MODELLING THE DYNAMICS OF CONJUNCTIVITIS ALONG THE TANO BASIN

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by

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DECLARATION AND CERTIFICATION

Student Declaration

I, Samuel Dateh **Ampofo** (UEMP1902021), hereby declare that this study was carried out and written by me, and that all sources of information have been acknowledged and that I am wholly responsible for any act that may infringe on the research ethics policies of this University.

Candidate's Signature: .... Date: ....

Supervisor's Certification

This study was carried out under the supervisory committee of supervisors in accordance with the guidelines on supervision of the graduate school.

Major Supervisor: Prof. Dominic Otoo, Ph.D

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ABSTRACT

Ghana’s Tano Basin continues to be a major public health hazard for conjunctivitis due to the introduction of adenovirus and herpes simplex virus into river bodies by activities of anthropogenic and zoogenic. The study objectives were to develope a mathematical model to explain the dynamics of conjunctivitis in the the Tano basin, the local analysis on the model was performed and behaviour of key parameters at different α values were investigated. In order to account for the effects of memory and non-locality, this work modelled the dynamics of conjunctivitis transmission using Caputo-Fabrizio differential equations. For numerical simulation, the Lagrange polynomial iterative scheme is applied. We examine the model at different α values, which span from 1 to 0.65, in order to evaluate the effect of memory degradation on the spread of disease. The findings demonstrate that the dynamics of the disease are considerably changed by lowering α values, with lower values (α < 0.80) displaying more oscillations and a lower peak incidence. It is also discovered that α affects the basic reproduction number R_0, with R_0 falling as alpha falls. According to the research, taking memory effects into consideration while analysing the spread of conjunctivitis can enhance model accuracy and guide sensible management measures.

DEDICATION

To my dear wife, Deborah Nana Gyamaa Bonsu, for your unflinching support and prayers.

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I am indebted to God Almighty, who has always been my guide and hope and has seen me through the completion of this program.

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Introduction

1.1 Overview

This chapter will look at background of the study, statement of the problem, the objective of the study, the methodology, significance of the study, the limitation and the organization of the work.

1.2 Background of the Study

1.2.1 Water Pollution

In recent years, there has been a significant rise in urban wastewater generation due to various factors, including population growth, industrial output, expansion of commercial activities, and alterations in water usage patterns. In spite of the implementation of countermeasures, the disposal of excess wastewater in many urban areas of developing countries continues to occur directly with or with-out adequate treatment, leading to a substantial decline in the quality of surface water bodies. The presence of water contamination poses a significant threat to the sustainability of urban systemsB. K. Mishra et al. (2017)

Rao (2007) defined Pollution as the deliberate or unintentional release of anthropogenic energy or substances into the environment, which possess the potential to pose risks to human well-being, inflict harm upon structures or facilities, impair living resources and ecological systems, or disrupt the lawful utilization of the environment.

Water, an essential component for sustaining life on our planet, is unfortunately subject to daily pollution, a problem that is progressively worsening. The contamination of water bodies occurs as a result of the discharge of neglected waste, filth, run-off from farming operations, and toxic metals Armah et al. (2014).

According to Baginda & Zainudin (2009), The degradation of air and water quality has a significant impact on the biota within the surrounding ecosystem. Ensuring access to clean and uncontaminated water is a fundamental necessity for both humans and animals. However, the presence of water pollutants in aquatic environments has become a prevalent issue, primarily due to anthropogenic activities and natural phenomena, leading to the phenomenon commonly referred to as water pollution. The utilization of water contaminated with water pollutants for domestic purposes, such as drinking and cooking, can lead to the transmission of waterborne diseases. Infectious diseases, including dysentery, malaria, typhoid, cholera, amoebiasis, hookworm, and giardiasis, can be acquired through the consumption of contaminated water sources such as rivers, lakes, streams, and groundwater for domestic purposes Gerba & Pepper (2019). Infections such as earache, cunjunctivitis (pink eye), hepatitis, gastroenteritis, rashes, and respiratory infections can be acquired by bathing in water contaminated with water contaminants, Belhassan (2021).

There is a collective concern among the government, local authorities, government agencies, and the general public regarding the water quality standards in Ghana, specifically pertaining to its rivers. Pollutants can potentially exert an indirect influence on a species by leading to the elimination of another species within the food web or food chain. This subsequent loss of a species can disrupt the trophic structure, resulting in an unstable ecological system. One of the most formidable challenges confronting society pertains to the environmental transformation resulting from pollution, which exerts significant influence on human lifestyles, biodiversity within habitats, and the enduring viability of aquatic species Siddiqua et al. (2020).

Eutrophication occurs when an excessive influx of nutrients, primarily resulting from anthropogenic activities such as agricultural practices, sewage disposal, or urban run-off, enters a water body. The proliferation of algae and other aquatic flora is facilitated by heightened nutrient concentrations, leading to the formation of a dense layer of vegetation on the surface of the water. During the process of decomposition and senescence, these plants gradually exhaust the oxygen content of the water, thereby posing a threat to the survival of fish and other organisms inhabiting the aquatic ecosystem. Eutrophication can lead to various ramifications, such as modifications in water quality, diminished biodiversity, and disruptions in the food chain. One potential consequence of the proliferation of algae and other plant species is the creation of ”dead zones,” characterized by insufficient oxygen levels to sustain aquatic organisms.

Moreover, the process of eutrophication can lead to the excessive growth of harmful algal blooms. These blooms have the potential to generate toxins that can adversely affect aquatic organisms and pose a significant risk to human well-being if consumed via contaminated seafood or water sources.

1.2.2 Conjunctivitis

Conjunctivitis, commonly referred to as pink eye, refers to the inflammatory or infectious condition affecting the conjunctiva. The conjunctiva is a delicate and translucent tissue layer that lines the inner surface of the eyelid and envelops the sclera, the white portion of the eye. The etiology of this condition encompasses viral, bacterial, allergenic, and irritant factors.The clinical manifestations of conjunctivitis may encompass ocular erythema, pruritus, a sensation of burning or stinging, lacrimation or purulent discharge, edema of the eyelids, photophobia, and visual impairment. The severity of symptoms and their impact on ocular health can vary depending on the underlying etiology, potentially affecting either unilateral or bilateral ocular involvement.

Conjunctivitis possesses the potential for contagion, particularly when instigated by viral or bacterial agents. Transmission occurs via direct contact with ocular secretions, contaminated surfaces, or personal articles such as towels or cosmetics. Viral conjunctivitis is frequently a self-limiting ailment that resolves spontaneously within a span of several days to a few weeks, without the manifestation of any enduring symptoms. Nevertheless, in certain instances, specifically among individuals with compromised immune systems, viral conjunctivitis may lead to complications or exhibit a prolonged duration. Viral conjunctivitis can result in corneal damage, leading to the development of scarring or other visual impairments as a long-term consequence or complication.

The presence of microorganisms or noxious chemicals in contaminated water has the potential to induce conjunctivitis, an inflammation of the conjunctiva that can result in eye infection or irritation. One instance where water that is contaminated with bacteria, viruses, or parasites can result in the development of bacterial or viral conjunctivitis is observed.Chemical substances, including chlorine, ammonia, and various pollutants commonly present in contaminated water, have the potential to impact the eye. These chemical substances have the potential to induce irritation and inflammation of the conjunctiva, resulting in the development of conjunctivitis.Individuals who engage in swimming activities within bodies of water that have been contaminated, such as lakes or rivers, face an elevated susceptibility to the development of conjunctivitis as a consequence of their exposure to these deleterious substances. Likewise, individuals who engage in the practice of utilizing water that is contaminated for the purpose of cleansing their face or hands are also susceptible to the potential development of conjunctivitis.

1.3 Study Area

The Tano basin's topography varies from 0 to 700 meters above sea level. The basin's climate is typified by double rainfall maxima because it lies partially in Ghana's wet, semi-equatorial and partially in its south-western equatorial climate zones. The majority of the vegetation in the Tano basin is made up of tropical evergreen forests, which make up 50 % of the forest reserve, 20 % of the Celtis-Triplochiton association, and 10% of rain forests. The Tano basin, which is a part of Ghana's southwest river system, receives between 1136.7 and 2156.0 mm of rainfall annually. The two rainfall seasons in the basin have their peaks in May/June and October/November. The region experiences yearly temperature fluctuations, with minimum and maximum values of 25.0 °C and 27.0 °C, respectively (Kesse, 1985).

This work took into consideration the people who resided in the Bono and Ahafo region along the Tano River basin. Abesim, Goaso, Mim, Hwidiem, and Achirensua were a few of the communities taken into consideration. Most of this population is reliant on the Tano River, either directly or indirectly. Illegal mining operations, farming practices by farmers, open defecation near riverbanks, and river washing are some of the factors that contaminate the water. Ghana's water resources are mostly found in these rural areas, where the majority of the population lives and works in agriculture and livestock rearing. Some of the improper practices carried out by these farmers, which lead to the pollution of these river bodies, include the improper application of fertilizers, weedicides, and pesticides; grazing of livestock in the water bodies; and open defecation along river banks. People may contract conjunctivitis as a result of feces that may harbor adenoviruses or herpes simplex viruses getting into water bodies. The goal of the study is to model the dynamics of bacteria conjunctivitis, which is thought to be responsible for 60–70% of cases of conjunctivitis in the study area.

When these faecal matter which may contain adenovirus or herpes simplex virus found itself into the water bodies, people may be infested with conjunctivitis.

The study will focus on modelling the dynamics of conjunctivitis caused by bacterial conjunctivitis which is said to account for about 60-70 percent of conjunctivitis in the study area.

1.4 Statement of the Problem

The majority of water sources in Ghana are presently contaminated due to anthropogenic and zoogenic activities. The introduction of adenovirus and herpes simplex virus into rivers can occur through the presence of human or animal fecal matter, which has the potential to harbor these viruses. This phenomenon can arise as a result of inadequate waste or sewage disposal, the run-off from agricultural or industrial activities, or unintentional releases of contaminated substances. Upon introduction into the aquatic environment, these viruses have the potential to infect other individuals who come into contact with the water that has been contaminated or consume it.

According to Sonone et al. (2020), Water pollution, specifically nutrient pollution (including nitrogen, urea, and phosphates), oil pollution, and chemical contamination (such as hydrocarbons, mercury, and pesticides), have been found to be associated with adverse health effects. These effects include the development of cancer and hormone-related issues, as well as damage to the liver and kidneys. Additionally, water pollution has been linked to slower rates of growth and development. Certain pollutants have been found to impede the growth and development of marine species, whereas others have been observed to induce adverse effects such as nervous system impairment, reduced reproductive capabilities, and mortality.

Human health is greatly impacted by water pollution, especially in areas where freshwater and surface water are the main sources of drinkable water. Bacteria, parasites, and viruses are examples of pathogenic microorganisms that proliferate in aquatic environments. Drinking this water encourages the spread of illnesses that are highly dangerous to people's health. There is a serious risk to human health when eating plants, animals, or aquatic life directly. Nowadays, people all across the world are more conscious of and concerned about water pollution. As a result, fresh concepts and methods for the sustainable use of water resources have emerged.

In spite of notable advancements in scientific and public health domains, infectious diseases continue to exert a substantial toll on human populations worldwide, resulting in heightened mortality and disability rates. Notably, these diseases exhibit a disproportionate impact on impoverished nations.

According to a recent survey, it has been estimated that infectious diseases are accountable for over 50 percent of human mortality in Sub-Saharan Africa. Furthermore, the sub-lethal consequences of these diseases exert significant burdens on both the quality of life and the economic progress of the region. Ezzati et al. (2002). The environment, biological traits of the pathogens, host susceptibilities to disease, and the impact of behavioral, cultural, and social patterns that can increase the host's exposure to the disease source and, in turn, increase its susceptibility to the disease are some of the factors that influence the dynamics of this interaction.

In order to safeguard the well-being of individuals residing in the Tano basin, it is imperative to effectively manage the dynamics of the disease to mitigate the risk of conjunctivitis infections. The development of a mathematical model is imperative for the analysis and comprehension of the dynamics associated with viral conjunctivitis, with the ultimate goal of effectively managing and mitigating the transmission of this disease. Numerous scholars and researchers have extensively examined the dynamics of conjunctivitis in order to acquire comprehensive insights into the diverse transmission models that have been proposed. Høvding (2008) paper, which was published in Acta Ophthalmologica in 2008, is a comprehensive review article that seeks to offer a comprehensive overview of acute bacterial conjunctivitis.This study seeks to offer a comprehensive analysis of the epidemiology, pathophysiology, clinical manifestation, diagnostic criteria, and therapeutic interventions pertaining to this prevalent ocular infection. The primary objective of the author's research appears to be the dissemination of up-to-date evidence-based practices pertaining to the diagnosis and treatment of acute bacterial conjunctivitis, with the intention of providing healthcare professionals and clinicians with valuable insights in this domain. The goal of this research is to create a mathematical model that examines the behavior of stability at various α values and describes the dynamics of bacterial conjunctivitis

1.5 Research Questions

The direction of the study will be determined by the following research questions:

1. Is it possible to create a mathematical model that explains the Tano Basin conjunctivitis dynamics?
2. Can a local stability analysis be performed on the developed model?
3. The behaviour of the model at different alpha (α) values.

1.6 Objective of the Study

The study's principal objectives are:

1. To develop a mathematical model to explain the dynamics of conjunctivitis is the Tano basin.
2. To perform local analysis on the developed model.
3. To study the behaviour of the key parameters in the model at different alpha (α) values.

1.7 Research Methodology

The research examined the SEITRS model, which is an expansion of the SEIR model that incorporates the notion of individuals who have recovered from infection becoming susceptible once more in the future. The researchers collected data pertaining to the disease and the population being investigated. The data was utilized for the purpose of parameterizing the SEITRS model and determining the initial values of the compartments. The Caputo-Fabrizio fractional derivative was employed to solve the equations governing the dynamics of the compartments. The model underwent validation through a process of comparing its output to empirical data from the real world, as well as other models that have been published in academic literature. An analysis was conducted on the outcomes of the simulations in order to derive conclusions regarding the effects of variations in the model as a result of variations in the alpha values.

1.8 Significance of the Study

The dynamics of conjunctivitis refer to the patterns and processes by which the disease is transmitted and develops over a period of time. There are several implications that can arise from studying the dynamics of conjunctivitis. These implications encompass:

1. The examination of viral diseases can facilitate the identification of risk factors and modes of transmission that have the potential to result in outbreaks, thereby aiding public health practitioners in their efforts. The aforementioned data is utilized for the purpose of formulating preventive strategies, including the implementation of vaccination initiatives, dissemination of health education campaigns, and establishment of infection control protocols within healthcare environments.

2. Gaining an understanding of the disease's impact on a population can facilitate the more efficient allocation of resources by policy makers. The potential measures encompass the allocation of financial resources towards the advancement of research and development pertaining to treatments and vaccines. Additionally, resources are allocated towards public health campaigns and the enhancement of emergency preparedness.

3. Through the examination of disease dynamics, mathematicians have the capacity to construct and enhance epidemiological models, which in turn can provide valuable insights to guide decision-making in public health policy.

4. The occurrence of conjunctivitis can lead to notable economic ramifications, including reduced productivity and heightened healthcare expenses, attributable to its widespread prevalence. Additionally, conjunctivitis possesses the capacity to induce absenteeism among school-age children. Gaining a comprehensive understanding of the underlying dynamics can assist policymakers in accurately assessing the economic ramifications of the disease and formulating effective strategies to alleviate its effects.

1.9 Limitation of the Study

One of the primary constraints encountered during the study pertained to data collection, as health practitioners at the different health posts lacked information regarding the various types of diseases. All cases were classified as either allergic or bacterial conjunctivitis, and patients were administered antibiotics, eye drops, or eye ointment. This approach resulted in the emergence of antibioticresistant bacteria, alongside undesirable side effects including allergic reactions and disturbance of the eye's normal microbial equilibrium.

1.10 Organization of the Study

The study's background, problem statement, scope, objective, methodology, significance, and limitations were all taken into consideration in this chapter. The remaining chapters will follow this format: An overview of the body of research on conjunctivitis is provided in Chapter 2. In Chapter Three, the techniques are covered. The results analysis and discussion are covered in Chapter 4. The study's conclusions and recommendations are presented in Chapter 5, which serves as its conclusion. A bibliography completes the work.

Chapter 2

Literature Review

2.1 Introduction

The utilization of mathematical modelling is of paramount importance in comprehending and examining diverse facets of infectious diseases. Through the application of mathematical methodologies, scholars have successfully examined the complicated nature of transmission dynamics, stability, and control strategies pertaining to infectious diseases, alongside their interconnectedness with various other factors. The primary research focus of our study lies in the mathematical modelling of infectious diseases, with a specific emphasis on conjunctivitis and the intricate dynamics of disease transmission. Mathematical models have been developed by researchers to elucidate the dynamics of conjunctivitis transmission within various populations, including public schools during specific seasonal periods Masoumnezhad et al. (2020).

