) &= 0 \\ \varphi l - \emptyset m &= 0 \end{aligned}\right\} \qquad (3.4.1)$$

Since we're working with the standard **PLH$Q^t Q^p S_m M$** epidemiological model basically a framework for tracking how smoking behaviors spread through a population. This model gives us two key equilibrium points to look at. The first is called the smoking-free equilibrium. In this scenario, there are no smokers at all in the population: no light smokers, no heavy smokers, no temporary quitters, no potential smokers, and no recovered or immune individuals all those groups are at zero($l = h = q^t = s_m = M = 0$). The second is the endemic equilibrium. Here, the smoking behavior persists within the population, meaning all those different compartments light smokers, heavy smokers, and temporary quitters, and so on have nonzero numbers of people in them($l, h,\ q^t, s_m, m \neq 0$).

Now, one important assumption we're making is that the birth rate and death rate are equal in other words, π = μ. This keeps the overall population size stable. So from this point forward, we'll be replacing the birth rate with the death rate in our equations.

3.4.1 Smoking Free Equilibrium Point

When we're looking at the smoking-free equilibrium, we're basically imagining a scenario where no smoking exists in the population at all. That means all the smoker-related compartments light smokers, heavy smokers, temporary quitters, and so on are empty. So when we plug that assumption into our equations, we set those values to zero. In other words, we substitute zero for all those variables to see what the system looks like when there's no smoking present.

Substituting $l = h = q^t = s_m = M = 0$ into equations (3.4.1) it becomes

$$\left.\begin{aligned} \pi - \beta p(0) - \epsilon_0 p(0) - \mu p = 0 \\ \beta p(0) - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)(0) = 0 \\ \alpha_2(0) - (\mu + d_2 + \gamma)(0) = 0 \\ \alpha_3(0) + \gamma\sigma(0) - (\eta + \mu + d_3)(0) = 0 \\ \epsilon_0 p - (\varphi l + \mu s_m) = 0 \\ \varphi l - \emptyset m = 0 \end{aligned}\right\}$$

$$\pi - \mu p = 0$$

$$p^* = \frac{\pi}{\mu} \qquad (but\ \pi = \mu)$$

$$p^* = 1.$$

Therefore, the SFE, state in which there is no infection (no smoking) in the society, is given by,

$$(p^*, l^*, h^*, q^{t^*}, s_m{}^*, m) = (1,0,0,0,0,0) \qquad (3.4.2)$$

3.4.2 Endemic Equilibrium Point (EE)

Now let's talk about the endemic equilibrium. This is the opposite of the smoking-free scenario it's the situation where smoking has taken hold and continues to exist within the population over time. In other words, the 'disease' (in this case, smoking behavior) persists and doesn't die out. To

figure out exactly what this looks like meaning, to find the actual numbers of people in each group (p, l, h, q^t, s_m, m) when the system is stable but smoking is still present we need to solve a set of six equations. These equations will give us the values for each compartment in this persistent-smoking scenario.

Then, from equation (3.4.1)

$$\frac{dp}{dt} = \pi - (\beta h + \epsilon_0 m + \mu)p = 0$$

$$h^* = \frac{\pi - p(\epsilon_0 m + \mu)}{\beta p} = \frac{\pi}{\beta p} - \frac{(\epsilon_0 m + \mu)}{\beta}$$

(3.4.3)

From equation (3.4.1)

$$\frac{dh}{dt} = \alpha_2 l - (\mu + d_2 + \gamma)h$$

$$\alpha_2 l - (\mu + d_2 + \gamma)h = 0$$

$$l^* = \frac{(\mu + d_2 + \gamma)h}{\alpha_2} = \frac{bh}{\alpha_2} \qquad (3.4.4)$$

Suppose $a = \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1$

$b = \mu + d_2 + \gamma$ $\qquad$ $c = \eta + \mu + d_3$

From equation (3.4.1)

$$\frac{dl}{dt} = \beta p h - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l$$

Then, $\beta p h - a l = 0$

$$\beta p h = a l \qquad (3.4.5)$$

Substitute $l^* = \frac{bh}{\alpha_2}$ from equation(3.4.4) in to equation(3.4.5), then

Then, $\beta ph = a\frac{bh}{\alpha_2}$

$$p^* = \frac{ab}{\alpha_{2\beta}} \tag{3.4.6}$$

From equation(3.4.3) , we have $h^* = \frac{\pi - p(\epsilon_0 m + \mu)}{\beta p}$

And From equation (3.4.1) we have $\varphi l - \emptyset m = 0$

Then, $m^* = \frac{\varphi l}{\emptyset}$ (3.4.7)

Substitute $l^* = \frac{bh}{\alpha_2}$ from equation(3.4.4) in to equation(3.4.7)

Now let's substitute $p^* = \frac{(\alpha_1+\alpha_2+\alpha_3+\mu+d_1)(\mu+d_2+\gamma)}{\alpha_2\beta}$ or $p^* = \frac{ab}{\alpha_2\beta}$ and $m^* = \frac{\varphi bh}{\alpha_2\emptyset}$ in to equation (3.4.3)

$$h^* = \frac{\alpha_2\pi(\alpha_2\beta\emptyset)}{ab(\alpha_2\beta\emptyset + \epsilon_0\varphi b)} - \frac{\alpha_2\emptyset\mu}{\alpha_2\beta\emptyset + \epsilon_0\varphi b}$$

$$= \frac{\alpha_2\emptyset}{\theta}\left(\frac{\pi}{p} - \mu\right) \tag{3.4.8}$$

Where $\theta = \alpha_2\beta\emptyset + \epsilon_0\varphi b$

From equation(3.4.4), we have $l = \frac{(\mu+d_2+\gamma)h}{\alpha_2} = \frac{bh}{\alpha_2}$

Now substitute $h^* = \frac{\alpha_2\pi(\alpha_2\beta\emptyset)}{ab(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{\alpha_2\emptyset\mu}{\alpha_2\beta\emptyset+\epsilon_0\varphi b} = \frac{\alpha_2\emptyset}{\theta}\left(\frac{\pi}{p} - \mu\right)$

Then, $l^* = \frac{b}{\alpha_2}\left[\frac{\alpha_2\pi(\alpha_2\beta\emptyset)}{ab(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{\alpha_2\emptyset\mu}{\alpha_2\beta\emptyset+\epsilon_0\varphi b}\right] = \frac{b\emptyset}{\theta}\left(\frac{\pi}{p} - \mu\right)$ (3.4.9)

From equation (3.4.1)

$$\frac{dq^t}{dt} = \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t$$

$$\alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t = 0$$

$$q^t = \frac{\alpha_3 l + \gamma\sigma h}{(\eta + \mu + d_3)}$$

$$q^t = \frac{\alpha_3 l + \gamma\sigma h}{c}$$

Now substitute $h^* \; and \; l^* \; from$ equations (3.4.7) and (3.4.8)

i.e.

$$(q^t)^* = \frac{\alpha_3\left(\frac{\pi(\alpha_2\beta\emptyset)}{a(\alpha_2\beta\emptyset + \epsilon_0\varphi b)} - \frac{b\emptyset\mu}{\alpha_2\beta\emptyset + \epsilon_0\varphi b}\right) + \gamma\sigma\left(\frac{\alpha_2\pi(\alpha_2\beta\emptyset)}{ab(\alpha_2\beta\emptyset + \epsilon_0\varphi b)} - \frac{\alpha_2\emptyset}{\alpha_2\beta\emptyset + \epsilon_0\varphi b}\right)}{c}$$

$$= \frac{\emptyset}{\theta c}(\alpha_3 b + \gamma\sigma\alpha_2)\left(\frac{\pi}{p} - \mu\right) \qquad (3.4.10)$$

Next substitute $l^* = \frac{b\emptyset}{\theta}\left(\frac{\pi}{p} - \mu\right)$in to equation (3.4.7)

That is $m^* = \frac{\varphi\left(\frac{\pi(\alpha_2\beta\emptyset)}{a(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{b\emptyset\mu}{\alpha_2\beta\emptyset+\epsilon_0\varphi b}\right)}{\emptyset} = \varphi\left(\frac{\pi(\alpha_2\beta)}{a(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{b}{\alpha_2\beta\emptyset+\epsilon_0\varphi b}\right)$

$$= \frac{\varphi b}{\theta}\left(\frac{\pi}{p} - \mu\right) \qquad (3.4.11)$$

From equation (3.4.1)

$$\epsilon_0 pm - (\varphi l + \mu s_m) = 0$$

$$s_m = \frac{\epsilon_0 pm - \varphi l}{\mu}$$

But from equation (3.4.6) and (3.4.8) $we \; have$

$$p^* = \frac{ab}{\beta\alpha_2} \text{ And } l^* = \frac{\pi(\alpha_2\beta\emptyset)}{a(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{b\emptyset\mu}{\alpha_2\beta\emptyset+\epsilon_0\varphi b} = \frac{b\emptyset}{\theta}\left(\frac{\pi}{p} - \mu\right)$$

$$\text{Then, } (s_m)^* = \frac{\epsilon_0\frac{ab}{\beta\alpha_2} - \varphi\left(\frac{\pi(\alpha_2\beta\emptyset)}{a(\alpha_2\beta\emptyset+\epsilon_0\varphi b)} - \frac{b\emptyset}{\alpha_2\beta\emptyset+\epsilon_0\varphi b}\right)}{\mu}$$

$$(s_m)^* = \frac{\epsilon_0 ab}{\beta\alpha_2\mu} - \frac{\varphi}{\mu}\left(\frac{\pi(\alpha_2\beta\emptyset)}{a(\alpha_2\beta\emptyset + \epsilon_0\varphi b)} - \frac{b\emptyset\mu}{\alpha_2\beta\emptyset + \epsilon_0\varphi b}\right)$$

$$= \frac{\varphi b}{\theta\mu}(\epsilon_0 p - \emptyset)\left(\frac{\pi}{p} - \mu\right) \qquad (3.4.12)$$

At the endemic state, the equilibrium point will be

$$(\ p^*, l^*, h^*, (q^t)^*, (s_m)^*, m^*\ ) = \left(\frac{ab}{\beta\alpha_2}, \frac{b\emptyset}{\theta}\left(\frac{\pi}{p^*} - \mu\right), \frac{\alpha_2\emptyset}{\theta}\left(\frac{\pi}{p^*} - \mu\right), \frac{\emptyset}{\theta c}(\alpha_3 b + \gamma\sigma\alpha_2)\left(\frac{\pi}{p^*} - \mu\right), \frac{\varphi b}{\theta\mu}(\epsilon_0 p - \emptyset)\left(\frac{\pi}{p^*} - \mu\right), \frac{\varphi b}{\theta}\left(\frac{\pi}{p^*} - \mu\right)\right)$$

(3.4.13)

Where $p^* = \frac{ab}{\beta\alpha_2}$

This exists when $\frac{\pi}{p^*} - \mu > 0$.

That is $p^* < \frac{\pi}{\mu}$

3.4.3 Basic Reproductive Number (Rs)

The reproduction number (R_s), is the average number of new smokers generated by a single smoker in a population of potential smokers in his /her whole smoking period. Suppose the matrix F states that a new infection has occurred, V^- states the transfer of individuals who come out of the first class to another class and matrix V^+ states the transfer of individuals who enter class one from another class. Equation (5) can be written in form $\dot{x} = f(x) = f_i(x) - v_i(x)$, with $v_i(x) = v^+{}_i(x) - v^-{}_i(x)$.

Let us now define matrices F and V by $F = \frac{\partial f_i}{\partial x_j}$ and $V = \frac{\partial f_i}{\partial x_j}$ respectively. Reproduction number

is defined in terms of next generation matrix as R_0 = ρ (FV^{-1}). ρ (FV^{-1}) denotes the spectral radius or magnitude of the largest eigenvalue of matrix FV^{-1}. The matrix of new infection term denoted by *F* and the non- singular matrix of remaining transfer term denoted by *V*. Based on the compartment figure (1) and equation (3.3.3) we get the exposed and infected compartments

$$\begin{cases} \dfrac{dl}{dt} = \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l \\ \dfrac{dh}{dt} = \alpha_2 l - (\mu + d_2 + \gamma)h \\ \dfrac{dq^t}{dt} = \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t \end{cases}$$

Let $x = (L, H, Q^t)^T$.Then the above system can be represented in matrix form as shown below

$$\frac{dx_i}{dt} = F_i(x) - V_i(x)$$

$$= \begin{pmatrix} \beta ph \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l \\ -\alpha_2 l + (\mu + d_2 + \gamma)h \\ -\alpha_3 l - \gamma\sigma h + (\eta + \mu + d_3)q^t \end{pmatrix}$$

$$= \begin{pmatrix} \beta ph \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} al \\ -\alpha_2 l + bh \\ -\alpha_3 l - \gamma\sigma h + cq^t \end{pmatrix}$$

Suppose a $= \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1$

b$= \mu + d_2 + \gamma$

c$= \eta + \mu + d_3$

For this system, write down matrix F which rate of new infection in different compartments, differentiated with respect to L, H and Q^t and evaluated at smoking –free equilibrium.

$$F_1 = \beta ph$$

$$F_2 = 0$$

$$F_3 = 0$$

$$\Rightarrow \qquad F = \begin{pmatrix} \frac{\partial F_1}{\partial L} & \frac{\partial F_1}{\partial H} & \frac{\partial F_1}{\partial Q^t} \\ \frac{\partial F_2}{\partial L} & \frac{\partial F_2}{\partial H} & \frac{\partial F_2}{\partial Q^t} \\ \frac{\partial F_3}{\partial L} & \frac{\partial F_3}{\partial H} & \frac{\partial F_3}{\partial Q^t} \end{pmatrix}$$

$$F = \begin{pmatrix} 0 & \beta p & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

But the smoking-free equilibrium is $(1, 0, 0, 0, 0, 0)$, then

$$F = \begin{pmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Now, let's write a new matrix V that defines rate of transfer of infective from one compartment to another

$$V_1 = (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l = al$$

$$V_2 = -\alpha_2 l + (\mu + d_2 + \gamma)h = -\alpha_2 l + bh$$

$$V_3 = -\alpha_3 l - \gamma\sigma h + (\eta + \mu + d_3)q^t = -\alpha_3 l - \gamma\sigma h + cq^t$$

$$\Rightarrow V = \begin{pmatrix} \frac{\partial V_1}{\partial L} & \frac{\partial V_1}{\partial H} & \frac{\partial V_1}{\partial Q^t} \\ \frac{\partial V_2}{\partial L} & \frac{\partial V_2}{\partial H} & \frac{\partial V_2}{\partial Q^t} \\ \frac{\partial V_3}{\partial L} & \frac{\partial V_3}{\partial H} & \frac{\partial V_3}{\partial Q^t} \end{pmatrix}$$

$$V = \begin{pmatrix} \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 & 0 & 0 \\ -\alpha_2 & \mu + d_2 + \gamma & 0 \\ -\alpha_3 & -\gamma\sigma & \eta + \mu + d_3 \end{pmatrix}$$

$$V = \begin{pmatrix} a & 0 & 0 \\ -\alpha_2 & b & 0 \\ -\alpha_3 & -\gamma\sigma & c \end{pmatrix}$$

Then, $|V| = abc$.

Now, let's find the inverse of the matrix V leads to

$$V^{-1} = \frac{1}{|V|} AdjV$$

Where $|V| = abc,\ AdjV = \left(C_{ij}\right)^T$

With $C_{ij} = \begin{pmatrix} bc & \alpha_2 c & \alpha_2\gamma\delta + \alpha_3 b \\ 0 & ac & a\gamma\delta \\ 0 & 0 & ab \end{pmatrix}$, then $AdjV = \begin{pmatrix} bc & 0 & 0 \\ \alpha_2 c & ac & 0 \\ \alpha_2\gamma\delta + \alpha_3 b & a\gamma\delta & ab \end{pmatrix}$

$$V^{-1} = \frac{1}{abc}\begin{pmatrix} bc & 0 & 0 \\ \alpha_2 c & ac & 0 \\ \alpha_2\gamma\delta + \alpha_3 b & a\gamma\delta & ab \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{a} & 0 & 0 \\ \frac{\alpha_2}{ab} & \frac{1}{b} & 0 \\ \frac{\alpha_2\gamma\delta + \alpha_3 b}{abc} & \frac{\gamma\delta}{bc} & \frac{1}{c} \end{pmatrix}$$

$$R_s = \rho(FV^{-1})$$

$$FV^{-1} = \begin{pmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}\begin{pmatrix} \frac{1}{a} & 0 & 0 \\ \frac{\alpha_2}{ab} & \frac{1}{b} & 0 \\ \frac{\alpha_2\gamma\delta + \alpha_3 b}{abc} & \frac{\gamma\delta}{bc} & \frac{1}{c} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{\alpha_2\beta}{ab} & \frac{\beta}{b} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

R_s is given by largest eigenvalue of this matrix.

$|A - \lambda I| = 0$ where A $= \begin{pmatrix} \frac{\alpha_2\beta}{ab} & \frac{\beta}{b} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$ and I is 3 by 3 identity matrix

Then $\begin{vmatrix} \frac{\alpha_2\beta}{ab} - \lambda & \frac{\beta}{b} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0$

$$\left(\frac{\alpha_2\beta}{ab} - \lambda\right)(\lambda^2) = 0$$

$$\Rightarrow \lambda^2 = 0 \text{ or } \frac{\beta\alpha_2}{ab} - \lambda = 0$$

From this matrix we have two eigenvalues $\lambda_1 \; and \; \lambda_2$ which are given by $\lambda_1 = 0 \; or \; \lambda_2 = \frac{\alpha_2\beta}{ab}$

Hence the eigenvalues of FV^{-1} are$\left\{0, \frac{\alpha_2\beta}{ab}\right\}$.

$$\Rightarrow R_s = \rho(FV^{-1})$$

l_2 is the dominant $(largest)$ eigenvalue and becomes the largest reproduction number R_s of the model is

$$\boldsymbol{R_s = \frac{\alpha_2\beta}{ab}} \qquad (3.4.14)$$

Where $a = \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1$

$$b = \mu + d_2 + \gamma$$

Now let's express the endemic equilibrium point in (3.4.12) using$\boldsymbol{R_s = \frac{\alpha_2\beta}{ab}}$.

$$(\; p^*, l^*, h^*, (q^t)^*, (s_m)^*, m^*\;) = \left(\frac{1}{R_s}, \frac{b\emptyset}{\theta}(R_s\pi - \mu), \frac{\alpha_2\emptyset}{\theta}(R_s\pi - \mu), \frac{\emptyset}{\theta c}(\alpha_3 b + \gamma\sigma\alpha_2)(R_s\pi - \right.$$

$$\left.\mu), \frac{\varphi b}{\theta\mu}(\epsilon_o p - \emptyset)(R_s\pi - \mu), \frac{\varphi b}{\theta}(R_s\pi - \mu)\right) (3.4.15)$$

$$R_s\pi - \mu = (R_s - 1)\left(\frac{\alpha_2\beta\pi - ab\mu}{\alpha_2\beta - ab}\right)$$

Then, this exist when $R_s - 1 > 0$

$$R_s > 1.$$

3.4.4 Stability Analysis of Smoking-Free Equilibrium (SFE)

To figure out whether the smoking-free and endemic equilibrium points are stable meaning, whether the system tends to return to these states after a disturbance. We'll use something called the Jacobean matrix, which comes from our set of equations. Essentially, we take the equilibrium points we've found (both the smoking-free and the endemic ones) and plug them into this matrix. From there, we solve the matrix equations to derive what are known as characteristic equations. These characteristic equations are key. They're what we analyze to determine the stability of each equilibrium point.

