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SIRT MATHEMATICAL MODEL OF INFLUENZA INFECTION AND ACCESSION OF ITS ANTIVIRAL DRUGS EFFICACY

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Declaration

We affirm that this submission is an original and entirely our own, presented to obtain an undergraduate degree. We have not included any material that has been published by anyone else for any other University degree unless we have given proper credit where due within the text.

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Dedication

We express our deepest gratitude to Almighty God for His continual love and protection throughout the course of this research work. Additionally, we dedicate this work to our supervisor, Dr. Gaston Edem Awashie, whose guidance and support have been invaluable. Lastly, we extend our heartfelt appreciation to our families for their unwavering concern, love and encouragement.

Abstract

Influenza remains a significant global health concern, affirming continuous efforts to evaluate the effectiveness of antiviral drugs. The emergent of Mathematical modeling as a valuable tool to comprehensively assess the impact of antiviral treatments on the dynamics of influenza infection. This study employs SIRT (Susceptible, Infected, Recovered, and Treated) modeling to investigate the efficacy of antiviral drugs in fighting influenza infections. The SIRT model incorporates vital epidemiological parameters to simulate the dynamics of influenza spread and the impact of antiviral interventions. By calibrating the model with data collected by other publishers on influenza infection.This research provides valuable insights into the optimal administration of antiviral drugs. The findings from this study can inform policymakers and healthcare professionals on evidence-based decision-making to relieve the burden of influenza and improve patient outcomes.

Acknowledgement

First and famous, we are so much grateful Almighty God for His grace and mercy, which gave us the strength and motivation to persevere and complete our work, even during times when we felt like giving up. However, we would not forget the relentless effort of our supervisor, Dr. Gaston Edem Awashie, for teaching us to be self-sufficient and not rely on hand-holding. We appreciate the constructive feedback and pressure he provided, which helped us improve our work. We also extend our appreciation to all the lecturers who taught us during our four-year stay in the Mathematics department at KNUST, for sharing their knowledge with us.

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Chapter 1

Introduction

1.1 Background Of Study

Influenza, also known as flu, stands as a global health challenge, perpetually imposing a substantial burden of morbidity and mortality on communities worldwide (WHO, 2021). Its popularity lies in its persistent ability to evolve, shape-shifting through genetic mutations and reassortment, often producing novel strains with the potential to evade both pre-existing immunity and medical interventions (CDC, 2020). The emergence of drug-resistant influenza strains further intensifies this difficult situation, underscoring the dire need for effective antiviral interventions (Baranovich et al., 2017).

Antiviral drugs represent indispensable weapons in our arsenal against influenza. They serve a dual purpose, aiding in both the treatment of infected individuals and the protection of those at risk of exposure. However, their effectiveness is far from uniform and depends on a complex interplay of factors, including the specific viral strain, the presence of drug-resistant variants, and the host's immune response (Paules and Subbarao, 2017). This variability highlights the necessity for a comprehensive understanding of the dynamics that govern the interaction between influenza viruses

and antiviral drugs.

To tackle this intricate problem, mathematical modeling has emerged as a potent and invaluable tool. Mathematical models, rooted in epidemiology and population biology, enable researchers to simulate and analyze the intricate interplay between viral dynamics, host immunity, and the impact of antiviral interventions (Longini et al., 2004).

One of the gravest concerns associated with influenza is the potential for a pandemic, which occurs when a novel influenza virus variant emerges, one to which the majority of the population lacks immunity due to its distinct genetic makeup (WHO, 2021). Such a virus can spread readily from person to person, leading to widespread illness and societal disruption. Interestingly, seasonal influenza strains may play a pivotal role in the genesis of pandemic viruses. When these seasonal strains introduce genetic elements into a new viral subtype, the stage is set for a pandemic to unfold (Krammer et al., 2018).

Furthermore, once a pandemic virus establishes itself within the human population, as seen with the pandemic A(H1N1) in 2009, it can transform into a seasonal virus, perpetually circulating and contributing to the annual influenza burden (WHO, 2021). This underscores the interconnectedness of pandemic and seasonal influenza and the importance of continuous monitoring and surveillance.

1.2 Problem Statement

Influenza possesses the potential to inflict severe illness and complications, particularly in vulnerable populations such as children under the age of five, pregnant women, and the elderly. As these groups are at higher risk of experiencing serious health outcomes due to influenza infection, it emphasizes the necessity of putting evidence-based procedures in place to handle influenza outbreaks efficiently. This involves not only accurate diagnosis and appropriate medical interventions but it highlights the need for

serious educational initiatives regarding antiviral medications and their established usefulness. Healthcare professionals can encourage prompt and informed decision-making, enhance outcomes, and have a better ability to lessen the effects of influenza outbreaks by raising public awareness of and comprehension of antiviral medications.As accurate information is essential for selecting the best course of therapy.

1.3 Objectives

The objectives of this study are:

- To develop a mathematical model using the SIRT.
- To simulate the dynamics of influenza infection in a population
- To show treatment-induced recovery helps to curb Influenza.

1.4 Methodology

This research employs qualitative approach to look into the transmission dynamics of influenza A and the impact of anti-viral drugs. However, qualitative analysis is applied in simulating a mathematical model known as SIRT. This model is applied to simulate the spread of the virus and predict the potential impact of anti-viral drugs on reducing its transmission. The combination of these approaches allows us to gain a comprehensive understanding of the transmission dynamics of influenza A virus and the effectiveness of anti-viral drugs.

1.5 Justification

The urgent requirement to comprehend influenza dynamics, antiviral drug efficacy, and their combined impact on disease transmission. Mathematical modeling offers a powerful tool to analyze complex interactions and provides insights that can guide public health authorities and researchers in optimizing strategies for influenza control and antiviral drug usage.

1.6 Organization Of Study

This research work consist of five chapters. Chapter one contains the introduction which comprises of the background of study, which highlights the essential work that has been done concerning the work. the problem statement , the objectives of the research, and methods used . In the chapter two, various researches that have been done by others pertaining to the work are reviewed.Chapter three, a detailed methodology used for the research. Chapter four contains the analysis and results that is attained after using the methods, and lastly Chapter five contain the conclusion and recommendations.

Chapter 2

Literature Review

2.1 Introduction

Mathematical modelling of influenza A virus dynamics and impact of anti-viral drugs can provide insights into transmission patterns and guide public health policies. This literature review aims to summarize the current state of research with regards to the topic of study.

2.2 Over View Of Concept

A study by Samuel Okyere et al (2012) presented SEIR epidemiological model of influenza A (H1N1) transmission in the Ashanti region using the regional data. They showed the pandemic potential of influenza A (H1N1) required decision makers to act in the face of uncertainties. They assumed that the population is constant and homogeneous mixing of people. They found disease free and endemic equilibrium with their stabilities with the aim of determining the threshold conditions under which the disease will die out or spread. They illustrated the outcome with numerical solutions

and their results indicates that 0.64% of the susceptible population needed to be vaccinated to control the spread of the disease.