The utilization of mathematical modelling has demonstrated its efficacy as a valuable instrument in the examination of infectious diseases from diverse vantage points. The utilization of this method has been employed for the examination of the transmission dynamics, stability, and control strategies pertaining to numerous infectious diseases, alongside its correlations with various other factors. These models offer both quantitative and qualitative insights into the behaviour of infectious diseases, thereby facilitating the development of efficacious prevention and control strategies. Additional investigation in this particular domain has the potential to enhance comprehension of infectious diseases and its implications for the overall well-being of the general population.

2.2 Conceptual Review

2.2.1 Infectious Diseases

Infectious diseases are caused by pathogenic microorganisms, which include bacteria, viruses, fungi, and parasites and can spread from one person to another Snieszko

(1974). Numerous pathways can lead to the spread of infectious diseases, such as direct physical contact, the dispersal of airborne droplets, the contamination of consumables or water sources, or vector-mediated transmission Connolly et al. (2021). The dynamics of infectious diseases Snieszko (1974) encompass the complex relationship between the pathogen's origin, its mode of transmission, and the vulnerability of the host population..

Recognizing the emergence and dissemination of infectious diseases holds significant importance for public health, as highlighted by Dumonteil & Herrera (2020). The correlation between biodiversity and the dynamics of infectious diseases is complex and poses difficulties in its quantification (Dumonteil & Herrera, 2020). The impact of reduced host biodiversity on pathogen transmission can exhibit diverse outcomes, as evidenced by studies indicating both heightened transmission (amplification effect) and diminished transmission (dilution effect) (Dumonteil & Herrera, 2020). The variability of responses can be influenced by the specific pathogens and environmental factors at play Dumonteil & Herrera (2020).

According to Jones & Brosseau (2015), the transmission of infectious diseases can occur through the inhalation of infectious aerosols. (Jones & Brosseau, 2015), mentioned that animals that are afflicted with diseases have the ability to produce infectious aerosols, which can subsequently be inhaled by animals that are susceptible to these diseases, resulting in the transmission of the diseases. Various factors, including the quantity of afflicted animals, the rate of ventilation, and the duration of exposure, have the potential to impact the concentration of infectious particles and the susceptibility of animals to infection (Jones & Brosseau, 2015).

The consideration of the incubation period and period of infectiousness holds significant importance in the management and control of communicable diseases Czumbel et al. (2018). The comprehension of these time intervals aids in the effective implementation of suitable exclusion strategies and the mitigation of disease transmission within environments such as educational institutions and facilities for the care of young children (Czumbel et al., 2018). Nevertheless, there is a discrepancy in the accessibility of data pertaining to these time periods among different diseases (Czumbel et al., 2018).

According to Cohen (2000), the understanding of emerging infectious diseases has undergone a transformation throughout the years. The scope of this phenomenon extends beyond infectious diseases and encompasses the growing occurrence and frequency of non-communicable diseases, such as obesity Frerot´ et al. (2018). The discipline of epidemiology has responded to these transformations by redirecting its attention from solely infectious diseases to encompass a wider spectrum of health conditions (Frerot et al., 2018).´

Drawing on relevant literature,The process by which pollutants or other dangerous materials cause water bodies to become contaminated or deteriorated is known as water pollution. The environment, human health, and aquatic ecosystems are all seriously endangered by the presence of this material. Chemical, biological, and physical pollutants all have the potential to significantly affect biodiversity and the spread of diseases that are transmitted through water, which is why water is polluted. In order to protect and conserve water resources, mitigation efforts include a variety of tactics like the installation of wastewater treatment systems, the adoption of sustainable practices, and the implementation of public awareness campaigns.

2.2.2 Water Pollution

The process by which water bodies, such as lakes, rivers, seas, and groundwater, get contaminated or deteriorated because of pollutants or other dangerous substances is known as water pollution Agrawal et al. (2010). Pollutants can enter water sources due to a variety of human activities, industrial processes, natural sources, or agricultural practices (Agrawal et al., 2010). In addition to having a detrimental effect on human health, water pollution can also have an adverse effect on aquatic ecosystems and the wider environment.

The phenomenon of water pollution in developing countries can be attributed to industrialization, as it is a significant contributing factor. This is primarily due to the practice of industries releasing their waste materials into water bodies, thereby resulting in the contamination of these aquatic environments S. Mishra et al. (2019). The contamination of water can lead to the dissemination of waterborne illnesses, including but not limited to diarrhoea, cancer, and hepatitis, thereby exerting substantial repercussions on global public health (S. Mishra et al., 2019).

Water pollution, as defined by Gattringer (2018), refers to the global phenomenon of plastic waste accumulation in marine environments. This accumulation has resulted in the widespread presence of microplastics and nanoplastics in water bodies. According to (Gattringer, 2018), it has been observed that plastic particles possess the ability to assimilate toxic organic pollutants and subsequently traverse the food chain. This phenomenon poses significant hazards to human well-being, thereby underscoring the imperative of eliminating these particles. From related literatures, Water pollution is the process by which water bodies become contaminated or degraded due to the presence of pollutants or harmful substances. The presence of this substance presents substantial hazards to aquatic ecosystems, human well-being, and the surrounding environment. Water pollution is caused by the presence of chemical, biological, and physical pollutants, all of which can have significant impacts on biodiversity and the transmission of waterborne diseases. Mitigation endeavours encompass a range of strategies, such as the implementation of wastewater treatment systems, adoption of sustainable practices, and execution of public awareness campaigns, aimed at safeguarding and conserving water resources.

2.2.3 Water Pollution and Infectiuos Diseases

Many academics have been interested in and concerned about the research and concern surrounding the link between infectious diseases and water pollution. Because contaminated water sources can act as reservoirs for pathogenic microorganisms that cause a variety of illnesses, water pollution has the potential to accelerate the spread of infectious diseases and waterborne illnesses. The connection between water pollution and infectious disease outbreaks has been the subject of numerous studies.. The scholarly article authored by Schwarzenbach et al. (2010) examines the pressing necessity for enhanced sanitation practices in developing nations as a means to combat waterborne illnesses. The authors conduct an examination of the primary categories of aquatic pollutants and their impacts on human well-being, with particular emphasis on chemical contamination, encompassing both inorganic and organic micropollutants.

The authors (Schwarzenbach et al., 2010) emphasize that persistent organic pollutants (POPs) are a significant contributor to enduring regional and local water contamination, alongside geogenic pollutants, mining activities, and hazardous waste sites. (Schwarzenbach et al., 2010) have identified agricultural chemicals and wastewater sources as factors that have shorter-term impacts on regional to local scales.

Emelko et al. (2019) carried out a study to look into the incidence of infectious disease outbreaks linked to drinking water consumption in Canada. Numerous pathogens have been linked to outbreaks of waterborne diseases, including Campylobacter, Giardia, Cryptosporidium, Norwalk-like viruses, Salmonella, and the hepatitis A virus. Emelko et al. (2019)discovered that a variety of water system types, including public, semi-public, and private systems, were linked to the outbreaks.

The study conducted by Suk et al. (2020) primarily examined the cascading consequences of post-disaster outbreaks, specifically those associated with water pollution. The study conducted by Suk et al. (2020) determined that the occurrence of intense precipitation plays a significant role in the initiation of disease epidemics. This phenomenon establishes interrelationships between water systems and various environmental components, ultimately resulting in the pollution of water reservoirs. Suk et al. (2020) reported that outbreaks can be attributed to various factors, including exposure to surface water or floodwater that is contaminated, contact with animal excreta, and the living conditions that follow a disaster.

The COVID-19 pandemic has also highlighted the role that water plays in the spread of illness. In their most recent work, SanJuan-Reyes et al. (2021) evaluated wastewater as a possible channel for the spread of SARS-CoV-2. The study's authors acknowledged wastewater-based epidemiology as a very useful technique for quickly identifying and forecasting the onset and spread of infectious illnesses and epidemics. SanJuan-Reyes et al. (2021) 2021 also stressed how the environment affects COVID-19 transmission and the possible health effects on people.

These studies provide evidence of the correlation between water pollution and the transmission of infectious diseases. The presence of pathogens and contaminants in water sources that have been polluted can result in the occurrence of outbreaks and the subsequent transmission of infectious diseases. The comprehension and resolution of water pollution are of utmost importance in the prevention and mitigation of infectious diseases linked to the contamination of water sources.

2.2.4 Mathematical Modelling of Infectious Disease

According to Abassian et al. (2020),), mathematical modeling is the process of using mathematical models to formulate and clarify real-world problems or scenarios. In order to improve understanding or make it easier to make decisions based on the mathematical framework, the process comprises representing a specific aspect of the tangible world using mathematical language, Brown & Ikeda (2019). A thorough methodology that incorporates empirical data from many domains and organizational levels is mathematical modeling Zinovyev (2015). The tool in question is used in a variety of fields, including engineering, systems biology, and mathematics education Zinovyev (2015); Dada & Mendes
(2011).

A mathematical model has been developed by researchers to study the dynamics of COVID-19 transmission. The models that are frequently used in this context usually have discrete compartments with susceptible, infected, and recovered individuals in them. Additionally, these models incorporate a number of variables, including the incident and immigration rates Din et al. (2020). By using nonlinear analysis and other pertinent techniques, researchers can investigate the qualitative aspects of the model, such as the existence of equilibriums and stability outcomes Din et al. (2020).

The utilization of mathematical modelling has become a prevalent and established approach in providing support for public health policy, with a specific emphasis on the domain of infectious disease control. Decision-makers at different hierarchical levels depend on mathematical models to inform their decisionmaking processes and formulate efficient strategies for disease control Kretzschmar (2020). According to Porgo et al. (2019), mathematical models offer significant contributions in understanding the effects of interventions, exposure risks, health outcomes, and health costs. These models play a crucial role in informing evidence synthesis and facilitating clinical decision-making.

Beyond the context of COVID-19, mathematical modeling is used in the field of infectious disease research. Moreover, this methodology has been applied to a number of infectious diseases, such as highly drug-resistant tuberculosis. The application of mathematical models has been utilized to investigate the outcomes of interventions implemented in healthcare facilities with the objective of reducing the spread of tuberculosis contracted in these environments(Porgo et al., 2019). These models' development has advanced significantly in recent decades, which has sped up their evolution. According to (Land et al., 2019) these models have proven to be accurate in predicting the frequency of various diseases, which makes it easier to identify strategies that work well for controlling disease.

Mathematical modelling serves as a potent instrument in comprehending the complicated nature of infectious disease dynamics. This tool enables researchers to effectively explain and forecast the dissemination of diseases, evaluate the consequences of interventions, and provide valuable insights for decision-making processes in the field of public health. The utilization of mathematical models has been widely employed in the examination of COVID-19, tuberculosis, and other contagious illnesses, yielding significant insights into the transmission of diseases and strategies for their containment. The incorporation of mathematical modelling with interdisciplinary approaches and computational tools serves to augment our comprehension of disease dynamics and bolster the implementation of evidence-based public health policies.

2.2.5 Mathematical modelling of infectious Disease using Caputo-Fabrizio fractional derivative

Pandey et al. (2022), modelled the dynamics of Covid-19 in India using CaputoFabrizio fractional derivative. They investigated the dynamics of the virus in the human population with the prediction of the size of epidemic and spreading time. The objective of their study was to estimate the effectiveness of various preventive tools adopted for COVID-19. Their work made use of the Genocchi collocation technique to investigate the proposed fractional mathematical model numerically via Caputo-Fabrizio fractional derivative. The Genocchi operational matrix was derived to

investigate various constant parameters' effect in the concerned fractional mathematical model. A compartmental model that included susceptible parameter, exposed parameter, infectious parameter, recovered parameter, and mortality parameter with a quarantined parameter was developed to analyse the dynamics of the covid-19 virus. The results obtained from their numerical concerned model were compared with real data of Covid-19 from cases in India. It was noticed that the memory system of Caputo-
Fabrizio derivative described the technical dynamics of transmission of COVID19 more accurately than the classical derivative.

Khan et al. (2021) investigated and did analysis on SARS-CoV-2 virus using fractional-order model that discuss the temporal dynamics of the SARS-CoV2 virus in a community. They used the SACR model to study the dynamics of the virus where the total population was divided into compartments susceptible(S), asymptomatic(A), symptomatic(C), and recovered(R) groups. The Caputo-Fabrizio fractional order was used to model the transmission dynamics of SARS-CoV-2 virus. They used the fixed point theory to show that the proposed model possesses a unique solution where the boundedness and positivity of the fractional-order model solutions were discussed. Analyses on the steady states of the model was performed and also sensitivity analysis of the basic reproductive number was explored.

Ullah et al. (2018) used the Caputo-Fabrizio derivative to model the dynamics
of hepatitis E. An SEIRP model was created by dividing the entire host population in order to examine the dynamics of the virus. To determine the existence and uniqueness of the outcomes connected to the model, they applied the fixed point theory. A. Atangana et al.'sAdams-Bashforthnumerical scheme was used to obtain the numerical solution and produce the graphical results.

A study on two fractional models in the Atangana-Baleanu and Caputo-Fabrizio senses was conducted by Kumar et al. (2023). They created two fractional-order models—the malaria model with Caputo-Fabrizio derivative and the malaria model with Atana-baleanu derivative—by extending an existing malaria model that takes into account the effects of malaria infection on mosquito biting behavior and human attractiveness. They created a model to simulate how malaria infection affects both human attractiveness and mosquito biting behavior. They demonstrated the global asymptotic stability of the distinct endemic equilibrium of the fractional and ordinary differential models using the Lyapunov theory. They demonstrated the global asymptotic stability of the distinct endemic equilibrium of the fractional and ordinary differential models using the Lyapunov theory. They established the existence of solutions, gave conditions for their uniqueness, and demonstrated the stability and convergence of numerical schemes using the fixed point theory. We employed the fractional-order γ method and the Adams-Bashforth method, respectively, to verify the efficacy of the employed approximation techniques.

A study using the Caputo-Fabrizio fractional derivative to model the human liver was conducted by Baleanu et al. (2020). They presented a novel fractional model of the human liver using the exponential kernel and the Caputo-Fabrizio derivative in their work. The existence of a unique solution was investigated using the fixedpoint theory and the Picard-Lindelof approach. For the numerical analyses on the fractional model, they employed the homotopy analysis transform method (HATM). Combining the homotopy analysis method with the conventional Laplace transform technique is known as the homotopy analysis transform method. Following a comparative study using actual clinical data, the new fractional model proved to be more effective than the previous integerorder model using regular time derivatives.

2.2.6 Stability Analysis of Mathematical Models

The analysis of stability in a mathematical model pertains to the investigation of the temporal behaviour and characteristics of the model's solutions. The process entails evaluating whether the solutions of the model converge towards a stable equilibrium or demonstrate oscillatory or chaotic behaviour Masoumnezhad et al. (2020). The analysis of stability offers valuable insights into the enduring behaviour of the system, facilitating comprehension of its dynamics and enabling predictions of the model's outcomes Almocera et al. (2021).

Understanding the dynamics of disease transmission and formulating successful control strategies in the field of infectious diseases depend heavily on stability analysis. Researchers use mathematical models to illustrate the dynamics of infectious disease transmission and assess the stability of different model equilibria, such as the endemic equilibrium and the disease-free equilibrium Masoumnezhad et al. (2020). Stability analysis helps determine whether the disease will become extinct or persist in the population over the long run. Stability analysis encompasses a range of methodologies, such as Lyapunov functions, Volterra-Lyapunov matrices, and bifurcation analysis, as discussed by Almocera et al. (2021) and Masoumnezhad et al. (2020). Lyapunov functions are mathematical functions utilized to assess the stability of a system by quantifying the rate of change of a specific property of said system Masoumnezhad et al. (2020). The utilization of Volterra-Lyapunov matrices is employed in the examination of equilibrium stability within mathematical models, as discussed by Masoumnezhad et al. (2020). The analysis of bifurcation investigates the alterations in the system's dynamics when parameters undergo variations, thereby identifying pivotal points where significant qualitative transformations take place Almocera et al. (2021).

The application of stability analysis to infectious disease models has been observed in a range of diseases, encompassing COVID-19 and tuberculosis. An instance where stability analysis has been employed is in the examination of the within-host dynamics of COVID-19, as well as in comprehending the interplay between the virus and the immune response Almocera et al. (2021). Through the process of analyzing the stability of the model, researchers are able to acquire valuable insights pertaining to the mechanisms by which the immune system effectively eliminates the pathogen, as well as the strategies employed by the virus to replicate and counteract immune responses Almocera et al. (2021). The analysis of stability in mathematical models of infectious diseases is a highly valuable tool that facilitates comprehension of the long-term dynamics of the system and enables the prediction of disease outcomes. This phenomenon aids in the determination of the disease's potential for long-term existence or eradication, while also offering valuable insights into the disease's dynamics and the efficacy of control measures. Through the application of analytical methods to assess equilibrium stability and the utilization of diverse mathematical approaches, scholars are able to acquire a more profound comprehension of the dissemination and management of contagious illnesses.