There are several approaches to the study of the stability of the flow of nonlinear systems, but for the purpose of this work, we shall restrict our work to Routh-Hurwitz stability criterion.

$$\left.\begin{aligned}
\frac{dp}{dt} &= \pi - \beta ph - \epsilon_0\, pm - \mu p = \pi - (\beta h + \epsilon_0\, m + \mu)p = \pi - \tau p, \tau = \beta h + \epsilon_0\, m + \mu \\
\frac{dl}{dt} &= \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l = \beta ph - al, a = \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 \\
\frac{dh}{dt} &= \alpha_2 l - (\mu + d_2 + \gamma)h = \alpha_2 l - bh, h = \mu + d_2 + \gamma \\
\frac{dq^t}{dt} &= \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t = \alpha_3 l + \gamma\sigma h - cq^t, c = \eta + \mu + d_3 \\
\frac{ds_m}{dt} &= \epsilon_0 pm - (\varphi l + \mu s_m) \\
\frac{dm}{dt} &= \varphi l - \emptyset m
\end{aligned}\right\}$$

Finding the Jacobean matrix equation (3.2.2) of the above equations becomes

$$J(p, l, h, q^t, s_m, m) =$$

$$\begin{pmatrix}
\frac{\partial}{\partial \mathrm{p}}(\pi - \tau \mathrm{p}) & \frac{\partial}{\partial \mathrm{l}}(\pi - \tau \mathrm{p}) & \frac{\partial}{\partial \mathrm{h}}(\pi - \tau \mathrm{p}) & \frac{\partial}{\partial \mathrm{q^t}}(\pi - \tau \mathrm{p}) & \frac{\partial}{\partial \mathrm{s_m}}(\pi - \tau \mathrm{p}) & \frac{\partial}{\partial \mathrm{m}}(\pi - \tau \mathrm{p}) \\
\frac{\partial}{\partial \mathrm{p}}(\beta ph - al) & \frac{\partial}{\partial \mathrm{l}}(\beta ph - al) & \frac{\partial}{\partial \mathrm{h}}(\beta ph - al) & \frac{\partial}{\partial \mathrm{q^t}}(\beta ph - al) & \frac{\partial}{\partial \mathrm{s_m}}(\beta ph - al) & \frac{\partial}{\partial \mathrm{m}}(\beta ph - al) \\
\frac{\partial}{\partial \mathrm{p}}(\alpha_2 \mathrm{l} - \mathrm{bh}) & \frac{\partial}{\partial \mathrm{l}}(\alpha_2 \mathrm{l} - \mathrm{bh}) & \frac{\partial}{\partial \mathrm{h}}(\alpha_2 \mathrm{l} - \mathrm{bh}) & \frac{\partial}{\partial \mathrm{q^t}}(\alpha_2 \mathrm{l} - \mathrm{bh}) & \frac{\partial}{\partial \mathrm{s_m}}(\alpha_2 \mathrm{l} - \mathrm{bh}) & \frac{\partial}{\partial \mathrm{m}}(\alpha_2 \mathrm{l} - \mathrm{bh}) \\
\frac{\partial}{\partial \mathrm{p}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) & \frac{\partial}{\partial \mathrm{l}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) & \frac{\partial}{\partial \mathrm{h}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) & \frac{\partial}{\partial \mathrm{q^t}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) & \frac{\partial}{\partial \mathrm{s_m}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) & \frac{\partial}{\partial \mathrm{m}}(\alpha_3 \mathrm{l} + \gamma\sigma \mathrm{h} - \mathrm{cq^t}) \\
\frac{\partial}{\partial \mathrm{p}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) & \frac{\partial}{\partial \mathrm{l}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) & \frac{\partial}{\partial \mathrm{h}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) & \frac{\partial}{\partial \mathrm{q^t}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) & \frac{\partial}{\partial \mathrm{s_m}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) & \frac{\partial}{\partial \mathrm{m}}(\epsilon_0 \mathrm{pm} - (\varphi \mathrm{l} + \mu \mathrm{s_m})) \\
\frac{\partial}{\partial p}(\varphi l - \emptyset m) & \frac{\partial}{\partial l}(\varphi l - \emptyset m) & \frac{\partial}{\partial h}(\varphi l - \emptyset m) & \frac{\partial}{\partial \mathrm{q^t}}(\varphi l - \emptyset m) & \frac{\partial}{\partial \mathrm{s_m}}(\varphi l - \emptyset m) & \frac{\partial}{\partial m}(\varphi l - \emptyset m)
\end{pmatrix}$$

$$J(p,l,h,q^t,s_m,m)=\begin{pmatrix}-(\beta h+\epsilon_0 m+\mu) & 0 & -\beta h & 0 & 0 & -\epsilon_0 p\\ \beta h & -a & \beta p & 0 & 0 & 0\\ 0 & \alpha_2 & -b & 0 & 0 & 0\\ 0 & \alpha_3 & \gamma\sigma & -c & 0 & 0\\ \epsilon_0 m & -\varphi & 0 & 0 & -\mu & \epsilon_0 p\\ 0 & \varphi & 0 & 0 & 0 & -\emptyset\end{pmatrix} \quad (3.4.16)$$

Local Stability Analysis of Smoking free Equilibrium Point

At the smoking free equilibrium, $p=1, l=h=q^t=s_m=m=0$, the Jacobian matrix become

$$J(1,0,0,0,0,0)=J(E_o)=\begin{pmatrix}-\mu & 0 & 0 & 0 & 0 & -\epsilon_0\\ 0 & -a & \beta & 0 & 0 & 0\\ 0 & \alpha_2 & -b & 0 & 0 & 0\\ 0 & \alpha_3 & \gamma\sigma & -c & 0 & 0\\ 0 & -\varphi & 0 & 0 & -\mu & \epsilon_0\\ 0 & \varphi & 0 & 0 & 0 & -\emptyset\end{pmatrix} \quad (3.4.17)$$

, Where J_{SFE} represents Jacobian matrix at smoking-free equilibrium. From here, we begin solving the matrix equation$J_{SFE}-\lambda I$. Therefore, $J_{SFE}-\lambda I$ implies that

$$\begin{pmatrix}-\mu & 0 & 0 & 0 & 0 & -\epsilon_0\\ 0 & -a & \beta & 0 & 0 & 0\\ 0 & \alpha_2 & -b & 0 & 0 & 0\\ 0 & \alpha_3 & \gamma\sigma & -c & 0 & 0\\ 0 & -\varphi & 0 & 0 & -\mu & \epsilon_0\\ 0 & \varphi & 0 & 0 & 0 & -\emptyset\end{pmatrix}-\begin{pmatrix}\lambda & 0 & 0 & 0 & 0 & 0\\ 0 & \lambda & 0 & 0 & 0 & 0\\ 0 & 0 & \lambda & 0 & 0 & 0\\ 0 & 0 & 0 & \lambda & 0 & 0\\ 0 & 0 & 0 & 0 & \lambda & 0\\ 0 & 0 & 0 & 0 & 0 & \lambda\end{pmatrix}$$

$$J_{SFE}-\lambda I=\begin{pmatrix}-\mu-\lambda & 0 & 0 & 0 & 0 & -\epsilon_0\\ 0 & -a-\lambda & \beta & 0 & 0 & 0\\ 0 & \alpha_2 & -b-\lambda & 0 & 0 & 0\\ 0 & \alpha_3 & \gamma\sigma & -c-\lambda & 0 & 0\\ 0 & -\varphi & 0 & 0 & -\mu-\lambda & \epsilon_0\\ 0 & \varphi & 0 & 0 & 0 & -\emptyset-\lambda\end{pmatrix}$$

$$det\,(J_{SFE}-\lambda I)=\begin{vmatrix}-\mu-\lambda & 0 & 0 & 0 & 0 & -\epsilon_0\\ 0 & -a-\lambda & \beta & 0 & 0 & 0\\ 0 & \alpha_2 & -b-\lambda & 0 & 0 & 0\\ 0 & \alpha_3 & \gamma\sigma & -c-\lambda & 0 & 0\\ 0 & -\varphi & 0 & 0 & -\mu-\lambda & \epsilon_0\\ 0 & \varphi & 0 & 0 & 0 & -\emptyset-\lambda\end{vmatrix}=0$$

$$det\,(J_{SFE} - \lambda\, I\,) = (\mu + \lambda)(a + \lambda)(b + \lambda)(c + \lambda)(\mu + \lambda)(\emptyset + \lambda) = 0$$

The characteristic equation becomes,

$$\begin{aligned} = \lambda^6 + (a\ +\ b\ +\ c\ +\ \emptyset)\,\lambda^5\ + (2\mu(a\ +\ b\ +\ c\ +\ \emptyset)\ +\ a\,b\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset \\ +\ c\,\emptyset)\,\lambda^4 + (\mu^2(a\ +\ b\ +\ c\ +\ \emptyset)\ +\ 2\mu(ab\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset \\ +\ c\,\emptyset))\ \lambda^3 + (\mu^2(ab\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset\ +\ c\,\emptyset)\ +\ 2\mu(a\,b\,c \\ +\ a\,b\ \emptyset\ +\ a\,c\,\emptyset\ +\ b\,c\,\emptyset))\,\lambda^2 + (\mu^2(a\,b\,c\ +\ a\,b\,\emptyset\ +\ a\,c\,\emptyset\ +\ b\,c\,\emptyset) \\ +\ 2\mu a\,b\,c\,\emptyset)\ \lambda + \mu^2 a\,b\,c\,\emptyset \end{aligned}$$

Expanding and arranging will give us

$$\left.\begin{array}{c} a_1 = a\ +\ b\ +\ c\ +\ \emptyset \\ a_2 = 2\mu(a\ +\ b\ +\ c\ +\ \emptyset)\ +\ a\,b\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset\ +\ c\,\emptyset \\ a_3 = \mu^2(a\ +\ b\ +\ c\ +\ \emptyset)\ +\ 2\mu(ab\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset\ +\ c\,\emptyset) \\ a_4 = \mu^2(ab\ +\ a\,c\ +\ a\,\emptyset\ +\ b\,c\ +\ b\,\emptyset\ +\ c\,\emptyset)\ +\ 2\mu(a\,b\,c\ +\ a\,b\ \emptyset\ +\ a\,c\,\emptyset\ +\ b\,c\,\emptyset) \\ a_5 = \mu^2\,(a\,b\,c\ +\ a\,b\,\emptyset\ +\ a\,c\,\emptyset\ +\ b\,c\,\emptyset)\ +\ 2\mu a\,b\,c\,\emptyset \\ a_6 = \mu^2\ a\,b\,c\,\emptyset \end{array}\right\} \qquad (3.4.18)$$

And we can write $a_6 = (R_s - 1)\left(\frac{(\mu ab)^2 c}{\alpha_2 \beta - ab}\right)$

The above characteristics becomes,

$$\lambda^6 + a_1\,\lambda^5\ + a_2\,\lambda^4 + a_3\ \lambda^3 + a_4\,\lambda^2 + a_5\ \lambda + a_6 = 0$$

Hurwitz matrix using the coefficients of $a_{\ i}$ where $i = 1, 2, \ldots\ldots 6$ is equal to

$$H_6 = \begin{pmatrix} a_1 & 1 & 0 & 0 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 \\ a_7 & a_6 & a_5 & a_4 & a_3 & a_2 \\ a_9 & a_8 & a_7 & a_6 & a_5 & a_4 \\ a_{11} & a_{10} & a_9 & a_8 & a_7 & a_6 \end{pmatrix} \qquad (3.4.19)$$

$$= \begin{pmatrix} a_1 & 1 & 0 & 0 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 \\ 0 & a_6 & a_5 & a_4 & a_3 & a_2 \\ 0 & 0 & 0 & a_6 & a_5 & a_4 \\ 0 & 0 & 0 & 0 & 0 & a_6 \end{pmatrix}$$

Where $a_j = 0$ if $j > n$

$$\begin{vmatrix} a_1 & 1 & 0 & 0 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 \\ 0 & a_6 & a_5 & a_4 & a_3 & a_2 \\ 0 & 0 & 0 & a_6 & a_5 & a_4 \\ 0 & 0 & 0 & 0 & 0 & a_6 \end{vmatrix}$$

Through a simple computation, we obtain that

$$a_6[2{a_1}^2a_2a_5a_6 + {a_1}^2{a_4}^2a_5 + a_1a_2a_3a_4a_5 + 2a_1a_4{a_5}^2 + a_2a_3{a_5}^2 - ({a_1}^3{a_6}^2 + a_1{a_2}^2{a_5}^2 + a_1a_2{a_3}^2a_6 + 3a_1a_3a_5a_6)] > 0 \quad (3.4.20)$$

According to the theorem of Routh-Hurwitz, it follows that all the roots of the characteristic's equation have negative real parts. Therefore, the smoking-free equilibrium is locally asymptotically stable.

3.4.5 Stability Analysis of the Endemic Equilibrium

For the stability analysis of the endemic equilibrium point, we will make use of the Jacobian Matrix of the model equations

$$\begin{cases} \frac{dp}{dt} = \pi - \beta ph - \epsilon_0\, pm - \mu p = 0 \\ \frac{dl}{dt} = \beta ph - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1)l = 0 \\ \frac{dh}{dt} = \alpha_2 l - (\mu + d_2 + \gamma)h = 0 \\ \frac{dq^t}{dt} = \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3)q^t = 0 \\ \frac{ds_m}{dt} = \epsilon_0 pm - (\varphi l + \mu s_m) = 0 \\ \frac{dm}{dt} = \varphi l - \emptyset m = 0 \end{cases} \quad (3.4.21)$$

$p^*, l^*, h^*, (q^t)^*, (s_m)^*$ and m^* Substituting these values into the Jacobian matrix equation (3.4.16) above and solving eigenvalue $\det(J - \lambda I) = 0$

$$J(p^*, l^*, h^*, (q^t)^*, (s_m)^*, m^*) = J_{EE=}\begin{pmatrix} -(\beta h^* + \epsilon_0 m^* + \mu) & 0 & -\beta h^* & 0 & 0 & -\epsilon_0 p^* \\ \beta h^* & -a & \beta p^* & 0 & 0 & 0 \\ 0 & \alpha_2 & -b & 0 & 0 & 0 \\ 0 & \alpha_3 & \gamma\sigma & -c & 0 & 0 \\ \epsilon_0 m^* & -\varphi & 0 & 0 & -\mu & \epsilon_0 p^* \\ 0 & \varphi & 0 & 0 & 0 & -\emptyset \end{pmatrix} \quad (3.4.22)$$

Therefore, $\det(J_{EE} - \lambda I) = 0$

$$\det(J_{EE} - \lambda I) = \begin{vmatrix} -(\beta h^* + \epsilon_0 m^* + \mu) - \lambda & 0 & -\beta h^* & 0 & 0 & -\epsilon_0 p^* \\ \beta h^* & -a - \lambda & \beta p^* & 0 & 0 & 0 \\ 0 & \alpha_2 & -b - \lambda & 0 & 0 & 0 \\ 0 & \alpha_3 & \gamma\sigma & -c - \lambda & 0 & 0 \\ \epsilon_0 m^* & -\varphi & 0 & 0 & -\mu - \lambda & \epsilon_0 p^* \\ 0 & \varphi & 0 & 0 & 0 & -\emptyset - \lambda \end{vmatrix} = 0 \quad (3.4.23)$$

Through a simple computation, we obtain that

$$(x + y\lambda + z\lambda^2 + \lambda^3)\left[\left((a+b)\lambda + \lambda^2\right)(\beta h^* + \epsilon_0 m^* + \mu + \lambda) + \alpha_2(\beta h^*)^2\right] = 0$$

Where $x = c\mu\emptyset, y = c\mu + \emptyset(c + \mu), \quad z = c + \mu + \emptyset$

Then the characteristics equation becomes

$$\lambda^6 + a_1\lambda^5 + a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6 = 0$$

Where $a_1 = \beta h^* + \epsilon_0 m^* + \mu + z$

$$a_2 = z(a+b)(\beta h^* + \epsilon_0 m^* + \mu + z)$$

$$a_3 = x + (y+z)(\beta h^* + \epsilon_0 m^* + \mu) + \alpha_2(\beta h^*)^2$$

$$a_4 = x(a + b + \beta h^* + \epsilon_0 m^* + \mu) + z\alpha_2(\beta h^*)^2$$

$$a_5 = x(a+b)(\beta h^* + \epsilon_0 m^* + \mu) + y\alpha_2(\beta h^*)^2$$

$$a_6 = x\alpha_2(\beta h^*)^2$$

The root of characteristics equation solved using Routh-Hurwitz stability criterion,

if $a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_5 > 0, a_6 > 0,$

Through a simple computation, we obtain that

$$a_6[2{a_1}^2 a_2 a_5 a_6 + {a_1}^2 {a_4}^2 a_5 + a_1 a_2 a_3 a_4 a_5 + 2a_1 a_4 {a_5}^2 + a_2 a_3 {a_5}^2 - ({a_1}^3 {a_6}^2 + a_1 {a_2}^2 {a_5}^2 + a_1 a_2 {a_3}^2 a_6 + 3a_1 a_3 a_5 a_6)] > 0$$

According to the theorem of Routh-Hurwitz, it follows that all the roots of the characteristic equation have negative real parts. Therefore, the endemic equilibrium is locally asymptotically stable.

3.5. Sensitivity analysis

The researcher conducted a sensitivity analysis of the fundamental reproduction number with regard to various parameters in this section. This is accomplished by applying the Normalized Forward Sensitivity Index (70) method. The relative change in a variable when a parameter is modified can be computed using the sensitivity indices. It is employed to identify the factors that most significantly impact (Rs) and on which different intervention strategies should be

concentrated. The ratio of the relative change in the variable to the relative change in the parameter is the definition of the forward sensitivity index of a variable with respect to a specific parameter. The variable can be described in terms of partial derivatives if it is a differentiable function of the given parameter. The forward sensitivity index of R_s with respect to the parameter k is defined as follows:

$$\omega_K^{R_S} = \frac{\partial R_S}{\partial k} . \frac{k}{R_S} \tag{3.4.24}$$

Chapter Four: Optimal Control

Fundamentally, the optimal control is concerned with the determination of the optimal means of controlling a system at a given time. Consider it as an attempt to appear at the optimal strategy which can bring you the most optimal results in accordance with a certain aim. Each control problem is associated with a cost function more or less a measure of our performance in terms of where the system is and what decisions we are making in between. What does an optimal control then really look like? It consists of the differential equations to plot the action of your control variables through time in order to maintain costs at a minimum. However, it is also possible to find this sweet spot in two main ways now. One is the Hamilton-Jacobi-Bellman equation as long as you can solve the equation; you have yourself a sufficient condition of optimality. The other is the maximum principle of Pontryagin (sometimes known as the minimum principle, again depending on your framing) which provides you with the conditions you need. Any of them can take you there they are only varying mathematical instruments with which to solve same fundamental problem how to arrive at the best possible choices in a dynamic system [59].

4.1 Introduction to Optimal Control

By optimization, we are just referring to the process of coming up with the most appropriate solution to an issue be it increased gains or reduced losses. In order to perform such an activity, researchers frequently resort to numerical mathematics, which is all about the creation of the quantitative ways of approaching the solution of mathematical problems with a close eye towards the errors and shortcomings. This in practice can usually imply the use of such tools as MATLAB to compute numerical algorithms that can estimate solutions to optimal control problems that is where optimal management theory comes in. Such methods found particular application in such areas as mathematical epidemiology, where scientists are attempting to learn the propagation and effect of infectious illnesses on populations. Their models are not merely found in books, however; they are in fact involved in the life of diagnosis, prevention, treatment, and management programs that take place in the real world to assist in decision making and saving lives [60, 61].

4.2 Optimal control problem

A standard OC problem requires, in a time t, with$t_0 \leq$ t $\leq t_f$, a performance index or cost functional (J[x(t),u(t)]), a set of state variables (x(t) ϵ X), and a set of control variables (u(t) ϵ U). The two are to be found for a given objective functional to be maximized, with u(t) being piecewise continuous. This chapter shall be developed in a style closely related to Lenhart and Workman [62].

Definition1 (Basic OC Problem in Lagrange formulation): An OC problem is in the form

$$max_u J[x(t), u(t)] = \int_{t_0}^{t_f} f\big(t, x(t), u(t)\big)dt$$

$$\text{Subject to } x' = g\big(t, x(t), u(t)\big) \tag{4.1}$$

$$x(t_0) = x_0, x(t_1)$$

The value of $x\big(t_f\big)$ is either fixed at or unrestrained, meaning it can be free. For our purposes, f and g will always be continuously differentiable functions in all t arguments. The control set U is assumed to be a Lebesgue measurable function. Thus, as the control(s) will always be piecewise continuous, the associated states will always be piecewise differentiable. We have always been looking for the maximum of a function. The maximization and minimization problems are reducible to each other by negating the cost functional: min{J}=-max{J}.

Formulation of optimal control problems

There are many different types of optimal control problems depending on the performance index, whether time is continuous or discrete, the types of constraints and the variables that can be chosen. The following elements are required in setting an optimal control problem correctly;

• a mathematical model that symbolize the system being controlled.

• a well-defined performance index to evaluate outcomes.

• a complete description of boundary conditions for states, as well as any restrictions that the states and controls must satisfy.

• a clear indication of variables that are free to vary.

Optimal Control Theory

In the 1950s, two mathematicians, Lev Pontryagin, and Richard Bellman, had invented what is now known as optimal control theory. It is basically a mathematical method of determining the most optimal ways of guiding or management of systems and it has come in quite handy when it comes to comparison of various strategies in fields such as diagnosis, prevention, treatment and management programs. Reading the literature, there is no deficit of sound research out there as to epidemic models, and how optimal control is applied to them. Optimal control is actually nothing more than a thought process as to the control strategies that allow us to determine not only how to control something, but how to control it in the optimal manner [63].