Another study by Ankamu Daniel et al (2015) formulated SEIR model for the $H1N1$ human transmission in the Brong Ahafo region of Ghana. Their method of solution proved that the mode has positive solution. They found the basic reproductive number to check the spread of the disease. They also carried out the stability analysis of the DFE and the endemic equilibrium. Sensitivity Analysis and herd humminity of the model was also established. They performed numerical simulation of the model and showed the result graphically. The result indicated that by 0.6% of the susceptible population needed to be vaccinated to curb down the disease in the region.

Also, Hakimeh Mohammadi et al (2020) studies the SEIR epidemic model for the spread of AH_1N_1 influenza using the Caputo-Fabrizio fractional-order derivative. They calculated the basic reproductive number, Equilibrium points and stability of the DFE. They used the Euler method to find the numerical simulation and represented the results diagrammatically. Also in the numerical section, they calculated the equilibrium points of the system and examined the behavior of the resulting functions. They calculated the results of the integer-order model and examined their difference with results of the fractional-order model.

Muostafa EL-Shaled et al (2011) took into account the fractional order SIRC linked to the evolution of influenza A sickness affecting the human population. The fundamental reproductive number R_o determines the model's qualitative dynamics.The asymptotic stability of disease-free and positive fixed points were thoroughly examined and numerical simulations were presented to support the findings. The rise in birth rate was found to increase with the proportion of susceptible.

Muhammad Kharis, Riza Afrifudin (2017) modeled Seasonal Influenza Epidemic In

Central Java With Treatment Action with (SIRT). They discussed mathematical model of seasonal influenza epidemic with treatment action. They got parameters from their survey at some districts in the central java in their research. The parameters include incubation period, infection period with treatment and without treatment and reinfection period. They analysed the existence of the equilibrium points and their stability. They also determined the basic reproductive number of the model. Lastly they did a numerical simulation to show the dynamics of the system based on the parameter's value that they got.

2.3 Conclusion:

This study therefore seeks to modify the SIRC model by Muostafa EL-Shaled et al (2011), by the inclusion of a therapy compartment instead of complications.Prevention should be prefered to remedy. Base on this fact, this research will simulate the dynamics of influenza virus and assess the efficacy of anti-viral drugs in managing the infection.

Chapter 3

Methodology

3.1 Introduction

In this research, we employ qualitative approach to look into the transmission dynamics of influenza A and the impact of anti-viral drugs. To complement our qualitative findings, a mathematical model known as SIRT is used to conduct quantitative analysis. This model is applied to simulate the spread of the virus and predict the potential impact of anti-viral drugs on reducing its transmission. The combination of these approaches allows us to gain a comprehensive understanding of the transmission dynamics of influenza A virus and the effectiveness of anti-viral drugs.

3.2 Model Development

This is a four compartmental model, SIRT model development. It is an SIR model with additional compartment for individuals in therapy T. The Therapy compartment was inculcated in order to assess the efficacy of antiviral drugs on the population subjects.

3.2.1 Assumptions

The model is developed on the basis of these assumptions:

- The population is dynamic .
- Recovery from illness does not confer full immunity.
- Every death is assumed to be a natural death.
- Infectious individuals may recover naturally or by treatment.

3.2.2 Compartmental Diagram

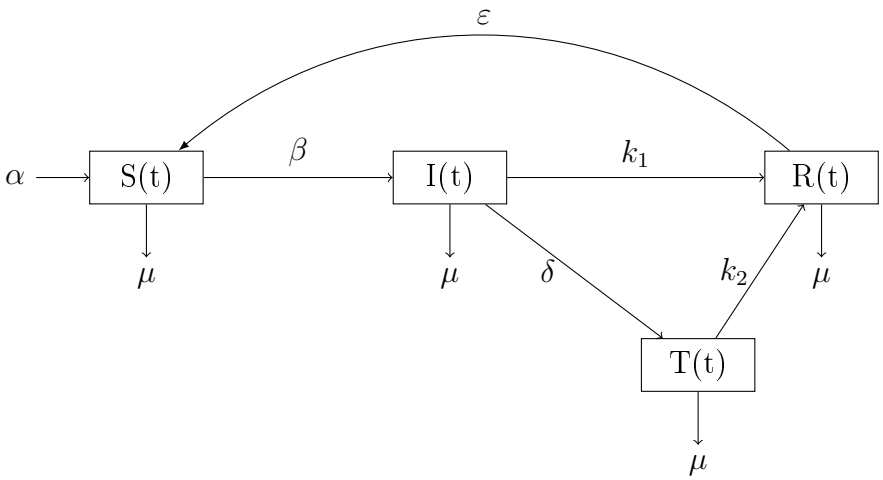

Figure 3.1: Compartmental model of SIRT

3.2.3 Definition of Parameters

Given the total population is 'N':

S(t): Number of susceptible individuals at time 't'

I(t): Number of infective persons at time 't'

R(t): Number of recovered individuals at time 't'

T(t): Number of persons under treatment at time 't'

Table 3.1: Definition of parameters

	Description
β	Contact rate between susceptible and infected individuals
κ_1	Natural recovery from infection
κ_2	Recovery rate from infection due to treatment
ε	Natural immunity loss
δ	Transition rate from infectivity to therapy
α	Recruitment rate of susceptible individual
μ	Rate of Natural death

3.2.4 Differential Equations

$\frac{dS}{dt} = \alpha N - \frac{\beta SI}{N} - \mu S + \varepsilon R$ . . . . . (1)

$\frac{dI}{dt} = \frac{\beta SI}{N} - (\mu + \delta + k_1)I$ . . . . . (2)

$\frac{dT}{dt} = \delta I - (\mu + k_2)T$ . . . . . (3)

$\frac{dR}{dt} = k_1 I + k_2 T - (\mu + \varepsilon)R$ . . . . . (4)

$S(t) + I(t) + T(t) + R(t) = N$ . . . . . (5)

3.2.5 Scalling differential equationa

$$S(t) + E(t) + I(t) + T^{\cdot}(t) + R(t) = N \quad \text{.....} \quad (5)$$

Dividing through by N

$$\frac{S(t)}{N} + \frac{I(t)}{N} + \frac{T^{\cdot}(t)}{N} + \frac{R(t)}{N} = 1 \quad \text{.....} \quad (6)$$

Let

$\frac{S(t)}{N} = s(t) \quad \Rightarrow S(t) = s(t)N \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (a)$

$\frac{I(t)}{N} = i(t) \quad \Rightarrow I(t) = i(t)N \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (b)$

$\frac{T(t)}{N} = t\cdot(t) \quad \Rightarrow T(t) = t\cdot(t)N \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (c)$

$\frac{R(t)}{N} = r(t) \quad \Rightarrow R(t) = r(t)N \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (d)$

Substituting equations (a),(b),(c),(d) in to equations 1,2,3,4 respectively

$\frac{N}{N}\frac{ds}{dt} = \frac{N}{N}(\alpha - \beta si - \mu s + \varepsilon r)$

$\frac{N}{N}\frac{di}{dt} = \frac{N}{N}(\beta si - (\mu + \delta + k_1)i)$

$\frac{N}{N}\frac{dt'}{dt} = \frac{N}{N}(\delta i - (\mu + k_2)t)$

$\frac{N}{N}\frac{dr}{dt} = \frac{N}{N}(k_1 i + k_2 t - (\mu + \varepsilon)r)$

Hence,

$\frac{ds}{dt} = \alpha - \beta si - \mu s + \varepsilon r \ldots\ldots\ldots\ldots (7)$

$\frac{di}{dt} = \beta si - (\mu + \delta + k_1)i \ldots\ldots\ldots\ldots (8)$

$\frac{dt'}{dt} = \delta i - (\mu + k_2)t \ldots\ldots\ldots\ldots (9)$

$\frac{dr}{dt} = k_1 i + k_2 t - (\mu + \varepsilon)r \ldots\ldots\ldots\ldots (10)$

$s(t) + i(t) + r(t) + t(t) \ldots\ldots\ldots\ldots\ldots (11)$

3.2.6 Positivity Analysis

The variables of the model formulated are non-negative since they describe the human population. Hence it is essential to prove that the solutions of the system's non-negative initial conditions will remain non-negative, $\forall t > 0$.

We, therefore, have the theorem:

Given that the initial condition of the systems are;

$S(0) > 0, \quad I(0) \geq 0, \quad T(0) \geq 0, \quad R(0) \geq 0$

The solutions $S(t)$, $I(t)$, $T(t)$, and $R(t)$ are non-negative for all $t > 0$

proof:

Considering the first equation using integrating factor;
$\frac{ds}{dt} = \alpha - \beta is - \mu s + \varepsilon r$

$\frac{ds}{dt} = \alpha - (\beta i - \mu)s + \varepsilon r$

$\frac{ds}{dt} = -(\beta i - \mu)s + \alpha + \varepsilon r$ integrating factor; $e^{\int_0^t (\beta i - \mu)dt}$

$s(t)e^{\int_0^t (\beta i - \mu)dt} = \int_0^t (\alpha + \varepsilon r)e^{\int_0^t (\beta i - \mu)dt}$

$s(t) = e^{-\int_0^t (\beta i - \mu)dt}\left[\int_0^t (\alpha + \varepsilon r)e^{\int_0^t (\beta i - \mu)dt}\right] > 0$

With the second equation;
$\frac{di}{dt} = \beta si - (\mu + \delta + k_1)i$

$\frac{di}{dt} = (\beta s - (\mu + \delta + k_1))i = \frac{di}{dt} = (\beta s - \mu - \delta - k_1))i$

integrating factor; $e^{\int_0^t \beta s - (\mu + \delta + k_1)dt}$

$i(t)e^{\int_0^t (\beta s - (\mu + \delta + k_1))dt} = \int_0^t 0.\ e^{\int_0^t (\beta s - (\mu + \delta + k_1))dt}$

$i(t) = \int_0^t 0dt$

$i(t) = C \geq 0$ 3rd equation;

$$\frac{dt'}{dt} = \delta i - (\mu + k_2)t$$

$$\frac{dt'}{dt} + (\mu + k_2)t = \delta i$$

integrating factor; $e^{\int_0^t (\mu+k_2)dt}$

$$t'(t)e^{\int_0^t (\mu+k_2)dt} = \int_0^t \delta i \ . \ e^{\int_0^t (\mu+k_2)dt} dt$$

$$t'(t) = \left[\int_0^t \delta i dt\right] \geq 0$$

Considering the last equation;

$$\frac{dr}{dt} = k_1 i + k_2 t - (\mu + \varepsilon)r$$

$$\frac{dr}{dt} + (\mu + \varepsilon)r = k_1 i + k_2 t$$

integrating factor; $e^{\int_o^t (\mu+\varepsilon)dt}$

$$r(t)e^{\int_o^t (\mu+\varepsilon)dt} = \left[\int_o^t (k_1 i + k_2 t)e^{\int_o^t (\mu+\varepsilon)dt}\right]$$

$$r(t) = \left[\int_o^t k_1 i dt\right] \geq 0$$

As shown above, in the various equations, it can be concluded therefore that all the solutions of the system with non-negative initial conditions will remain non-negative for all time $t > 0$

3.2.7 Disease Free Equilibrium Point

At the disease free equilibrium point $(s, i, r, t) = (s^*, i^*, r^*, t^{\cdot *})$

$s \geq 0, \quad e = 0, \quad i = 0, \quad r = 0, \quad t = 0$

$$\frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = \frac{dr}{dt} = \frac{dt^{\cdot}}{dt} = 0$$

Impling that,

$\frac{ds}{dt} = \alpha - \beta si - \mu s + \varepsilon r.....(i)$

$\Rightarrow \alpha - \beta si - \mu s + \varepsilon r = 0$

$\Rightarrow \alpha - \mu s = 0$

$\Rightarrow s = \frac{\alpha}{\mu}$

Hence disease free equilibrium point, $(s^*, i^*, t'^*, r^*) = (\frac{\alpha}{\mu}, 0, 0, 0)$

3.2.8 Endemic Equilibrium Point

Let $(\mu + \delta + k_1) = a_1, \qquad \mu + k_2 = a_2, \qquad \mu + \varepsilon = a_3$

Now,

$\alpha - \beta si - \mu s + \varepsilon r = o. \ldots\ldots (1)$

$\beta si - a_1 i = 0. \ldots\ldots (2)$

$\delta i - a_2 t = 0. \ldots\ldots (3)$

$k_1 i + k_2 t - a_3 r = 0. \ldots\ldots (4) \quad s^{**} = \frac{a_1}{\beta}$

from eqn (3)

$\delta i + a_2 t \qquad \Rightarrow i = \frac{a_2 t}{\delta} \ldots\ldots (*)$ from eqn (4)

$k_1 i + k_2 t = a_3 r$

$$\frac{k_1 i - a_3 r}{-k_2} = \frac{-k_2 t}{-k_2} \qquad \Rightarrow t = \frac{-k_1 i + a_3 r}{k_2} \ldots\ldots (*)$$

from eqn (1)

$$\frac{\alpha + \beta s i - \mu s}{-\varepsilon} = \frac{-\varepsilon r}{-\varepsilon} \qquad \Rightarrow r = \frac{\beta s i + \mu s - \alpha}{\varepsilon} \ldots\ldots (*)$$

putting , $\frac{a_1}{\beta}$, in place of , s, in the above;

$$r = \frac{\beta(\frac{a_1}{\beta}) i + \mu(\frac{a_1}{\beta}) - \alpha}{\varepsilon}$$

$$r = \frac{a_1 i + \frac{\mu a_1}{\beta} - \alpha}{\varepsilon} \qquad \Rightarrow \frac{(\frac{\beta a_1 i + \mu a_1 - \beta\alpha}{\beta})}{\varepsilon}$$

$$r = \frac{\beta a_1 i + \mu a_1 - \beta\alpha}{\beta\varepsilon} \ldots\ldots (*)$$

Again from eqn (4)

$$k_1 \left(\frac{a_2 t}{\delta}\right) + k_2 t - a_3 \left[\frac{\beta a_1 i + \mu a_1 - \beta\alpha}{\beta\varepsilon}\right] = 0$$

$$\frac{k_1 a_2 t}{\delta} + k_2 t - \frac{a_3}{\beta\varepsilon} \left[\frac{a_1 \beta a_2 t}{\delta\varepsilon} + \mu a_1 - \alpha\beta\right] = 0$$

$$\frac{k_1 a_2 t}{\delta} + k_2 t - \frac{a_1 a_2 a_3}{\delta\varepsilon} - \frac{\mu a_1 a_3}{\beta\varepsilon} + \frac{\alpha a_3}{\varepsilon} = 0$$

$$t \left[\frac{k_1 a_2}{\delta} + k_2 - \frac{a_1 a_2 a_3}{\delta\varepsilon}\right] = \frac{a_1 a_3 \mu}{\beta\varepsilon} - \frac{\alpha a_3}{\varepsilon}$$

$$t\left[\frac{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}{\delta\varepsilon}\right] = \frac{1}{\varepsilon}\left[\frac{a a_3 \mu - \alpha\beta a_3}{\beta}\right]$$

$$t = \frac{1}{\varepsilon}\left[\frac{a_1 a_3 \mu - \alpha\beta a_3}{\beta}\right] \times \left[\frac{\delta\varepsilon}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right]$$

$$t = \frac{\delta(a_1 a_3 \mu - \alpha\beta a_3)}{\beta(\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3)}$$

$$t^{**} = \frac{\delta}{\beta}\left[\frac{a_1 a_3 \mu - \alpha\beta a_3}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right]$$

$$\text{From } i = \frac{a_2 t}{\delta} \qquad \Rightarrow \frac{a_2\delta}{\beta\delta}\left[\frac{a_1 a_3 \mu - \alpha\beta a_3}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right]$$

$$i^{**} = \frac{a_2}{\beta}\left[\frac{a_1 a_2 \mu - \alpha\beta a_3}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right]$$

$$r^{**} = \frac{k_1 i - k_2 t}{a_3}, \text{ where}$$

$$i = \frac{a_2}{\beta}\left[\frac{a_1 a_2 \mu - \alpha\beta a_3}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right] \text{ and}$$

$$t = \frac{\delta}{\beta}\left[\frac{a_1 a_3 \mu - \alpha\beta a_3}{\varepsilon k_1 a_2 + \delta\varepsilon k_2 - a_1 a_2 a_3}\right]$$

$$s^{**} = \tfrac{\mu+\delta+\kappa_1}{\beta}$$

$$t^{**} = \tfrac{\delta}{\beta}\left[\frac{(\mu+\delta+\kappa_1)(\mu+\varepsilon)\mu - \alpha\beta(\mu+\varepsilon)}{\varepsilon\kappa_1(\mu+\kappa_2) + \delta\varepsilon\kappa_2 - [(\mu+\delta+\kappa_1)(\mu+\kappa_2)(\mu+\varepsilon)]}\right]$$

$$i^{**} = \tfrac{\mu+\kappa_2}{\beta}\left[\frac{(\mu+\delta+\kappa_1)(\mu+\kappa_2)\mu - \alpha\beta(\mu+\varepsilon)}{\varepsilon\kappa_1(\mu+\kappa_2) + \delta\varepsilon\kappa_2 - [(\mu+\delta+\kappa_1)(\mu+\kappa_2)(\mu+\varepsilon)]}\right]$$

$$r^{**} = \frac{\kappa_1 a_2(a_1 a_2 \mu - \alpha\beta a_3) - \kappa_2\delta(a_1 a_2 \mu - \alpha\beta a_3)}{\beta a_3(\varepsilon\kappa_1 a_2 + \delta\kappa_2 - a_1 a_2 a_3)}$$

$r^{**} =$

$$\frac{\kappa_1(\mu+\kappa_2)\left[(\mu+\delta+\kappa_1)(\mu+\kappa_2)\mu-\alpha\beta(\mu+\varepsilon)\right]-\kappa_2\delta\left[(\mu+\delta+\kappa_1)(\mu+\kappa_2)\mu-\alpha\beta(\mu+\varepsilon)\right]}{\beta(\mu+\varepsilon)\left[\varepsilon\kappa_1(\mu+\kappa_2)\delta\varepsilon\kappa_2-(\mu+\delta+\kappa_1)(\mu+\kappa_2)(\mu+\varepsilon)\right]}$$

3.2.9 Basic Reproductive Number

Considering the disease classes:

$\frac{di}{dt} = \beta si - (\mu+\delta+k_1)i$

$\frac{dt'}{dt} = \delta i - (\mu+\kappa_2)t$

Where βsi is the contact rates for infectivity

$$\mp = \begin{bmatrix} \beta si \\ 0 \end{bmatrix}^{....H}_{....G}$$

and

$$\nu = \begin{bmatrix} -(\mu+\delta+\kappa_1)i \\ \delta i-(\mu+k_2)t \end{bmatrix} = \begin{bmatrix} -ai \\ \delta i - bt \end{bmatrix}^{....H}_{....G}$$

Where $a = (\mu+\delta+\kappa_1)$ and $b = (\mu+\kappa_2)$

Let the first rows from each of the matrices $\mp$ and ν be H and the second rows be G.

From $\mp$,

$$F = \begin{bmatrix} \frac{\delta H}{\delta i} & \frac{\delta H}{\delta t\cdot} \\ \frac{\delta G}{\delta i} & \frac{\delta G}{\delta t\cdot} \end{bmatrix} = \begin{bmatrix} \beta s & 0 \\ 0 & 0 \end{bmatrix}$$

From ν,

$$V = \begin{bmatrix} \frac{\delta H}{\delta i} & \frac{\delta H}{\delta t\cdot} \\ \frac{\delta G}{\delta i} & \frac{\delta G}{\delta t\cdot} \end{bmatrix} = \begin{bmatrix} -a & 0 \\ \delta & -b \end{bmatrix}$$

$$V^{-1} = \frac{1}{ab}\begin{bmatrix} -b & 0 \\ \\ -\delta & -a \end{bmatrix} = \begin{bmatrix} -\frac{1}{a} & 0 \\ \\ -\frac{\delta}{ab} & -\frac{1}{b} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \beta s & 0 \\ \\ 0 & 0 \end{bmatrix}\begin{bmatrix} -\frac{1}{a} & 0 \\ \\ -\frac{\delta}{ab} & -\frac{1}{b} \end{bmatrix} = \begin{bmatrix} -\frac{\beta s}{a} & 0 \\ \\ 0 & 0 \end{bmatrix}$$

$$FV^{-1}(s^*, i^*, t'^*, r^*) = \begin{bmatrix} -\frac{\beta(1)}{a} & 0 \\ \\ 0 & 0 \end{bmatrix} = \begin{bmatrix} -\frac{\beta}{a} & 0 \\ \\ 0 & 0 \end{bmatrix}$$

$$\rho FV^{-1} = \begin{vmatrix} -\frac{\beta}{a} - \lambda & 0 \\ \\ 0 & -\lambda \end{vmatrix} = -\lambda\left[-(\frac{\beta}{a} + \lambda)\right] = 0$$

$\lambda_1 = 0$ and $\lambda_2 = \left|\frac{\beta}{a_1}\right| = \frac{\beta}{(\mu+\delta+k_1)}$

3.2.10 Sensitivity Analysis

Hence the Basic Reproduction number $R_o = \rho FV^{-1} = \frac{\beta}{(\mu+\delta+k_1)}$

$R_o = \rho FV^{-1} = \frac{\beta}{(\mu+\delta+k_1)}$

Sensitivity Analysis Using the forward sensitivity index

$$R_o = \frac{\beta}{(\mu + \delta + k_1)}$$

$$\psi_\beta^{R_o} = \frac{\partial R_o}{\partial \beta} . \frac{\beta}{R_o}$$

$$\frac{\partial R_o}{\partial \beta} = \frac{1}{(\mu + \delta + k_1)}$$

$$\psi_\beta^{R_o} = \frac{1}{(\mu + \delta + k_1)} \cdot \frac{\beta(\mu + \delta + k_1)}{\beta}$$

$$\psi_\beta^{R_o} = 1$$

$$\psi_\mu^{R_o} = \frac{\partial R_o}{\partial \mu} . \frac{\mu}{R_o}$$

Let $u = \beta \qquad v = (\mu + \delta + k_1)$

$$u' = 0 \qquad v' = 1$$

$$\frac{vu' - uv'}{v^2}$$

$$\frac{(\mu + \delta + k_1)(0) - \beta(1)}{(\mu + \delta + k_1)^2}$$

$$\psi_\mu^{R_o} = \frac{-\beta}{(\mu + \delta + k_1)^2} \cdot \frac{\mu(\mu + \delta + k_1)}{\beta}$$

$$\psi_\mu^{R_o} = \frac{-\mu}{(\mu + \delta + k_1)}$$

$$\psi_\delta^{R_o} = \frac{\partial R_o}{\partial \delta} \cdot \frac{\delta}{R_o}$$

$$\Rightarrow \frac{0 - \beta}{(\mu + \delta + k_1)^2} \cdot \frac{\delta(\mu+\delta+\kappa_1)}{\beta} = \frac{-\delta}{(\mu+\delta+\kappa)} \; \psi_{k_1}^{R_o} = \frac{-\beta}{(\mu + \delta + k_1)^2} \cdot \frac{k_1(\mu + \delta + k_1)}{\beta}$$

$$\psi_{k_1}^{R_o} = \frac{-k_1}{(\mu + \delta + k_1)}$$

3.2.11 Stability Analysis at disease free equilibrium point

Linearizing by the Jacobian matrix:

$$J(s^*, i^*, t^*, r*) = \begin{bmatrix} \frac{\partial f}{\partial s} & \frac{\partial f}{\partial i} & \frac{\partial f}{\partial t} & \frac{\partial f}{\partial r} \\ \frac{\partial g}{\partial s} & \frac{\partial g}{\partial i} & \frac{\partial g}{\partial t} & \frac{\partial g}{\partial r} \\ \frac{\partial h}{\partial s} & \frac{\partial h}{\partial i} & \frac{\partial h}{\partial t} & \frac{\partial h}{\partial r} \\ \frac{\partial k}{\partial s} & \frac{\partial k}{\partial i} & \frac{\partial k}{\partial t} & \frac{\partial k}{\partial r} \end{bmatrix}$$

$J(s^*, i^*, t^*, r^*)$

$$= \begin{bmatrix} -(\beta i + \mu)s & -\beta s & 0 & \varepsilon \\ \beta i & \beta\delta - (\mu + \delta + k_1) & 0 & 0 \\ 0 & \delta & -(\mu + k_2) & 0 \\ 0 & k_1 & k_2 & -(\mu + \varepsilon) \end{bmatrix}$$

$J(s^*, i^*, t^*, r^*)$

$$= \begin{bmatrix} -\mu & -\beta s & 0 & \varepsilon \\ 0 & \beta - (\mu + \delta + k_1) & 0 & 0 \\ 0 & \delta & -(\mu + k_2) & 0 \\ 0 & k_1 & k_2 & -(\mu + \varepsilon) \end{bmatrix}$$

Let $\beta - (\mu + \delta k_1) = a \qquad \mu + k_2 = b \qquad \mu + \varepsilon = c$

$$J(s^*, i^*, t^*, r^*) = \begin{bmatrix} -\mu & -\beta & 0 & \varepsilon \\ 0 & a & 0 & 0 \\ 0 & \delta & -b & 0 \\ 0 & k_1 & k_2 & -c \end{bmatrix}$$

$$\left| J - \lambda I \right| = \begin{vmatrix} -\mu - \lambda & -\beta & 0 & \varepsilon \\ 0 & a - \lambda & 0 & 0 \\ 0 & \delta & -b - \lambda & 0 \\ 0 & k_1 & k_2 & -c - \lambda \end{vmatrix}$$

$$-\mu-\lambda\begin{vmatrix} a-\lambda & 0 & 0 \\ \delta & -b-\lambda & 0 \\ k_1 & k_2 & -c-\lambda \end{vmatrix}+\beta\begin{vmatrix} 0 & 0 & 0 \\ 0 & -b-\lambda & 0 \\ 0 & k_2 & -c-\lambda \end{vmatrix}+$$

$$0\begin{vmatrix} 0 & a-\lambda & 0 \\ 0 & -\delta & 0 \\ 0 & k_2 & -c-\lambda \end{vmatrix}-\varepsilon\begin{vmatrix} 0 & a-\lambda & 0 \\ 0 & -\delta & -b-\lambda \\ 0 & k_1 & k_2 \end{vmatrix}=0$$

$$-(\mu+\lambda)(a-\lambda)\begin{vmatrix} -b-\lambda & 0 \\ k_2 & -c-\lambda \end{vmatrix}=0$$

$$\Rightarrow -(\mu+\lambda)(a-\lambda)(-b-\lambda)(-c-\lambda)=0$$

$$\Rightarrow (-\mu-\lambda)[(a-\lambda)(-b-\lambda)(-c-\lambda)]=0$$

$$\lambda_1=-\mu \quad \lambda_2=a \quad \lambda_3=-b \quad \lambda_1=-c$$

$$\Rightarrow \lambda_1=-\mu \quad \lambda_2=\beta-(\mu+\delta+\kappa_1) \quad \lambda_3=-(\mu+\kappa_2) \quad \lambda_1=-(\mu+\varepsilon)$$

From the values of the parameter, we know that $\beta<(\mu+\delta+\kappa_1)$. Hence λ_2 is also negative

Therefore, the system is **stable**

3.2.12 Stability at the Endemic equilibrium point

since

$J(s^*,i^*,t^*,r^*)=$

$$\begin{bmatrix} \frac{\partial u}{\partial s} & \frac{\partial u}{\partial i} & \frac{\partial u}{\partial t'} & \frac{\partial u}{\partial r} \\ \frac{\partial v}{\partial s} & \frac{\partial v}{\partial i} & \frac{\partial v}{\partial t'} & \frac{\partial v}{\partial r} \\ \frac{\partial w}{\partial s} & \frac{\partial w}{\partial i} & \frac{\partial w}{\partial t'} & \frac{\partial w}{\partial r} \\ \frac{\partial z}{\partial s} & \frac{\partial z}{\partial i} & \frac{\partial z}{\partial t'} & \frac{\partial z}{\partial r} \end{bmatrix}$$

At the endemic state $(s, i, t', r) = (s^{**}, i^{**}, t'^{**}, r^{**})$

$$= \begin{bmatrix} -(\beta i + \mu) & -\beta s & 0 & \varepsilon \\ \beta i & \beta s - (\mu + \delta + \kappa_1) & 0 & 0 \\ 0 & \delta & -(\mu + \kappa_2) & 0 \\ 0 & \kappa_1 & \kappa - 2 & -(\mu + \varepsilon) \end{bmatrix}$$

Let $s^{**} = x, \quad i^{**} = y, \quad t'^{**} = p, \quad r^{**} = q$

$$\left| J(s^{**}, i^{**}, t'^{**}, r^{**}) - \lambda I \right| =$$

$$\begin{vmatrix} -(\beta y + \mu) - \lambda & -\beta x & 0 & \varepsilon \\ \beta y & \beta x - (\mu + \delta + \kappa_1) - \lambda & 0 & 0 \\ 0 & \delta & -(\mu + \kappa_2) - \lambda & 0 \\ 0 & \kappa_1 & \kappa_2 & -(\mu + \varepsilon) - \lambda \end{vmatrix} = 0$$

Let $z_1 = \beta y + \mu, \qquad z_2 = \beta x - (\mu + \delta + \kappa_1),$

$z_3 = (\mu + \kappa_2), \qquad z_4 = (\mu + \varepsilon)$

$$\begin{bmatrix} -z_1 & \beta x & 0 & \varepsilon \\ \beta y & z_2 & 0 & 0 \\ 0 & \delta & -z_3 & 0 \\ 0 & \kappa_1 & \kappa_2 & -z_4 \end{bmatrix}$$

$$\left|J - I\lambda\right| = \begin{vmatrix} -z_1 - \lambda & \beta x & 0 & \varepsilon \\ \beta y & z_2 - \lambda & 0 & 0 \\ 0 & \delta & -z_3 - \lambda & 0 \\ 0 & \kappa_1 & \kappa_2 & -z_4 - \lambda \end{vmatrix}$$

$$(-z_1 - \lambda) \begin{vmatrix} z_2 - \lambda & 0 & 0 \\ \delta & -z_3 - \lambda & 0 \\ \kappa_1 & \kappa_2 & -z_4 - \lambda \end{vmatrix}$$

$$-\beta y \begin{vmatrix} \beta x & 0 & \varepsilon \\ \delta & z_3 - \lambda & 0 \\ \kappa_1 & \kappa_2 & -z_4 - \lambda \end{vmatrix} \quad = \quad 0$$

$$(-z_1-\lambda)(z_2-\lambda)(-z_3-\lambda)(-z_4-\lambda)-\beta y[\beta x(-z_3-\lambda)(-z_4-\lambda)+\varepsilon*(\delta*\kappa_2+z_3\kappa_1+\lambda*\kappa_1)] = 0$$

Now, distributing the factors and simplifying further:

$$(-1)^4(z_1z_2z_3z_4+z_1z_2z_3\lambda+z_1z_2z_4\lambda+z_1z_2\lambda^2-z_1z_3z_4\lambda-z_1z_3\lambda^2-z_1z_4\lambda^2-z_1\lambda^3-z_2z_3z_4\lambda-z_2z_3\lambda^2-z_2z_4\lambda^2-z$$

$$(z_1z_2z_3z_4+z_1z_2z_3\lambda+z_1z_2z_4\lambda+z_1z_2\lambda^2-z_1z_3z_4\lambda-z_1z_3\lambda^2-z_1z_4\lambda^2-z_1\lambda^3-z_2z_3z_4\lambda-z_2z_3\lambda^2-z_2z_4\lambda^2-z_2\lambda^3+$$

Now, move all terms to one side of the equation:

$$z_1z_2z_3z_4+z_1z_2z_3\lambda+z_1z_2z_4\lambda+z_1z_2\lambda^2-z_1z_3z_4\lambda-z_1z_3\lambda^2-z_1z_4\lambda^2-z_1\lambda^3-z_2z_3z_4\lambda-z_2z_3\lambda^2-z_2z_4\lambda^2-z_2\lambda^3+z$$

$$z_1z_2z_3z_4+z_1z_2z_3\lambda+z_1z_2z_4\lambda+z_1z_2\lambda^2-z_1z_3z_4\lambda-z_1z_3\lambda^2-z_1z_4\lambda^2-z_1\lambda^3-z_2z_3z_4\lambda-z_2z_3\lambda^2-z_2z_4\lambda^2-z_2\lambda^3+z$$

$$\lambda^4+(-z_1-z_2+z_3+z_4)\lambda^3+(z_1Z_2-z_1z_3-z_1z_4-z_2z_3-z_2z_4+z_3z_4+\beta^2xy)\lambda^2+(z_1z_3+z_1z_2z_4-z_1z_3z_4-z_2z_3z_4-$$

From the auxiliary equation above, the coefficients of λ^4 and λ^3 are positive values while that of λ^2, λ and the constant coefficient are of negative values.

Hence, by the Routh-Hurwitz criterion the system is **unstable** .

3.3 Data Collection

The table below provides a summary of sensitivity analysis results for various model parameters and are listed along with their sensitivity index and corresponding values. The sensitivity index of the various parameters were computed previously. Sensitivity index indicates the magnitude and direction of parameter impact on the model output. The sources of the parameter values are indicated in the table as well.

Table 3.2: Parameter values, Sensitivity index and their respective Sources

Parameter	Sensitivity Index	Value	Source
α	0	0.0088	Ankamu Daniel et al,2015
β	+ 1	0.1508	Ankamu Daniel et al,2012
μ	-0.109	0.0044	Ankamu Daniel et al,2012
κ_1	-0.0004	0.00015	Muhammad Khariset al,2017
κ_2	0	0.36	Muhammad Khariset al,2017
ε	0	0.00274	Muhammad Khariset al,2017
δ	-0.9888	0.4	Rwezanra et al, 2009

3.3.1 Numerical Solution

USING THE FORWARD FINITE DIFFERENCE METHOD

$\frac{dS}{dt} = \alpha N - \frac{\beta SI}{N} - \mu S + \varepsilon R$ . . . . . (1)

$\frac{dI}{dt} = \frac{\beta SI}{N} - (\mu + \delta + k_1)I$ . . . . . (2)

$\frac{dT}{dt} = \delta I - (\mu + k_2)T$ . . . . . (3)

$\frac{dR}{dt} = k_1 I + k_2 T - (\mu + \varepsilon)R$ . . . . . (4)

The forward finite difference formula is commonly used to approximate the derivative of

a function at a given point. The formula is given by:

$$f'(x) \approx \frac{(f(x+h) - f(x))}{h}$$

The Taylor series expansion of a function f(x) around the point x is given by:

$$f(x+h) = f(x) + hf'(x) + \frac{h^2}{2}f''(x) + ...$$

$$f(x+h) - f(x) = hf'(x) + O(h) + ...$$

DISCRETIZING DERIVATIVES

First, we discretize the equations in time using the forward difference for the time derivative:

$$\frac{dS}{dt} \approx \frac{S_{i+1} - S_i}{\triangle t} \quad .....(a)$$

$$\frac{dI}{dt} \approx \frac{I_{i+1} - I_i}{\triangle t} \quad .....(b)$$

$$\frac{dT}{dt} \approx \frac{T_{i+1} - T_i}{\triangle t} \quad .....(c)$$

$$\frac{dR}{dt} \approx \frac{R_{i+1} - R_i}{\triangle t} \quad .....(d)$$

Substituting equations (a),(b),(c) and (d) into the differential equations;

$$(1) \qquad \frac{(S_{i+1} - S_i)}{\triangle t} = \alpha N - \frac{\beta S_i I_i}{N} - \mu S_i + \varepsilon R_i$$

$$(2) \qquad \frac{(I_{i+1} - I_i)}{\triangle t} = \frac{\beta S I}{N} - (\mu + \delta + \kappa_1)$$

$$(3) \qquad \frac{(T_{i+1} - T_i)}{\triangle t} = \delta T_i - (\mu + \kappa_2) T_i$$

$$(4) \qquad \frac{(R_{i+1} - R_i)}{\triangle t} = \kappa_1 I_i + \kappa_2 T_i - (\mu + \varepsilon) R_i$$

The discritized equations becomes a system of algebraic equations as follows:

$$S_{i+1} = S_i + \triangle t \left[\alpha N - \frac{\beta S_i I_i}{N} - \mu S_i + \varepsilon R_i \right]$$

$$I_{i+1} = I_i + \triangle t \left[\frac{\beta S_i I_i}{N} - (\mu + \delta + \kappa_1) I_i \right]$$

$$T_{i+1} = T_i + \triangle t \left[\delta T - (\mu + \kappa_2) T_i \right]$$

$$R_{i+1} = R_i + \triangle t \left[\kappa_1 I + \kappa_2 T - (\mu + \varepsilon) R_i \right]$$

Chapter 4

Results Analysis and Discussion

4.1 Numerical Simulation

From the analysis of the model, the following were deduced: (i)At disease free equilibrium

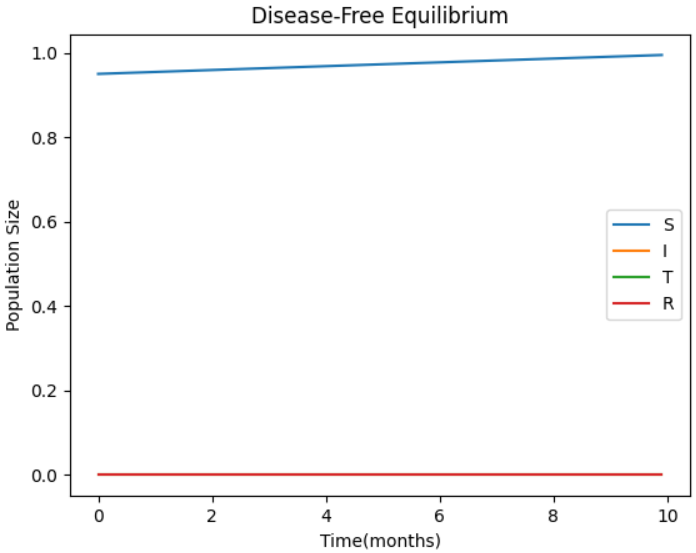

Figure 4.1: Numerical Simulation at Disease free Equilibruim

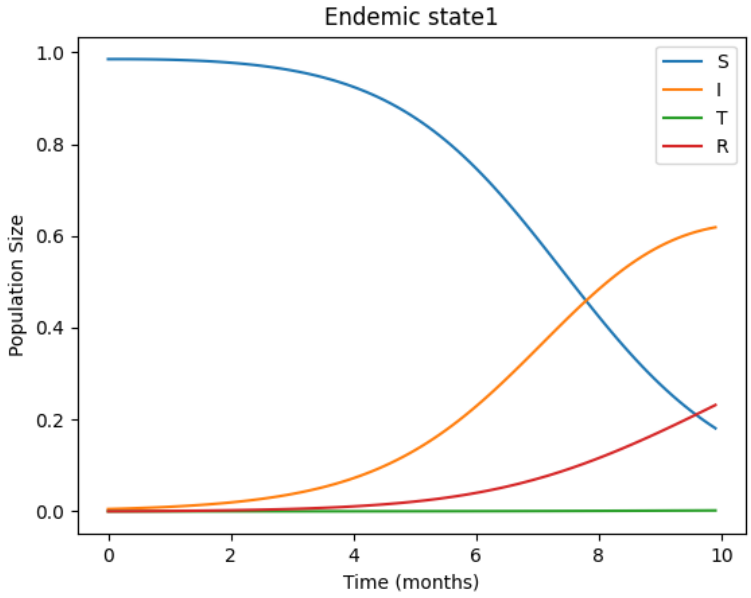

Figure 4.2: Numerical Simulation at Endemic Equilibruim

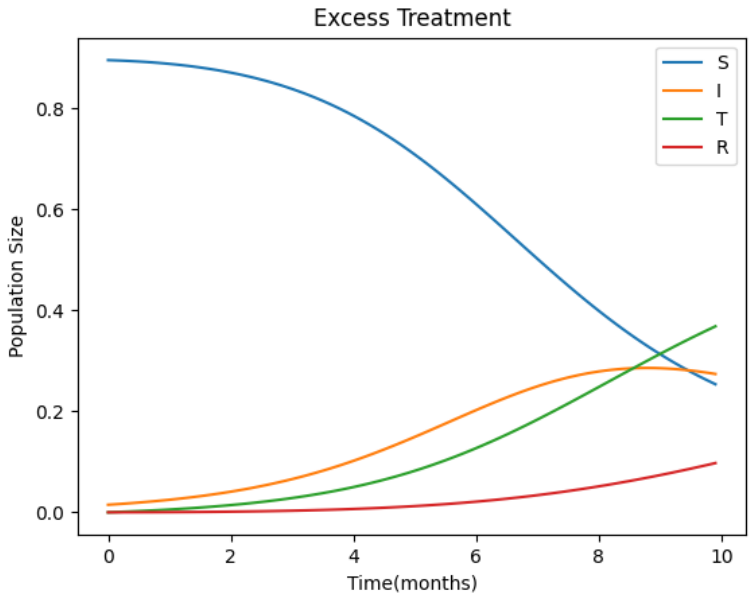

Figure 4.3: Simulation of Treatment-induce Recovery

4.2 Results Analysis

The basic reproductive number,R0 < 1 at the disease-free quilibrium point. Implying that the population is predominantly free from the infection, there is no spread of the virus. At the endemic equilibrium point, Ro > 1, implying that the infection is going to persist over time.The basic reproductive number, Ro depends on transmission rate, natural death and recovery rate, and the rate at which infectives transition into treatment.

Using the parameter values, we simulated for a period of 10 months for interaction between susceptible, infected, treated and recovered. From graph 1. It can be observed that from time 0 to the 10th months, the proportion of the susceptible class keep increasing while infected, treated and recovered compartment converges towards zero over the period.

From figure 3.3: It can be seen that susceptible decreases exponentiallly. Infected class increases from drastically, Recovered increases, as well as Treated compartment at the end of 10 months.

In figure 3.4: We studied the model behavior when treatment(δ) is smaller than natural recovery rate $\kappa_1(\delta < \kappa_1)$ by varying $\kappa_1 = 0.1 \quad \delta = 0.01 \quad \kappa_2 = 0.05$.
We observed that infected class slowly decreased from 0.05 to 0.03, Recovery increases from 0.005 to 0.05, Susceptible increased from 0.10 to 0.13 and Treated class remain zero throughout the 10 months.

In figure 3.5: We also studied the behavior of the model. When treatment rate(δ) is greater than natural recovery rate (κ_1) $\delta > \kappa_1$ by varying $\delta = 3.0$ and $\kappa_1 = 0.01$. We observed that treatment compartment increased rapidly from 0.000 to 0.026 for the first one month, two weeks and reduced slowly for the next 4 months, 2 weeks and from 0.026 to 0.012 and still reduced very slowly from 0.012 to 0.002 for the next 4 months.

Infected class initially reduced rapidly from 0.050 to 0.004 for the first 2 months, still reduces slowly to zero for the next 2 months stayed at zero for the rest of the time.

Susceptible class increased from 0.100 to 0.160 over the time.
Recovery slowly increased for 1st week and rapidly increased for next 3 months, 3 weeks and increased from 0.021 to 0.050 for the rest of the time.
We noted from graph 3 that both treatment and infected classes decreases to zero very fast while treated class stay at zero and infected is far greater than zero graph 4.

From figure 3.2: we can say that the susceptible increases from 0.10, the infected decreases from 0.05 to 0, the treated and the recovered class remains at equilibrium as time increases.

From figure 3.3, we can see that susceptible decreases from 0.6 to 0.35, the infected class increases from 0.05 to 0.25, the treated class increases from 0 to 0.3 and the recovered class increases from 0 to 0.20 as time increases. From graph 3, the susceptible increases from 0.10 to 0.14, the infected class decreases from 0.05 to 0.04, the treated class remains at equilibrium and recovered class increases from 0.005 to 0.06 as the parameters varies.

From figure 3.5, the susceptible increases from 0.10 to 0.175, the infected class decreases from 0.050 to 0 and treated class increases from 0 to 0.25 and decreases to 0 and the recovered class increases from 0.001 to 0.050 as the parameter varies.

4.3 Discussion

The research presents a four-compartmental SIRT model, an extension of the classical SIR model, aiming to assess the efficacy of antiviral drugs on the population during an infectious disease outbreak. The model assumes a homogeneous mixture of people, constant population size, and recovery from the disease does not provide full immunity.

It uses differential equations as well as numerical simulations to determine the rate of change in each compartment, including contact rates, recovery rates, transition rates, natural immunity loss rates, and recruitment rates. The model derives the disease-free equilibrium point, where all individuals are susceptible and no one is infected, under treatment, or recovered. The basic reproductive number (Ro) is determined, and the stability analysis at the disease-free equilibrium point indicates stability. The stability analysis at the endemic equilibrium point depends on parameter values. The study provides a mathematical model for understanding infectious disease dynamics in the presence of antiviral treatment, exploring the disease-free and endemic equilibrium states, basic reproductive number, and sensitivity to parameter variations.

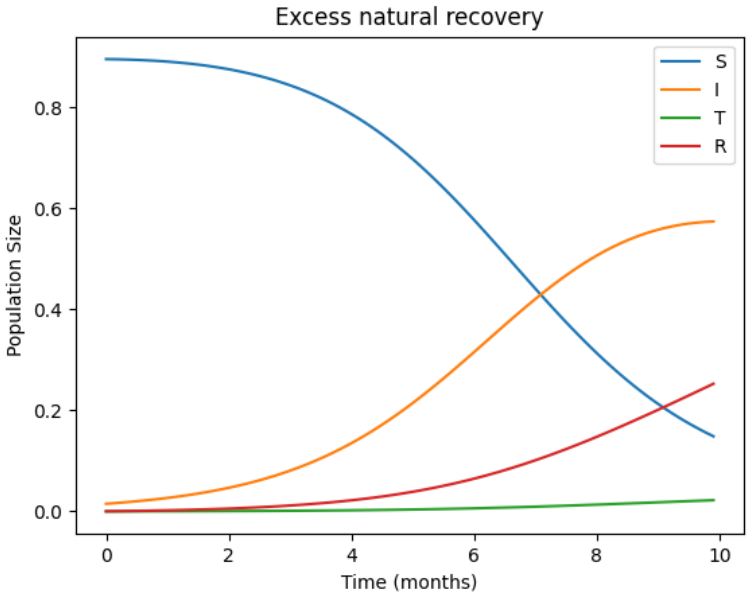

Figure 4.4: Simulation of Natural Recovery

Chapter 5

Conclusion And Recommendations

5.1 Conclusion

The research work presents a four-compartmental model, the SIRT model, to assess the efficacy of antiviral drugs on a population subjected to an infectious disease. The model is based on several assumptions, including a constant population size, natural death, and the possibility of recovery through natural means or treatment. The model's differential equations and stability analysis were derived to understand the dynamics of the disease in different compartments.

Based on the analysis of the model, the following conclusions can be drawn:

Disease-Free Equilibrium: The model predicts a disease-free equilibrium point where the entire population is susceptible, and no infections are present. This equilibrium point is stable, indicating that without external factors, the disease would not spread in the population.

Endemic Equilibrium: The model also identifies an endemic equilibrium point where the disease persists in the population. The stability analysis shows that the endemic

equilibrium can be stable, indicating that the disease can persist over time with the right conditions.

Basic Reproductive Number (Ro): The basic reproductive number (Ro) was calculated to measure the disease's potential to spread in a completely susceptible population. The Ro value helps to understand the disease's transmission dynamics and provides valuable insights into control measures.

The SIRT model presented in the research work provides valuable information for understanding infectious disease dynamics and assessing the efficacy of antiviral drugs. By utilizing the model's findings and recommendations, public health efforts should be optimized to control and prevent the spread of infectious diseases more effectively.

5.2 Recommendations

Antiviral Drug Efficacy: The model's inclusion of a therapy compartment allows for the assessment of antiviral drugs' efficacy on the population. Further research and clinical trials should be conducted to evaluate and validate the effectiveness of specific antiviral drugs in reducing the disease's impact on the population.

Public Health Interventions: The model provides valuable information on the disease's transmission dynamics and stability points. Public health authorities can utilize this information to design targeted intervention strategies to control and prevent the disease's spread. These interventions may include vaccination campaigns, early detection and treatment, and public health awareness programs.

Sensitivity Analysis: The sensitivity analysis conducted in the research work highlights the impact of individual parameters on the disease's transmission. This information can guide policymakers and healthcare professionals in prioritizing and allocating resources

effectively to mitigate the disease's impact.

Continuous Surveillance: Given the dynamic nature of infectious diseases, continuous surveillance and data collection are crucial for accurate modeling and decision-making. Governments and health organizations should establish robust surveillance systems to monitor disease prevalence, transmission rates, and antiviral drug effectiveness.

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