2.2.7 Mathematical Modelling of Conjunctivitis

Ogunmiloro (2020) presents a mathematical model that explains the dynamics of conjunctivitis transmission within the human host population. The equilibrium with and without conjunctivitis are the two equilibrium points that the model takes into account; each equilibrium point was dependent on the basic reproduction number R_0. The analysis's primary concern is the equilibrium points' stability. When R_0 is ¡ 1, it was discovered that the equilibrium point, which stands for the absence of conjunctivitis, is asymptotically stable both locally and globally.

Verma et al. (2019) used comparative analysis of time series models to study the estimation of conjunctivitis disease. They adopted the use of time series forecasting techniques in response to future events, then separated the model into compartments including state-of-the-art time series models, such as Exponential Smoothing, Random Forest, Neural Network, and Auto Arima, and then compared the results. After analysis, they calculated error metrics using Mean Square Error, Mean Absolute Error, Root Mean Square Error, and Auto Correlation Function. Their analysis's findings demonstrated that Random Forest outperforms Neural Network, Auto Arima, and Exponential Smoothing when error metrics drop.

Nana-Kyere et al. (n.d.) used the stochastic optimal control model to study the dynamics of hemorrhagic conjunctivitis disease. The dynamic tool system was used to examine the potency of epidemic spread. They assessed the system's stability and found that the epidemic would spread unchecked if the fundamental reproduction number was higher than one. They used Pontryagin's Maximum Principle to reformulate the optimal control issue and evaluate the efficacy of the control solutions. They used a numerical solution to evaluate the best control techniques, and the results indicated a decrease in the number of exposed and sick people. Using deterministic methods with stochastic perturbations, the model version of stochastic was inferred. Using numerical simulations, they demonstrated how the dynamics of the models and the pandemic phenomenon differed.

In their study, Uchenna et al. (2019) put forth a theoretical framework to examine the transmission dynamics of acute conjunctivitis within public schools during the harmattan season. The researchers conducted both analytical and numerical analyses to investigate this phenomenon. The model is redefined as an optimal control problem, incorporating the impact of adequate sanitation and educator training. The Maximum Principle is utilized to derive the essential conditions for the existence of optimal control. The fundamental reproductive number was derived through the utilization of the next generation matrix and spectral radius, resulting in a value that is less than unity upon computation. The findings demonstrate a concurrence between the analytical and numerical solutions. Viriyapong & Khedwan (2019) developed a mathematical model to examine the effects of treatment control and isolation of conjunctivitis epidemic-infected patients on conjunctivitis stability. By computing the fundamental reproduction number, they were able to determine the theoretical analysis of the model and utilise it to show that the equilibrium point model is stable. Once more, they used Pontryagin's Maximum Principal to include the treatment control variable and carry out the optimal control. The findings imply that conjunctivitis was lessened when the treatment control strategy and the isolation of afflicted people were combined.

In order to assess the effects of an education campaign on the spread of conjunctivitis disease, Nana-Kyere et al. (2024)) created a deterministic mathematical model to study the disease's dynamics. The endemic equilibrium and the disease-free equilibrium of the mathematical model for hemorrhagic conjunctivitis disease were found to be distinct and stable after a local stability analysis was carried out. In order to examine the rate at which the epidemic is spreading, the current study used a dynamical system, concentrating on the best control model for hemorrhagic conjunctivitis. According to the analysis done, there is a chance that the epidemic will spread to every member of the population if the basic reproduction number is exceeded.

In a research published in 2006, Chowell et al. (2006) created a mathematical model to represent an acute hemorrhagic conjunctivitis outbreak. Individuals who were susceptible to the disease, those who were infected and able to spread the disease, those who had been diagnosed and reported as cases, and those who had recovered from the infection were all represented by separate compartments in the model. The study looked at how underreporting and behavioral modifications affected the rate at which acute hemorrhagic conjunctivitis spread throughout Mexico.

2.3 Emperical Studies

Empirical studies encompass the systematic gathering and examination of data acquired through direct observation or experimentation. They offer insights that are grounded in evidence and make valuable contributions to the advancement of knowledge across multiple disciplines. Empirical research is of utmost importance in comprehending real-world phenomena, validating theories, and guiding decision-making procedures.

Empirical studies pertain to research that is grounded in direct observation or experiential evidence, as opposed to being solely reliant on theoretical frameworks or speculative conjecture. Empirical studies encompass the systematic collection and analysis of data in order to examine hypotheses, make observations, and derive conclusions pertaining to phenomena that exist in the real world Alvesson & Karreman (2011).¨

According to Alvesson & Karreman (2011), empirical studies are characterized¨ by the utilization of empirical methods, which facilitate the direct collection of data from the real world. This approach enables researchers to draw informed and evidence-based conclusions. Empirical studies are of paramount importance in the generation of knowledge, the validation of theories, and the facilitation of informed decision-making across diverse academic disciplines.

2.3.1 Previous Studies on Infectious Diseases

The study conducted by Huang et al. (2020) examined infectious diseases from multiple perspectives, such as the influence of infectious diseases on market behaviour and the correlation between financial ratios and abnormal returns within the biotechnology sector during disease outbreaks. Zhou (2012) emphasized the significance of intersectoral collaboration and the adoption of a ”One Health - One World” approach in the realm of infectious disease research. Similarly, Tao et al. (2022) explored the role of procalcitonin in the diagnosis and monitoring of infectious diseases.

In Zhou (2012) study titled ”Prioritizing research for One Health- one world,” the author examines the factors contributing to the prevalence of infectious diseases among impoverished populations. The report emphasizes the potential of scientific and technological advancements in addressing the control and management of these diseases. The authors propose the adoption of a ”One Health - One World” framework as a means to establish a fresh strategic trajectory for infectious disease research. This approach underscores the importance of intersectoral collaboration. To develop suitable public health interventions for infectious diseases that disproportionately impact impoverished populations, it is imperative to consider various research perspectives. These perspectives encompass health systems, environmental and social factors, agriculture, basic research and innovation, as well as patterns and gaps in research funding. The current landscape of clinical research in the field of infectious diseases primarily centers around three key domains: the advancement of targeted diagnostic tests, particularly leveraging contemporary molecular diagnostic methodologies; the detection and characterization of co-infections, specifically bacterial infections arising as secondary complications to viral infections; and the surveillance of disease progression throughout treatment regimens and the utilization of antibiotics. Procalcitonin is involved in the diagnosis and monitoring of infectious diseases.

2.3.2 Previous Studies on Conjunctivitis

The development of the S-E-I-R (Susceptible-Exposed-Infected-Recovered) mathematical model, which explains the dynamics of conjunctivitis transmission in the human population, is the main topic of Ogunmiloro (2020) Two equilibrium points, the equilibrium without conjunctivitis and the equilibrium with conjunctivitis, are shown in the analyzed model. These equilibrium points depend on the value of the fundamental reproduction number R_0. The study looked at the built model system's stability characteristics at the equilibrium solutions from both a local and global standpoint. The findings show that the equilibrium solution corresponding to the absence of conjunctivitis is both locally and globally asymptotically stable when the value of $R_0 is < 1$. Additionally, it can be said that the equilibrium state with conjunctivitis present is locally asymptotically stable when $R_0 > 1$. To determine the relative importance of the conjunctivitis model's parameter values in relation to the condition's prevalence, a sensitivity analysis was performed on the model's parameter values. The researcher created an optimality system and used the forward-backward Runge-Kutta algorithm in the computer program MATLAB to solve it numerically. It was possible to solve the system with and without controls. The results showed that every control strategy has a unique role to play in the elimination of conjunctivitis. In their research paper from 2024, Nana-Kyere et al. (2024) provide a model that clarifies the dynamics of hemorrhagic conjunctivitis disease transmission. The potential for epidemic spread is analyzed using the dynamical system framework. The study found that when the basic reproduction number is greater than one, there is a greater chance of the epidemic spreading.

Pediatric patients with bacterial conjunctivitis were the subject of research by Patel et al. (2007)in their study titled "Clinical Features of Bacterial Conjunctivitis in Children." In 2007, the research was released in the academic emergency medicine journal. The goal of this study, which was carried out in a pediatric hospital emergency room, was to give a thorough description of the clinical traits that have the best predictive value for children's bacterial conjunctivitis Patel et al. (2007).

2.4 Conclusion/Summary

Ogunmiloro (2020) constructed the S-E-I-R mathematical model to examine the dynamics of conjunctivitis spread in the human population based on previously reviewed research. The researcher created an optimality system and used the forward-backward Runge-Kutta algorithm in the computer program MATLAB to solve it numerically.

The S-E-I-R model was introduced in Nana-Kyere et al. (2024)researchpaper from 2024. Ordinary differential equations were used to write and solve the mathematical differential equations governing the conjunctivitis model's dynamics.

This study aims to develop a seven compartmental model taken into consideration symptomatic and asymptomatic compartment to analyze the dynamics of conjunctivitis. A fractional differential derivative would be used to assess the efficacy of educational interventions in enhancing knowledge and preventing the spread of the disease.

Chapter 3
Methodology

3.1 Introduction

Important ideas and theorems needed for this research project are defined in this chapter. The Caputo-Fabrizio fractional derivative is a non-local, non-singular fractional derivative, which means that it doesn't involve the singularity issues that are found in the classic Riemann-Liouville and Caputo fractional derivatives. This type of fractional derivative is defined using the exponential function, which gives it a non-singular nature. This chapter would focus on the Caputo-Fabrizio fractional derivative, positivity and variant regions, equilibrium and stability analysis of endemic and disease-free environments, the RouthHurwitz criterion, Lyapunov stability and numerical simulations are among the ideas and theorems to be covered.

3.2 Fractional Derivative

A subfield of mathematics known as fractional calculus studies the extension of differentiation and integration to non-integer orders. Numerous fields, such as mathematics, engineering, and physics, have found use for it Ding et al. (2023), Understanding and using fractional calculus require a grasp of the definition of a fractional derivative. When modelling and analysing real-world issues involving complicated dynamics, long-term memory, or non-standard behaviour, fractional derivatives offer a potent tool. Even though fractional calculus is a specialized area of study, it has shown to be helpful in comprehending and resolving issues that conventional calculus is unable to sufficiently handle.

3.3 The Caputo-Fabrizio Fractional Differential Equation

The principle of differentiation can be expanded from integers to fractions, resulting in the complex and fascinating discipline of fractional calculus. Although the Riemann-Liouville and Caputo definitions of fractional derivatives are known and acknowledged, they have singularities that might cause problems. In order to tackle these problems, the Caputo-Fabrizio fractional differential equation was proposed, distinguished by its non-singular properties. The historical development of fractional calculus can be traced back to several centuries ago, with notable contributions from mathematicians such as Leibniz, Liouville, and Riemann. Fractional calculus finds extensive applications in several domains including physics, engineering, economics, and biology.

Fractional differential equations (FDEs) are differential equations that involve derivatives of non-integer (or fractional) order. Classical fractional differential equations (FDEs) feature kernels that exhibit power-law decay, resulting in singularities. The Caputo-Fabrizio fractional derivative is defined without the presence of a singular kernel, which distinguishes it significantly from its classical predecessors. More precisely, it is specified utilizing an exponential function to guarantee a behaviour that is not singular.

3.3.1 Lemma

Let $u(t)$ be a function such that its second order derivative $u''(t)$ exists and is integrable on [0,t] for a given t. The Caputo-Fabrizio fractional derivative of order α (where 0 ¡ α ¡ 1) is related to the classical second derivative $u''(t)$ by:

$$D^{\alpha}u(t) = \frac{1 - e^{-\alpha t}}{\Gamma(2-\alpha)} \int_0^t e^{-s} u''(s)ds, \tag{3.1}$$

where, α is the order of the fractional derivative (with 0¡α¡1) and Γ represents the gamma function.

3.4 Mathematical Preliminaries on the Caputo-Fabrizio Fractional Derivative

The definitions that follow will be used to support the existence, uniqueness, and positivity of the Caputo-Fabrizio (CF) model that is the subject of this work's analysis.

3.4.1 Definition 1

The fractional derivative in Caputo-Fabrizio sense for the function $\chi \in H^1(a, b)$, b ¿ a, $\tau \in [0, 1]$ defined as

$$D_t^{\tau}(\chi(t)) = \frac{M(\tau)}{1-\tau}\int_a^t \chi'(x)exp\left[-\tau\frac{t-x}{1-t}\right]dx \quad (3.2)$$

$M(t)$ is the normalized function satisfying $M(0) = M(1) = 1$. For the case when $\chi \notin H^1(a, b)$ the above Caputo-Fabrizio derivative can be express as

$$D_t^{\tau}(\chi(t)) = \frac{M(\tau)}{1-\tau}\int_a^t (\chi(t)-\chi(x))exp\left[-\tau\frac{t-x}{1-t}\right]dx \quad (3.3)$$

3.4.2 Remark 1

If $\alpha=\frac{1-\tau}{\tau}\in[0,\infty)$, $\tau=\frac{1}{1+\alpha}\in[0,1]$, then equation (3.2) can be written as

$$D_t^{\alpha}(\chi(t)) = \frac{N(\alpha)}{\alpha}\int_a^t \chi'(x)exp\left[-\frac{t-x}{\alpha}\right]dx, \; N(0) = N(\infty) = 1 \quad (3.4)$$

Moreover,

$$\lim_{\alpha\to 0}\frac{1}{\alpha}exp\left[-\frac{t-x}{\alpha}\right] = \delta(x-t) \quad (3.5)$$

The integral regarding to Caputo-Fabrizio derivative is defined as

3.4.3 Definition 2

Let $0 < \tau < 1$, and consider the fractional derivative given below

$$D_t^{\tau}(\chi(t)) = g(t) \quad (3.6)$$

Then the corresponding integral of fractional order τ is expressed as

$$I_t^{\tau}(\chi(t)) = \frac{2(1-\tau)}{(2-\tau)M(t)}g(t) + \frac{2\tau}{2-\tau}M(t)\int_0^1 g(s)ds, t \geq 0 \quad (3.7)$$

3.4.4 Remark 2

Using the result

$$\frac{2}{2M - \tau M(t)} = 1 \tag{3.8}$$

Which gives $M(\tau) = \frac{2}{2-\tau}, 0 < \tau < 1$, the authors give the new Caputo-Fabrizio fractional derivative of order 0¡τ¡1 which is defined as

$$D_t^{\alpha}(\chi(t)) = \frac{1}{1-\tau}\int_0^t \left[-\tau\frac{t-x}{1-t}\right] dx \tag{3.9}$$

3.5 Existence and Uniqueness of the system of Caputo-Fabrizio differential equation

The existence and uniqueness of solutions for differential equations are critical subjects in mathematical analysis. When dealing with fractional differential equations, especially those defined using the Caputo-Fabrizio derivative, these subjects become even more intricate due to the non-local and non-singular nature of the derivative.

Let's consider a system of Caputo-Fabrizio fractional differential equations:

$$D^{\alpha}U(t) = F(t, u(t)) \tag{3.10}$$

Where:

D^{α} represents the Caputo-Fabrizio fractional derivative of order α(0¡α¡1)
$U(t)$ is a vector of unknown functions.

$F(t, u(t))$ is a vector-valued function representing the system.

The existence and uniqueness of the solution to this system can be established under certain conditions on F.

1. $F(t, u)$ is continuous in both its arguments and satisfies a Lipschitz condition in u, that is, there exists a Lipschitz constant L such that:

$$\| F(t, u_1) - F(t, u_2) \| \leq L \| (u_1 - u_2) \tag{3.11}$$

For all u_1 and u_2 and for all t

2. There exist a constant M such that

   $\| F(t,u) \| \leq M$

   For all u and for all t

   Then, there exists a unique solution u(t) to the system of Caputo-Fabrizio fractional differential equations on a closed interval [0,T].

3.6 Positivity and variant region of Caputo-Fabrizio differential equation

The Caputo-Fabrizio fractional differential equation possesses distinct problems and properties in comparison to classical differential equations and other fractional differential equations, owing to its non-singular nature. An attribute frequently analyzed in the field of differential equations is the ”positivity” of solutions. Essentially, this term describes the characteristic of a system where, if the starting circumstances are greater than or equal to zero, the solutions of the differential equation will also be greater than or equal to zero for all subsequent times.