A special form of optimization problem called an optimal control problem is one whose aim is not merely to determine a unique, best value, and therefore cannot be considered an optimal control problem, but rather to determine a entire function which is an optimizer. That is, we are attempting to determine an optimal course of the controls of a dynamic system along with the states of the system with respect to time, all in an effort to minimize the so-called performance index (which is essentially the value of how you are doing).

In this arrangement, there are two important players. State variable is defined as the present position of the system it is a snapshot of the position of things. Instead, the control aspect is what you are actually adjusting which is the way data is recorded, processed, transmitted, to control the system. These two functions combined define the behavior of the system and the way you proceed to find that optimum control strategy. By these definitions, a simple optimal control problem may be defined. We shall call this fundamental problem our standard problem (SP).

$$\text{Standard } max_u J[x(t), u(t)] = \int_{t_0}^{t_f} f\big(t, x(t), u(t)\big)dt$$

$$\text{Subject to } x' = g\big(t, x(t), u(t)\big) \qquad (4.2)$$

$$x(t_0) = x_0, \qquad x(t_1)$$

In optimal control problems we are seeking the so-called optimal control, denoted u, which is the function that maximizes our objective, J(u), in the most optimal way. In this specific configuration, we are assuming that the upper limit or restriction of the control is there is none that will restrict its freedom to assume any value that we require. At this point, the control is no

longer in isolation. It is dependent on both the time (t) and the condition of the system, which we monitor with the state equation x(t). The control-state relationship is characterized by equations basically, there is a relationship between the control you choose and the resulting state path of which we may write x(u) to emphasize this dependency. Strictly speaking, x is actually a time-dependent function; however, it is convenient to write x(u) to remind ourselves that it is a path that the state takes that depends on the choice of control that we make.

The equations also enumerate where the system has to begin and also the ending of the boundaries of the system as well as any restrictions the state must obey. Here we are establishing it in a manner that the state will have the freedom to develop as time goes by with no artificial boundaries being placed.

Fundamentally, our optimal control problem is associated with a series of requirements that should be fulfilled. In mathematics these necessary conditions are what we refer to as requirements that have to be met in order to have a solution, but satisfying the requirements does not ensure that you are dealing with the correct solution. In order to solve our specific problem (also known as SP), there are specific necessary conditions that we are required to deal with. They were calculated already in the 1950s by the mathematician Lev Pontryagin and his Moscow colleagues in the work that prefigured much of the modern development of the optimal control field [64].

Pontryagin initially introduced ad -joint function to be added to the differentiation equation of the objective functional. These functions serve the same purpose as the Lagrange multipliers of multivariable calculus. The conditions required to be able to solve the basic problems are obtained by the so-called Hamilton, H, provided by equation.

$$H(t,P,L,H,Q^t,Q^t,S_M,M) = f(t,P,L,H,Q^t,Q^p,S_M,M,u) + \sum_1^7 \lambda_i g_i(t,P,L,H,Q^t,Q^t,S_M,M,u)$$

Here λ denotes the ad joint and is dependent on t, x, and u. Using this, Pontryagin's determined that the following conditions are satisfied by the optimal control, denoted as u^*, when the Hamiltonian is nonlinear in u.

$$\frac{\partial(H)}{\partial(u)} = 0 \; at \; u = u^* \Longrightarrow f_u(t,x,u) + \lambda g(t,x,u) = 0 \Longrightarrow Optimality\ condition$$

$$\lambda' = \frac{-\partial(H)}{\partial(x_i)} \Longrightarrow \lambda' = -(f_x + \lambda g_x) \Longrightarrow adjoint\ condition$$

$$\lambda(t_f) = 0 \Longrightarrow transversality\ condition$$

$$x' = g(t, x, u)$$

$x(t_0) = x_0$ Dynamics of the state Equation

With these conditions, there is now a process on how to solve the standard problem defined by SP.

Optimal control application

In light of these variables, the control group (u_1) aims to prevent the susceptible population or potential smoker from becoming smokers by providing counseling, therapy, and other value-added sessions to help them avoid developing a smoking habit and deteriorating their own health.

The second control variable (u_2) is the government's prohibition against smoking, especially in public areas. This will be a mandatory measure for the smoking section. It has been noticed that light smokers who smoke in public settings tend to give up when they come into touch with law enforcement officers. In other words, by increasing the number of law enforcement officers in public areas, the proportion of light smokers will likely decline, and fewer people will likely decide to give up smoking.

The control group (u_3), on the other hand, is concentrated on implementing treatment initiatives such as self-help programs, pharmaceutical and counseling combinations, and nicotine replacement therapy. These controls were added with the purpose of observing whether there is a noteworthy decrease in the number of chain smokers and whether there is a noteworthy increase in the number of people who give up smoking.

On the interval $[0, t_f]$, where t_f is the fixed period to which these controls are applied, these two control functions are limited or are bounded and Lebesgue integrable. It is presumed that the two controls fall within the range of 0 to 1. This assumption is reinforced by the fact that these controls are applied according to their percentage of effect. Values zero and one indicate that no intervention methods are proposed or implemented, respectively, and that every effort is being made to put these policies into effect.

Thus, we have the following optimal control problem to minimize the objective functional

$$J(u_1, u_2, u_3) = \int_0^T \left(A_1 P + A_2 H + A_3 L + \frac{1}{2}(B_1 {u_1}^2 + B_2 {u_2}^2 + B_3 {u_3}^2) \right) dt \qquad (4.3)$$

$PLHQ^tQ^tS_mM$ Model with optimal control

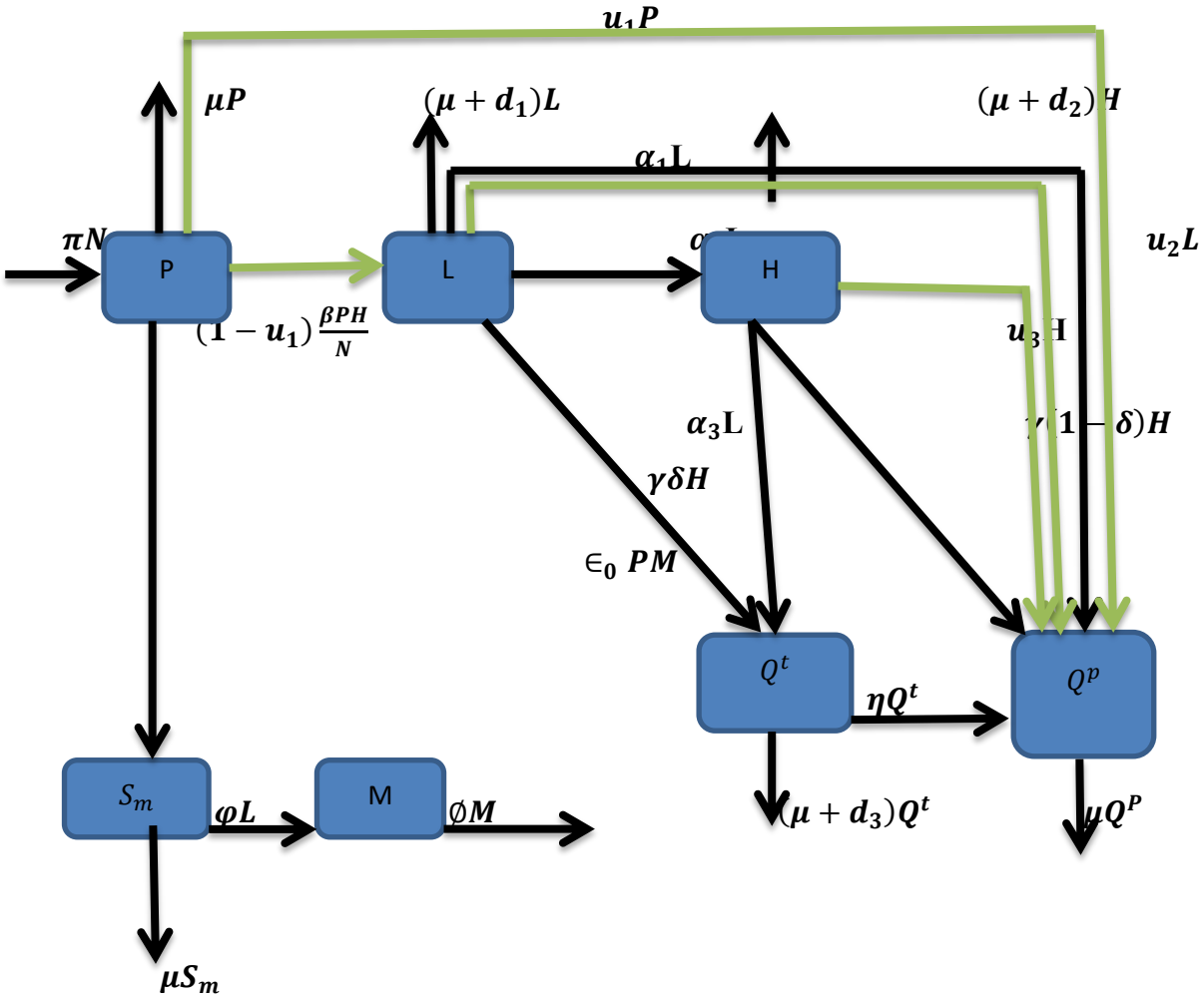


Figure 5: Tobacco Transmission Diagram with optimal control

$PLHQ^tQ^tS_mM$ Model Equation with optimal control

The optimal control problem is stated as follows in light of the aforementioned presumptions.

$$\left.\begin{aligned}
\frac{dP}{dt} &= \pi N - (1-u_1)\,\frac{\beta PH}{N} - \frac{\epsilon_0 PM}{N} - u_1 P \\
\frac{dL}{dt} &= (2-u_1)\,\frac{\beta PH}{N} - (\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2)L \\
\frac{dH}{dt} &= \alpha_2 L - (\mu+d_2+\gamma+u_3)H \\
\frac{dQ^t}{dt} &= \alpha_3 L + \gamma\sigma H - (\eta+\mu+d_3)Q^t \\
\frac{dQ^p}{dt} &= \alpha_1 L + \gamma(1-\sigma)H + \eta Q^t - \mu Q^p + u_1 P + u_2 L + u_3 H \\
\frac{dS_m}{dt} &= \frac{\epsilon_0 PM}{N} - (\varphi L + \mu S_m) \\
\frac{dM}{dt} &= \varphi L - \emptyset M
\end{aligned}\right\} \qquad (4.4)$$

We can linearized this in to

$$\left.\begin{aligned}
\frac{dp}{dt} &= \pi - (1-u_1)\beta ph\ - \epsilon_0\, pm - u_1 p \\
\frac{dl}{dt} &= (1-u_1)\beta ph - (\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2)l \\
\frac{dh}{dt} &= \alpha_2 l - (\mu+d_2+\gamma+u_3)h \\
\frac{dq^t}{dt} &= \alpha_3 l + \gamma\sigma h - (\eta+\mu+d_3)q^t \\
\frac{dq^p}{dt} &= \alpha_1 l + \gamma(1-\sigma)h + \eta q^t - \mu q^p + u_1 p + u_2 l + u_3 h \\
\frac{ds_m}{dt} &= \epsilon_0\, pm - (\varphi l + \mu s_m) \\
\frac{dm}{dt} &= \varphi l - \emptyset m
\end{aligned}\right\}$$

Table 3: Description of state variables with control variables

Parameters	Definitions
π	Rate of recruitment/birth rate
β	Rate of conversion from nonsmoker to smoker
α_1	The rate of conversion from light smoker to permanent quit
α_2	The rate of conversion from light smokers to heavy smoker
α_3	The rate of conversion from light temporary quit smoker
σ	The probability of conversion from smoker to temporarily quit
$\gamma\delta$	The rate probability convert into temporarily quit smoking
μ	Natural death rate
d_1	Death rate of smokers in the light smokers
d_2	Death rate of smokers in the heavy smokers
d_3	Death rate of smokers in the temporary light smokers
ϵ_0	Rate at which potential smoker aware about the ill effects of smoking and remains in the awareness media compartment

$\emptyset$	Rate of addition of media campaigns that depends on number of smokers
φ	Decay rate of media campaigns
$1-\delta$	The probability of conversion from smoker to permanent quit smokers
η	The rate of conversion from temporary quit to permanent quit
R_s	Smoking generation number
u_1	control variable counseling with media education
u_2	control variable ban on smoking particularly in public places
u_3	Control variable implementing treatment initiatives

Steps in formulating optimal system of an optimal control problem

1. Formulate the Hamiltonian for the problem

$$\mathrm{H}(t, x, u_1, u_2, \lambda) = f(t, x, u_1, u_2) + \lambda g(t, x, u_1, u_2)$$

2. Write the ad- joint differential equation, transversality boundary condition, and optimality condition.

$$\lambda' = \frac{-\partial H}{\partial(x)} \Rightarrow \lambda' = -(f_x + \lambda g_x) \Rightarrow adjoint\ function$$

$$\lambda(t_f) = 0 \Rightarrow transversality\ condition$$

$\frac{-\partial H}{\partial(x)} = 0$. At $u_1 = u_1{}^*, u_2 = u_2{}^* \Rightarrow f_u(t, x, u_1, u_2) + \lambda g(t, x, u_1, u_2) = 0$

$$\Rightarrow Optimality\ condition$$

3. Solve for u^* in terms of x^* and λ
4. After finding the optimal states and ad joint, solve for the optimal control.

4.3 Optimal control problem

If $x(t)$ represents the group of individuals($potential\ smokers$) to be educated using media to prevent the susceptible population or potential smoker from becoming smokers by providing counseling, therapy, and other value-added sessions to help them avoid developing a smoking habit and deteriorating their own health and $\big(u_1(t), u_2(t), u_3(t)\big)\,\epsilon\,(u_1, u_2, u_3)$ represents the control where the control sets $u_1, u_2\ and\ u_3$. The control function $u(t)$ with $0 \leq u_1(t), u_2(t), u_3(t) \leq 1$ represents the fraction of susceptible or potential smoker and heavy smokers or chain smokers that requires counseling and treatment. In addition to this ban smoking

in public places and then the light smokers will tend to quit smoking in public places. When the values of $u_1(t), u_2(t) and\ u_3(t)$ are close to one the control methods failure is too low or with high implementation costs where as a value zero signifies that no intervention strategies are put-forth and put into action.

The optimal control problem is to minimize the multiple objectives cost functional J to be minimized considering the costs of control methods of susceptible or potential smokers, light smokers and the heavy smoking individuals given by:

$$J(u_1, u_{2,}\, u_3) = \int_0^T \left(A_1 P + A_2 L + A_3 H + \frac{1}{2}(B_1 {u_1}^2 + B_2 {u_2}^2 + B_3 {u_3}^2) \right) dt$$

Subject to

$$\left.\begin{aligned} \frac{dp}{dt} &= \pi - (1-u_1)\beta p h\ - \epsilon_0\, pm - u_1 p \\ \frac{dl}{dt} &= (1-u_1)\beta p h - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 + u_2) l \\ \frac{dh}{dt} &= \alpha_2 l - (\mu + d_2 + \gamma + u_3) h \\ \frac{dq^t}{dt} &= \alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3) q^t \\ \frac{dq^p}{dt} &= \alpha_1 l + \gamma(1-\sigma) h + \eta q^t - \mu q^p + u_1 p + u_2 l + u_3 h \\ \frac{ds_m}{dt} &= \epsilon_0\, pm - (\varphi l + \mu s_m) \\ \frac{dm}{dt} &= \varphi l - \emptyset m \end{aligned}\right\}$$

, **Where** $A_1, A_2\ and\ A_3$ are balancing cost factors due the size of potential smokers, light smokers and heavy or chain smokers respectively.

B_1, B_2, B_3 Represent the "weight" attached on the cost of control methods.

4.3.1 Pontryagin's Maximum Principle (PMP)

***Theorem*4. 1. (*Pontryagin's maximum principle*)**

If $u^*(t)$ and $x^*(t)$ are optimal for the problem

$$max_u J\big(x(t), u(t)\big), where\ J\big(x(t), u(t)\big) = max_u \int_{t_0}^{t_f} f\big(t, x(t), u(t)\big) dt$$

$$\text{Subject to } \begin{cases} \frac{dx}{dt} = g\big(t, x(t), u(t)\big) \\ x(t_0) = x_0 \end{cases}$$

Then there exist a piecewise differential ad-joint variable $\lambda(t)$ such that $H\big(t, x^*(t), u^*(t), \lambda(t)\big) \leq H\big(t, x^*(t), u^*(t), \lambda(t)\big)$. For all controls u at each time t, where the Hamiltonian H is given by

$$H\big(t, x^*(t), u^*(t), \lambda(t)\big) = f\big(t, x(t), u(t)\big) + \lambda(t) g\big(t, x(t), u(t)\big)$$

And $\begin{cases} \lambda'(t) = \frac{\partial H(t, x^*(t), u^*(t), \lambda(t))}{\partial x} \\ \lambda(t_f) = 0 \end{cases}$

Pontryagin's Maximum Principle (PMP) for the model

From the definition of Hamiltonian which stated,

$$H(t, P, L, H, Q^t, Q^p, S_M, M, u_1, u_2) = f(t, P, L, H, Q^t, Q^p, S_M, M, u_1, u_2) + \sum_{i=1}^{7} \lambda_i g_i\,(t, P, l, h, Q^t, Q^p, S_M, M, u_1, u_2)$$

$$= f(t, P, L, H, Q^t, Q^p, S_M, M, u_1, u_2) + \lambda_1 \frac{dP}{dt} + \lambda_2 \frac{dL}{dt} + \lambda_3 \frac{dH}{dt} + \lambda_4 \frac{dQ^t}{dt} + \lambda_5 \frac{dQ^p}{dt} + \lambda_6 \frac{dS_m}{dt} + \lambda_7 \frac{dM}{dt}$$

$$H(t, P, L, H, Q^t, Q^p, S_M, M, u_1, u_2, u_3) = A_1 P + A_2 L + A_3 H + \frac{1}{2}(B_1 {u_1}^2 + B_2 {u_2}^2 + B_3 {u_3}^2)$$

$$+ \begin{pmatrix} \lambda_1(\pi - (1-u_1)\beta p h \ - \in_0 pm - u_1 p) \\ +\lambda_2\big((1-u_1)\beta p h - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 + u_2) l\big) \\ +\lambda_3(\alpha_2 l - (\mu + d_2 + \gamma + u_3) h\,) \\ +\lambda_4(\alpha_3 l + \gamma\sigma h - (\eta + \mu + d_3) q^t) \\ +\lambda_5(\alpha_1 l + \gamma(1-\sigma) h + \eta q^t - \mu q^p + u_1 p + u_2 l + u_3 h) \\ +\lambda_6\big(\in_0 pm - (\varphi l + \mu s_m)\big) \\ +\lambda_7(\varphi l - \emptyset m) \end{pmatrix}$$

4.3.2 The ad-joint function for each state variable (P, L, H, Q^t,Q^p, S_m, M)

Where λ'_is are the ad-joint variables (i = 1, 2, 3, 4, 5, 6, 7). The ad-joint variables are written in the form of differential equations as follows:

- Ad-joint function with respect to P

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial P} = -A_1 + (\lambda_1 - \lambda_2)(1-u_1)\beta h + (\lambda_1 - \lambda_5) u_1 + (\lambda_1 - \lambda_6) \in_0 m$$

- Ad-joint function with respect to L

$$\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial L}$$

$$= -A_2 + (\lambda_2 - \lambda_5)\alpha_1 + (\lambda_2 - \lambda_3)\alpha_2 + (\lambda_2 - \lambda_4)\alpha_3 + (\mu + d_1)\lambda_2 + (\lambda_2 - \lambda_5) u_2 + (\lambda_6 - \lambda_7)\varphi$$

- Ad-joint function with respect to H

$$\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial H} = -A_3 + (\lambda_1 - \lambda_2)(2 - u_1)\,\beta p + (\mu + d_2 + \gamma + u_3)\lambda_3 + \gamma\sigma(\lambda_5 - \lambda_4) - (\gamma + u_3)\lambda_5$$

- Ad-joint function with respect to Q^t

$$\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial Q^t} = (\lambda_4 - \lambda_5)\eta + \lambda_4(\mu + d_3)$$

- Ad-joint function with respect to Q^p

$$\frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial Q^p} = \lambda_5\mu$$

- Ad-joint function with respect to S_m

$$\frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial S_m} = \lambda_6\mu$$

- Ad-joint function with respect to M

$$\frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial \mathrm{M}} = \lambda_7\emptyset$$

$$Then,$$

$$\left.\begin{aligned}
\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial P} &= -A_1 + (\lambda_1 - \lambda_2)(1-u_1)\beta h + (\lambda_1 - \lambda_5)u_1 + (\lambda_1 - \lambda_6)\in_0 m \\
\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial L} &= -A_2 + (\lambda_2 - \lambda_5)\alpha_1 + (\lambda_2 - \lambda_3)\alpha_2 + (\lambda_2 - \lambda_4)\alpha_3 + (\mu + d_1)\lambda_2 + (\lambda_2 - \lambda_5)u_2 + (\lambda_6 - \lambda_7)\varphi \\
\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial H} &= -A_2 + (\lambda_2 - \lambda_5)\alpha_1 + (\lambda_2 - \lambda_3)\alpha_2 + (\lambda_2 - \lambda_4)\alpha_3 + (\mu + d_1)\lambda_2 + (\lambda_2 - \lambda_5)u_2 + (\lambda_6 - \lambda_7)\varphi \\
\frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial Q^t} = (\lambda_4 - \lambda_5)\eta + \lambda_4(\mu + d_3) \\
\frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial Q^p} = \lambda_5\mu \\
\frac{d\lambda_6}{dt} &= -\frac{\partial H}{\partial S_m} = \lambda_6\mu \\
\frac{d\lambda_7}{dt} &= -\frac{\partial H}{\partial \mathrm{M}} = \lambda_7\emptyset
\end{aligned}\right\} \quad (4.7)$$

4.3.3 Optimality condition

Let $\tilde{p},\ \tilde{l}, \tilde{h}, \widetilde{q^t}, \widetilde{q^p}, \widetilde{s_m}, \widetilde{m}$ be the optimum values of P, L, H, Q^t, Q^p, S_m *and* M respectively. Let $\widetilde{\lambda_1}, \widetilde{\lambda_2}, \widetilde{\lambda_3}, \widetilde{\lambda_4}, \widetilde{\lambda_5}, \widetilde{\lambda_6}, \widetilde{\lambda_7}$ be the solutions of system of equations (4.7) we state and prove the following theorem.

Theorem4.2. There exist optimal controls $u^*_1, u^*_2, u^*_3 \epsilon \Omega$ such that $J(u_1, u_2, u_3)$ *subject to extended system of equations* (4.5).

Proof. We demonstrate this theorem using [65]. We note that the controls in this instance are not negative. In order to minimize the issue, the objective functional in (u_1, u_2, u_3) has the

required convexity, which is satisfied. By definition, the set of control variables $u_1, u_2, u_3 \epsilon\ \Omega$ is closed and convex. The functional $A_1P + A_2L + A_3H + \frac{1}{2}(B_1{u_1}^2 + B_2{u_2}^2 + B_3{u_3}^2)$ is convex on Ω, and the state variables are bounded. Given that there are optimal controls for minimizing the functional subject to systems (4.5) and (4.7), we may deduce the necessary conditions to discover the optimal solutions in the following manner by applying Pontryagin's Maximum Principle [65].

Assuming that (z, u) is the best solution to an optimal control problem, it follows that a non-trivial vector function $\lambda = \lambda_1, \lambda_2, \ldots, \lambda_n$ satisfies the following conditions:

$$\frac{dz}{dt} = \frac{\partial H(t,z,u,\lambda)}{\partial \lambda}, \quad 0 = \frac{\partial H(t,z,u,\lambda)}{\partial u} \text{ at } u^* \quad \frac{d\lambda}{dt} = \frac{\partial H(t,z,u,\lambda)}{\partial z}$$

Theorem 4.3.The optimal controls that minimize J across the region Ω are ${u^*}_1, {u^*}_2, {u^*}_3$ and given by

$${u_1}^* = min\{1, max(0, \widetilde{u_1})\}$$

$${u_2}^* = min\{1, max(0, \widetilde{u_2})\}$$

$${u_3}^* = min\{1, max(0, \widetilde{u_3})\}$$

Where $u_1 = \frac{(\lambda_2 - \lambda_1)\beta ph}{B_1} + \frac{(\lambda_1 - \lambda_5)p}{B_1}$

$$u_2 = \frac{(\lambda_2 - \lambda_5)l}{B_2}$$

$$u_3 = \frac{(\lambda_3 - \lambda_5)h}{B_3}$$

Proof: We prove this theorem by using [70] and Theorem 4.2.

Using the optimality condition:

$$\frac{\partial H}{\partial u_1} = 0\ at\ u_1 = {u_1}^*,\ \frac{\partial H}{\partial u_2} = 0\ at\ u_2 = {u_2}^* \text{ and } \frac{\partial H}{\partial u_3} = 0\ at\ u_3 = {u_3}^*$$

Then, $\frac{\partial H}{\partial u_1} = B_1u_1 + (\lambda_1 - \lambda_2)\beta ph + (\lambda_5 - \lambda_1)P = 0$

$$u_1 = \frac{(\lambda_2 - \lambda_1)\beta ph}{B_1} + \frac{(\lambda_1 - \lambda_5)p}{B_1} = \widetilde{u_1}$$

$$\frac{\partial H}{\partial u_2} = B_2 u_2 - \lambda_2 l + \lambda_5 l = 0$$

$$u_2 = \frac{(\lambda_2 - \lambda_5) l}{B_2} = \widetilde{u_2}$$

$$\frac{\partial H}{\partial u_3} = B_3 u_3 + \lambda_5 h - \lambda_3 h = 0$$

$$u_3 = \frac{(\lambda_3 - \lambda_5) h}{B_3} = \widetilde{u_3}$$

Again, the lower bound is 0 and upper bound is 1 for the controls u_1, u_2 and u_3. This implies that $u_1 = u_2 = u_3 = 0$ if $\widetilde{u_1}, \widetilde{u_2}\ and\ \widetilde{u_3} < 0$ and also $u_1 = u_2 = u_3 = 1\ if\ \widetilde{u_1}, \widetilde{u_2}\ and\ \widetilde{u_3} \geq 1$, otherwise $u_1 = {u_1}^*, u_2 = {u_2}^*$ and$u_3 = {u_3}^*$.

Therefore, for these controls${u_1}^*$, ${u_2}^*$ and ${u_3}^*$ we get optimum values of J

$$u_1 = \frac{(\lambda_2 - \lambda_1)\beta p h}{B_1} + \frac{(\lambda_1 - \lambda_5) p}{B_1}$$

$$u_2 = \frac{(\lambda_2 - \lambda_5) l}{B_2}$$

$$u_3 = \frac{(\lambda_3 - \lambda_5) h}{B_3}$$

4.3.4 Analytical Process

The Forward Backward Sweep method or FBS as it is abbreviated is one of the primary methods we will be considering. The reason is that it works by sweeping forward and backwards through time which is why it is called that way. It works as follows: The algorithm begins by making a preliminary guess at the control function. Having this conjecture, it then solves the state equations by taking time forward (t_0) to the final (t_f) time. It then reverses and solves the adjoint equations that are reversed moving backwards and all the way back to t0. It is easy to imagine it as going through the problem in both directions to have a full picture. When the state and adjoint functions have been calculated, the algorithm changes the control according to what it has learned, according to the principles of the standard problem we are dealing with. Then it tests whether the solutions to the state, control, and adjoint functions have converged basically, that is, have converged within a margin of error (or tolerance) that we predetermine. In case they are yet to fully line up, the algorithm uses the updated control and repeats the entire process. It repeats itself until all the things come within the acceptable range. When it does, it terminates and sends the ultimate approximations of the state, adjoint and control functions and they are our solution.

1. Construction of the problem Hamiltonian. It is possible to define the Hamilton H that depends on the equations provided, using the state variables and the control variables. It generally takes the form:
2. H=H (x , u, λ, t)

   Where x denotes the state variables, u denotes the control variable and λ denotes the ad-joint variables.
3. Formulate the ad-joint differential equation, transversality boundary condition and the optimality condition in the form of three unknowns, u^*, x^*, and λ. The ad-joint equations can be obtained by the equation above.

   From the given equations, we have the ad-joint equations:

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial P} \qquad \frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial Q^p}$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial L} \qquad \frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial S_m}$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial H} \qquad \frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial \mathrm{M}}$$

$$\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial Q^t}$$

4. Use the optimality equation Hu = 0 to solve for u^* in terms of x^* and λ.
5. Solve the two differential equations for $x^*$and λ with two boundary conditions.
6. After finding the optimal state and ad-joint, solve for the optimal control using the formula derived by step (3).

If it is possible to solve for the optimal control in terms of x^* and λ, then the formula for u^* is called the characterization of the optimal control. The state equation and ad-joint equations together with the characterization and boundary conditions are called the optimality system.

4.3.5 Backward-forward Sweep Method

The method of the forward-backward sweep is a technique of a famous book by Suzanne Lenhart and Workman [62]. Here's how it works: We begin by making a guess of the first initial guess of the control variable basically, an estimating of what the optimum control should appear. We can solve two equations simultaneously there, state equations proceeding (at the start to the finish) and adjoint equations proceeding (at the finish to the start). It is as though addressing the issue on both sides at the same time. After you do that, you take the new information in the states and adjoints and substitute them in your control estimate and put them in the formula that defines the

control. Then we rinse and repeat of running the entire process over and over again, with a better estimate of control each time, until the solution finally comes to rest. And then we see that we are getting our answer.

Chapter 5: Model Analysis and Results

5.1 Introduction

In this chapter, we are going to estimate the parameter values that will be used for the analysis. The values will be substituted into equation obtained in chapter three and chapter four to get the exact value for the basic reproductive number. Besides, parameter values will be substituted into the various equations derived in chapter 3 to get their actual values. Sensitivity analysis will also be carried out on the parameter values to investigate their impact on study results. The study was conducted based on data obtained from the questionnaire made in Mekelle town.

5.2 Parameter Estimation

The mathematical models that we have discussed would in fact mirror the real-life trends of smoking in such locations as Mekelle Town, provided we give them real-life values. To obtain those values we have to consider the behavior of smoking in real life. One of the most significant aspects: when a person begins to smoke, he or she does not tend to give up in the nearest future. This is because tobacco has nicotine and we all know that this is an addictive substance. Individuals may be addicted in a span of years before they can consider quitting. We gathered primary information within Mekelle Town in order to come with an accurate study as possible. We have created a structured questionnaire (the full version of this questionnaire is found in the appendix) and provided it in the community. The questions were well designed in such a way that they would not only capture the issue of whether an individual smokes or not, but also of what type of a smoker he or she is since all smokers are not the same. We categorized the respondents as follows in accordance with international standards and guidelines as provided by such sources as the CDC and LOINC [71] namely:

- Potential Smoker: This is a person who is not a smoker, but who is vulnerable or susceptible to beginning smoking probably because of social pressures, curiosity or other risk factors.

- Aware of Smoking: Informed people who might or might not have smoked themselves on the risks of smoking. These are the ones who have made a conscious decision to avoid smoking after being informed about the dangers of smoking.

- Light Smoker: A light smoker is one who smokes on a daily basis but who takes few cigarettes, that is, less than 5-10 cigarettes a day in a national health survey. They can produce less evidence of nicotine addiction than heavier smokers.

- A heavy smoker: A daily smoker who smokes more cigarettes each day (usually a pack or more a day) and tends to be more addicted to nicotine.

- Temporary Quit Smoker: This is a person who used to smoke previous to the present time but has since ceased smoking temporarily but can also relapse. This is similar to the former smoker group of the epidemiological research, except their quit attempt is not necessarily permanent.

- Permanent Quit Smoker: A former smoker that has been able to quit and has remained smoke-free in the long term that has no intention to relapse. These categories allowed us to map real people onto the compartments in our mathematical model, making the simulation much more meaningful.

The researcher selected four of them as sites of high population density within Mekelle, which are Kedamay Woyane, Hadinet (22), Kwiha, and Hawelti and used it to collect a study on smoking behavior. All of the places were chosen to represent a specific layer of the population: Kedamay Woyane as a general cross-section of the residents, Kwiha as working-class diversity and Hawelti as residential point of view. This was aimed at gathering information at the locations where social interaction- and subsequently the incidence of smoking habits- is realistically practiced, so that the sample was varied and reflected the population of the city, to make the mathematical model derived after this a more realistic.

The researcher administered questionnaires to 100 individuals each in each of the four study areas Kedamay Woyane, Hadinet (22), Kwiha, and Hawelti which added up to 400 people in Mekelle Town. The questionnaire, which is enclosed in the appendix, was constructed in such a way that it will put the people into one of internationally recognized smoking groups: potential smokers, aware non-smokers, light smokers, heavy smokers, temporary and permanent quitters. The researcher also used different locations around markets, health facilities, streets to which people lived and around commercial areas to approach people in order to have a true picture of

the community. The reasoning was not hard: in case the mathematical model presupposes the spread of smoking by means of social contacts, then the most appropriate location to collect the data is where people really meet. Finally, the 400 replies of four major neighborhoods provided the researcher with a grounded, representative picture of Mekelle one that makes the model more reflective of the real-life dynamics.

Table4: *This table shows number of individuals in the Nonsmoker/Potential smoker, light smoker, heavy smoker, temporarily quit smokers, permanently quit smoker and the aware population.*

Smoking status	Population in each division
Nonsmoker/potential smoker P	155
Light smoker L	84
Heavy smoker H	70
Temporary quit smoker Q^t	28
Permanently quit smokerQ^p	17
Aware population S_m	46
Total	400

In addition to this we have found the following data

Table5: Describes the removal rate

Smoking status	Population in each division
Removal from light smoker to Q^t	25
Removal from heavy smoker to Q^t	3
Removal from light smoker to Q^p	8
Removal from heavy smoker to Q^p	3
Removal from Q^t to Q^p	6

Real Parameter Estimation

To identify the equilibrium points of the system and the stability analysis of it according to the actual data obtained at the research place we summarized the parameters observed in the dynamical system by the table below.

1. Transmission rate (β)

$$\beta = \frac{L}{N} = contact\ rate\ , where\ N\ is\ total\ population$$

This indicates the rate at which nonsmokers become light smokers or exposed

2. Removal Rate (γ) from heavy smokers

$\gamma = \frac{Q^t + Q^p}{N}$ Or $\frac{Number\ of\ quits}{Total\ smokers}$

$$This\ indecates\ the\ rate\ at\ which\ smokers\ quit$$

3. Rate of conversion light smokers to heavy smokers (α_2)

$$\alpha_2 = \frac{H}{L}$$

4. Rate of Removal from $Q^t\ to\ Q^p\ (\eta)$

$$\eta = \frac{Q^p}{Q^t}(transition\ from\ temporarly\ to\ permanent\ quitting\ )$$

5. Probability of conversion in to $Q^t\ and\ Q^p\ (\sigma)$

$$\sigma = \frac{Q^t + Q^p}{H}(Probability\ of\ transmission\ out\ of\ the\ heavy\ smokers)$$

6. Probability convert to temporary quit from the Heavy smoker $(\gamma\sigma)$

$$\gamma\sigma = \gamma.\sigma(transition\ from\ heavy\ smoker\ to\ temporary\ quit)$$

7. Probability convert to permanent quit from the Heavy smoker $\gamma(1-\sigma)$

$$\gamma(1-\sigma) = \gamma(1 - probability\ of\ convert\ to\ Q^p)(transition\ to\ permanent)$$

8. **The rate of conversion from light smoker to permanent quit(α_1)**

$$\alpha_1 = \frac{Number\ of\ \textbf{Removal from light smoker to}\ Q^p}{Number\ of\ light\ smokers}$$

9. **The rate of conversion from light smoker to temporary quit smoker(α_3)**

$$\alpha_3 = Removal\ from\ light\ smoker\ to\ temporary\ quit$$
$$= \frac{\textbf{Number of Removal from light smoker to}\ Q^t}{Number\ of\ Light\ smokers}$$

10. Rate at which potential smoker aware about the ill effects of Smoking and remains in the awareness media compartment(η)

$$\epsilon_0 = \frac{Aware\ population}{Total\ population\ asked}$$

Table 6: *This table shows parameter estimation based on the data obtained by Table 4*

Parameters	Values	Descriptions
π	0.0296[$Assumed$]	Rate of recruitment/birth rate
β	0.5454	Rate of conversion from potential smoker to light smoker
α_1	0.095	The rate of conversion from light smoker to permanent quit
α_2	0.833	The rate of conversion from light smokers to heavy smoker
α_3	0.2976	The rate of conversion from light smoker to temporary quit smoker
σ	0.5	The probability of conversion from heavy smoker to temporarily quit smoker
γ	0.2922	Rate of removal from heavy to temporary and permanent quit
$\gamma\sigma$	0.1461	The rate probability converts into temporarily quit smoking
μ	0.0075[$Assumed$]	Natural death rate
d_1	0.005[$Assumed$]	Death rate of smokers in the light smokers
d_2	0.008[$Assumed$]	Death rate of smokers in the heavy smokers
d_3	0.003[$Assumed$]	Death rate of smokers in the temporary quit smokers
$\in_0$	0.115	Rate at which potential smoker aware about the ill effects of smoking and remains in the awareness media compartment
$\emptyset$	0.06 [$Assumed$]	Rate of addition of media campaigns that depends on number of smokers
φ	0.0007[$Assumed$]	Decay rate of media campaigns
$1-\sigma$	0.5	The probability of conversion from heavy smoker to permanent quit smokers
η	0.607	The rate of conversion from temporary quit to permanent quit
$\gamma(1-\sigma)$	0.1461	The rate of conversion from heavy smoker to permanent quit smokers
R_s	1.19255	Smoking generation number

5.3 Equation of the PLH$Q^tQ^pS_mM$ Model with the Parameters

The value of the parameter estimates from table5 are substituted in equation (3.3.3) to obtain

$$\left.\begin{aligned}
\frac{dp}{dt} &= 0.0\mathbf{296} - \mathbf{0.5454}ph - \mathbf{0.115}pm - \mathbf{0.0075}p \\
\frac{dl}{dt} &= \mathbf{0.5454}ph - (\mathbf{0.095} + \mathbf{0.833} + \mathbf{0.2976} + \mathbf{0.0075} + \mathbf{0.005})l \\
\frac{dh}{dt} &= \mathbf{0.833}l - (\mathbf{0.0075} + \mathbf{0.008} + \mathbf{0.2922})h \\
\frac{dq^t}{dt} &= \mathbf{0.0075}l + \mathbf{0.1461}h - (\mathbf{0.607} + \mathbf{0.0075} + \mathbf{0.003})q^t \\
\frac{dq^p}{dt} &= \mathbf{0.095}l + \mathbf{0.1461}h + \mathbf{0.607}q^t - \mathbf{0.0075}q^p \\
\frac{ds_m}{dt} &= \mathbf{0.115}pm - \mathbf{0.0082}s_m \\
\frac{dm}{dt} &= \mathbf{0.0007}l - \mathbf{0.06}m
\end{aligned}\right\}$$

5.4 Basic Reproduction Number (R_s)

By (3.4.14) the basic reproduction number is given by;

$\boldsymbol{R_s = \frac{\alpha_2 \beta}{ab}}$, where a $= \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1$ and b$= \mu + d_2 + \gamma$

Then $\boldsymbol{R_s = 1.19255}$

Since the reproductive number, $\boldsymbol{R_s = 1.19255 > 1}$, smoking will spread in Mekelle town.