3.6.1 Positivity of the Caputo-Fabrizio differential equation

3.6.1.1 Theorem (positivity)

Consider the Caputo-Fabrizio fractional differential equation of the form:

$D^{\alpha}u(t) = F(t, u(t))$ with $u(0) \geq 0$, and suppose that $F(t, u)$ satisfies:

1. $f(t, 0) = 0$ for all t

2. $f(t, u)$ is non-decreasing in u for all t Then, the solution $u(t)$ of the differential equation remains non-negative for all $t \geq 0$.

3.6.1.2 Proof

1. Assume that $u(t_1) < 0$ for some $t_1 > 0$ and $u(t) \geq 0$ for all t in the interval $[0, t_1)$

2. The non-decreasing property of $f(t, u)$ would imply that the Caputo-Fabrizio deravitive, $D^{\alpha}u(t) = F(t, u(t))$ becomes negative at $t = t_1$

3. This would contradict the definition of the Caputo-Fabrizio derivative given the assumed properties of f and the initial condition.

3.7 Invariant Region for the Caputo-Fabrizio Fractional Differential Equation

3.7.1 Invariant Region

For the Caputo-Fabrizio fractional differential equation: $D^{\alpha}u(t) = F(t, u(t))$

let's define a region R such that if $u(0)$ lies in R, then $u(t)$ remains in R for all $t \geq 0$ if the following conditions are satisfied:

1. $f(t, u)$ is continuous and bounded within the region R
2. The boundary of R, denoted as ϑR, ensures that trajectories are either tangential to it or directed into R.

3.8 Positivity and boundedness of Caputo-Fabrizio Differential Equation

Positivity and boundedness are fundamental notions that have significant implications in the analysis of differential equations. A positive solution refers to a solution that remains non-negative throughout its domain, assuming nonnegative initial conditions. Conversely, boundedness guarantees that the answer does not exhibit unbounded growth, meaning it stays inside a finite range.

Under specific conditions, one can obtain these features for the Caputo-Fabrizio fractional differential equation.

3.8.1 Boundedness of the Caputo-Fabrizio Fractional Differential Equation

3.8.2 Boundedness

Consider the Caputo-Fabrizio fractional differential equation of the form:

$D^{\alpha}u(t) = F(t, u(t))$, suppose $f(t, u)$ has the following properties:

1. $f(t, u)$ is continuous in u for all t.

2. There exist a constant M such that for all u and t, $|f(t, u)| \leq M(1 + |u|)$ Then, for any bounded initial condition, the solution $u(t)$ remains bounded for all $t \geq 0$.

3.8.2.1 The Lyapunov-like Function Method for Caputo-Fabrizio Fractional Differential Equation

Given a Caputo-Fabrizio fractional differential equation of the form

$D^{\alpha}u(t) = F(t, u(t))$, we are interested in the stability of the equilibrium solution u^* (i.e., $f(t, u^* = 0)$.

3.8.2.2 Definition:

A scalar function $V(t, u(t))$ is called a Lyapunov-like function if: 1. It is continuously differentiable

2. $V(t, u^*) = 0$ and $V(t, u(t)) > 0$ for all $u(t) \neq u^*$.

3. The fractional derivative $D^{\alpha}V(t, u(t))$, is negative semi-definite.

If such a function exists, then the equilibrium solution u^* is said to be stable in the Lyapunov sense.

3.9 The Basic Reproductive Number of Conjunctivitis

In the field of epidemiology, the fundamental reproductive number, or R_0, is a vital quantity. The basic reproduction number, a unitless value, is the average number of secondary cases of conjunctivitis that develop over the course of an infected person's lifetime of illnesses when they are introduced into a community of susceptible hosts. An infectious disease has the ability to spread throughout the population when the value of $R_0 > 1$. On the other hand, the disease is anticipated to worsen and ultimately vanish if $R_0 < 1$. The next generation matrix operator approach is used to compute the R_0.

3.10 Equilibrium and Stability Analysis of Fractional Differential Equations

3.10.1 Equilibrium Points

Equilibrium points (or steady-states) are solutions to the Fractional Differential Equation that don't change over time. To find them for a general CaputoFabrizio Fractional Differential Equation of the form:

$$ {}^{CF}_{t}D^{\alpha}x(t) = f(x(t)) \tag{3.12} $$

Set ${}^{CF}_{t}D^{\alpha}x(t) = 0$ and solve for x. The solutions are the equilibrium points, x^*.

3.10.2 Stability Analysis

Stability can be assessed using various methods

3.10.2.1 Linearization

If $f(x)$ is continuously differentiable, you can perform a Taylor expansion about the equilibrium point x^*. Retaining only the linear terms and discarding higherorder terms provides a linear approximation:

$$ {}^{CF}_{t}D^{\alpha}\delta(t) = f'(x^*)\delta(t) \tag{3.13} $$

Where $\delta(t) = x(t) - x^*$ represents a small perturbation from the equilibrium. The behavior of this linear system can guide the stability of the original nonlinear system.

3.10.2.2 Lyapunov Stability

By Computing the Caputo-Fabrizio fractional derivative of the Lyapunov function:

$$ {}^{CF}D^{\alpha}V(t, x(t)) \quad (3.14)\ {}_{t} $$

The direction and magnitude of this derivative provide insights into the behavior of trajectories in the vicinity of the equilibrium.

If ${}^{CF}{}_{t}\quad D^{\alpha}V(t, x(t)) \leq 0$, for all $x(t)$ in the neighborhood of x^* (except at x^*), and ${}^{CF}{}_{t}D^{\alpha}V(t, x(t)) = 0$ if and only if $x(t) = x^*$, then the equilibrium x^* is Lyapunov stable.

If ${}^{CF}{}_{t}D^{\alpha}V(t, x(t)) \leq 0$, for all $x(t)$ in the neighborhood of x^* (except at x^*), then the equilibrium x^* is Lyapunov asymptotically stable.

If there exists a region around x^* where ${}^{CF}{}_{t}D^{\alpha}V(t, x(t)) > 0$, then the equilibrium is unstable.

3.11 Routh- Hurwitz Criterion

The Routh-Hurwitz criterion helps one to estimate, without actually calculating, the sign of the roots of the polynomial. A matrix's eigenvalues can be constructed from the roots of the matrix's characteristic polynomial.

Consider the polynomial of degree n with real coefficients

$P(\lambda) = \lambda^n + m_1\lambda^{n-1} + \cdots + m_{n-1}\lambda + m_n$

3.11.0.1 Preposition

If the real part of all the roots of $P(\lambda)$ is strictly negative, then all the coefficients of m must be strictly greater than zero.

3.11.0.2 Proof

Let $\lambda_1, \lambda_2, \dots, \lambda_k$ be the (negative) real roots of $P(\lambda)$ and let $\alpha_1 \pm i\beta_1, \alpha_2 \pm i\beta_2, \dots, \alpha_h \pm i\beta_h$ be the complex conjugate roots, with $\alpha_2 < 0, \alpha_1 < 0, \dots, \alpha_h < 0$. Then we have $P(\lambda) = (\lambda - \lambda_1)^{\mu_1}, \dots (\lambda - \lambda_k)^{\mu_k}$

$P(\lambda) = (\lambda - (\alpha_1 + i\beta_1))^{\nu_1}.(\lambda - (\alpha_1 - i\beta_1))^{\nu_1}. \dots .(\lambda - (\alpha_h + i\beta_h))^{\nu_h}.(\lambda - (\alpha_h - i\beta_h))^{\nu_h}$

Each pair of linear variables in which the complex roots appear can be replaced by a unique factor of degree 2. $\lambda_2 + p_1\lambda + q_1, \dots, \lambda^2 + p_h\lambda + q_h$

Where $\alpha_1 < 0, \alpha_2 < 0, \dots, \alpha_h < 0$ and all the coefficients of $p_1, \dots, p_h, q_1, \dots, q_h$ are positive. we find that all the coefficients are positive by obtaining the expanded form of the polynomial.

3.11.0.3 Theorem

Consider the characteristic polynomial

$$P(\lambda) = \lambda^n + m_1\lambda^{n-1} + \cdots + m_{n-1}\lambda + m_n \tag{3.15}$$

Where, the coefficients m_i are real constants, such that $i = 1, 2, \dots, n$ and λ is the identity matrix. The eigenvalues from Hurwitz matrices considering the coefficients m_i of $m_3\ m_2\ m_1$ and

, n . . . . m_n

0 0 0 · m_1 1 0

m_5 m_4 m_3 m_3 m_2

1 0 · ·

m_1 m_2 m_1 · · 0

m_3 3 · · 0

0

$$(3.18) H_1 = (m_1), H_2 = \begin{pmatrix} m_1 & 1 \\ \end{pmatrix}, H_3 =$$

m_5 m_4 m

$H =$

Where $m_j = 0$ if $j > n$. If all of the roots of the polynomial $P(\lambda)$ are negative or have negative real part if and only if the determinant of all Hurwitz matrices are positive, det $H_j) > 0, j = 1, 2, \cdots, n$

When $n = 2$, the Routh-Hurwitz Criteria simplify

$$\det H_2 = \begin{vmatrix} m_1 & 1 \\ 0 & m_2 \end{vmatrix} = m_1 m_2 > 0 \text{ or } m_1 > 0 \text{ and } m_2 > 0.$$

For a polynomial of degree n = 2, 3 and 4, the Routh-Hurwitz criteria are summarized as follows:

$n = 2 : m_1 > 0$ and $m_2 > 0$

$n = 3 : m_1 > 0, m_2 > 0, m_3 > 0$ and $m_1 m_2 > m_3$

$n = 4 : m_1 > 0, m_2 > 0, m_3 > 0, m_4 > 0$ and $m_1 m_2 m_3 > m_3^2 + m_1^2 m_4$

3.12 Numerical Solutions

Numerical simulation in mathematical modelling is the process of discretising the issue domain and solving the resulting algebraic equation using computing techniques to approximate the solution of a model, usually a distinct equation or system of equations. MATLAB codes for Lagrange interpolation iteration scheme is used to find the effect of memory degradation on the model.

Chapter 4 Discussions and Analysis on the model

4.1 Conceptual Model for the Study

The study is guided by the conceptual model indicated in Figure 4.1. The model was derived from the SEIR baseline model. Additional compartments such as symptomatic, asymptomatic and treated individuals were incorporated.

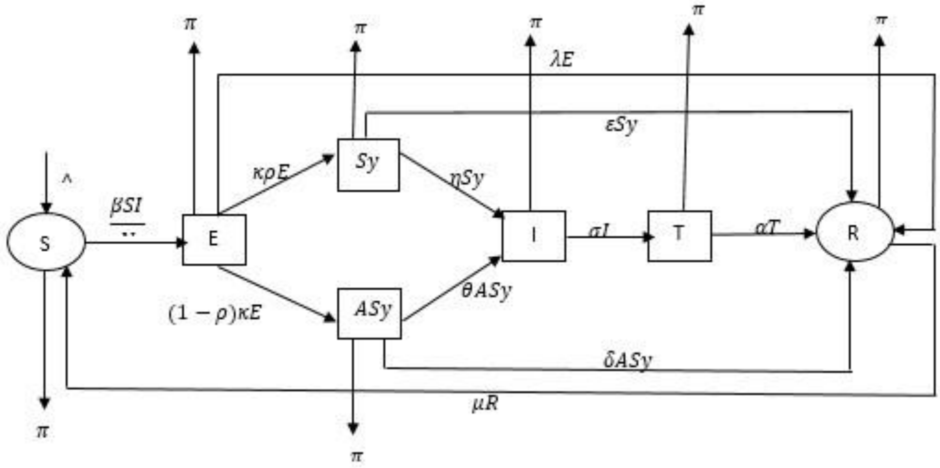

Figure 4.1: Conceptual Model of the Study

Susceptible (S): In the context of conjunctivitis, the susceptible individuals are those who are currently healthy but at risk of contracting the disease. This group includes individuals who have not been previously exposed to the specific pathogen causing conjunctivitis.

Exposed (E): The exposed compartment represents individuals who have been

Variables	Description
S	Susceptible Population
E	Exposed Population
Sy	Symptomatic Population
Asy	Asymptomatic Population
I	Infected Population

T	Treated Population
R	Recovered Population

Table 4.1: **Description of state variables of the model**

exposed to the conjunctivitis-causing pathogen but are not yet infectious. In the case of conjunctivitis, exposure could occur through contact with contaminated surfaces, respiratory droplets, or direct contact with infected individuals. Not all exposures will lead to infection, as the individual's immune response may prevent the development of the disease.

Asymptomatic: Asymptomatic individuals have been exposed to the pathogen and are capable of spreading the disease, even though they do not show any symptoms. This compartment accounts for the transmission of the disease by individuals who are infected but asymptomatic.

Infectious (I): The infectious compartment includes individuals who have contracted conjunctivitis and are capable of spreading the disease to susceptible individuals. In the context of conjunctivitis, this stage represents individuals who are actively experiencing symptoms and have a contagious form of the disease.

Treated: Treated population are individuals who have sought medical attention and received treatment for conjunctivitis are placed in this compartment. Treatment may include antibiotics or antiviral medications, depending on the cause of the conjunctivitis. Treated individuals are no longer infectious.

Recovered/Removed (R): In this compartment, we consider individuals who have recovered from conjunctivitis and are no longer infectious. Conjunctivitis is often a self-limiting condition, and individuals in this compartment have either naturally cleared the infection or have received appropriate treatment.

The addition of Asymptomatic to the model is to cater for the spread of disease through Asymptomatic carriers. Conjunctivitis can manifest as either symptomatic or asymptomatic. Asymptomatic carriers of the disease can play a significant role in its transmission because they may not be aware of their infection but can still spread it to others through direct or indirect contact with their eyes Huang et al. (2020); Al-Arosi et al. (2021). Asymptomatic individuals can therefore contribute to the spread of the disease, and including them in the model allows for a more comprehensive assessment of the potential impact of interventions.

Also, the inclusion of Treatment (T) is to cater for individuals who seek medical care and receive treatment for their conjunctivitis. Treatment can significantly affect the duration of infectiousness and may reduce the likelihood of transmission to others. This is important because it reflects a real-world scenario where individuals with conjunctivitis may receive medical care. Including a Treatment compartment allows for the evaluation of the impact of treatment on disease progression and transmission dynamics. This is particularly relevant for conjunctivitis, where timely treatment can reduce the duration of symptoms and infectiousness.

The flow of individuals between these compartments is governed by a system of differential equations, which describe how the number of individuals in each compartment changes over time. The equations are based on parameters.

Parameter	**Description**
Λ	Recruitment of susceptible individuals
$\frac{\beta SI}{N}$	Transmission rate of the disease from infected to susceptible individuals
β	The rate of infection of susceptible individuals
ρ	Fraction of expose individuals showing symptoms
$(1-\rho)$	Fraction of expose individuals not showing symptoms
λ	Rate at which the exposed recover
ξ	Recovery rate for symptomatic individual
δ	Recovery rate for the asymptomatic individual
η	Rate at which the symptomatic progress to the infected
ϑ	Rate at which the asymptomatic progress to the infected
σ	Treatment rate
α	Recovery rate of treated individuals
μ	Rate of individuals returning to the susceptible state

κ	Rate at which individuals move from the expose compartment

Table 4.2: **Description of parameters of the model**

The basic equations governing the model are therefore stated as:

$$\begin{cases} \frac{dS}{dt} = \Lambda + \mu R - \frac{\beta SI}{N} - \pi S \\ \frac{dE}{dt} = \frac{\beta SI}{N} - (\lambda + \kappa + \pi)E \\ \frac{dSy}{dt} = \kappa \rho E - (\eta + \xi + \pi)Sy \\ \frac{dAsy}{dt} = (1 - \rho)\kappa E - (\vartheta + \pi + \delta)Asy \\ \frac{dI}{dt} = \eta Sy + \theta Asy - (\sigma + \pi)I \\ \frac{dT}{dt} = \sigma I - (\alpha + \pi)T \\ \frac{dR}{dt} = \alpha T + \eta Sy + \delta Asy + \lambda E - (\mu + \pi)R \end{cases} \tag{4.1}$$

Where $N = S + E + Sy + Asy + I + T + R$, with initial conditions $(S(0), E(0), Sy(0), ASy(0), I(0), T(0), R(0)) \in R^7_+$.

4.2 Basic Properties of the model

We demonstrate that the parameters in the model and the state variable must be positive and limited for all $t \geq 0$.

Let a total population $N(t) = S(t) + E(t) + Sy(t) + Asy(t) + I(t) + T(t) + R(t)$ and taking the time derivative of $N(t)$ along solutions of the model equation (4.1),

$$\frac{dN}{dt} = \Lambda - \pi N \tag{4.2}$$

Hence, equation (4.2) shows there are changes in the population known as the population dynamics.