5.5 Equilibrium Point and Stability

5.5.1 Stability Analysis of Smoking-Free Equilibrium (SFE)

From equation(3.4.2), *the SFE is* $(p^*, l^*, h^*, q^{t^*}, s_m{}^*, m) = (1,0,0,0,0,0)$

and equation (3.4.18) the coefficients of the characteristic equation are

$$where\ a = \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1$$
$$b = \mu + d_2 + \gamma$$
$$c = \eta + \mu + d_3$$
$$a_1 = a + b + c + \emptyset$$
$$a_2 = 2\mu(a + b + c + \emptyset) + a\,b + a\,c + a\,\emptyset + b\,c + b\,\emptyset + c\,\emptyset)$$
$$a_3 = \mu^2(a + b + c + \emptyset) + 2\mu(ab + a\,c + a\,\emptyset + b\,c + b\,\emptyset + c\,\emptyset)$$
$$a_4 = \mu^2(ab + a\,c + a\,\emptyset + b\,c + b\,\emptyset + c\,\emptyset) + 2\mu(a\,b\,c + a\,b\,\emptyset + a\,c\,\emptyset + b\,c\,\emptyset)$$
$$a_5 = \mu^2\,(a\,b\,c + a\,b\,\emptyset + a\,c\,\emptyset + b\,c\,\emptyset) + 2\mu a\,b\,c\,\emptyset$$
$$a_6 = \mu^2\,a\,b\,c\,\emptyset$$

Then,

$$a_1 = 0.095 + 0.833 + 0.2976 + 0.0075 + 0.005 + 0.0075 + 0.008 + 0.2922 + 0.607 + 0.0075 + 0.003 + 0.06$$
$$= 2.2233$$
$$a_2 = 2 \times 0.0075(2.2233) + (0.095 + 0.833 + 0.2976 + 0.0075 + 0.005)(0.0075 + 0.008 + 0.2922)$$
$$+ (0.095 + 0.833 + 0.2976 + 0.0075 + 0.005)(0.607 + 0.0075 + 0.003)$$
$$+ (0.095 + 0.833 + 0.2976 + 0.0075 + 0.005)0.06$$
$$+ (0.0075 + 0.008 + 0.2922)(0.607 + 0.0075 + 0.003) + (0.0075 + 0.008 + 0.292)0.06$$
$$+ (0.607 + 0.0075 + 0.003)0.06$$
$$=0.0333+0.38 + 0.7645 + 0.074 + 0.19 + 0.055 = 1.4968$$
$$a_3 = (0.0075)^2(1.2381) + 2 \times 0.0075(0.38 + 0.7645 + 0.074 + 0.19 + 0.055) = 0.022$$
$$a_4 =0.00008+0.0288 = 0.02888$$
$$a_5 =0.0001+0.03739 = 0.03749$$
$$a_6 = 0.0000007$$

$a_1 > 0$ $\quad$ $a_4 > 0$

$a_2 > 0$ $\quad$ $a_5 > 0$

$a_3 > 0$ $\quad$ $a_6 > 0$

And $a_1a_2 - a_3 = 3.305 > 0$, $a_1a_2a_3 - a_2^2a_4 - a_3^2 = 0.007593 > 0$

$$a_6[2a_1^2a_2a_5a_6 + a_1^2a_4^2a_5 + a_1a_2a_3a_4a_5 + 2a_1a_4a_5^2 + a_2a_3a_5^2 - (a_1^3a_6^2 + a_1a_2^2a_5^2 + a_1a_2a_3^2a_6 + 3a_1a_3a_5a_6)] = 0.000000000264 > 0$$

The Jacobian matrix at the smoking free equilibrium $(1,0,0,0,0,0)$ is

$$J(1,0,0,0,0,0) = \begin{pmatrix} -0.0075 & 0 & 0 & 0 & 0 & -0.115 \\ 0 & -1.2381 & 2.96 & 0 & 0 & 0 \\ 0 & \mathbf{0.833} & -0.3077 & 0 & 0 & 0 \\ 0 & \mathbf{0.2976} & \mathbf{0.1461} & -0.6175 & 0 & 0 \\ 0 & \mathbf{-0.0007} & 0 & 0 & -0.0075 & 0.115 \\ 0 & \mathbf{0.0007} & 0 & 0 & 0 & -0.06 \end{pmatrix}$$

The corresponding characteristic equation becomes

$$\lambda^6 + 2.2233\lambda^5 + 1.4968\lambda^4 + 0.022\lambda^3 + 0.02888\lambda^2 + 0.03749\lambda + 0.0000007 = 0$$

Using the Routh Hurwitz stability sufficient condition, all the roots of the equation are negative; the SFE is locally asymptotically stable. This implies that the system will approach the equilibrium state as time progresses to infinity and this demonstrates the notion that the smoking free equilibrium point is steady (there is no smoking).

5.5.2 Stability Analysis of Endemic Equilibrium Point (EE)

From (3.4.15) the Endemic Equilibrium was given by

$$(\ p^*, l^*, h^*, (q^t)^*, (s_m)^*, m^*\ ) =$$

$$\left(\frac{1}{R_s}, \frac{b\emptyset}{\theta}(R_s\pi-\mu), \frac{\alpha_2\emptyset}{\theta}(R_s\pi-\mu), \frac{\emptyset}{\theta c}(\alpha_3 b+\gamma\delta\alpha_2)(R_s\pi-\mu), \frac{\varphi b}{\theta\mu}(\epsilon_o p-\emptyset)(R_s\pi-\mu), \frac{\varphi b}{\theta}(R_s\pi-\mu)\right)$$

$$(\ p^*, l^*, h^*, (q^t)^*, (s_m)^*, m^*\ ) = (0.8385, 0.018827, 0.05092, 0.0211, 0.000006, 0.0002)$$

Then $(q^p)^* = 0.07$

Then the Jacobean matrix at the smoking present equilibrium point of equation

$$J_{EE} = \begin{pmatrix} -(\beta h^* + \epsilon_0 m^* + \mu) & 0 & -\beta h^* & 0 & 0 & -\epsilon_0 p^* \\ \beta h^* & -a & \beta p^* & 0 & 0 & 0 \\ 0 & \alpha_2 & -b & 0 & 0 & 0 \\ 0 & \alpha_3 & \gamma\sigma & -c & 0 & 0 \\ \epsilon_0 m^* & -\varphi & 0 & 0 & -\mu & \epsilon_0 \\ 0 & \varphi & 0 & 0 & 0 & -\emptyset \end{pmatrix} \text{ is}$$

$$J(0.8385, 0.018827, 0.05092, 0.0211, 0.000006, 0.0002)$$

$$= \begin{pmatrix} -0.035 & 0 & -0.02727 & 0 & 0 & -0.096 \\ 0.02727 & -1.2381 & 0.457 & 0 & 0 & 0 \\ 0 & 0.833 & -0.3077 & 0 & 0 & 0 \\ 0 & 0.2976 & 0.1461 & -0.6175 & 0 & 0 \\ 0.000023 & -0.0007 & 0 & 0 & -0.0075 & 0.115 \\ 0 & 0.0007 & 0 & 0 & 0 & -0.06 \end{pmatrix}$$

The corresponding characteristic equation is $\lambda^6 + 0.72\lambda^5 + 0.76\lambda^4 + 0.026\lambda^3 + 0.015\lambda^2 + 0.00087\lambda + 0.0000065 = 0$.

And from the calculations above,

$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_5 > 0, a_6 > 0$ and through a simple computation, we obtain that $a_6[2{a_1}^2 a_2 a_5 a_6 + {a_1}^2 {a_4}^2 a_5 + a_1 a_2 a_3 a_4 a_5 + 2a_1 a_4 {a_5}^2 + a_2 a_3 {a_5}^2 - ({a_1}^3 {a_6}^2 + a_1 {a_2}^2 {a_5}^2 + a_1 a_2 {a_3}^2 a_6 + 3a_1 a_3 a_5 a_6)] > 0$. That is $0.0001(0.000246496 - 0.00011458806) > 0$

This shows that the EE is locally asymptotically stable. This means that smoking tobacco will spread in Mekelle Town.

5.6 Sensitivity Analysis

Sensitivity analysis can be useful for the purpose of understanding of the relationships between input and output variables in a system or model.

To find the sensitivity of each parameter for R_s, we will employ $equation(3.4.14)$ and equation$(3.4.24)$. Where $R_s = \frac{\alpha_2 \beta}{ab}$ and $a = \alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1, b = \mu + d_2 + \gamma$

On the basis of parameter values β =0.5454, $\alpha_1 = 0.095, \alpha_2 = 0.833, \alpha_3 = 0.2976, d_1 = 0.005,$ $d_2 = 0.008, \gamma = 0.2922, \eta = 0.607$ and μ = 0.0075 we have the sensitivity indices of the form

$$S_\beta = \frac{\beta}{Rs}\frac{\partial Rs}{\partial \beta} = 1 > 0$$

$$S_{\alpha_1} = \frac{\alpha_1}{Rs}\frac{\partial Rs}{\partial \alpha_1} = \frac{-\alpha_1}{\alpha_1+\alpha_2+\alpha_2+\mu+d_1} = -0.0767 < 0$$

$$S_{\alpha_2} = \frac{\alpha_2}{Rs}\frac{\partial Rs}{\partial \alpha_2} = \beta - \frac{-\alpha_2}{\alpha_1+\alpha_2+\alpha_2+\mu+d_1} = 1.2154 > 0$$

$$S_{\alpha_3} = \frac{\alpha_3}{Rs}\frac{\partial Rs}{\partial \alpha_3} = \frac{-\alpha_3}{\alpha_1+\alpha_2+\alpha_2+\mu+d_1} = -0.24 < 0$$

$$S_{d_1} = \frac{d_1}{Rs}\frac{\partial Rs}{\partial d_1} = \frac{-d_1}{\alpha_1+\alpha_2+\alpha_2+\mu+d_1} = -0.004 < 0$$

$$S_{d_2} = \frac{d_2}{Rs}\frac{\partial Rs}{\partial d_2} = \frac{-b_2}{\mu+d_2+\gamma} = -1 < 0$$

$$S_\gamma = \frac{\gamma}{Rs}\frac{\partial Rs}{\partial \gamma} = \frac{-\gamma}{\mu+d_2+\gamma} = -0.95 < 0$$

$$S_\mu = \frac{\mu}{Rs}\frac{\partial Rs}{\partial \mu} = \frac{\mu}{ab}\left(d_2 + \gamma - (\alpha_1 + \alpha_2 + \alpha_3 + d_1)\right) = -0.018 < 0$$

Parameters with negative indices reduce the spread of smoking when increased, while those with positive indices accelerate it. These indices help identify which factors most influence the reproduction number.

Then, it is evident that more potential smokers are becoming heavy smokers due to an increase in effective contact rate (β), which raises the R_s value and the rate at which light smokers become heavy smokers (α_2).

5.7 Numerical Simulations of the Model

A numerical simulation of the model is conducted to find out the dynamics of smoking in the human population. The simulations were conducted using MATLAB's ode45. The initial Conditions used were P(0)= 155, L(0)= 84, H(0)=70, $Q^t = 28, Q^p = 17$, $S_m = 46$ and M(0)=20[$Assumed$].

So dividing each of the terms by the total population of, N(0)=400, then we have p(0)= 0.3875 ,l(0)= 0.21 ,h(0)=0.175, $q^t(0) = 0.07, q^p(0) = 0.0425, s_m(0) = 0.115$ and m(0)=0.05($Assumed$).

NUMERICAL ANALYSIS

The numerical analysis is obtained with the help of the smoking generation number graphs which is based on the real parameters identified and presented in Table 5. This section allows us to determine the parameter of the smoking dynamic that is sensitive. We will assume that the rate of transmission of nonsmokers into the class of light smoker (contact rate) is the control parameter and all other aspects are held constant. The result is a graphical plot of the generation of the number of smoking versus the contact rateβ.

Rate of Transmission of Nonsmoker into light smoker Class (Contact Rate) β

Where $R_s = \frac{\alpha_2\beta}{(\alpha_1+\alpha_2+\alpha_3+\mu+d_1)(d_2+\mu+\gamma)}$

$R_s(\beta) = \frac{0.833\beta}{(1.2381)(0.3077)} = 2.18658\beta$

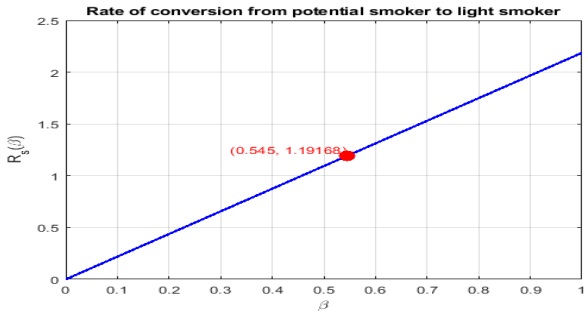


Figure 6: Rate of conversion from potential smoker to light smoker

Figure7: This graph shows that the reproduction number R_s > 1 for the transmission rate β > 0.5454. This indicates that when the contact rate β surpasses 0.5454, the smoking habit spreads throughout society.

Rate of Conversion light smoker into heavy smoker Class α 2

$$R_s(\alpha_2) = \frac{0.5454\alpha_2}{(0.4051+\alpha_2)(0.3077)}$$

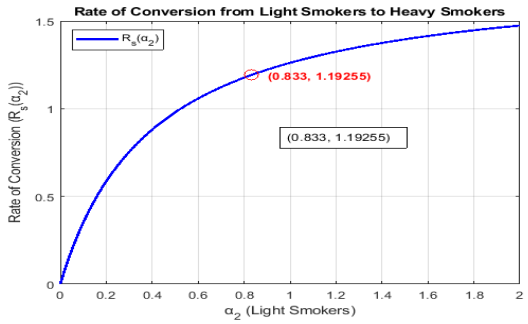


Figure 7: Rate of conversion from light smoker to heavy smoker

Figure8: In this graph, the conversion rate ($\alpha_2 > 0.833$) has a reproduction number R_s be above 1. This will mean that the higher the conversion rate of α_2, the higher the smoking habit in the society.

The rate of conversion of exposed/light smoker to permanent quit smoker measured in units of per one (α_1) can be assumed to be the control parameter. It is graphed as a1/ number of the smoking generation versus the conversion rate α_1.

Rate of Conversion light smoker into permanent quit smoker Class α_1

$$R_{\alpha_1} = \frac{0.4543182}{(1.1431 + \alpha_1)(0.3077)}$$

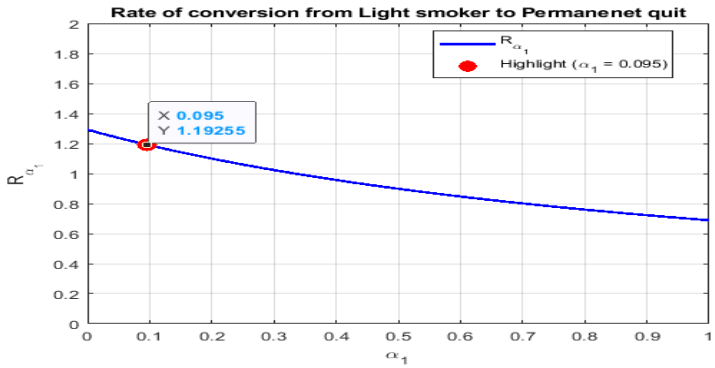


Figure 8:Rate of conversion from light smoker to permanent quit

Figure9: This graph indicates that the conversion rate $R_S < 1$. $\alpha_1 > 0.095$. This implies that, a conversion rate a1 beyond 0.095 will make the smoking habit extinct in the society.

When all the other factors are held constant, we may use as the control parameter α_3, which is the rate at which exposed/light smoker becomes temporary quit smoker. The rate of conversion $\alpha_3$versus the number of smokers of the generation is plotted graphically by

$$R(\alpha_3) = \frac{0.4543182}{(0.9405 + \alpha_3)(0.3077)}$$

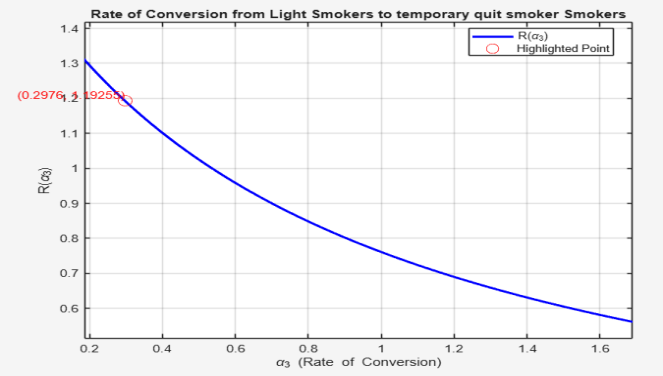


Figure 9: Rate of conversion from light smoker to temporary quit smoker

Figure10: This graph shows that the reproduction number $R_s < 1$ for the conversion rate $\alpha_3 >$ **0.2976**. This indicates that when the conversion rate α_3 surpasses **0.2976**, the smoking habit becomes dies out in society.

Let's assume that d_1, the light smoking causes mortality rate, is the control parameter and that the other values remain constant. The smoking generation number verses the smoking-related death rate is derived by a graphical representation.

$$R_{d_1} = \frac{0.4543182}{(1.2331 + d_1)(0.3077)}$$

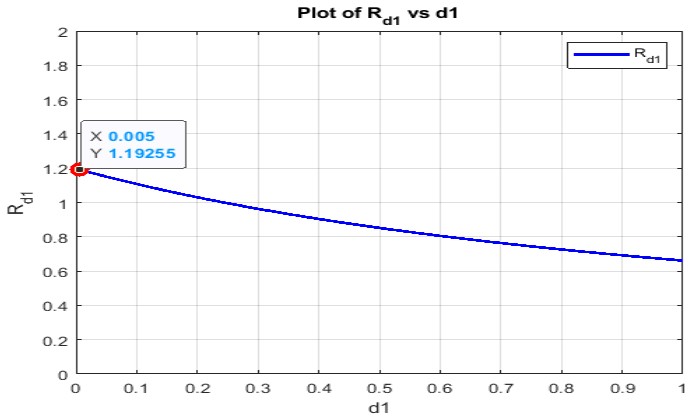


Figure 10: Light smoking causes mortality rate

Figure11: This graph shows that the reproduction number $R_s < 1$ for the light smoking causes mortality rate $d_1 >$ **0.005**. This indicates that when the conversion rate α_3 surpasses **0.005**, the smoking habit becomes dies out in society.

Let's assume that d_2, the heavy smoking causes mortality rate, is the control parameter and that the other values remain constant. The smoking generation number verses the smoking-related death rate is derived by a graphical representation.

$$R_{d_2} = \frac{0.4543182}{(1.2381)(d_2 + 0.2997)}$$

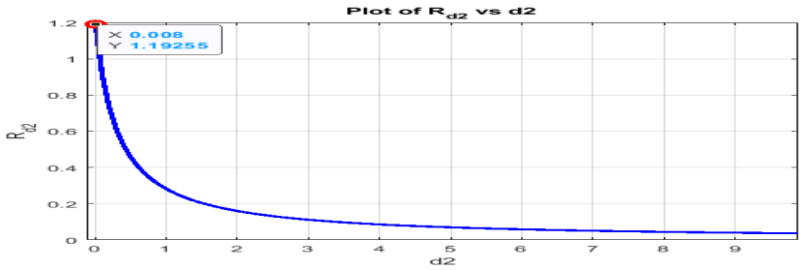


Figure 11: Heavy smoking causes mortality rate

Figure12: This graph shows that the reproduction number $R_s < 1$ for the heavy smoking causes mortality rate $d_2 >$ **0.008**. This indicates that when the conversion rate α_3 surpasses **0.005**, the smoking habit becomes dies out in society.

Let's assume that the natural death rate, or μ, is the control parameter and that the other parameters remain constant. The figure that shows the relationship between the number of smokers and the natural death rate (μ) is produced by.

Natural Death Rate

$$R_{s\mu} = \frac{0.4543182}{(1.2306 + \mu)(\mu + 0.3002)}$$

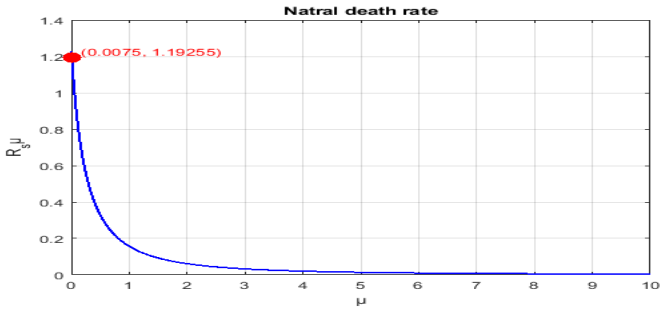


Figure 12: Natural death mortality rate

Figure13: This graph shows that the reproduction number $R_s < 1$ for the natural death mortality rate $\mu >$ **0.0075**. This indicates that when the conversion rate μ surpasses **0.0075**, the smoking habit becomes dies out in society.

Rate of Removal from heavy Smoker to Temporarily Quit and Permanent Quit Smokers

In this case, we are going to take the control parameter as which is the Rate of removal of the heavy smoker into temporary quit smokers(γ) and permanent quit smokers and all the other parameters are held constant. The graphical representation of the number of smoking generation against the Rate of remove heavy smoker to temporarily quit and permanent quit smoker permanent quit heavy smoker is obtained by

$$R_\gamma = \frac{0.4543182}{(1.2381)(\gamma + 0.0155)}$$

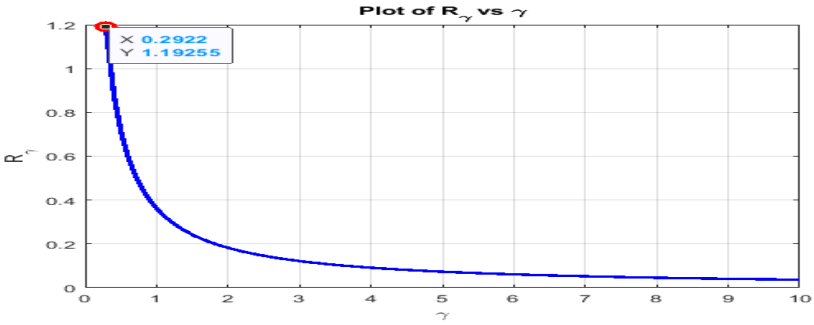


Figure 13: Rate of removal from heavy to temporary and permanent quit

Figure14: Based on this graph, we can see that the reproduction number $R_s < 1$ occurs when the rate of smokers being removed to temporarily and permanently quit is $\gamma > 0.2922$. Accordingly,

smoking cessation becomes extinct in society when the rate of heavy smokers quitting to both temporary and permanent cessation smokers γ above 0.2922.

Comparison of the light smoker versus awareness programme

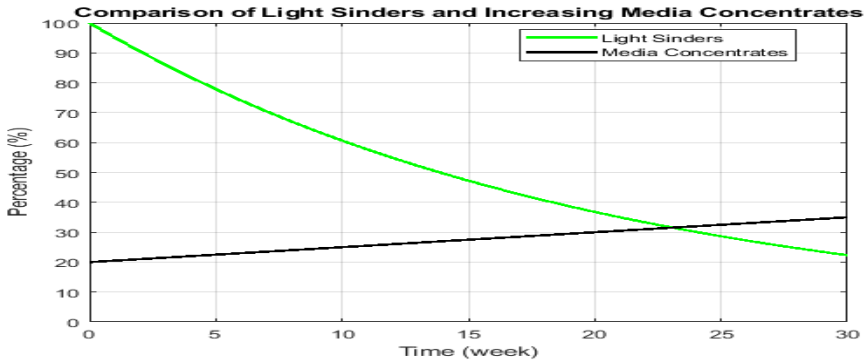


Figure 14: Comparison of light smoker versus awareness programme

Figure15: The chart will give a pictorial demonstration of the effect of augmented media campaigns on the number of light smokers in the long run. Through the observation of these trends, we will be able to determine the effectiveness of the awareness programs in decreasing the smoking behavior and whether some further measures should be taken to bring about more favorable results.

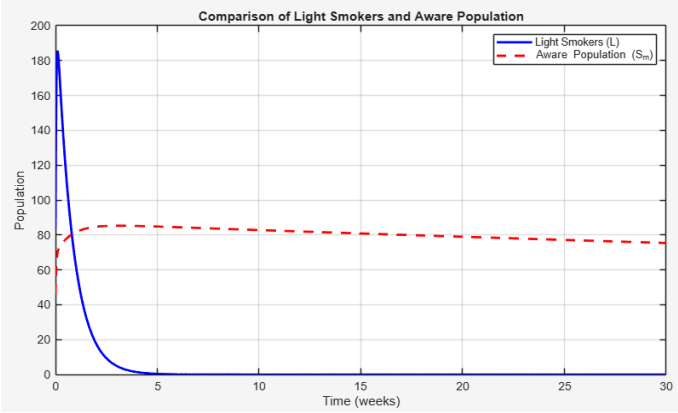


Figure 15: Comparison of light smoker and aware population

Figure16: The simulation may demonstrate how the behavior of smoking can be changed by alterations in media campaigns (modeled by the aware population). With the successfulness of the media campaigns, there will be more people who are aware of their smoking conditions and hence, fewer light smokers in the future. The graph gives a numerical illustration of the effect of growing media campaigns to the light smokers at a given time. Through such trends, you are able

to evaluate how effective the awareness programs are in lowering smoking behavior and then decide whether more needs to be put in place to ensure better results are realized.

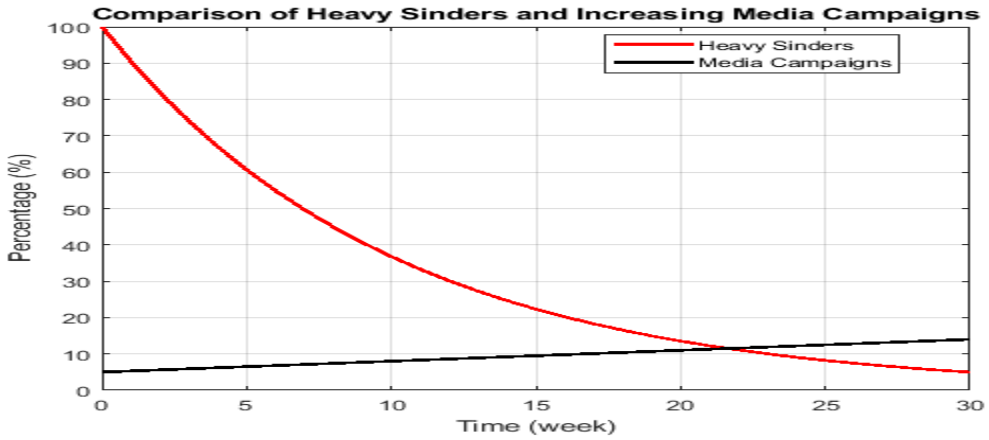


Figure 16: Comparison of the heavy smokers versus awareness programme

Figure17: The graph will give an insight on the influence of the growing number of media campaigns on the number of heavy smokers with time. The study of these trends allows evaluating the success of the public health awareness in preventing heavy smoking and identifying the necessity of the use of some other strategies to improve the results.

Comparison of the heavy smoker versus aware population

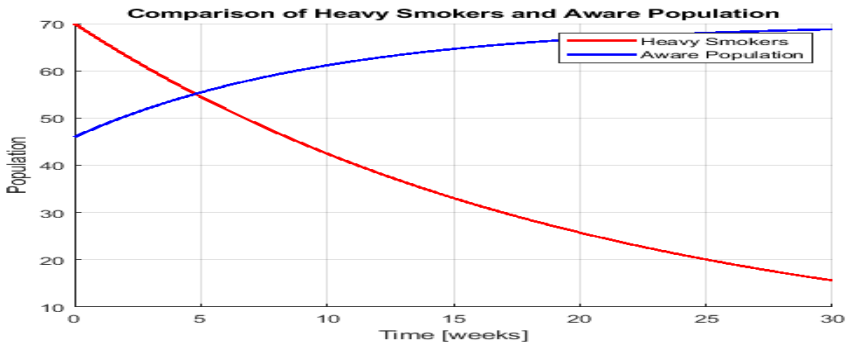


Figure 17: Comparison of heavy smokers and aware population

Figure18: The graph is very effective and demonstrates the dependence between the raising awareness and the decline in the smoking rates. This dynamic demonstrates the significance of continuous learning and intervention measures in population health towards fighting smoking and the risks associated with it.

Optimal control Analysis and Results

Here, we will analytically discuss and numerically analyze the effect of the optimal control strategies such as counseling, Ban smoking in public area and treatment. In order to determine the adjoint variable in the given optimal control problem we shall proceed in the steps of Forward-Backward sweep algorithm. Here is how to approach it,

Step1: Formulate the Optimal Control Problem. Minimizing the cost functional is:

$$J(u_1, u_{2,} u_3) = \int_0^T \left(A_1 P + A_2 L + A_3 H + \frac{1}{2}(B_1 {u_1}^2 + B_2 {u_2}^2 + B_3 {u_3}^2) \right) dt$$

Subject to the system dynamics:

$$\left.\begin{aligned}
\frac{dP}{dt} &= \pi N - (1-u_1)\frac{\beta PH}{N} - \frac{\epsilon_0 PM}{N} - u_1 P \\
\frac{dL}{dt} &= (1-u_1)\frac{\beta PH}{N} - (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 + u_2)L \\
\frac{dH}{dt} &= \alpha_2 L - (\mu + d_2 + \gamma + u_3)H \\
\frac{dQ^t}{dt} &= \alpha_3 L + \gamma\sigma H - (\eta + \mu + d_3)Q^t \\
\frac{dQ^p}{dt} &= \alpha_1 L + \gamma(1-\sigma)H + \eta Q^t - \mu Q^p + u_1 P + u_2 L + u_3 H \\
\frac{dS_m}{dt} &= \frac{\epsilon_0 PM}{N} - (\varphi L + \mu S_m) \\
\frac{dM}{dt} &= \varphi l - \emptyset M
\end{aligned}\right\}$$

The initial values are obtained from the real is presented as in the Table below.

Table Initial value

p_0	l_0	h_0	${q^t}_0$	${q^p}_0$	s_{m0}	m_0
0.3875	0.21	0.175	0.07	0.0425	0.115	$0.05[Assumed]$

And the assumed Initial values for $u_1 = 0.1, u_2 = 0.1, u_3 = 0.1$

Step 2: Forward Sweep Resolve the state equations by an adequate numerical process (such as RK4) of the dynamics in the above description. In order to solve our optimal control problem forward in time by Runge-Kutta 4^{th} order (RK4) approach, we formulated our equations. The following is a step-by-step instruction on the implementation of the RK4 method of the given system dynamics.

Step1: Define the RK4 Method

The RK4 approach is the computation of intermediate slopes the state in the subsequent time step. The general form is:

For state variable x:

$$k_1 = f(t_n, x_n)$$

$$k_2 = f\left(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_1\right)$$

$$k_3 = f\left(t_n + \frac{h}{2}, x_n + \frac{h}{2}k_2\right)$$

$$k_4 = f(t_n + h, x_n + hk_3)$$

$$k_{n+1} = x_n + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4)$$

Step2: Implement the RK4 Method for Each state Equation

We have assumed the initial conditions and constants; the integration can be implemented as follow numerically.

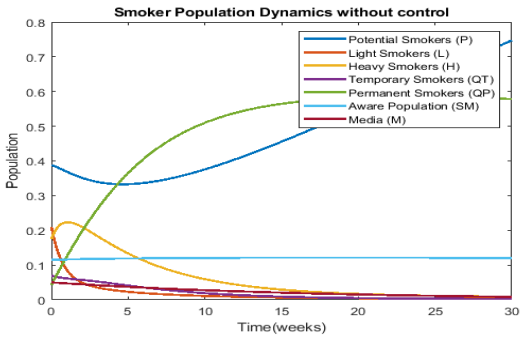


Figure 18: Smoker Population Dynamics without Control

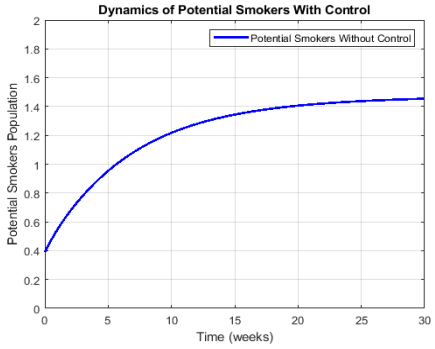


Figure 19: Dynamics of potential Smokers without Control

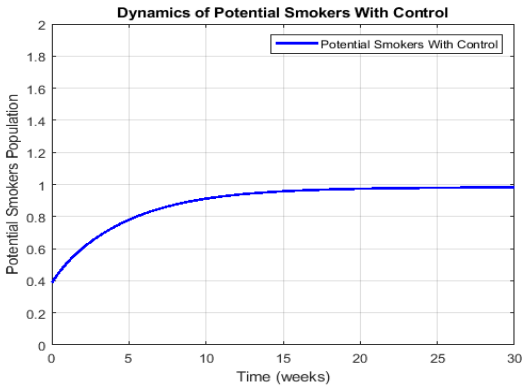


Figure 20: Dynamics of potential Smokers with control

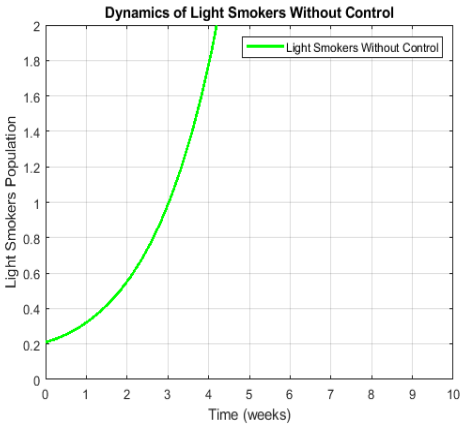


Figure 21: Dynamics of Light smokers without control

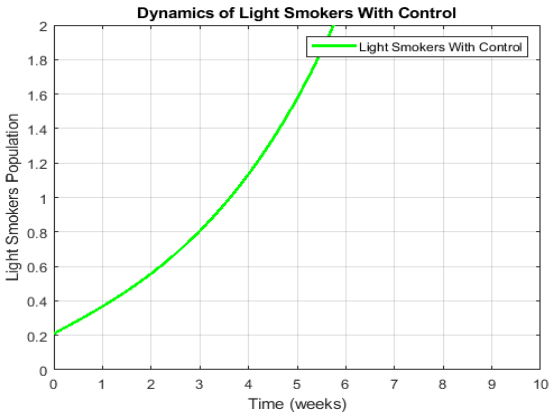


Figure 22: Dynamics of light smokers with Control

The graph shows that the measures taken to minimize the number of light smokers are very efficient and their population will reduce significantly in the long run. This is an indication of the relevance of proper public health policies in controlling smoking habits.

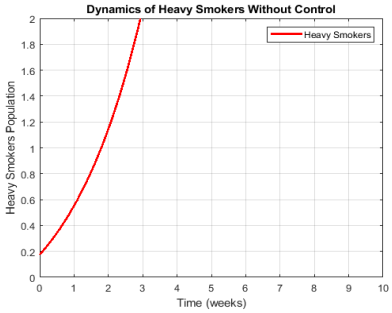


Figure 23: Dynamics of Heavy Smokers without Control

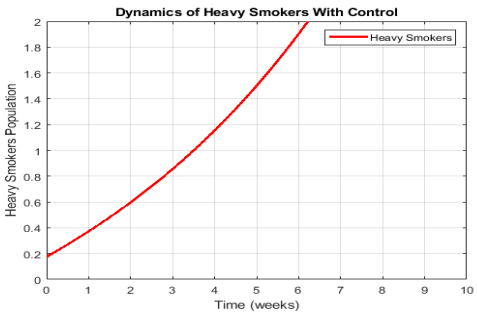


Figure 24: Dynamics of Heavy Smokers with Control

This means that the heavy smoking population can substantially be reduced by applying effective optimal control measures as shown in the graph. The decrease and the stabilization at a low level

indicate not only the instant success but also the possibility of the continuation of the achieved results in smoking cessation campaigns. The long-term sustainability of these outcomes as well as the factors that led to the effectiveness of the control strategies might be further analyzed.

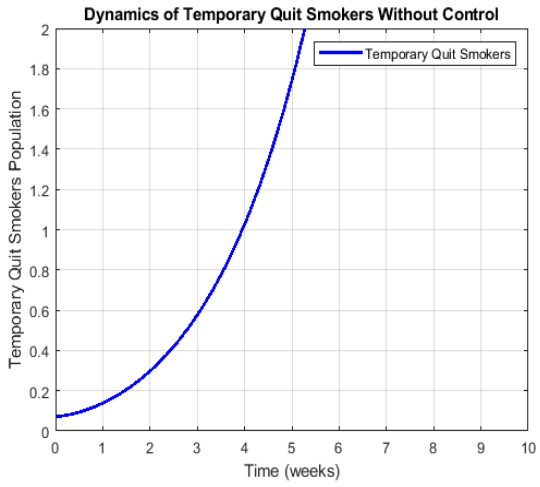


Figure 25: Dynamics of Temporary Quit Smokers without Control

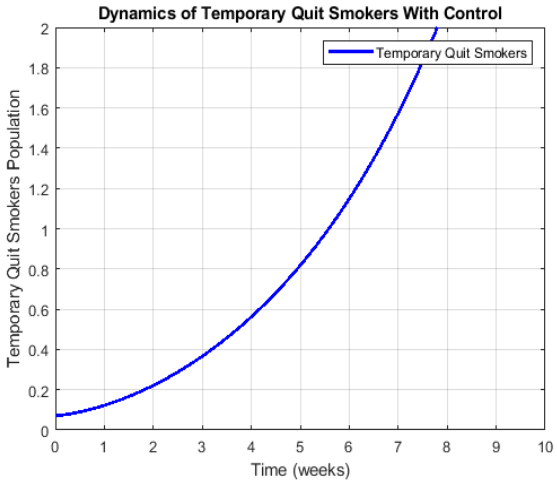


Figure 26: Dynamics of Temporary Quit Smokers with Control

The plot is that the optimal control measures can drive to a stabilized population of temporary quit smokers. The progressive increase and eventual stabilization are signs of success in the short run and a possibility of continued influence in the smoking cessation efforts.

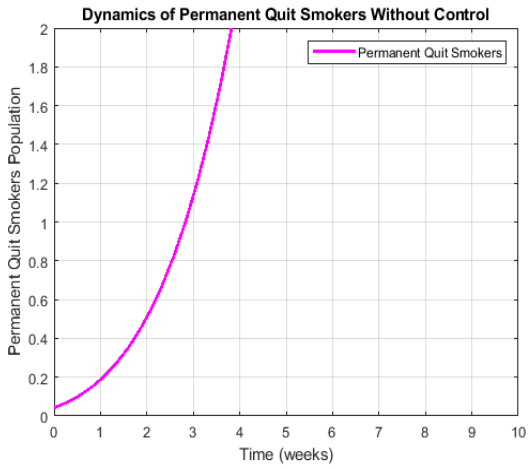


Figure 27: Dynamics of Permanent Quit Smokers with control

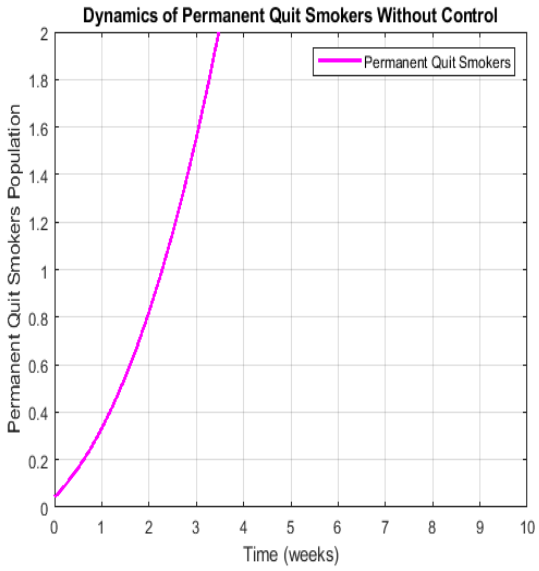


Figure 28: Dynamics of permanent Quit Smokers without Control

The plot is effective in demonstrating how the number of permanent quit smokers changes with time under optimal control with both initial success and stabilization in the end.

Step4: Compute the ad-joint variables

- $\lambda_5(t) = \lambda_{5(0)} e^{\mu t}$
- $\lambda_6(t) = \lambda_{6(0)} e^{\mu t}$
- The solution for

$$\boldsymbol{\lambda_4(t) = -\lambda_{5(0)}\eta.\frac{1}{\mu-c}e^{\mu t} + \left(\lambda_4(0) + \lambda_{5(0)}\eta.\frac{1}{\mu-c}\right)e^{Ct}}\textbf{, where}$$

$$\mathbf{C = \lambda_4(0) + \lambda_{5(0)}\eta.\frac{1}{\mu-c}e^{\mu t} = -3.4467}$$

- $\boldsymbol{\lambda_7(t) = \lambda_{7(0)}e^{\emptyset t}}$
- $\lambda_3(t) = \dfrac{-A_3+\left(\frac{\beta H}{N}\left[\frac{(A_1+A_2)(2-u_1)}{2+\frac{\epsilon_0 M}{N}-u_1} - \frac{A_2(2-u_1)}{\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2}\right]\right)+\gamma\sigma(\lambda_5-\lambda_4)-(\sigma+u_3)\lambda_5}{Z}$

$$\mathrm{Z} = \mu + \alpha_2 + \gamma + u_3 + \frac{(2-u_1)\alpha_2}{\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2}$$

- $\lambda_1(t) = \dfrac{A_1+\frac{A_2+\lambda_3\alpha_2+\left[-\eta\lambda_{5(0)}\frac{1}{\mu-c}e^{\mu t}+\left(\lambda_{4(0)}+\eta\lambda_{5(0)}\frac{1}{\mu-c}e^{\mu t}\right)e^{ct}\right]\alpha_3+\lambda_{5(0)}e^{\mu t}(\alpha_1+u_2)-\lambda_{6(0)}e^{\mu t}\emptyset}{\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2}(2-u_1)\frac{\beta H}{N} + \lambda_{5(0)}e^{\mu t}u_1 - \frac{\epsilon_0 M}{N}\lambda_{6(0)}e^{\mu t}}{\left(2+\frac{\epsilon_0 M}{N}-u_1\right)}$
- $\lambda_2(t) = \dfrac{A_2+\lambda_3\alpha_2+\left[-\eta\lambda_{5(0)}\frac{1}{\mu-c}e^{\mu t}+\left(\lambda_{4(0)}+\eta\lambda_{5(0)}\frac{1}{\mu t-c}e^{\mu t}\right)e^{ct}\right]\alpha_3+\lambda_{5(0)}e^{\mu t}(\alpha_1+u_2)-\lambda_{6(0)}e^{\mu t}\emptyset}{\alpha_1+\alpha_2+\alpha_3+\mu+d_1+u_2}$

Take initial values

$\lambda_{1(0)} = 1$ $\lambda_{4(0)} = 1$

$\lambda_{2(0)} = 1$ $\lambda_{5(0)} = 1$ $\lambda_{7(0)} = 1$

$\lambda_{3(0)} = 1$ $\lambda_{6(0)} = 1$

Then for $t = 200$

$$\lambda_{5(200)} = 4.4817$$

$$\lambda_{6(200)} = 4.4817$$

$$\lambda_{7(200)} = e^{12}$$

$$\lambda_4(200) = 4.5097$$

$$\lambda_3(200) = -1.7071$$

$$\lambda_2(200) = -0.603$$

$$\lambda_1(200) = -1.191$$

Objective Function: In this study, the objective of the optimal control is to minimize the level of potential smokers, light smokers, heavy smokers and the charge used in implementing the three control measures.

$$J(u_1, u_{2,}\, u_3) = \int_0^T \left(A_1 P + A_2 L + A_3 H + \frac{1}{2}(B_1 {u_1}^2 + B_2 {u_2}^2 + B_3 {u_3}^2) \right) dt$$

$$J(u_1, u_{2,}\, u_3) = \int_0^T 309.015\, dt = 0.78755T$$

Assume $A_1 = A_2 = A_3 = B_1 = B_2 = B_3 = 1$ and $u_1 = u_2 = u_3 = 0.1$

Then, at $T = 200$

$$J(u_1, u_{2,}\, u_3) = 0.7875 \times 200 = 157.5$$

The ad-joint equations play a vital role in the determination of optimal control procedures and may be resolved by the same numerical techniques as in the case of the state equations. The optimal control inputs $u_1, u_{2,}\, u_3$ which will minimize the cost functional *J* can be obtained by solving these ad-joint equations and the state equations.