4.3 Feasibility of the model

The feasibility of the model describes the region in which the solution of the system of equation (4.1) is biological meaningful.

4.3.0.1 Theorem

Assuming the validity of equation (4.1), all model solutions in the system of equation (4.1) with initial conditions in R^7_+ approach and remain in the compact set Ω as t → ∞. Next, the model's positive invariant set, or feasible solution, is provided by

$$\Omega = \left\{(S, E, Sy, Asy, I, T, R) \in R^7_+ : N(t) \le \frac{\Lambda}{\pi}\right\}$$

4.3.0.2 Proof

From the equation (4.2) where changes of N leads to change of all variables in the population, we have $\frac{dN}{dt} = \Lambda - \pi N(t)$

The equation then becomes

$$\frac{dN}{dt} = \Lambda - \pi N(t)$$

$$\frac{dN}{dt} \le 0 \text{ if } N(t) \ge 0$$

Solving the first order ordinary differential equation by integrating factor,

$$\frac{d}{dt}\left[e^{\pi t}N(t)\right] \le e^{\pi t}\Lambda$$

$$e^{\pi t}N(t) \le \frac{e^{\pi t}\Lambda}{\pi} + c$$

$$N(t) \le \frac{\Lambda}{\pi} + ce^{-\pi t}$$

At $t = 0$, $N(0) = N_0$

$$N_0 = \frac{\Lambda}{\pi} + c$$

$$c = N_0 - \frac{\Lambda}{\pi}$$

$$e^{-\pi t} \le \frac{\Lambda}{\pi} + (N_0 - \frac{\Lambda}{\pi})e^{-\pi t}$$

$$N(t) \le \frac{\Lambda}{\pi}(1 - e^{-\pi t}) + N_0 e^{*\pi t}$$

Hence, at $t \to \infty$

$$N(t) \le \frac{\Lambda}{\pi}$$

Hence, the feasible solution is given by $\Omega = \{(S, E, Sy, Asy, I, T, R) \in R^7{}_+ :$ $N(t) \leq \frac{\Lambda}{\pi}\}$

4.4 Positivity of the Solution

4.4.0.1 Theorem

The set $(S(t), E(t), Sy(t), Asy(t), I(t), T(t), R(t))$ being the solution of the state system (4.1) with parameters which are not negatives is positive with the initial condition given by; $\{S_0 \geq 0, E_{\geq}0, Sy_0 \geq 0, ASy_0 \geq 0, I_0 \geq 0, T_0 \geq 0, R_0 \geq 0\}$

4.4.0.2 Proof

From equation (4.1), we state that

$\frac{dS}{dt} \geq \frac{-\beta SI}{N} - \pi_S$

$\frac{dS}{dt} \geq -(\frac{-\beta I}{N} + \pi$

—)S

$dSS \geq -(\_\_-N\beta I + \pi)dt$

Taking integral of both sides

$\ln |S| \geq -(\frac{-\beta I}{N} + \pi)t + c$

$S \geq ce^{-(\frac{\beta I}{N}+\pi)}{}_{dt}$

$S(t) \geq ce^{-(\frac{\beta I}{N}++\pi)}{}_t$

At $t = 0$

$S(0) \geq ce^0$

$\therefore c \leq S(0)$

And since

$(\_{}^{\beta}{}_{N}{}^{I} + \pi) > 0$, $S(t) \geq 0$ Again

$\frac{dE}{dt} \geq -(\lambda + \pi + \kappa)$

— E $^{dE}{}_E \geq$

$-(\lambda + \pi + \kappa)dt \ \ln |E| \geq -(\lambda +$

$\pi + \kappa)t + c$

$E \geq ce^{-(\frac{\beta I}{N}+\gamma+\pi)}{}_{dt}$

$E(t) \geq ce^{-(\lambda+\pi+\kappa)t}$

At $t = 0$

$E(0) \geq ce^{0}c$

$\leq E(0)$

$\therefore E(t) \geq E(0)e^{-(\lambda+\kappa+\pi)E}$

And since

$(\lambda + \pi + \kappa) > 0, E(t) \geq 0$

Also

$\frac{dSy}{dt} \geq -(\xi + \eta + \pi)Sy$

$\frac{dSy}{Sy} \geq -(\xi + \eta + \pi)dt \ \ln |Sy| \geq$

$-(\xi + \eta + \pi)t + c$

$Sy(t) \geq ce^{-(\xi+\eta+\pi)t}$

At $t = 0$

$Sy(0) \geq ce^{0}c$

$\leq Sy(0)$

$\therefore \ Sy(t) \ \geq \ Sy(0)e^{-(\xi+\eta+\pi)t}$ And

since

$(\xi + \eta + \pi) > 0, Sy(t) \geq 0$

Also

$\frac{dAsy}{dt} \geq -(\vartheta + \delta + \pi)Asy$

$\frac{dAsy}{Asy} \geq -(\vartheta + \delta + \pi)dt \ \ln |Asy| \geq$

$-(\vartheta + \delta + \pi)t + c$

$Asy(t) \geq ce^{-(\vartheta+\delta+\pi)t}$

At $t = 0$

$Asy(0) \geq ce^{0}c$

$\leq Asy(0)$

$\therefore Asy(t) \geq Asy(0)e^{-(\vartheta+\delta+\pi)t}$

And since

$(\vartheta + \delta + \pi) > 0, Asy(t) \geq 0$

Also

$\frac{dI}{dt} \geq -(\sigma + \pi)$

$\frac{dI}{I} \geq -(\sigma + \pi)dt$ $\ln |I| \geq$

$-(\sigma + \pi)t + c$

$I(t) \geq ce^{-(\sigma+\pi)t}$

At $t = 0$

$I(0) \geq ce^{0} c$

$\leq I(0)$

$\therefore I(t) \geq I(0)e^{-(\sigma+\pi)t}$

And since

$(\sigma + \pi) > 0, I(t) \geq 0$

Also

$\frac{dT}{dt} \geq -(\alpha + \pi)$

$\frac{dT}{T} \geq -(\alpha + \pi)dt$ $\ln |T| \geq$

$-(\alpha + \pi)t + c$

$T(t) \geq ce^{-(\alpha+\pi)t}$

At $t = 0$

$T(0) \geq ce^{0} c$

$\leq T(0)$

$\therefore T(t) \geq I(0)e^{-(\alpha+\pi)t}$

And since

$(\alpha + \pi) > 0, T(t) \geq 0$

Also

$\frac{dR}{dt} \geq -(\mu + \pi)$

$\frac{dR}{R} \geq -(\mu + \pi)dt$

$\ln |R| \geq -(\mu + \pi)t + c$

$R(t) \geq ce^{-(\mu+\pi)t}$

At $t = 0$

$R(0) \geq ce^0 c$

$\leq R(0)$

$\therefore R(t) \geq I(0)e^{-(\mu+\pi)t}$

And since

$(\mu + \pi) > 0, R(t) \geq 0$

4.5 Analysis of the Model

The basic reproduction number, Local Stability of the Disease-Free Equilibrium, global Stability of the Disease-Free Equilibrium, existence and uniqueness analysis of the solution of the model are carried out.

4.5.1 Existence of Disease-Free Equilibrium Point

The method of finding equilibrium solutions is straightforward, whereby equating the dynamical system (4.1) to zero and solving the system for the state variables give the disease free equilibrium (E_0).

$$E_0 = \{S^0, E^0, Sy^0, ASy^0, I^0, T^0, R^0\} = \{\tfrac{\Lambda}{\pi}, 0, 0, 0, 0, 0, 0\}$$

4.5.1.1 Proof:

$Let\ \frac{dS}{dt} = 0$ and $E = Sy = Asy = I = T = R = 0$

$0 = \Lambda + \mu R - \frac{\beta SI}{N} - \pi S$

$\pi S = \Lambda\ \pi S = \Lambda$

$S = \frac{\Lambda}{\pi}$

The endemic equilibrium (E_E) is proofed by equating the equations in the model to zero and making S, E, Sy, Asy, I, T, R the subjects.

$$E_E = (S^*, E^*, Sy^*, ASY^*, I^*, T^*, R^*)$$

$$S^* = \frac{N(\Lambda+\mu R^*)}{\beta I^*+N\pi}$$

$$E^* = \frac{\beta S^* I^*}{N(\lambda+\kappa+\pi)}$$

$$Sy^* = \frac{\kappa\rho E^*}{(\xi+\eta+\pi)}$$

$$Asy^* = \frac{(1-\rho)\kappa E^*}{(\vartheta+\delta+\pi)}$$

$$I^* = \frac{\eta Sy^* + \vartheta Asy^*}{(\sigma+\pi)}$$

$$T^* = \frac{\sigma I^*}{(\alpha+\pi)}$$

$$R^* = \frac{\alpha T^* + \xi Sy^* + \delta Asy^* + \lambda E^*}{(\mu+\pi)}$$

4.5.2 Basic Reproductive Number

The state-of-the-art matrix approach is used to compute the basic reproductive. According to Osman et al. (2020),), the basic reproduction number in a dynamical system determines the disease's state over time. It is employed to forecast the disease equilibrium's stability. When calculating the basic reproduction number, a value greater than one indicates that there is potential for the infection to spread. Conversely, if the outcome is less than one, it suggests that the disease could potentially be contained with some effort and prevented from spreading. The next-generation matrix is defined as $K = FG^{-1}$ and $R_0 = \rho FG^{-1}$, where ρFG^{-1} indicates the spectral radius of FG^{-1}, as stated by Diekmann et al. (2010) and Otoo et al. (2021) the next-generation matrix is defined as $K = FG^{-1}$ and $R_0 = \rho(FG^{-1})$, where $\rho(FG^{-1})$ denotes the spectral radius of FG^{-1}. We only take into account the infectious compartments in the differential equation system in (4.1) using the next-generation matrix.

$$\begin{cases} \frac{dE}{dt} = \frac{\beta SI}{N} - (\lambda+\kappa+\pi)E \\ \frac{dSy}{dt} = \kappa\rho E - (\xi+\eta+\pi)Sy \\ \frac{dAsy}{dt} = (1-\rho)\kappa E - (\vartheta+\delta+\pi)Asy \\ \frac{dT}{dt} = \sigma I - \alpha T - \pi T \end{cases} \tag{4.3}$$

If f is the count of emerging infection moving into the system and g is the count of infections exiting the system, then

$$
f = \begin{pmatrix} \frac{\beta S I}{N} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad \text{and } g = \begin{pmatrix} (\lambda + \kappa + \pi)E \\ (\xi + \eta + \pi)Sy - \kappa E \\ (\theta + \delta + \pi)_{Asy} - (1-\rho)\kappa E \\ (\sigma + \pi)I - \eta Sy - \theta_{Asy} \\ (\alpha + \pi)T - \sigma I \end{pmatrix}
$$

The Jacobian matrix of *f* and *g* are obtained as

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

But at the disease free equilibrium, there is no exposure. Therefore, no infection and hence no treatment and recovery.

$N = S + E + Sy + Asy + I + T + R$

$N = S + 0 + 0 + 0 + 0 + 0 + 0$

$N = S$

Therefore,

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

$(\lambda + \kappa + \pi)$ 0 0 $\frac{\beta S}{N}$ 0

$-\kappa\rho$ $(\xi + \eta + \pi)$ 0 0 0

$$\begin{vmatrix} \frac{1}{\lambda+\kappa+\pi} & 0 & 0 & 0 & 0 \\ \frac{\kappa\rho}{(\lambda+\kappa+\pi)(\xi+\eta+\pi)} & \frac{1}{(\lambda+\kappa+\pi)} & 0 & 0 & 0 \\ \frac{(1-\rho)\kappa}{(\lambda+\kappa+\pi)(\theta+\delta+\pi)} & 0 & \frac{1}{(\theta+\delta+\pi)} & 0 & 0 \\ \frac{\theta(\xi+\eta+\pi)(1-\rho)\kappa+\eta(\kappa\rho)(\theta+\delta+\pi)}{(\sigma+\pi)(\lambda+\kappa+\pi)(\xi+\eta+\pi)(\theta+\delta+\pi)} & \frac{\eta}{(\xi+\eta+\pi)(\sigma+\pi)} & \frac{\theta}{(\sigma+\theta)(\theta+\delta+\pi)} & \frac{1}{\sigma+\pi} & 0 \\ \frac{\sigma\{(\theta(\xi+\eta+\pi)(1-\rho)\kappa+\eta\kappa\rho(\theta+\delta+\pi))\}}{(\alpha+\pi)(\lambda+\kappa+\pi)(\xi+\eta+\pi)(\theta+\delta+\pi)(\sigma+\pi)} & \frac{\sigma\eta}{(\xi+\eta+\pi)(\alpha+\pi)(\sigma+\pi)} & \frac{\sigma\theta}{(\alpha+\pi)(\theta+\delta+\pi)(\sigma+\pi)} & \frac{\sigma}{(\alpha+\pi)(\alpha+\pi)} & \frac{1}{(\alpha+\pi)} \end{vmatrix}$$

$G =$ $-(1-\rho)\kappa$ 0 $(\vartheta+\delta+\pi)$ $(\sigma+\pi)$ 0 0

0 $-\eta$ $-\vartheta$ $-\sigma$ $(\alpha+\pi)$

0 0 0

The basic reproductive number R_0 is given as $R_0 = (FG^{-1})$. From the relation FG^{-1}, the inverse of G, G^{-1} was calculated as

$G^{-1} =$

Computing the product FG^{-1},

$$FG^{-1} = \begin{vmatrix} \frac{\beta[\theta(\xi+\eta+\pi)(1-\rho)\kappa+\eta\kappa\rho(\theta+\delta+\pi)]}{(\sigma+\pi)(\lambda+\kappa+\pi)(\xi+\eta+\pi)(\theta+\delta+\pi)} & \frac{\beta\eta}{(\alpha+\pi)(\eta+\xi+\pi)} & \frac{\beta\theta}{(\sigma+\pi)(\theta+\delta+\pi)} & \frac{\beta}{\sigma+\pi} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{vmatrix}$$

By selecting the dominant eigenvalue of FG^{-1}, the basic reproductive number is

$$R^0 = \frac{\beta[\theta(\xi+\eta+\pi)(1-\rho)\kappa+\eta\kappa\rho(\theta+\delta+\pi)]}{(\sigma+\pi)(\lambda+\kappa+\pi)(\xi+\eta+\pi)(\theta+\delta+\pi)}$$

4.5.3 Local Stability of the Disease-Free Equilibrium

4.5.3.1 Proof:

The Jacobian matrix of the dynamical system (4.1) at the disease free equilibrium (DFE) point $E_0 = \{S^0, E^0, Sy^0, ASy^0, I^0, T^0, R^0\} = \{\frac{\Lambda}{\pi}, 0, 0, 0, 0, 0, 0\}$, but since at the disease free equilibrium, $S = N = \frac{\Lambda}{\pi}$ is given by

$$J(E_0) = \begin{bmatrix} -\pi & 0 & 0 & 0 & & -\beta & 0 & \mu \\ 0 & -(\lambda+\kappa+\pi) & 0 & 0 & & \beta & 0 & 0 \\ 0 & \rho\kappa & -(\eta+\xi+\pi) & & & 0 & 0 & 0 \\ 0 & (1-\rho)\kappa & 0 & -(\theta+\delta+\pi) & & 0 & & \\ 0 & 0 & \eta & \theta & -(\sigma+\pi) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma & 0 & 0 & \\ 0 & \lambda & \xi & \delta & 0 & -(\alpha+\pi) & 0 & \\ & & & & & \alpha & -(\mu+\pi) & \end{bmatrix}$$

The corresponding characteristic equation for the eigen-values τ is $|JE_0 - \tau| = 0$

$(-\pi-\tau)(-\mu-\pi-\tau)(-\tau-\alpha-\pi)\{(-(\lambda+\kappa+\pi)-\tau)(-(\xi+\eta+\pi)-\tau)(-(\vartheta+\delta+\pi)-\tau)(-\sigma-\pi-\tau) - \beta[\rho\kappa(-(\vartheta+\delta+\pi)-\tau)+\vartheta(-(\xi+\eta+\pi)-\tau)(1-\rho)\kappa] = 0\}$

Hence,

$(-\pi-\tau)(-\mu-\pi-\tau)(-\tau-\alpha-\pi) = 0$ $\tau_1 = -\pi$,

$\tau_2 = -\mu-\pi$, $\tau_3 = -\alpha-\pi$

Also,

$$\begin{bmatrix} -(\lambda+\kappa+\pi)-\tau & 0 & 0 & \beta \\ \kappa\rho & -(\xi+\eta+\pi)-\tau & 0 & 0 \\ (1-\rho)\kappa & 0 & -(\theta+\delta+\pi)-\tau & 0 \\ & \eta & \theta & -(\sigma+\pi)-\tau \end{bmatrix}_0$$