Step5: **Backward sweep**

The ad-joint equations are given as follow

$$\left.\begin{aligned}
\frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial P} = -A_1 + (\lambda_1 - \lambda_2)(2 - u_1)\,\frac{\beta H}{N} + (\lambda_1 - \lambda_5)u_1 + (\lambda_1 - \lambda_6)\frac{\epsilon_0\, M}{N} \\
\frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial L} = -A_2 + (\alpha_1 + \alpha_2 + \alpha_3 + \mu + d_1 + u_2)\lambda_2 - (\lambda_3\alpha_2 + \lambda_4\alpha_3) - \lambda_5(\alpha_1 + u_2) + (\lambda_6 - \lambda_7)\varphi \\
\frac{d\lambda_3}{dt} &= -\frac{\partial H}{\partial H} = -A_3 + (\lambda_1 - \lambda_2)(2 - u_1)\,\frac{\beta P}{N} + (\mu + d_2 + \gamma + u_3)\lambda_3 + \gamma\sigma(\lambda_5 - \lambda_4) - (\sigma + u_3)\lambda_5 \\
\frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial Q^t} = \lambda_4 c - \lambda_5\eta \\
\frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial Q^p} = \lambda_5\mu \\
\frac{d\lambda_6}{dt} &= -\frac{\partial H}{\partial S_m} = \lambda_6\mu \\
\frac{d\lambda_7}{dt} &= -\frac{\partial H}{\partial \mathrm{M}} = \lambda_7\emptyset
\end{aligned}\right\}$$

The terminal values of each ad-joint variables is given as follow

$$\lambda_{5(200)} = 4.4817$$

$$\lambda_{6(200)} = 4.4817$$

$$\lambda_{7(200)} = e^{12}$$

$$\lambda_4(200) = 4.5097$$

$$\lambda_3(200) = -1.7071$$

$$\lambda_2(200) = -0.603$$

$$\lambda_1(200) = -1.191$$

Strategy A: Ideal Use Control (u_1, u_2, u_3) of people

These processes begin with a rough estimate of the control variable. The state equations are then solved in forward time direction and the ad joint equations solved in reverse time direction. This is the procedure continued until convergence occurs, with the characterization of the control being updated with the new values of states and ad joints. ODE solver was used to solve the state and adjoint system and is a Runge-Kutta fourth order algorithm written in MATLAB. Epidemiological parameters are estimated based on the information obtained in the region of a study.

Strategy A:-Best Use Control (u_1, u_2, u_3) on individuals. The control variable $u_1, u_2, and\ u_3$ are utilized in order to maximize the objective function. In the Approach A, the quantity of individuals having been infected at a short period is reduced with the help of u_1, u_2, u_3. Relative to Strategy A where there is no control model, all numbers represent the control variables u_1, u_2, u_3 (representing potential smokers, light smokers, and heavy smokers) as minimizing them. In the meantime, recovered people or permanent smokers are on the rise within a short span.

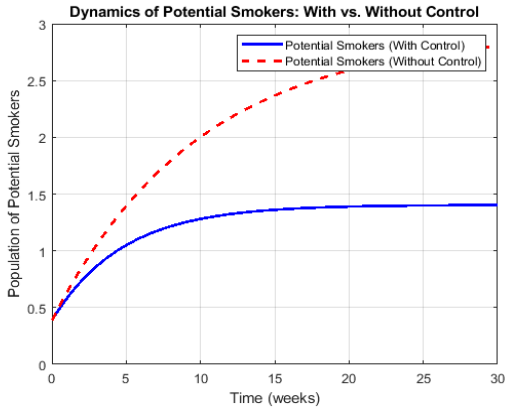


Figure 29: Dynamics of potential smokers with and without control

Figure30: showcases the numeric resolution of potential smokers / vulnerable individuals that are controlled and those that are not. It demonstrates that the number of vulnerable people can significantly decrease when the control methods are implemented, and both reach the stable positions at the end of the control time. Based on the data, media education significantly

decreases the quantity of those infected into the population, and vulnerable people are also minimized, which affects the dynamics of the population positively.

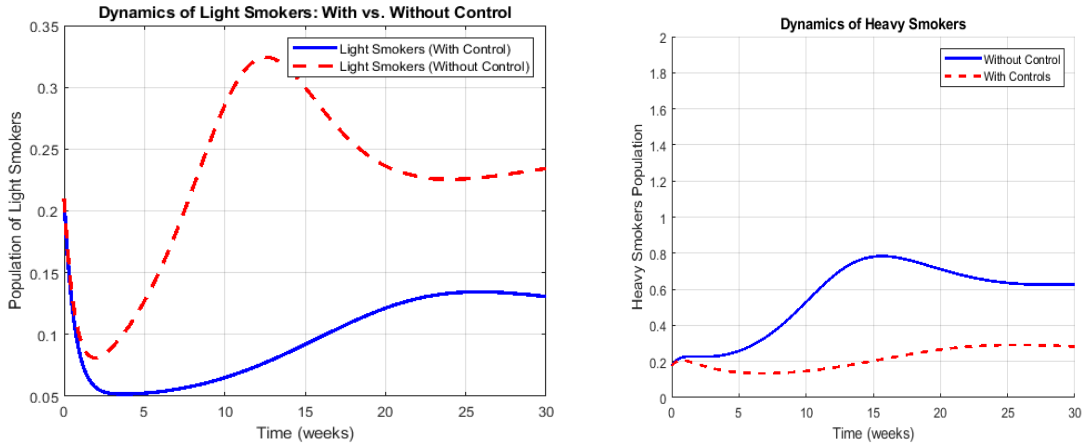


Figure 30: Comparison (L, H) individuals under with optimal control situation and without control

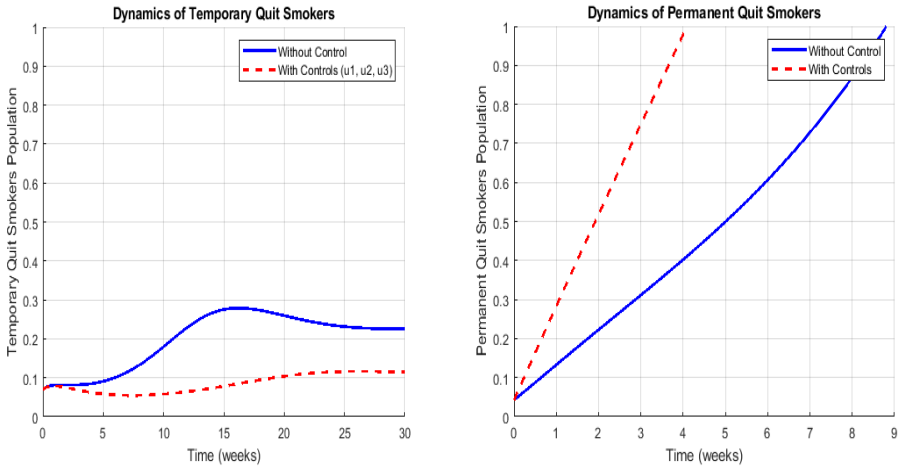
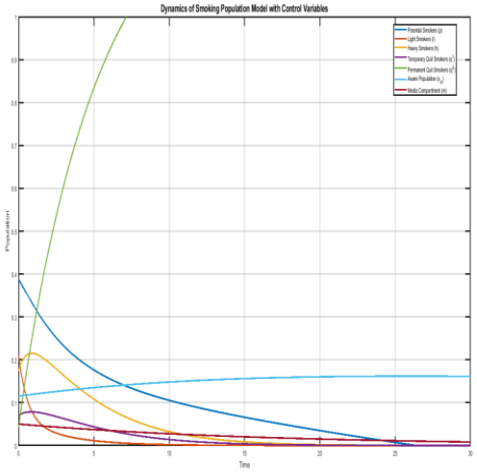
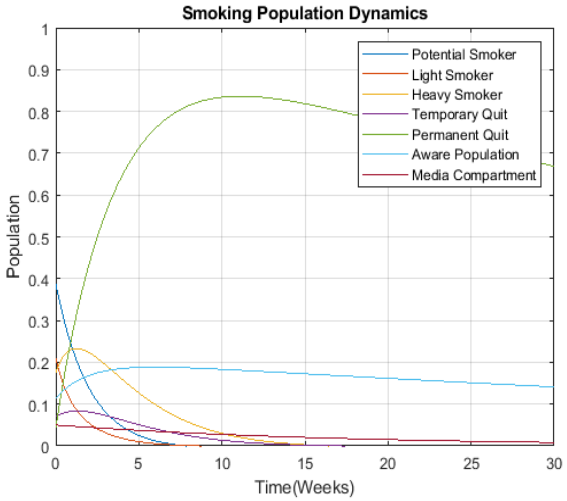


Figure 31: Comparison (Q^t, Q^P)) individuals under with optimal control situation and without control

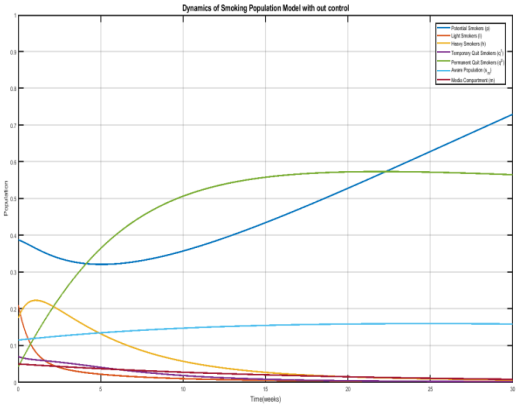
Figure32: The numerical solution demonstrates a large drop in light smokers, heavy smokers, and temporary quit smoking in case of the control measures. Media-based education, banning of smoking in public places, treatment of heavy smokers greatly cut the number of infective people in the population.





(A) PLH$Q^tQ^pS_mM$ With optimal Control When $u_1 = 0.1, u_2 = 0.1$ and $u_3 = 0$

PLH$Q^tQ^pS_mM$ With optimal Control when $u_1 = 0.3\ u_2 = 0.3\ and\ u_3 = 0$



(B) PLH$Q^tQ^pS_mM$ Without optimal Control

Figure 32: PLH$Q^tQ^pS_mM$ With optimal Control and without optimal Control

Figure33: show the numerical solution of controlled and uncontrolled PLH$Q^tQ^pS_mM$ model. From figure 33(A): Potential smoker, Light smoker, Heavy smoker and Temporary quit smoker individuals were minimize slowly. This means smoking tobacco without any control increasing potential smoker, light smoker and Heavy smoker individuals. But on the other figure 33 (B) show the numerical solution of controlled PLH$Q^tQ^pS_mM$ model we observe that the infectious individuals decrease significantly when control strategies are implemented and recovered or

permanent quit individuals increasing at the end of the control period. The results clearly show using different controlling methods given to the potential smoker, light smoker and Heavy smokers.

Strategy B:-Optimal Use Control (u_1, u_2) on individuals

The objective function is optimized through the application of the control variables u_1, u_2. In strategy B using u_1, u_2 that indicates the number of potential smokers, light smokers, and heavy smokers is reduced, but the number of smokers who permanently stop increases after a brief period of time in comparison to the two and three control models on approach B. Each of the following figures displays the control variables u_1, u_2.

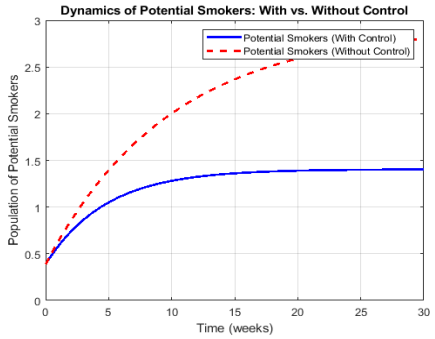


Figure 33: Comparison of potential smoker individuals

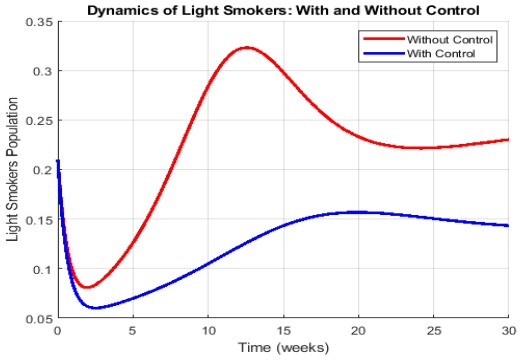


Figure 34: Comparison of light smoker individuals

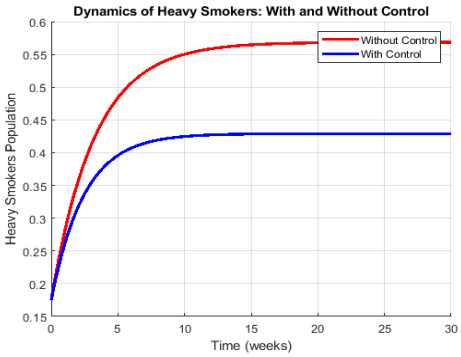


Figure 35: Dynamics of Heavy smokers with and without control

Strategy C:- Optimal Use Control $u_1$on individuals: The control variables u_1 applicable in the optimization of the objective. The formula of strategy C with $u_1$implying number of infected individuals minimizing with a small time interval is: All the figures below demonstrate that control variable u(1) susceptible, vaccination and exposed individuals minimize but recovered individual increasing small time interval compared to that in two and three control model on strategy C.

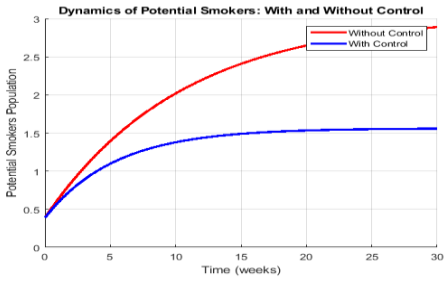


Figure 36: Comparison of potential smoker individuals

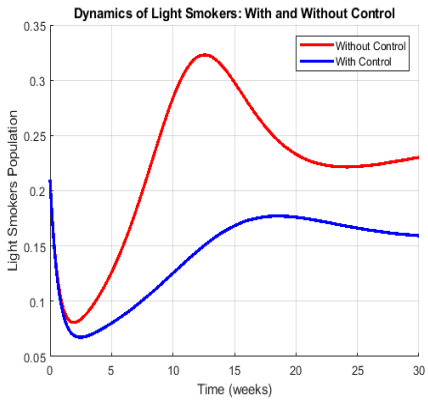


Figure 37: Comparison of light smoker individuals

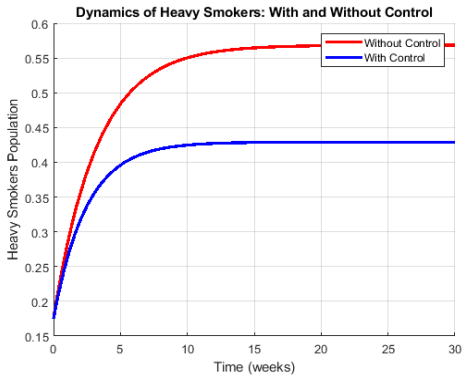


Figure 38: Comparison of heavy smoker individuals

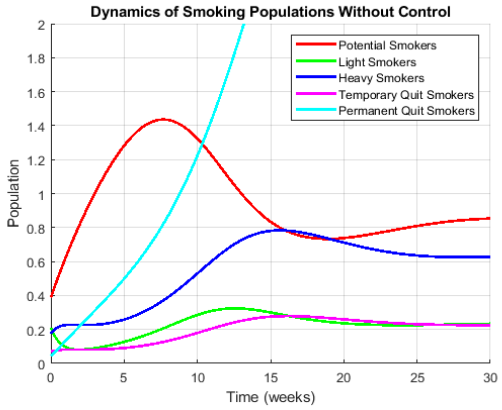


Figure 39: $\mathbf{PLHQ^tQ^pS_mM}$ without control

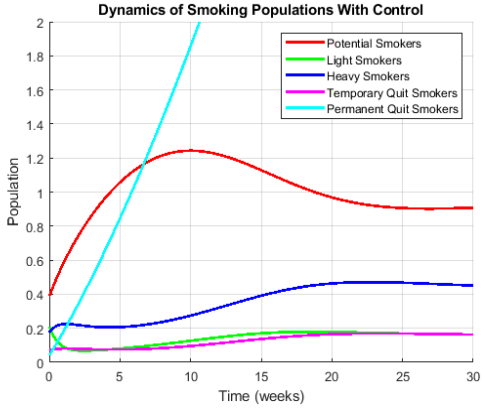


Figure 40: $\mathbf{PLHQ^tQ^pS_mM}$ with control

From figures (37-41) shows the potential smoker and light smoker individuals were decrease slower than that of strategy A(with the two optimal control). The recovered individuals were increasing slower than having one u_1 optimal control variable. u_1 was controled variable used to

minimize the susceptible individuals. Finlay to get effective solution for minimize the infected individual by smoking tobacco the more perfectible method is use three optimal control than with only one control variable(u_1)

Strategy D:-Optimal Use Control u_3 upon people. The control variables u_3 were utilized to minimize the objective. Strategy C with u_3 that is number of infected individuals minimize with small time interval. All the figures below represent the control variable u_3 potential smokers, the light and heavy smokers minimize but recovered individual increasing small time interval on strategy D with two and three control model.