Representing the matrix as

$$A = \begin{bmatrix} a-\tau & 0 & 0 & \beta \\ b & -c-\tau & 0 & 0 \\ d & 0 & -f-\tau & 0 \\ 0 & g & \theta & -h-\tau \end{bmatrix}$$

$$|A-\tau| = 0$$

$$\begin{vmatrix} a-\tau & 0 & 0 & \beta \\ b & -c-\tau & 0 & 0 \\ d & 0 & -f-\tau & 0 \\ 0 & g & \theta & -h-\tau \end{vmatrix} = 0$$

$\tau^4 + (h+a+c+f)\tau^3 + (ca+fa+ha+fc+hc+fh)\tau^2 + (fca + hca + fha + fhc - \beta bg - \beta\vartheta d)\tau + (fhca - \beta bgf - \beta\vartheta cd) = 0$

We had, $\tau^4 + [(\sigma + \pi) + (\lambda + \kappa + \pi) + (\vartheta + \delta + \pi) + (\xi + \eta + \pi)]\tau^3 + [(\vartheta + \delta + \pi)(\sigma + \pi) + (\alpha + \pi)(\xi + \eta + \pi) + (\vartheta + \delta + \pi)(\xi + \eta + \pi) + (\sigma + \pi)(\lambda + \kappa + \pi) + (\lambda + \kappa + \pi)(\vartheta + \delta + \pi) + (\lambda + \kappa + \pi)(\xi + \eta + \pi)]\tau^2 + [(\xi + \eta + \pi)(\vartheta + \delta + \pi)(\sigma + \pi) + (\lambda +\kappa + \pi)(\vartheta + \delta + \pi)(\sigma + \pi) + (\lambda +\kappa + \pi)(\xi + \eta + \pi)(\sigma + \pi) + (\lambda+\kappa\pi)(\xi +\eta +\pi)(\vartheta +\delta+\pi) - \beta(\kappa -\kappa\rho)(\vartheta) - \beta(\rho\kappa)(\eta)]\tau + (\lambda+\kappa +\pi)(\xi + \eta\pi)(\vartheta + \delta + \pi)(\sigma + \pi) - \beta\rho\kappa\eta(\vartheta + \delta + \pi) - \beta(\kappa - \kappa\rho)(\vartheta)(\xi + \eta + \pi) = 0$

This is of the form $a_0\tau^4 + a_1\tau^3 + a_2\tau^2 + a_3\tau + a_4$

According to the Routh-Hurwitz criterion, since $a_0 = 1$, $a_1 > 0$ and $a_2 > 0$, τ_4 and τ_5 will have negative real part as roots. Analysis is therefore carried out on a_3 and a_4.

Ruth-Hurwitz matrix

$$\begin{bmatrix} a_1 & a_3 & 0 & 0 \\ a_0 & a_2 & a_4 & 0 \\ 0 & a_1 & a_3 & 0 \\ 0 & a_0 & a_2 & a_4 \end{bmatrix}$$

$H_1 = [a_1]\ a_1$

> 0

$$H_2 = \begin{bmatrix} a_1 & a_3 \\ a_0 & a_2 \end{bmatrix}$$

$(a_1a_2 - a_0a_3) > 0$

$$H_3 = \begin{bmatrix} a_1 & a_3 & 0 \\ a_0 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{bmatrix}$$

$a_1(a_2a_3 - a_1a_4) - a_3(a_0a_3 - 0) > 0$

$a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$

$$H_4 = \begin{bmatrix} a_1 & a_3 & 0 & 0 \\ a_0 & a_2 & a_4 & 0 \\ 0 & a_1 & a_3 & 0 \\ 0 & a_0 & a_2 & a_4 \end{bmatrix}$$

$a_1a_2a_3a_4 - a_4^2a_1^2 - a_0a_3^2a_4 > 0$

$[(a_1a_2 - a_3)a_3 - a_1^2a_4]a_4 > 0$

For $H_1 = a_1 > 0$

$H_1 = (h + a + c + f) > 0$

For $H_2 = (a_1a_2 - a_0a_3) > 0$

$H_2 = (h + a + c + f)(ca + fa + ha + fc + hc + fh) - (fca + hca + fha + fhc - \beta bg - \beta\vartheta d) > 0$

By simplifying $H_2 > 0$ as required

For $H_3 = a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$

By expanding and simplifying, $H_3 > 0$ as required.

For H_4, it follows that $H_4 = [(a_1a_2 - a_3)a_3 - a_1^2a_4]a_4 > 0$. Since H_3 is positive and a_4 is positive, thus $H_4 = H_3a_4 > 0$.

Hence, $H_4 > 0$ as required.

4.6 The Caputo-Fabrizio Model Properties and Analysis

The Caputo-Fabrizio derivative to the conjunctivitis model is applied to equation (4.1) and is given by:

$$D_t^{\tau}S = \Lambda + \mu R - \frac{\beta SI}{N} - \pi S$$

$$D_t^{\tau}E = \frac{\beta SI}{N} - (\lambda + \kappa + \pi)E$$

$$D_t^{\tau}Sy = \kappa\rho E - (\eta + \xi + \pi)Sy$$

$$D_t^{\tau}Asy = (1 - \rho)\kappa E - (\vartheta + \pi + \delta)Asy$$

$$D_t^{\tau}I = \eta Sy + \vartheta Asy - (\sigma + \pi)I$$

$$D_t^{\tau}T = \sigma I - (\alpha + \pi)T$$

$$D_t^{\tau}R = \alpha T + \eta Sy + \delta Asy + \lambda E - (\mu + \pi)R$$

Where τ represents the fractional order $0 < \tau < 1$ and the initial conditions of the fractional order are $(S(0), E(0), Sy(0), ASy(0), I(0), T(0), R(0)) \in R^7_+$.

4.6.1 Existence and Uniqueness of a Solution of the Model by the Caputo-Fabrizio Operator

A fixed-point result is used to verified the existence and uniqueness of the solution of the model. The system is now written as;

$$^{CF}_{0}D^{\alpha}_{t}S(t) = F_1(t, S)$$

$$^{CF}_{0}D^{\alpha}_{t}E(t) = F_1(t, E)$$

$${}_0^{CF}D_t^{\alpha} Sy(t) = F_1(t, Sy)$$
$${}^{CF}D^{\alpha}{}_0 \; {}_t Asy(t) = F_1(t, Asy)$$
$${}^{CF}D^{\alpha}{}_0 \; {}_t I(t) = F_1(t, I)$$
$${}^{CF}D^{\alpha}{}_0 \; {}_t T(t) = F_1(t, T)$$
$${}_0^{CF}D_t^{\alpha} R(t) = F_1(t, R)$$

Applying the Caputo – Fabrizio operator, the system becomes;

$$S(t) - S(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_1(t,S) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_1(\eta, S) d\eta$$
$$E(t) - E(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_2(t,E) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_2(\eta, E) d\eta$$
$$Sy(t) - Sy(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_3(t,Sy) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_3(\eta, Sy) d\eta$$
$$Asy(t) - Asy(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_4(t,Asy) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_4(\eta, Asy) d\eta$$
$$I(t) - I(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_5(t,I) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_5(\eta, I) d\eta$$
$$T(t) - T(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_6(t,T) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_6(\eta, T) d\eta$$
$$R(t) - R(0) = \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} F_7(t,R) + \frac{2\alpha}{(2-\alpha)} \int_0^t F_7(\eta, R) d\eta$$

We prove that F_1 to F_7 satisfy Lipschitz continuity and contraction.

4.6.1.1 Theorem:

$$\| F_1(t,S) - F_1(t,S^*) \| = \| \Lambda + \mu R(t) - \frac{\beta S(t) I(t)}{N} - \pi S(t) - (\Lambda + \mu R(t) - \frac{\beta S^*(t) I(t)}{N} - \pi S^*(t)) \|$$

$$= \| -\frac{\beta S(t) I(t)}{N} - \pi S(t) + \frac{\beta S^*(t) I(t)}{N} - \pi S^*(t) \|$$
$$= \| -\frac{\beta I(t)}{N}(S(t) - S^*(t)) - \pi(S(t) - S^*(t)) \|$$

$$\leq \frac{\beta}{N} \| I(t) \| \| S(t) - S^*(t) \| + \pi \| S(t) - S^*(t) \|$$
$$\leq (\frac{\beta}{N} h_1 + \pi) \| S(t) - S^*(t) \|$$
$$\leq L_1 \| S(t) - S^*(t) \|$$

Where, $L_1 = \frac{\beta}{N} + \pi$ and $h_1 \leq \| I(t) \|$

In the same way, the Lipschitz continuity and contraction were shown for F_2 to F_7 where L_2 to L_7 were obtained respectively as their Lipschitz constants.

$$\| F_2(t,E) - F_2(t,E^*) \| = \| \frac{\beta S(t) I(t)}{N} - (\lambda+\kappa+\pi)E(t) - (\frac{\beta S^*(t) I(t)}{N} - (\lambda+\kappa+\pi)E^*(t)) \|$$

$$= \| -(\lambda+\kappa+\pi)E(t) + (\lambda+\kappa+\pi)E^*(t) \|$$

$$= \| -(\lambda+\kappa+\pi) \| E(t) - E^*(t) \|$$

$$= \| (\lambda+\kappa+\pi) \| E(t) - E^*(t) \|$$

Where, $L_2 = (\lambda+\kappa+\pi)$

$$\| F_3(t,Sy) - F_3(t,Sy^*) \| = \| \kappa\rho E(t) - (\xi+\eta+\pi)Sy(t) - (\kappa\rho E(t) - (\eta+\xi+\pi)Sy^*(t)) \|$$

$$= \| -(\xi+\eta+\pi)Sy(t) + (\xi+\eta+\pi)Sy^*(t) \|$$

$$= \| -(\xi+\eta+\pi) \| Sy(t) - Sy^*(t) \|$$

$$= \| (\xi+\eta+\pi) \| Sy(t) - Sy^*(t) \|$$

Where, $L_3 = (\xi+\eta+\pi)$

$$\| F_4(t,Asy) - F_4(t,Asy^*) \| = \| (1-\rho)\kappa E(t) - (\vartheta+\delta+\pi)Asy(t) - ((1-\rho)\kappa E(t) - (\vartheta+\delta+\pi)Asy^*(t)) \|$$

$$= \| -(\vartheta+\delta+\pi)Sy(t) + (\xi+\eta+\pi)Sy^*(t) \|$$

$$= \| -(\vartheta+\delta+\pi) \| Asy(t) - Asy^*(t) \|$$

$$= \| (\vartheta+\delta+\pi) \| Asy(t) - Asy^*(t) \|$$

Where, $L_4 = (\vartheta+\delta+\pi)$

$$\| F_5(t,I) - F_5(t,I^*) \| = \| \eta Sy(t) + \vartheta Asy(t) - (\sigma+\pi)I(t) - (\eta Sy(t) + \vartheta Asy(t) - (\sigma+\pi)I^*(t)) \|$$

$$= \| -(\sigma+\pi)I(t) + (\sigma+\pi)I^*(t) \|$$

$$= \| -(\sigma+\pi) \| I(t) - I^*(t) \|$$

$$= \| (\sigma+\pi) \| I(t) - I^*(t) \|$$

Where, $L_5 = (\sigma+\pi)$

$\| F_6(t, T) - F_6(t, T^*) \| = \| \sigma I(t) - (\alpha + \pi)T(t) - (\sigma I(t) - (\sigma + \pi)T^*(t)) \|$

$$= \| -(\alpha + \pi)T(t) + (\alpha + \pi)T^*(t) \|$$

$$= \| -(\alpha + \pi) \| T(t) - T^*(t) \|$$

$$= \| (\alpha + \pi) \| T(t) - T^*(t) \|$$

Where, $L_6 = (\alpha + \pi)$

$\| F_7(t, T) - F_7(t, T^*) \| = \| \alpha T(t) + \xi Sy(t) + \delta Asy(t) + \lambda E(t) - (\mu + \pi)R(t) -$ $(\alpha T(t)\xi Sy(t) + \delta Asy(t) + \lambda E(t) - (\mu + \pi)R^*(t)) \|$

$$= \| -(\mu + \pi)R(t) + (\mu + \pi)R^*(t) \|$$

$$= \| -(\mu + \pi) \| R(t) - R^*(t) \|$$

$$= \| (\mu + \pi) \| R(t) - R^*(t) \|$$

Where, $L_7 = (\mu + \pi)$

To show the existence of the solution, the following theorem was proven.

4.6.1.2 Theorem:

The solution exists if there exist t_1 such that the following inequality is true,

$$\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)}L_i + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_i < 1, i = 1, \ldots, 7$$

4.6.1.3 Proof

Recursively, we have

$$\| q_{1n}(t) \| \leq \| S_n(0) \| \left[\frac{2\ 1\ \ \alpha}{(2-\alpha)M(\alpha)}L_1 + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_1\right] \quad (-) \quad n$$

$$\| q_{2n}(t) \| \leq \| E_n(0) \| \left[\frac{2\ 1\ \ \alpha}{(2-\alpha)M(\alpha)}L_2 + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_2\right], \quad (-) \quad n$$

,

$$\| q_{3n}(t) \| \leq \| Sy_n{}^{(0)} \| \left[\frac{}{(2-\alpha)M(\alpha)}L_3 + \frac{i}{(2-\alpha)M(\alpha)}L_3\right]_{2(1-\alpha)} \quad 2\alpha t \quad n,$$

$\| q_{4n}(t) \| \leq \| Asy_n(0) \|$ h(2−2(α1)−Mα()α)L_4 + (2−2αα)tM$_i$(α)L_4in ,

$$\| q_{5n}(t) \| \leq \| I_n(0) \| \left[\frac{2\ 1\ \ \alpha}{(2-\alpha)M(\alpha)}L_5 + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_5\right] \quad (-) \quad n$$
$$\| q_{6n}(t) \| \leq \| T_n(0) \| \left[\frac{2\ 1\ \ \alpha}{(2-\alpha)M(\alpha)}L_6 + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_6\right],$$
$$\| q_{7n}(t) \| \leq \| R_n(0) \| \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)}L_7 + \frac{2\alpha t_i}{(2-\alpha)M(\alpha)}L_7\right], \quad (-) \quad n$$

n

.

Hence, the solutions are existed and continuous. To demonstrate that the above functions construct the solutions, consider;

$S(t) - S(0) = S_n(t) - K_{1n}(t)$

$E(t) - E(0) = E_n(t) - K_{2n}(t)$

$Sy(t) - Sy(0) = Sy_n(t) - k_{3n}(t)$

$ASy(t) - ASy(0) = Asy_n - k_{4n}(t)$

$I(t) - I(0) = I_n(t) - K_{5n}(t)$

$T(t) - T(0) = T_n(t) - K_{6n}(t)$

$R(t) - R(0) = R_n(t) - K_{7n}(t)$

Hence,

$$\| K_{1n}(t) \| = \| \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)}(F_1(t, S_{n-1}) - F_1(t, S_{n-2})) + \frac{2\alpha}{(2-\alpha)M(\alpha)} \int_0^t (F_1(\eta, S_{n-1}) - F_1(\eta, S_{(n-2)}))d\eta \|$$

$$\leq \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} \| F_1(t, S_{n-1}) - F_1(t, S_{n-2}) \| + \frac{2\alpha}{(2-\alpha)M(\alpha)} \| \int_0^t (F_1(\eta, S_{n-1}) - F_1(\eta, S_{n-2}))d\eta \|$$
$$\leq \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)}L_1 \| S - S_{n-1} \| + \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)}L_1 \| S - S_{n-2} \|_t$$

Hence, carrying out the procedure

$$\| K_{1n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)}\right]^{n+1} L_1^{n+1} K$$

At $t = t_1$, we get

$$\| K_{1n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)}\right]^{n+1} L_1^{n+1} K$$

Taking the limit as $n \to \infty$, we have

$\| k_{1n}(t) \| \to 0$

Similarly,

$$\| K_{2n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)}\right]^{n+1} L_2^{n+1} K$$

At $t = t_1$, we get

$$\| K_{2n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)}\right]^{n+1} L_2^{n+1} K$$

Taking the limit as $n \to \infty$, we have

$\| k_{2n}(t) \| \to 0$

$\| K_{3n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)} \right]^{n+1} L_3^{n+1} K$

At $t = t_1$, we get

$\| K_{3n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)} \right]^{n+1} L_3^{n+1} K$

Taking the limit as $n \to \infty$, we have

$\| k_{3n}(t) \| \to 0$

$\| K_{4n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)} \right]^{n+1} L_4^{n+1} K$

At $t = t_1$, we get

$\| K_{4n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)} \right]^{n+1} L_4^{n+1} K$

Taking the limit as $n \to \infty$, we have

$\| k_{4n}(t) \| \to 0$

$\| K_{5n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)} \right]^{n+1} L_5^{n+1} K$

At $t = t_1$, we get

$\| K_{5n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)} \right]^{n+1} L_5^{n+1} K$

Taking the limit as $n \to \infty$, we have

$\| k_{5n}(t) \| \to 0$

$\| K_{6n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)} \right]^{n+1} L_6^{n+1} K$

At $t = t_1$, we get

$\| K_{6n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)} \right]^{n+1} L_6^{n+1} K$

Taking the limit as $n \to \infty$, we have

$\| k_{6n}(t) \| \to 0$

$\| K_{7n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t}{(2-\alpha)M(\alpha)} \right]^{n+1} L_7^{n+1} K$

At $t = t_1$, we get

$\| K_{7n}(t) \| \leq \left[\frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} + \frac{2\alpha t_1}{(2-\alpha)M(\alpha)} \right]^{n+1} L_7^{n+1} K$

Taking the limit as $n \to \infty$, we have

$\| k_{7n}(t) \| \to 0$

Finally, the uniqueness of the solution is shown by assuming there exists some solutions say,

$S^1(t)$, $E^1(t)$, $Sy^1(t)$, $Asy^1(t)$, $I^1(t)$, $T^1(t)$ and $R^1(t)$, then

$\| S(t) - S^1(t) \| \{1 - \frac{2(1-\alpha)}{(2-\alpha)M(\alpha)} L_1 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_1\} \leq 0$

The following theorem completes the result.

4.6.2 Theorem

If $\{1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_1 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_1\} \leq 0$

then the solution is unique.

4.6.3 Proof

Consider

$\| S(t) - S^1(t) \| (1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_1 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_1 \leq 0)$

Since,

$(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_1 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_1 > 0)$

Then, $\| S(t) - S^1(t) \| = 0$

This implies

$S(t) = S^1(t)$

Same analysis is done to the remaining functions.

$\| E(t) - E^1(t) \| (1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_2 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_2 \leq 0)$

Since,

$(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_2 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_2 > 0)$

Then, $\| E(t) - E^1(t) \| = 0$

This implies

$E(t) = E^1(t)$

$\| Sy(t) - Sy^1(t) \| (1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_3 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_3 \leq 0)$

Since,

$(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_3 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_3 > 0)$

Then, $\| Sy(t) - Sy^1(t) \| = 0$

This implies

$Sy(t) = Sy^1(t)$

$\| Asy(t) - Asy^1(t) \| (1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_4 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_4 \leq 0)$

Since,

$(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)}L_4 - \frac{2\alpha t}{(2-\alpha)M(\alpha)}L_4 > 0)$

Then, $\| Asy(t) - Asy^1(t) \| = 0$

This implies

$Asy(t) = Asy^1(t)$

$$\| I(t) - I^1(t) \| \left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_5 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_5 \leq 0\right)$$

Since,

$$\left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_5 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_5 > 0\right)$$

Then, $\| I(t) - I^1(t) \| = 0$

This implies

$I(t) = I^1(t)$

$$\| T(t) - T^1(t) \| \left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_6 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_6 \leq 0\right)$$

Since,

$$\left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_7 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_7 > 0\right)$$

Then, $\| T(t) - T^1(t) \| = 0$

This implies

$T(t) = T^1(t)$

$$\| R(t) - R^1(t) \| \left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_7 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_7 \leq 0\right)$$

Since,

$$\left(1 - \frac{(2(1-\alpha)}{(2-\alpha)M(\alpha)} L_7 - \frac{2\alpha t}{(2-\alpha)M(\alpha)} L_7 > 0\right)$$

Then, $\| R(t) - R^1(t) \| = 0$

This implies

$R(t) = R^1(t)$

4.7 Numerical Discussions

The model's numerical simulation, analysis, and graphical depiction of the fractional order system at various alpha values are all demonstrated. The model's parameters are investigated using simulation. We obtained some interesting results in the conjunctivitis infection model by employing non-integer parameters. The figures 4.2 - 4.8 provide us the graphical representations at different α values. MATLAB code for Langrange polynomial interpolation iteration scheme is used to find numerical simulation for the conjunctivitis model.

The parameter values involved in the formulated system are provided below;

Parameter	Description	Values	Source

Λ	Recruitment of susceptible individuals	200	Estimated
β	The rate of infection of susceptible individuals	0.567	Fitted
ρ	Fraction of expose individuals showing symptoms	0.201	Fitted
π	Natural death rate	0.09	Assumed
λ	Rate at which the exposed recover	0.038	Fitted
ξ	Recovery rate for symptomatic individual	0.14	Fitted
δ	Recovery rate for the asymptomatic individual	0.15	Fitted
η	Rate at which the symptomatic progress to the infected	0.181	Fitted
ϑ	Rate at which the asymptomatic progress to the infected	0.223	Fitted
σ	Treatment rate	0.601	Fitted
α	Recovery rate of treated individuals	0.417	Fitted
μ	Rate of individuals returning to the susceptible state	0.45	Fitted
κ	Rate at which individuals move from the expose compartment	0.234	Fitted

Table 4.3: **Parameters of conjunctivitis**

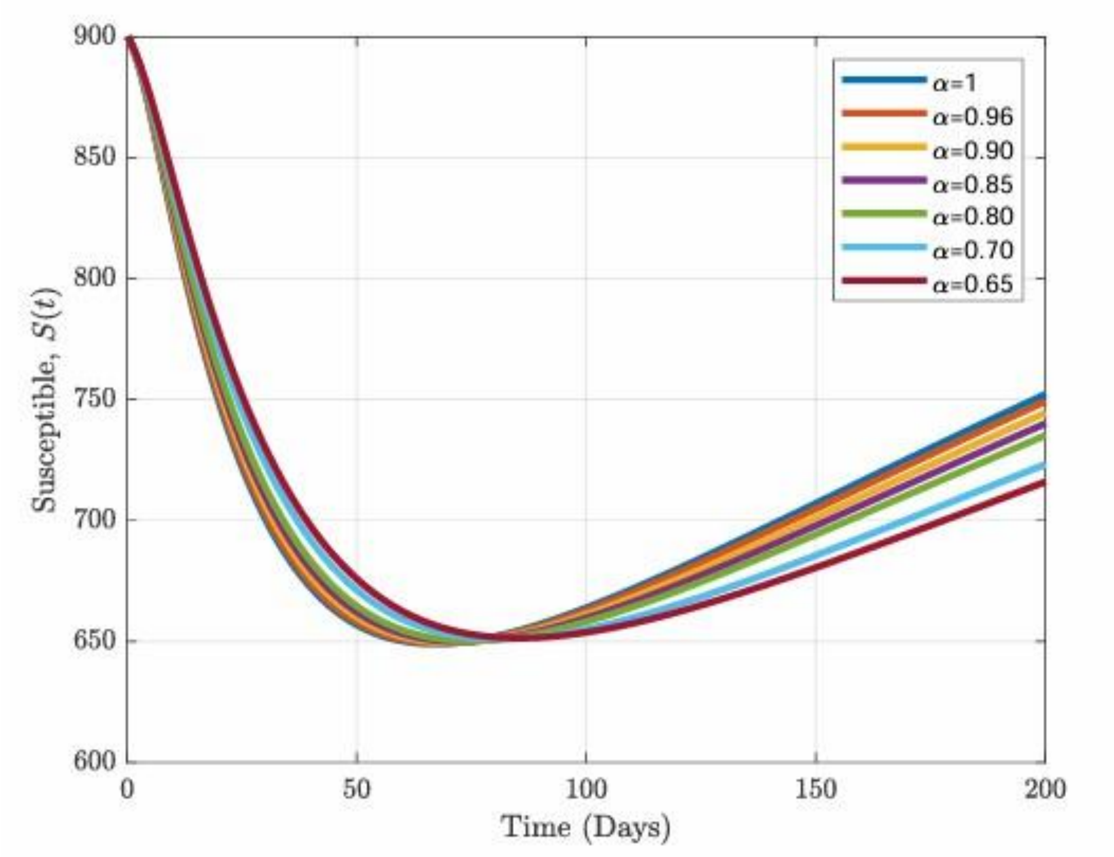

Figure 4.2: plot of susceptible individual

From Fig. 4.2, the graph exhibits a pattern that is typical of disease dynamics, a steep initial decrease in the number of susceptible people, a trough, and then a steady increase in their numbers over time. The early stages of the outbreak, when the disease spreads and infects a sizable portion of the populace, are correlated with the initial sharp decline. As the outbreak spreads, fewer people are susceptible because they either contract the infection or become immune, which causes the graph to trough. The number of susceptible people gradually starts to rise again as infections decrease and people either recover or stop being contagious. This is indicative of a resurgence of susceptible people brought on by things like new babies or waning immunity in the population.

The number of susceptible individuals declines sharply at first and reaches its lowest point faster in the classical model without memory effects. This is in contrast to the fractional models. This is consistent with the usual behaviour in classical epidemiological models, in which the susceptible population rapidly declines in the early stages of an outbreak due to a relatively high infection rate.

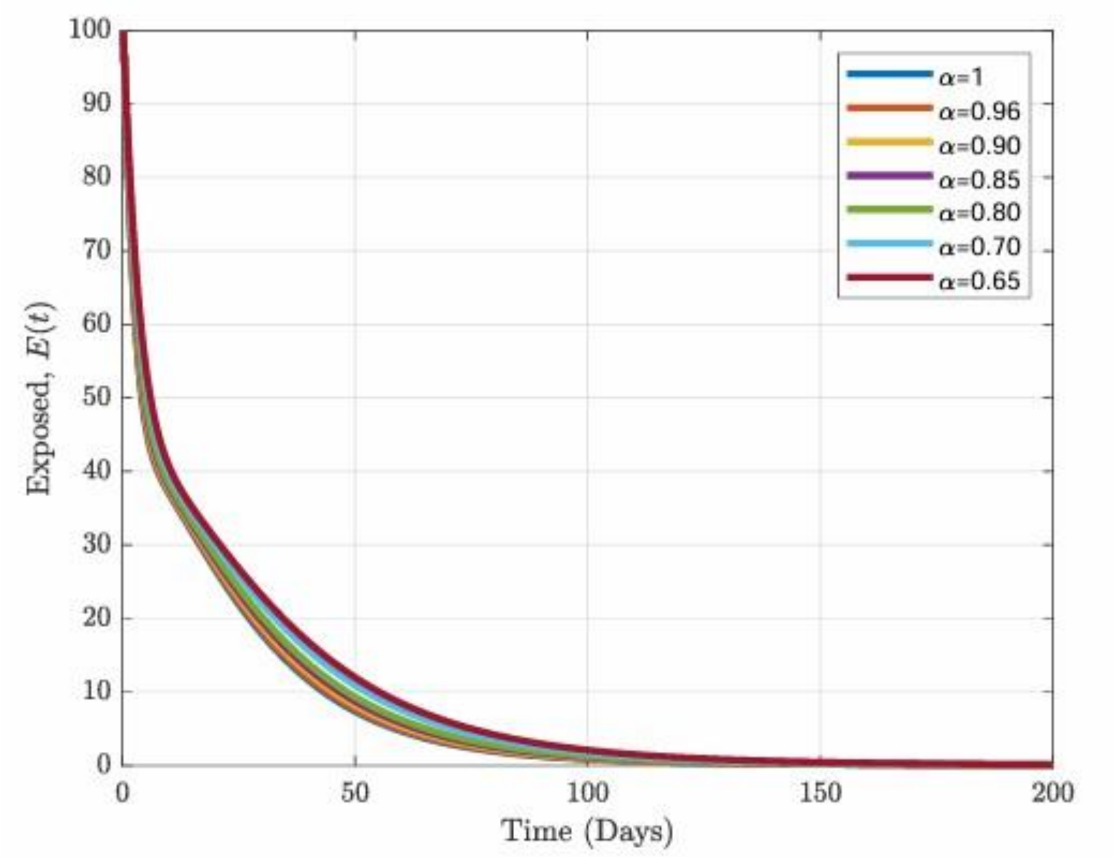

Figure 4.3: plot of Expose individual

In fig 4.3, even though the rate of decline varies across different α values, by day 150 to 200, all curves eventually converge toward zero exposed individuals. This suggests that the number of people exposed will eventually decline and there won't be any latent infections left in the population, regardless of the strength of the memory effect. The speed at which this resolution takes place is the crucial difference. The exposed population declines more quickly in models with higher alpha values, but the sustained influence of previous exposure states causes the decline to occur more gradually in models with lower alpha values.

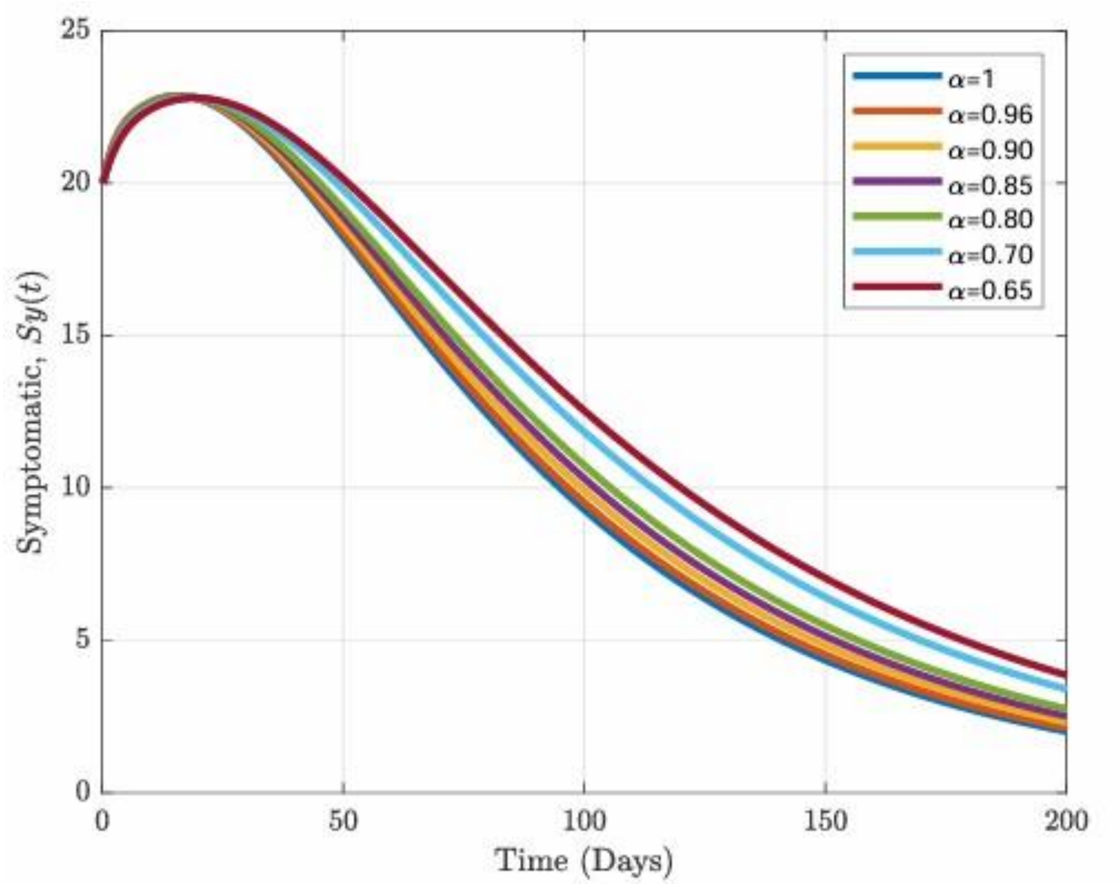

Figure 4.4: plot of symptomatic individuals

From fig 4.4, while the dynamics of the initial infection differ based on the α values, by day 150 to 200, all curves eventually converge toward zero symptomatic individuals. This convergence suggests that the outbreak will eventually end and there won't be any symptomatic individuals in the population, even if memory effects have any influence. The length of the symptomatic phase and the rate of recovery, however, are where the main differences lie. Models with lower α values show that symptomatic individuals stay in the population for longer periods of time, which may be a result of memory effects delaying the outbreak's resolution.

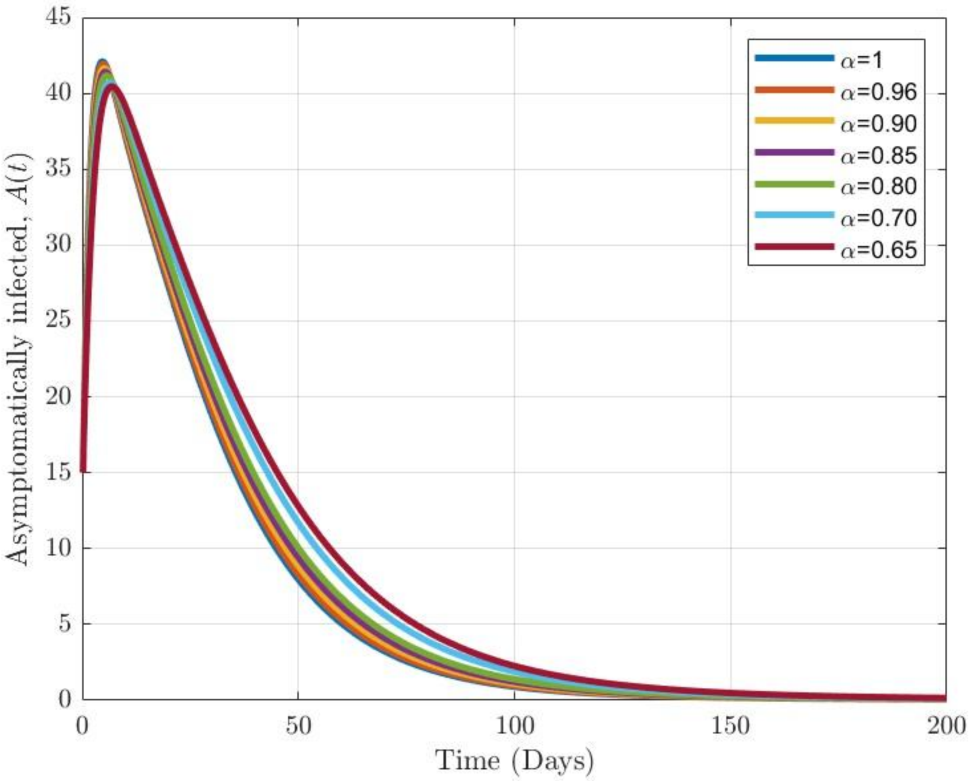

Figure 4.5: plot of Asymptomatic individual

From Fig 4.5, even though the dynamics of infection vary in terms of timing and speed for various α values, by day 150 to 200, all curves eventually converge towards zero. This implies that the population will eventually resolve the outbreak of asymptomatic individuals, irrespective of the extent of memory effect represented by the fractional derivative. But given that memory effects can have a substantial impact on the timing and duration of an outbreak, the variations in the peak and decline rates highlight how crucial it is to include them in disease models.

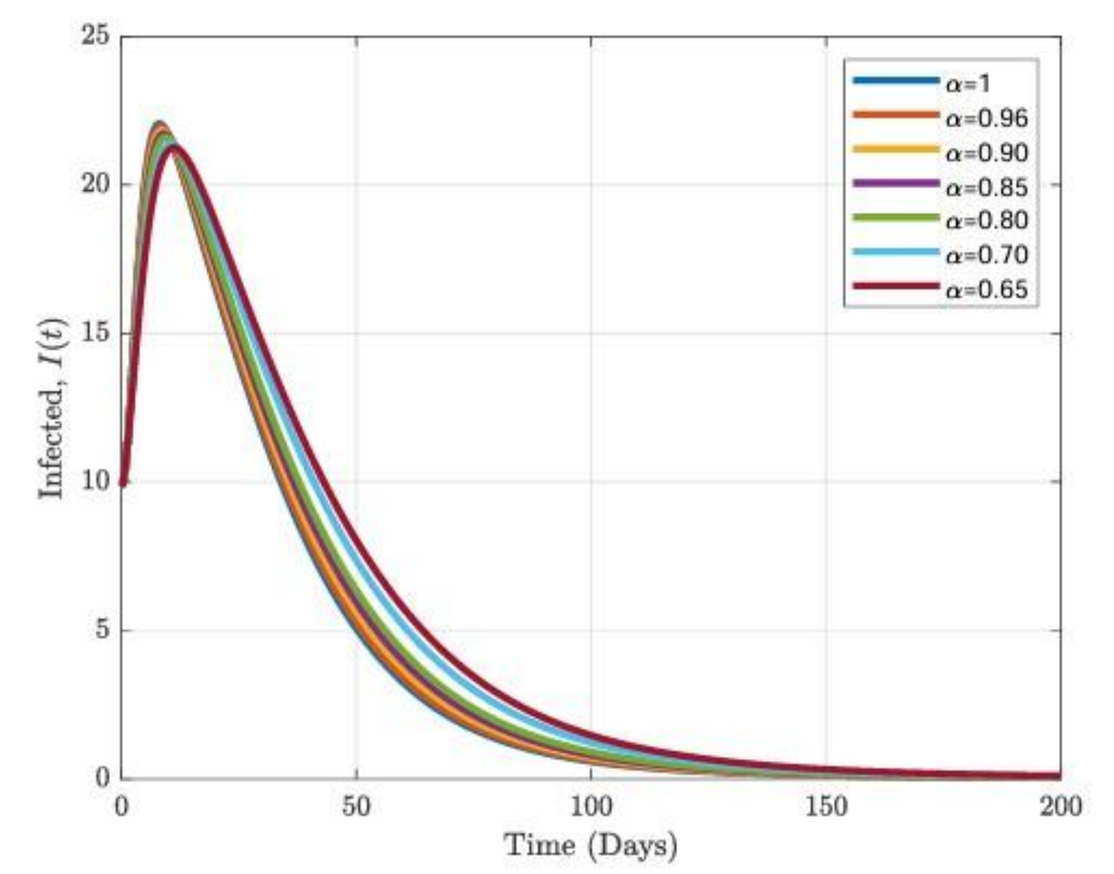

Figure 4.6: plot of infected individual

From Fig 4.6, depending on the value of α, the infection dynamics vary greatly in the early stages of the outbreak; however, by day 150 to 200, all curves eventually converge towards zero infected individuals. This implies that the outbreak eventually ends, leaving the population free of active infections, regardless of the extent of memory effect represented by the fractional derivative. However, the value of α has a strong influence on the time it takes to reach this state and the severity of the outbreak, as indicated by the height of the peak. A shorter outbreak resolution period is indicated by lower alpha values, whereas higher alpha values signify a longer infection period.

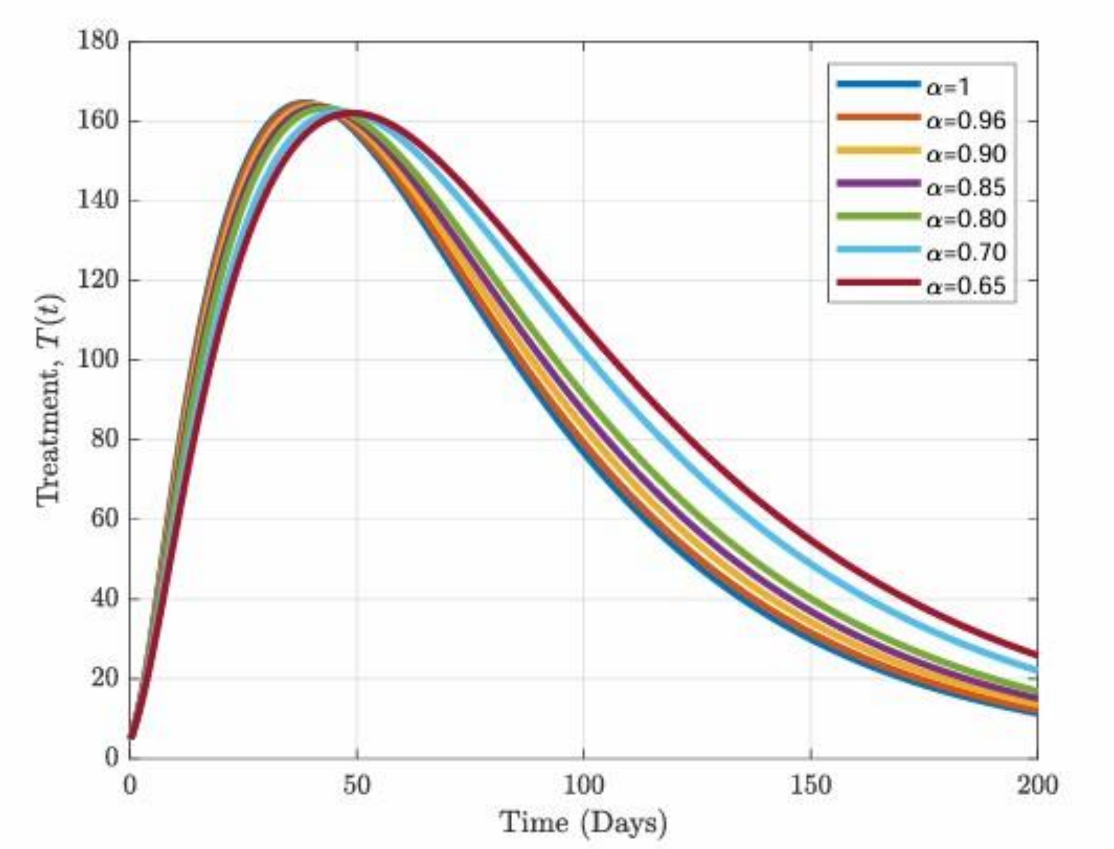

Figure 4.7: plot of Treatment

From Fig. 4.7, the curve exhibits the fastest increase in treatment numbers for $\alpha = 1$, which corresponds to the classical, integer-order derivative. It peaks approximately on day 30. This implies that treatment needs increase quickly and peak earlier in scenarios where past disease states have little bearing on current dynamics. This kind of behaviour is common during acute outbreaks, when the infection spreads quickly and causes a spike in the number of people seeking care quickly. Following the peak, the curve gradually falls as the infection is under control and fewer people need medical attention. The treatment curves show slower growth as the α values decrease, corresponding to stronger memory effects. In contrast to the classical model $\alpha = 1$, the curves for $\alpha = 0.96$ rise more gradually and peak slightly later. This suggests that the system's memory controls the rate of new infections and, consequently, the course of treatment, when past disease states impact current dynamics. The curve for $\alpha = 0.65$ where the treatment peak is both later and lower than for higher alpha values. This suggests that treatment demand increases more slowly and decreases more gradually in scenarios where memory is highly dependent.

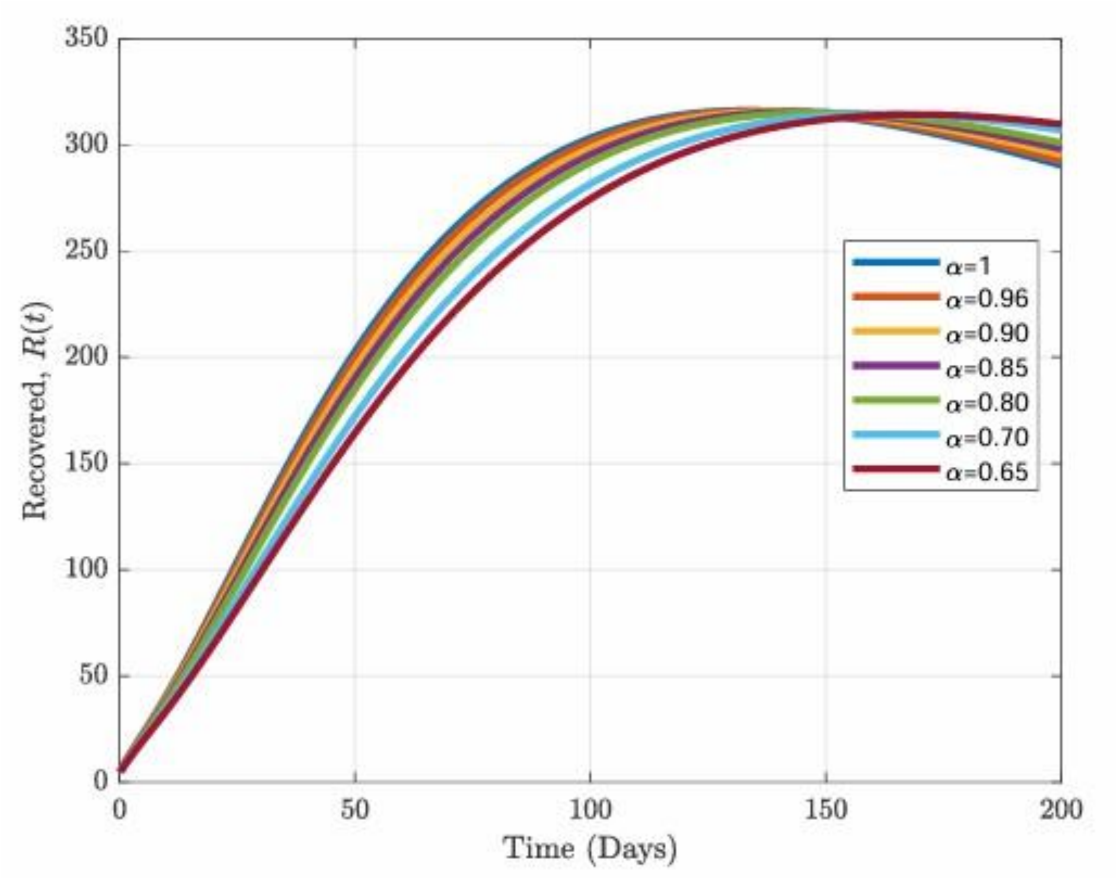

Figure 4.8: plot of Recovered Individuals

From Fig. 4.8, the graph for $\alpha = 1$ illustrates the classical recovery model, in which the steep initial slope indicates a rapid change in the number of recovered individuals during the early stages of the infection. This curve quickly reaches a plateau, indicating that most people heal in a comparatively short amount of time. This behaviour is consistent with conventional epidemiological models, which hold that the influence of previous disease states is negligible and that, once suitable interventions, like treatment or natural immunity, are in place, the infection can resolve quickly in the population.

The differences in recovery dynamics caused by various α values shed light on the characteristics of conjunctivitis outbreaks in the Tano basin. Recovery is faster and indicates that the disease progresses in a more straightforward, predictable manner, probably responding well to medical interventions or natural immunity, for higher α values. On the other hand, there is a slower recovery for lower α values emphasizing the necessity of longer-term or ongoing public health interventions. Under such circumstances, scenarios where environmental contamination, like waterborne pathogens, or delayed responses in treatment and containment lead to longer-lasting outbreaks may be represented by the strong memory effects modelled by the fractional derivative. This emphasizes how crucial it is to take historical data and prior infections into account.

Chapter 5
Conclusion and Recommendation

5.1 Introduction

A conjunctivitis model with a Caputo-Fabrizio derivative was successfully analysed. The model parameters employed in numerical simulations from 2019 to 2023 were parameterised using data from Holy Family Hospital and St. Elizabeth Hospital, two significant hospitals in Ghana's Bono East and Ahafo regions, respectively. By assuming various α values for the fractional parameter, we were able to obtain numerical findings and offer a thorough discussion. The existence, uniqueness, and model solution were demonstrated using fixed theory. The graphical findings show that, in comparison to the integer order derivative, the newly proposed derivative for the model yields flexible results that may be more useful. In order to confirm the theoretical findings, illustrate the impact of model parameters, and emphasise the effect of conjunctivitis infection, numerical simulations of the model are finally displayed.

5.2 Conclusion

This study has successfully achieved its objectives by developing a mathematical model to explain the dynamics of conjunctivitis in the the Tano basin, local analysis on the model was performed and behaviour of key parameters at different α values were investigated.

The study found that lower values of α (i.e., closer to 0.65) result in more pronounced memory effects, meaning that individuals in the population retain the influence of previous states for longer periods. This leads to slower recovery times, prolonged outbreaks, and oscillations in the number of infected individuals over time. In practical terms, this implies that when the memory effect is strong, individuals who have been exposed to conjunctivitis or have recovered are still susceptible to reinfection or may remain infectious for longer durations. This results in the disease persisting in the population for extended periods, increasing the risk of recurrent outbreaks. The persistence of infection also suggests that environmental factors, such as continuous exposure to contaminated water, exacerbate the problem by repeatedly exposing individuals to the pathogens that cause conjunctivitis. On the other hand, higher values of α (closer to 1) reduce the memory effect, meaning that the influence of past disease states diminishes more rapidly. This leads to quicker recovery rates, shorter disease cycles, and more predictable patterns of infection. When α is high, the system tends to stabilize faster, as individuals recover more fully and are less likely to experience reinfection in the short term. This behaviour indicates that higher α values could be associated with more effective medical treatments, improved immunity in the population, or reduced exposure to the environmental sources of infection, such as cleaner water supplies.

5.3 Recommendation

- The study only considered the dynamics of the model without focusing on intervention strategies, it is therefore recommended that, future studies could consider effective strategies to control bacterial conjunctivitis.

- The asymptomatic nature of the disease makes the spread easy, it is therefore advised that routine screening in schools, healthcare and work places should be regular.

- It is highly recommended that, future works should incorporate environmental and socioeconomic factors into the model.

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