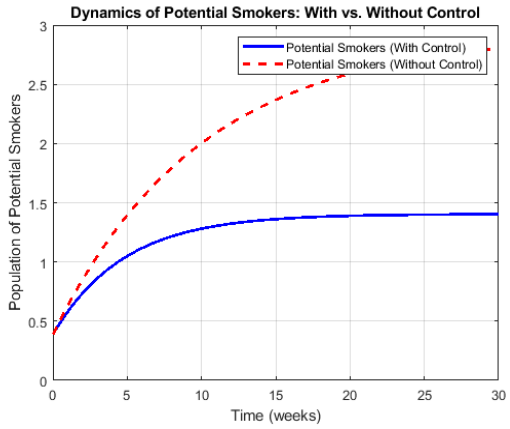


Figure 41: Comparison of potential smoker individuals

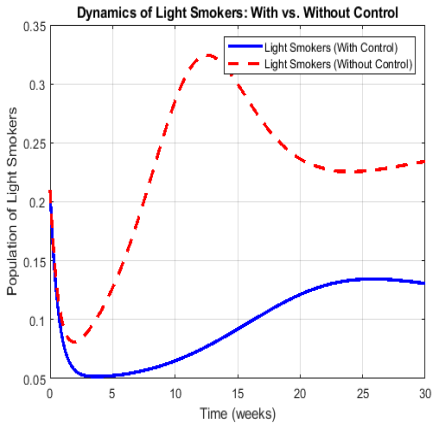


Figure 42: Comparison of light smoker individuals

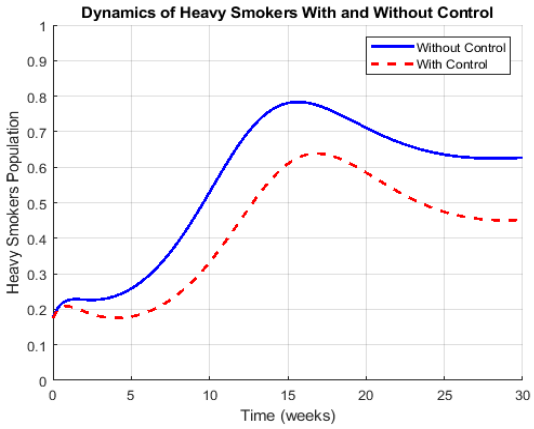


Figure 43: Comparison of heavy smoker individuals

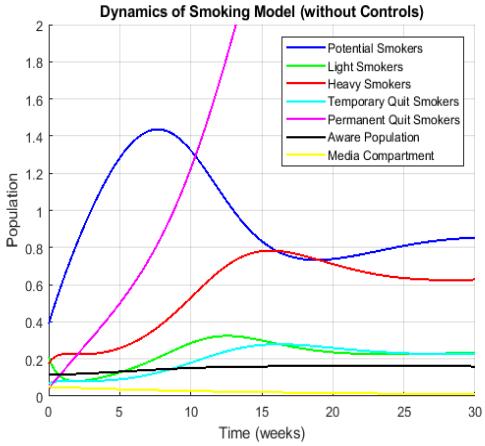


Figure 44: $PLHQ^tQ^pS_mM$ without control

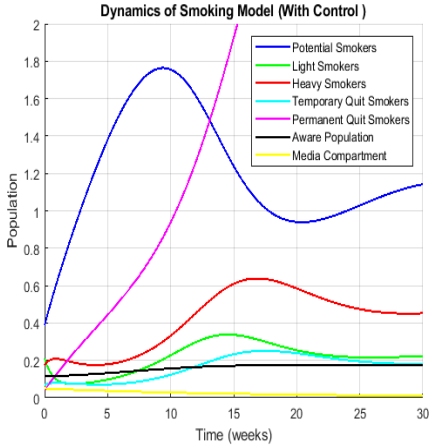


Figure 45: $PLHQ^tQ^pS_mM$ with control

From figures(42-46) shows the susceptible and infected individuals were decrease slower than that of strategy A(with the two optimal control). The recovered individuals were increasing

slower than having one u_3 optimal control variable. u_3 Was control variable used to minimize the susceptible individuals. Finlay to get effective solution for minimize the infected individual by smoking tobacco the more perfectible method is use three optimal control than with only one control variable (u_3).

Chapter 6: Conclusion and Recommendation

Conclusion

The paper formulated and developed a mathematical model to comprehend how smoking behavior works in Mekelle Town, Ethiopia, using primary data obtained through a sample of 400 respondents who were sampled in four of the closely populated zones. The analysis of the model was based on the parameter estimation that was determined after considering real-life data.

It was estimated that the basic reproduction number R_s= 1.19255 is larger than 1. This serious observation shows that the smoking is already spreading among the population of the Mekelle Town and has become a long-term health problem in the population. The smoking-free equilibrium and the endemic equilibrium were both discovered to be locally asymptotically stable to establish that even though a smoke-free state is in theory stable, the modern dynamics has the population in the endemic equilibrium in which smoking continues.

The sensitivity analysis showed the main causes of the smoking epidemic:

The effective contact rate (β) and rate at which light smokers transform into heavy smokers (α_2) are factors that increase the rate of smoking diffusion.

Some of the factors which decrease the spread of smoking include increase in quitting rates by light smokers (α_1, α_3), increase in removal rates by heavy smokers (γ), and the mortality rates attributed to smoking (d_1, d_2).

These results were supported by the numerical simulations that show that once the transmission parameters surpass critical values, smoking turns endemic, whereas the growth in the rate of cessation can eventually cause smoking to be suppressed in the society.

Above all, optimal control analysis considered three intervention measures, i.e. counseling/awareness programs (u_1), prohibited smoking in the open spaces (u_2), and treatment of serious smokers u_3). The findings are conclusive that a combination of all the three control measures (Strategy A) is the most effective. Such combined strategy will greatly decrease the number of potential smokers, light smokers, and heavy smokers and raise the number of permanent quitters. Single-control strategies (u_1 or u_3 only) demonstrated slower improvement, and it confirms that a multi-pronged intervention strategy is required to be used to improve it.

Finally, this study has made solid arguments that smoking is a persistent health issue among the people of Mekelle Town. Nevertheless, as the model shows, properly structured multi-faceted intervention programs that incorporate an awareness campaign, restriction of smoking in the community, and cessation management can help to reverse this trend successfully. The findings provide policymakers and government health officers with evidence based information when investing resources in strategies that are the most effective in smoking cessation.

Recommendation

In accordance with the results of the given study, the following recommendations may be suggested: Since the model indicates that awareness on media can be important in determining whether to smoke or to avoid it, one of these scenarios that may be explored by the local health planners in Mekelle with regard to future preventive measures is how awareness campaigns may be formulated or reinforced. Moreover, fiscal policy might also be examined by policy makers on different levels that may, in turn, be used to discourage smoking initiation and promote cessation as this kind of economic repelling has proven effective in other areas. According to the optimal control analysis, awareness + counseling and treatment support may be worthwhile. Future research might explore in cases of such combined interventions are ready in the current Mekelle health services. Specifically, since addiction is a complex concept, further studies can also draw inspiration in investigating the correlation between smoking and drinking since alcohol can greatly lead to the aggravation of smoking habits and likelihood of relapse in individuals wishing to quit. The paper has pointed out the significance of data. Assuming that local health authorities in Mekelle can gather and keep some basic data concerning smoking prevalence and issues linked to smoking, then such data would be useful to future studies in the field. To the researcher who is interested in developing this work, the model can be taken in a number of ways. Other elements like peer pressure, economic status, or cessation programs are other factors that can be included to fully paint the picture. Another approach to the dynamics of smoking intensity, such as the shift to light smoker, gradual movement between initial awareness of risks to permanent cessation would also be interesting. Similar modeling methods can also be used on other towns or substances and find comparisons based on such comparisons. Lastly, this research is presented as a modest contribution to the research on the smoking dynamics in Mekelle. Hopefully, the results can somehow help in the sustained discourse of public health issues within the society, albeit in a small manner.

Reference

1. Sweanor, D. T., Rose, . Christine Ann , Hilton, . Matthew J. and Henningfield, . Jack (2024, May 19). smoking. Encyclopedia Britannica. https://www.britannica.com/topic/smoking-tobacco
2. Armani C, Landini L, Leone A. Interactive effect of cigarette smoking and gene variants for predisposing to cardiovascular disease. Curr Pharm Des. 2010;16(23):2531-8. doi: 10.2174/138161210792062885. PMID: 20550507.
3. Chase, M. W., Christenhusz, M. J., Conran, J. G., Dodsworth, S., Medeiros de Assis, F. N., Felix, L. P., & Fay, M. F. (2018). Unexpected diversity of Australian tobacco species (Nicotiana section Suaveolentes, Solanaceae). *Curtis's botanical magazine*, *35*(3), 212-227.
4. Henningfield, J. , Sweanor, . David T. , Rose, . Christine Ann and Hilton, . Matthew J. (2024, May 19). Smoking. Encyclopedia Britannica. https://www.britannica.com/topic/smoking-tobacco
5. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, World Health Organization, & International Agency for Research on Cancer. (2004). *Tobacco smoke and involuntary smoking* (No. 83). Iarc.
6. Lee, S., Ling, P. M., & Glantz, S. A. (2012). The vector of the tobacco epidemic: tobacco industry practices in low and middle-income countries. *Cancer Causes & Control*, *23*, 117-129.
7. Office of the Surgeon General (US); Office on Smoking and Health (US). The Health Consequences of Smoking: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2004. 4, Respiratory Diseases. Available from: https://www.ncbi.nlm.nih.gov/books/NBK44694/
8. Emmanuel Appoh Andam(2013).Analysis of transmission dynamics of tuberculosis (tb) Using differential equations:a case study of Amansie West district,Ghana,Master Thesis, University of Kwame Nkrumah, Ghana.
9. Haines, C., & Crouch, R.(2007).Mathematical modeling and applications: Ability and Competence frameworks. In W. Blum, P. L. Galbraith, H. Henn, & M. Niss (Eds.), Modeling and applications in mathematics education: The 14th ICMI study,NY: Springer,(pp. 417-424), New York.
10. Lesh, R., & Doerr, H. M.(2003a).Foundations of a models and modeling perspective on Mathematics teaching, learning, and problem solving. In R. Lesh, & H. M. Doerr (Eds.), Beyond constructivism: Models and modeling perspectives on mathematics problem solving, learning, and teaching,(pp. 3-33),Mahwah, NJ: Lawrence Erlbaum.
11. Hethcote, H.W.(2000).The Mathematics of Infectious Disease and the basic epidemiology models, SIAM REVIEW,42(4),599-653.
12. Aron, J.L.(2006).Mathematical Modeling: The Dynamics of Infection (Nelson, K.E., & Williams, C.M., Eds.). In Infectious Disease Epidemiology: Theory and Practice.(2nd Ed.Chap.6, MA: Jones & Bartlett Publishers.

13. Bernoulli,D.(1760). Essai dune nouvelle analyse de la mortalite causee par la petite verole,et des avantages de linoculation pour la prevenir. Histoire de lAcad. Roy. Sci. (Paris) avec Mem. des Math. et Phys. and Mem., 00:145. English translation:Rev. Med.Virol.14,275-288(2004).
14. Ross R.(1911).The Prevention of Malaria,London.
15. Kermack, W.O. & McKendrick, A.G.(1927).A contribution to the mathematical theory of epidemics,Proc R Soc Lond;A115:700−21.
16. Bailey, N.T.J.(1975).The Mathematical Theory of Infectious Disease,2nd ed.,New York: Hafner.
17. Feng Z, Castillo-Chavez C, Capurro AF.(2000).A model for tuberculosis with exogenous Reinfection. Theoretical Population Biology,57:235-247.
18. Acevedo-Estefania, C., Torre, C., Cintron-Arias, A., Hernandez-Suarez, C., & Nshinyabakobeje, S. (2002). A Stochastic Approach for Modeling Treatment and Diagnosis of Attention Deficit Disorder.
19. Labzai, A., Balatif, O., & Rachik, M. (2018). Optimal control strategy for a discrete time smoking model with specific saturated incidence rate. *Discrete Dynamics in Nature and Society*, *2018*(1), 5949303.
20. Zeb, A., Bibi, F., & Zaman, G. (2015). Optimal control strategies in square root dynamics of smoking model. *International Journal of Scientific World*, *3*(1), 91-97.
21. Hussain, T., Awan, A. U., Abro, K. A., Ozair, M., & Manzoor, M. (2021). A mathematical and parametric study of epidemiological smoking model: a deterministic stability and optimality for solutions. *The European Physical Journal Plus*, *136*, 1-23.
22. Sofia, I. R., Bandekar, S. R., & Ghosh, M. (2023). Mathematical modeling of smoking dynamics in society with impact of media information and awareness. *Results in Control and Optimization*, *11*, 100233.
23. Khyar, O., Danane, J., & Allali, K. (2021). Research Article Mathematical Analysis and Optimal Control of Giving up the Smoking Model.
24. Ullah, A., Sakidin, H., Shah, K., Hamed, Y., & Abdeljawad, T. (2024). A mathematical model with control strategies for marijuana smoking prevention. *Electronic Research Archive*, *32*(4), 2342-2362.
25. Burns, D. M. (1991). Cigarettes and cigarette smoking. *Clinics in chest medicine*, *12*(4), 631-642.
26. Gately, I. (2007). *Tobacco: a cultural history of how an exotic plant seduced civilization*. Open Road+ Grove/Atlantic.
27. Strzelak, A., Ratajczak, A., Adamiec, A., & Feleszko, W. (2018). Tobacco smoke induces and alters immune responses in the lung triggering inflammation, allergy, asthma and other lung diseases: a mechanistic review. *International journal of environmental research and public health*, *15*(5), 1033.
28. Csordas, A., & Bernhard, D. (2013). The biology behind the atherothrombotic effects of cigarette smoke. *Nature Reviews Cardiology*, *10*(4), 219-230.

29. Smoking causes Cardiovascular, H. (2011). Disease)• Passive smoking.
30. Cepeda-Benito, A., Reynoso, J. T., & Erath, S. (2004). Meta-analysis of the efficacy of nicotine replacement therapy for smoking cessation: differences between men and women. *Journal of consulting and clinical psychology*, *72*(4), 712.
31. Sakthisankaran, S. M., Sakthipriya, D., & Swamivelmanickam, M. (2024). Health Risks Associated with Tobacco Consumption in Humans: An Overview. *Journal of Drug Delivery and Therapeutics*, *14*(5), 163-173.
32. Hethcote, H.W.(2000).The Mathematics of Infectious Disease and the basic epidemiology models, SIAM REVIEW,42(4),599-653
33. World Health Organization. Non-communicable diseases: key facts. Geneva: World Health Organization; 2018.
34. Chan, M. (2010). Global status report on no communicable diseases. *World Heal Organ*.
35. WHO. Tobacco: Key facts. Geneva, Switzerland: World Health Organization; 2020. Updated 27 May 2020. Available from: https://www.who.int/news-room/fact-sheets/detail/tobacco
36. Ezzati M, Lopez AD. Estimates of global mortality attributable to smoking in 2000. Lancet. 2003;362(9387):847–52.
37. Warren CW, Asma S, Lee J, Lea V, Mackay J. The global tobacco Surviellance system, The GTSS Atlas: CDC Foundation; 2009.
38. WHO. Global Status report on non-communicable diseases. 2010.
39. Williams J, Allen L, Wickramasinghe K, Mikkelsen B, Roberts N, Townsend N. A systematic review of associations between non-communicable diseases and socioeconomic status within low-and lower-middle-income countries. J Glob Health. 2018;8(2).
40. Di Cesare M, Khang Y-H, Asaria P, Blakely T, Cowan MJ, Farzadfar F, et al. Inequalities in non-communicable diseases and effective responses. Lancet. 2013;381(9866):585–97.
41. Juma PA, Mapa-Tassou C, Mohamed SF, Mwagomba BLM, Ndinda C, Oluwasanu M, et al. Multi-sectoral action in non-communicable disease prevention policy development in five African countries. BMC Public Health. 2018;18(1):1–11.
42. WHO. Preventing CHRONIC DISEASES a vital investment. Geneva: World health Organization; 2005.
43. Drope JSN, Cahn Z, Drope J, Hamill S, Islami F, Liber A, et al. The tobacco atlas: Ethiopian factsheet. Atlanta, Georgia, USA: American Cancer Society and Vital Strategies; 2016.
44. EDHS. Ethiopian Demographic and Health Survey. 2011.
45. Defar A, Getachew T, Teklie H, Bekele A, Gonfa G, Gelibo T, et al. Tobacco use and its predictors among Ethiopian adults: a further analysis of Ethiopian NCD STEPS survey-2015. Ethiop J Health Dev. 2017;31(1):331–9.
46. DACA. A report on global youth tobacco survey (GYTS) conducted in secondary school in Addis Ababa, Ethiopia. 2005.
47. Tu,P.N.V.(1994).Dynamical System An Introduction with Applications in Economic and Biology, Springer-Verlag, Heidelberg, Germany.

48. Ayodeji. O. Adebiyi(2016).Mathematical modeling of the population dynamics of tuberculosis, Master Thesis, University of the Western Cape, South Africa.
49. Emmanuel Appoh Andam(2013).Analysis of transmission dynamics of tuberculosis (tb) Using differential equations: a case study of Amanze West district, Ghana, Master Thesis, University of Kwame Nkrumah, Ghana.
50. Diekmann, O., Heester Beek J. A. P. , Metz J. A. J.(1990).On the definition and the Computation of the basic reproduction ratio R0 in models for infectious diseases in Heterogeneous populations. Math. Biol. 28,no. 4, 365-382.
51. Van den Driessche P.,Watmough J.,(2002).Reproduction numbers and sub-threshold Endemic Equilibrium for compartmental models of disease transmission. Mathematical Biosciences; 180:29-48.
52. P. van den Driessche, J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," Math. Biosci. 180 (2002), pp. 29–48.
53. C. Castillo-Garsow, G. Jordan-Salivia, and A. Rodriguez Herrera, " Mathematical models for the dynamics of tobacco use, recovery, and relapse," Technical Report Series BU-1505-M, Cornell University, Ithaca, NY, USA, 1997.
54. O. Sharomi, A. B. Gumel, "Curtailing smoking dynamics: A mathematical modeling approach," Applied Mathematics and Computation 195 (2008) 475–499.
55. G. Zaman, "Qualitative behavior of giving up smoking model"; Bulletin of the Malaysian Mathematical Sciences Society (2) (2011) 403-415.
56. Z. Alkhudhari, S. Al-Sheikh, S. Al-Tuwairqi, "The effect of heavy smokers on the dynamics of a smoking model," International Journal of Differential Equations and Applications, 14, No. 4 (2015), pp. 1311-2872.
57. A. Matins, "Smoking as Epidemic Modeling and Simulation Study," American Journal of Applied Mathematics Volume 5, Issue 1, (2017), pp. 31-38 .
58. WHO, Tobacco, https://www.who.int/news-room/fact-sheets/detail/tobacco (accessed 2019-05-24).
59. Sebastian, E., & Victor, P. (2017). Optimal control strategy of a discrete time SVIR Epidemic model with immigration of invectives. *Int. J. Pure Appl. Math*, *113*(8), 55-63.
60. Rose, G. R. (2015). Numerical methods for solving optimal control problems.
61. Breuer, F., Castillo-Chavez, C., & Castillo-Chavez, C. (2012). *Mathematical models in population biology and epidemiology* (Vol. 2, No. 40). New York: springer.
62. Lenhart, S., & Workman, J. T. (2007). *Optimal control applied to biological models*. Chapman and Hall/CRC.
63. Becerra, V. M. (2008). Optimal control. *Scholar pedia*, *3*(1), 5354.
64. Yoshida, N., & Hara, T. (2007). Global stability of a delayed SIR epidemic model with density dependent birth and death rates. *Journal of Computational and Applied Mathematics*, *201*(2), 339-347.
65. Pontryagin, L. S. (2018). *Mathematical theory of optimal processes*. Routledge.

66. Mekelle Population 2021. "World Population Review". *World Population review*
67. Aberra, Yohannes (2007). "Mäqälä". In Uhlig, Siegbert (ed.). *Encyclopaedia Aethiopica*. Wiesbaden: Harrassowitz Verlag.
68. Conrad, T. O., Ferrer, E., Mietchen, D., Pusch, L., Stegmüller, J., & Schubotz, M. (2024). Making Mathematical Research Data FAIR: Pathways to Improved Data Sharing. *Scientific Data*, *11*(1), 676.
69. Adhana, M. A., & Mekonnen, T. T. (2019). A mathematical model analysis of smoking tobacco in the case of Haremaya town; Ethiopia. *Int. J. Res. Stud. Sci. Eng. Technol*, *6*(2), 14-24.
70. Chitnis N, Hyman JM, Cushing JM. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model.Bull Math Biol 2008;70(5):1272–96.
71. Stuber, J., Galea, S., & Link, B. G. (2008). Smoking and the emergence of a stigmatized social status. *Social science & medicine*, *67*(3), 420-430.
72. Brauer, F., Van den Driessche, P., & Allen, L. J. (2008). *Mathematical epidemiology* (Vol. 1945, pp. 3-17). J. Wu (Ed.). Berlin: Springer.
73. Keeling, M. J., & Rohani, P. (2008). *Modeling infectious diseases in humans and animals*. Princeton university press.

Appendix

Tobacco Use Questionnaire

Demographic Information:

Age: _____

Gender: _____

Ethnicity: _____

Educational Level: _____

Occupation: _____

Section 1: Smoking Behavior

1. Do you currently smoke tobacco?

   Yes

   No

If yes, at what age did you start smoking?

2. How many cigarettes do you smoke per day?

   1-5

   6-10

   11-20

   More than 20

3. Have you ever tried to quit smoking?

   Yes

   No

If yes, what methods did you use to quit? (Select all that apply)

4. Nicotine Replacement Therapy

Prescription Medications

Counseling

Other: _____

Section 2: Influencing Factors

5. Do any of your family members smoke?

   Yes

   No

6. How often do you see people smoking in your community?

   Rarely

   Sometimes

   Often

7. Do you feel peer pressure to smoke?

   Yes

   No

8. How does alcohol use affect your smoking habits?

   Increases my smoking

   Decreases my smoking

   No effect

Section 3: Awareness and Attitudes

9. Are you aware of the health risks associated with smoking?

   Yes

   No

10. How effective do you think anti-smoking campaigns are?

    Very effective

    Somewhat effective

    Not effective

11. Would you support policies that increase taxes on tobacco products?

    Yes

    No

12. What resources would help you quit smoking? (Select all that apply)

    Support groups

    Educational materials

Access to cessation programs

Other: _____

Section 4: Additional Comments

13. Please share any additional comments or thoughts regarding smoking and tobacco use: