

**KWAME NKRUMAH UNIVERSITY OF SCIENCE AND
TECHNOLOGY, KUMASI**



**The analysis of vaccination and treatment of Measles diseases
Described by a Fractional order SIR epidemiological model**

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A THESIS SUBMITTED TO THE DEPARTMENT OF MATHEMATICS,
KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY IN
PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD
OF M.PHIL APPLIED MATHEMATICS

May 12, 2014

Declaration

I hereby declare that this submission is my own work towards the award of the M. Phil degree and that, to the best of my knowledge, it contains no material previously published by another person nor material which had been accepted for the award of any other degree of the university, except where due acknowledgment had been made in the text.

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Dedication

I dedicate this work to God Almighty and my family.

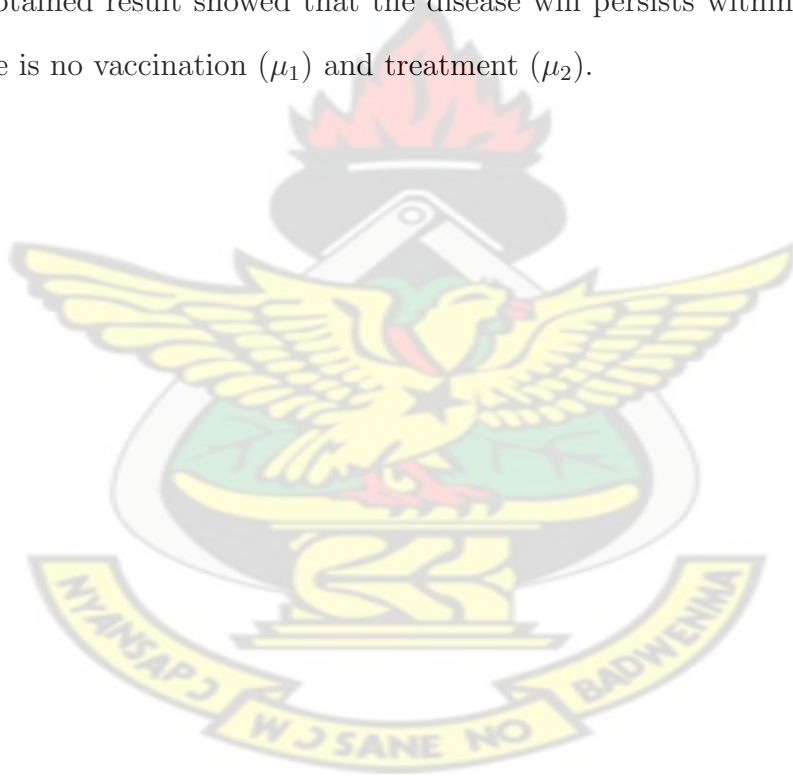
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Abstract

In this work, a fractional order SIR model with vaccination (μ_1) and treatment (μ_2) is formulated to describe measles disease. Firstly, the method of solution shows that the model possess non-negative solutions as desired in any population dynamics. The basic reproductive number is established, and a thorough analysis is carried out to study the stability of the equilibrium points. Numerical solutions are presented to illustrate the stability analysis using Generalized Euler method. Graphical results are presented and discussed.

The obtained result showed that the disease will persists within the population if there is no vaccination (μ_1) and treatment (μ_2).



Acknowledgments

My humble gratitude goes to God Almighty for his guidance and confidence that has enabled me to complete this work successfully.

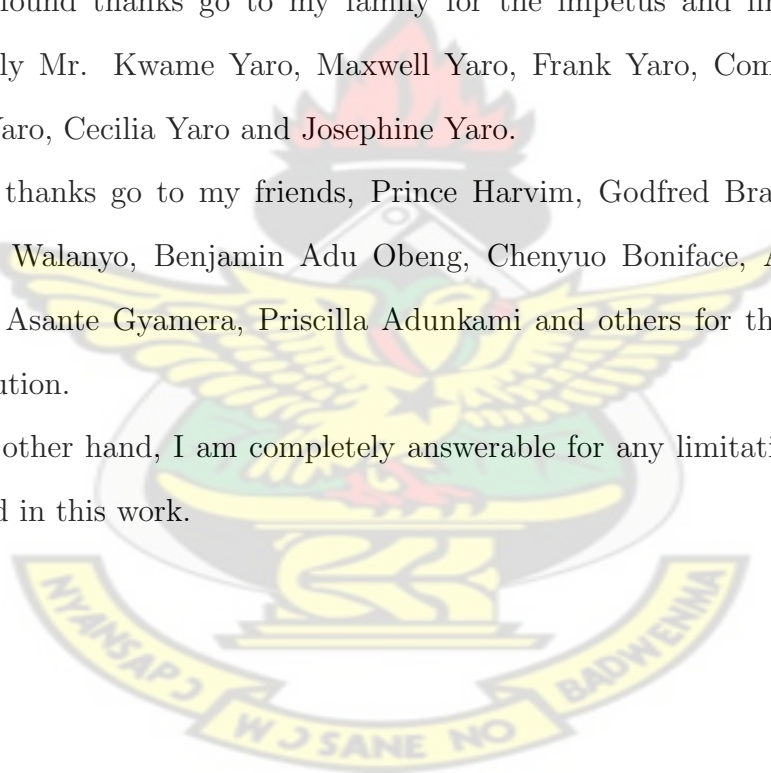
I wish to express my most heartily gratitude and thanks to my supervisor, Dr. A. Y. Omari-Sasu for the valuable and useful discussion which invaluablely improved this thesis.

I am also very grateful to Dr. Peter Yirenkyi and Nana Kena Frimpong for their valuable suggestions and corrections for this thesis.

My profound thanks go to my family for the impetus and financial support, especially Mr. Kwame Yaro, Maxwell Yaro, Frank Yaro, Comfort Yaro, Mrs. Hawa Yaro, Cecilia Yaro and Josephine Yaro.

Special thanks go to my friends, Prince Harvim, Godfred Bratuo, Akuamoah Saviour Walanyo, Benjamin Adu Obeng, Chenyuo Boniface, Afrifa Twumasi, Samuel Asante Gyamera, Priscilla Adunkami and others for their support and contribution.

On the other hand, I am completely answerable for any limitation that may be detected in this work.



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Chapter 1

Introduction

1.1 Background of study

Mathematics has shown to be a valuable tool in epidemiology. Mathematical modeling in epidemiology provides understanding of the basic mechanisms that influence on the theory and practice of diseases and in the process it suggests control strategies.

Worldwide, measles vaccination has been very effective, reducing measles deaths by 78% from an estimated 562 400 death in 2000 to 122 000 in 2012 (WHO, 2012). Although global incidence has been significantly reduced through vaccination, measles still remains an important public health problem. Since vaccination coverage is not uniformly high worldwide; measles is estimated to have caused 614 000 global deaths in 2002, with more than half of measles deaths occur in sub-Saharan Africa as stated by Ousmane (2006).

Measles is an infectious disease highly contagious through person-to-person transmission mode, with greater than 90% secondary attack rates among susceptible persons. It is the first and worst eruptive fever occurring during childhood.

Measles is a respiratory disease caused by a virus called paramyxovirus. This virus is spread through the respiratory route and contained in the millions of tiny droplets that come out of the nose and mouth when a measles carrier coughs or sneezes. One can catch measles by breathing in these droplets or, if the droplets have settled on a surface, by touching the surface and then placing the hands near

the nose or mouth. The measles virus can survive on surfaces for a few hours. As a result, it can spread rapidly in a susceptible population. Infected people carry the virus in their respiratory tract before they get sick, so they can spread the disease without being aware of it.

The symptoms of the measles appear ten to fourteen days after a person is infected with the measles virus. When a person becomes infected with the measles virus, it begins to multiply within the cells that line the lungs and the back of the throat. The virus can also spread to the lymph glands, bone marrow, liver, eyes, thymus, tonsils, spleen, skin and brain. The symptoms include; fever, sore throat, cough, sore eyes, red watery eyes, vomiting, runny nose, loss of appetite and fatigue. The skin rash appears within three to five days from the onset of the symptoms.

The consequences of a person infected with measles are; ear infection, pneumonia, diarrhoea, convulsions, meningitis, conditions affecting blood clotting and deaths.

There is no treatment that can kill the virus but focusing on supportive care can relieve symptoms, this includes; getting plenty of water, and medication to control fever or pain.

Mathematical modeling in epidemiology provides new aspect of understanding the spread of measles disease and it suggested control strategy but most of these dynamic has been limited to ordinary differential equations. In recent years, it has turned out that many phenomena in different fields can be described very successfully by the model using fractional order differential equations. This motivated the application of the fractional order differential equation approach to model measles disease.

1.2 Problem Statement

Measles is described as one of the most communicable diseases of humans and a major cause of childhood deaths. The global attempts to reduce this infection aim to achieve regular vaccination coverage of at least 90% in every district throughout the world. So far, this attempt has resulted in a remarkable decline in deaths.

Even though this incidence has been importantly reduced through vaccination, measles still remains an important public health problem.

Although, there have been a number of work over the years on measles, but the emphasis has been on integer-order differential equation. Also ordinary differential equation do not take into account of memory effect. But recently, it has been observed that using non-integer order or fractional order differential equations to model real life phenomena in different fields can be described very successful. To the best of our knowledge, no works has been contributed to analysis for a SIR model of fractional order differential equation for describing measles diseases. This situation therefore motivated us to apply the fractional order SIR differential equation to effectively model and analyze the disease.

1.3 Objectives of the study

The objectives of this work includes;

- 1.To propose a fractional order SIR differential equation model.
- 2.To analyze the dynamics or mathematical behavior of the solution of the model.
- 3.To Study the effect of vaccination and treatment of the disease.

1.4 Methodology

The method employed in this thesis includes;

- A look at the basic concept and definitions of fractional differential equation. This data was obtained from internet, journals, articles and books.
- A fractional order SIR model of the disease was formulated as a system of differential equations and the equilibrium points (steady states) were determined.
- The reproductive number and the stability analysis of the equilibrium points was also determined.
- Simulation using MATLAB.

1.5 Justification

Focusing on the epidemiology of modeling diseases in Ghana, several mathematicians have tried to find solution to analyze and control the measles infection but all has been restricted to ordinary differential equation.

But recently, much attention has been given to fractional order differential equation for modeling most infectious disease, since it possess memory and also helps to reduce the errors arising from the neglected parameters in modeling real life problems.

In this situation, it is very vital if we look within the field of fractional order differential equation in modeling the disease.

This thesis provides model epidemiology that can help to analyze any infectious disease effectively. It contributes in the area of academics since we provide method of solving the model by Laplace transformation. It also enables researchers and students to develop interest in modeling any infectious disease or real life phenomena by non-integer order.

1.6 Thesis Organization

This thesis comprises of five chapters. Chapter one reviews the background of the study, statement of the problem, the objectives of the thesis, the methodology applied in the study, as well as the justification and the organization of the thesis.

Chapter two consists of the review of relevant literature governing the theory. It took us through some of the various method of modeling infectious diseases and its control.

In chapter three we formulate the fractional order SIR model of the disease transmission. The methods of solution, equilibrium points or steady states, as well as the stability analysis and the reproductive number were determined.

Chapter four comprise of analysis and results. We determined the parameters of the differential equations and numerical simulation of the model equations by setting initial conditions.

Chapter five, the final chapter contains the conclusions and the recommendations of the thesis.

Chapter 2

Literature Review

2.0.1 Introduction

In this chapter we reviewed the work of other researchers related to the topic.

2.0.2 Literature Relevant to this thesis

Most mathematical modeling of infectious diseases has been restricted to the use of a system of integer-order ordinary differential equations. But of late, fractional calculus has been widely applied in many fields, for instance many mathematicians and researchers have tried to use fractional calculus to model real life process.

First of all, considering the topic "A Predictor-Corrector Approach for the Numerical Solution of Fractional Differential Equations" by Diethelm et al. (2002). They discuss an Adams-type predictor-corrector method for the numerical solution of fractional differential equations. The method may be used both for linear and non-linear problems and it may be extended to equations involving more than one differential operator too. They discuss an algorithm for the numerical solution of differential equations of fractional order, set with suitable initial conditions. They identified two possible ways forward, but stated that as to which of these two or some other approach would be more appropriate. The first option they identified is to decrease the step size h used as the dimension of the system increases. The second approach was to remove those zeros (that do not contribute to the overall solution, so that the numerical solution gets stuck at the initial value for the first approximately) by replacing the plain *PECE* structure

by $P(EC)^M E$ version with a sufficiently large M (where M is any given number of times i.e iterations). They also increased the accuracy of the numerical solution by using the Richardson extrapolation idea. They concluded that there is the need to use much smaller values for the step size (h) in the case the order is greater than one ($\alpha > 1$) before we can see that the asymptotic behavior really sets in. This would normally correspond to the situation that the coefficients of the leading terms are small in magnitude compared to the coefficients of the higher-order terms.

Antonio et al. (2008) stated an application of the SIR model to epidemics in Portugal. They applied the SIR model to study the evolution of Measles and Hepatitis C in Portugal using data from 1996 until 2007. They observed that the annual data shows lower volatility than the monthly data and they found a better fitting for the former data translated into higher correlation coefficients. Their data from the Portuguese health system was too scarce to make possible predictions for the evolution of virus epidemics, even though the particular cases of Hepatitis C seem to be the one with complete data. In this case, they used their previous results to forecast the number of infected individuals for the four subsequent years using polynomial interpolation of 4th degree. They conclude that even though the SIR model is quite simple and obtaining the 1996-2007 data was scarce, it gave them some useful insight about the evolution of the Measles and Hepatitis C viruses.

Hashim et al. (2009) presented homotopy analysis method for fractional initial-value problems. In their paper, they applied the homotopy analysis method to solve linear and non-linear fractional initial-value problems (fIVPS). They described the fractional order derivatives by Caputo's sense. They also obtained an exact and approximate analytical solution of fIVPs. The Taylor series expansion was employed to avoid the difficulties with radical non-linear terms. The reliabil-

ity of HAM and the decrease in computations gave HAM a wider applicability. They also demonstrated that ADM (Adomian decomposition method) is a special case of HAM. Their results showed that, applying the procedure indicate high accuracy and efficiency of the approach.

Ionescu et al. (2009) presented fractional-order models for the input impedance of the Respiratory system. They discuss the use of fractional order models for characterizing the input impedance of the human respiratory system in relation to its fractal structure. They stated that a comparison with the well-inherited fractional order model from the various specialized literature and recent published hot-stone articles will situate their results. Based on available model structures from literature and their investigations, four fractional order models were compared on two sets of impedance data; healthy and COPD (Chronic Obstructive Pulmonary Disease). Their results indicate that the two models broadly used in the clinical studies and reported in the specialized literature were suitable for frequencies lower than $15Hz$. However, if the range of frequencies is higher, two fractional orders in the model structure are necessary, in order to capture the frequency dependence of the real part in the complex respiratory impedance. They concluded that, since the real part may both decrease and increase within the evaluated frequency interval, there is the need for both fractional order derivative and fractional order integral parameters.

Also deserving mention is the work of Srivastava and Rai (2009). They presented an approximate analytical solution of 3D fractional micro-scale heat equation using modified homotopy perturbation method. In their article, they used modified homotopy perturbation method (HPM) to find approximate solution of three-dimensional (3D) time fractional micro-scale heat transport equation. This transport equation was at first transformed into equation of two functions. The first one have both, the first order fractional time derivative and second order

space derivatives, whereas the second one have only second order space derivative. They then applied the modified HPM on first and corresponding increments in second function obtain by Taylor's expansion for equal time step homotopically and for solution, couple them. The obtained solution was compared with the previous numerical result for integer order time derivative and they found out that the procedure was easy, accurate and user friendly in comparison to previous work.

Zeng and Yang (2010) presented a fractional order HIV Internal viral dynamics model. They established a fractional order model to describe HIV internal viral dynamics involving highly active antiretroviral treatment (HAART) effect. They proved that the model possess a non-negative solutions as preferred in any population dynamics. A detailed analysis to study the stability of the equilibrium points was carried out. They also applied the PECE (Predict, Evaluate, Correct, Evaluate) method to obtain their numerical simulations of the model, and approximate solutions were displayed for step size 0.007 with different order of $0.6 \leq \alpha \leq 1$. Their numerical result shows that the uninfected cells predominate. They concluded that FODE can give another option to model viral dynamics. Since fractional order models possess memory, FODE gives a more realistic way to model viral dynamics.

Demirci et al. (2011) presented a fractional order SEIR model with density dependent death rate. They introduce a fractional order SEIR epidemic model with vertical transmission, where the death rate of the population is density dependent, and they also assumed that there exists an infection related death rate. They show the existence of non-negative solutions of the model and also gave a detailed stability analysis of the equilibrium points. They gave a numerical solutions of the system of fractional differential equations using an Adam's-type predictor Corrector method. They observed that although the equilibrium points

are the same for both integer order and fractional order models, but the solution of the fractional order model tends to the fixed point over a longer period of time. Finally, they concluded by saying that when dealing with real life problems, the order of the system can be determined by using the collected data.

Arafa et al. (2011) presented fractional order model of Human T-cell Lymphotropic virus I (HTLV-I) infection of $CD4^+$ T-cells. They introduce a fractional-order into a model of (HTLV-I) infection of $CD4^+$ T-cells. They gave approximate and analytical solutions to their model using a Generalized Euler method (GEM). They describe the fractional derivatives in the Caputo sense. Their results indicate that varying the values of k (the rate at which uninfected cells are contacted by actively infected cells) and k_1 (the rate of infection of T-cells with virus from actively infected infected cells) will alter the number of uninfected $CD4^+$ T-cells, infected cells, and leukemic cells. Their numerical results also indicate that increasing the value of k and k_1 makes the number of healthy $CD4^+$ T-cells decreases dramatically whereas the numbers of latently infected cells and leukemic cell increase substantially. They also show that a decrease in R_0 decreases the parameter k . Their results show that the solution continuously depends on the time- fractional derivative.

On optimal singular controls for a general SIR-model with vaccination and treatment was presented by Ledzewicz and Schattler (2011). They considered a general SIR-model with vaccination and treatment as a multi-input optimal control problem over a fixed time horizon. They analysed the structure of singular controls, but was still remains to complete the analysis by determining the structure of possible concatenations with bang-bang controls in order to determine an optimal synthesis of controlled trajectories. Based on their computations, it was anticipated that the optimal treatment schedule will be bang-bang, most likely with just one switch from u_{max} to $u = 0$ (where u is a control parameter that

represents the rate at which susceptible individuals are vaccinated and it takes values in a solid interval $0 \leq u \leq u_{max}$) whereas they expect a singular regimen for the optimal vaccination strategy.

Yusuf and Benyah (2012) presented Optimal control of vaccination and treatment for an SIR epidemiological model. They considered an SIR model with variable size population and formulated an optimal control problem subject to the model with vaccination and treatment as controls. Their main aim was to find the optimal combination of vaccination and treatment strategies that will minimize the cost of the two control measures as well as the number of infectives. The analysis of the model show that the disease free equilibrium is globally asymptotically stable if the basic reproduction ratio is less than one while the endemic equilibrium exists and it is globally asymptotically stable whenever the basic reproduction ratio is greater than one. They used Pontryagin's maximum principle to characterize the controls and derived the optimality system. They solved the resulting optimality system numerically. Their results confirm that the optimal combination of vaccination and treatment approach required to achieve the set objective will depend on the relative cost of the control measures. In conclusion, the results indicate that the case where it is more expensive to vaccine than to treat, resources should be invested in treating the disease until the disease prevalence begins to fall. This option, does not decrease the number of susceptible quickly enough, but rather result in an overall increase in the infected population. On the other hand, if it is more expensive to treat than to vaccine, then more resource should be put into vaccination. This case rather resulted in a rapid decrease in the susceptible as well as an appreciable decrease in the number of infectives. However, the case where both measures are equally expensive showed that the optimal way to derive the epidemic towards eradication within the specified period is to use more of the vaccination control and less of the treatment control initially to derive the epidemic to below certain threshold after

which we can then apply less of vaccination control and more of the treatment control.

Arqub and El-Ajou (2012) presented the solution of the fractional epidemic model by homotopy analysis method. In their article, they examined the accuracy of the homotopy analysis method (HAM) for solving the fractional SIR model order problem of the spread of a non-fatal disease in a population. The HAM provides a simple way to adjust and control the convergence region of the series solution by introducing an auxiliary parameter. Their HAM also provided an analytical approximate solution in terms of an infinite power series. They gave a numerical solution to the system of equations. They observed that the HAM has successfully been applied to find the approximate solution of fractional SIR model. Their scheme shows importance of choice of convergence control parameter to guarantee the convergence of the solution. They concluded that, higher accuracy can be achieved using HAM by evaluating more components of the solution.

Rida et al. (2012) showed the effect of the environmental parameter on the Hantavirus infection through a fractional-order SI model. They presented a fractional-order model of the Hantavirus infection in terms of simple differential equations involving the mice population. They study that the effect of changes in ecological conditions and diversity of habitats can be observed by varying the value of the environmental parameter k . They used a generalized Euler method (GEM) to obtain an analytic approximate solution of the model. The GEM was implemented to describe the effect of carrying capacity k , which they used as a control parameter in the model of Hantavirus pulmonary syndrome (HPS) described by the fractional model. They presented their results by numerical solutions, and the result shows that the solution continuously depends on the time-fractional derivative and on the values of the parameters. They concluded that the population of infected mice m_i , will reduce to zero after 5 years regardless of the initial number

and the population of susceptible mice m_s on the other hand will approach a steady state of 5 years. Their results also show that when $\alpha \rightarrow 1$ the solution of the fractional model $(m_s)_\alpha$ and $(m_i)_\alpha$, reduce to the standard solution m_s and m_i .

Abdullah (2013) published Simulations of Hirschsprung's Disease Using Fractional Differential Equations. He examined a model of cell invasion focusing on the wave front of the neural crest (NC) cells in the case of Hirschsprung's disease (HSCR). He used fractional differential equations based upon two basic cell functions for simulation of the model. It was based on fractional trapezoidal numerical scheme to simulate the mathematical model in a one-dimensional setting. From his fractional trapezoidal numerical results, he concluded that, if host NC-derived cells and donor NC-derived cells migrate in opposite directions, then these cell vanguards might obstruct each other. Also, he observed from the results that, at the donor-host interface, neither donor nor host NC cells proliferate, once maximum capacity is reached. His results also confirmed that anomalous diffusion caused the proliferation of NC cells to slow down. He therefore, stated that the growing gut is not able to fully colonize within a specific time frame hence resulting in a Hirschsprung's disease-like state.

The Fractional order SIR and SIRS epidemic models with variable population size was showed by El-Saka (2013). In his work he dealt with the fractional-order SIR and SIRS epidemic models with constant recruitment rate, mass action incidence and variable population size. He analyzed the stability of the equilibrium points. He used an Adams-type predictor-corrector method for the numerical solution of the fractional integral equation. For the derivation of the method he replaced the original fractional-order SIRS epidemic model by equivalent fractional integral equations and then apply the PECE method. The results of the numerical solution showed that in the fractional order case the peak of the infection is reduced but the disease take a longer time to be eradicated. He concluded

that the differential equations can help us to reduce the errors arising from the neglected parameters in modeling real life problem, and he argue that the fractional order models are at least as good as integer order ones in modelling biological, economic and social systems where memory effects are important.

Radi et al. (2013) published the effect of vaccination on the dynamics of childhood diseases described by a fractional SIR epidemic model. They introduce fractional-order into the SIR model that monitors the temporal dynamics of a childhood disease in the presence of preventive vaccine. They used a Generalized Euler method (GEM) to obtain an analytic approximate solution of the model. The results show that the solution continuously depends on the time-fractional derivative and on the values of the parameters. It was observed that the population of the susceptible group decreases with time while that of the removed group gradually increases due to inclusion of vaccinated susceptible group, hence indicating that the entire population remains disease free with all the time and the endemic equilibrium remains stable. They concluded that the use of GEM for solving their SIR model shows that GEM is a good tool in solving biological systems.

Numerical behavior of a fractional order HIV/AIDS epidemic model was published by Mohammed and Nemat (2013). In their paper, they investigate a fractional order HIV/AIDS (FOHA) epidemic model with treatment. They firstly, represent the FOHA system as an equivalent system of ordinary differential equations. In their second step, they solved the system obtained in the first step by using the fourth order Runge-Kutta method. They solved the FOHA system with $0.8 \leq \alpha < 1$. They also analyzed the equilibrium points and stability of the system of equations. The simulation results from the numerical behavior of the FOHA show the effectiveness of the method.

Kuhnert et al. (2013) presented Simultaneous reconstruction of evolutionary his-

tory and epidemiological dynamics from viral sequences with the birth-death SIR model. They adapt a birth-death sampling model, which allows for serially sampled data and rate changes over time to estimate epidemiological parameters of the underlying population dynamics in terms of a compartmental susceptible-infected-removed (SIR) model. Their approach resulted in a phylodynamic method that enables the joint estimation of epidemiological parameters and phylogenetic history, and the method provides separate information on incidence and prevalence of infections. The Birth-Death SIR method (BDSIR) was applied to five human immunodeficiency virus type 1 clusters sampled in the United Kingdom (UK) between 1993 and 2003. The estimated basic reproduction number ranges from 1.9 to 3.2 among the clusters. Their results mean that the local epidemics arose from the introduction of infected individuals into population of between 900 and 3000 effectively susceptible individuals, albeit with wide margins of uncertainty. All the clusters show a turn down in the growth rate of the local epidemic in the middle or end of the 90's. The results also show that the effective reproductive number of cluster 1 drops below one around 1994, with the local epidemic having almost run its course by the end of the samples period. For the other four clusters the effective reproductive number also decreases in excess of time, but stays above 1. They concluded that, the BDSIR model provides the capacity to simultaneously reconstruct evolutionary processes with their underlying host population dynamics from viral sequence data and in particular the inferred parameters allow us to make statements about the future fate of the epidemic.

Multi-species SIR Models from a Dynamical Bayesian Perspective was presented by Zhuang et al. (2013). They proposed a dynamical Bayesian hierarchical SIR (HSIR) model, to capture the stochastic or random nature of an epidemic process in a multi-species SIR (with the recovered becoming susceptible again) dynamical setting, under hidden mass-balance constraints, which they call $MSIR_B$.

Different from a classic multi-species SIR model, which they call $MSIR_C$, their approach imposes mass balance on the underlying true counts rather than improperly, on the noisy observations. In addition, the $MSIR_B$ model can capture the discrete nature of, as well as uncertainties in, the epidemic process. In their case study for influenza in poultry and swine, they simulated datasets from $MSIR_B$ and a hierarchical version of the $MSIR_C$ model, respectively. From the simulation results, they observe the significance of incorporating between species transmission into the modelling. Furthermore, they see the advantages of the $MSIR_B$ model in accounting for uncertainties in the epidemic process while retaining the easily interpretable MSIRS flow that underlies the $MSIR_C$ model.

Anderson (2013) presented SIR Epidemic on a configuration model network. They study Susceptible-Infectious-Recovered epidemics on configuration model networks, for which they look at a closed population without births, deaths and migration. On that population they look at an SIR epidemic, which divides the population into three different states: susceptible, infectious and recovered. How disease spreads through the population depends strongly on the relations between infectious and susceptible individuals. By constructing a configuration model network it was possible for them to investigate when the epidemic may become large and when it will stay small with probability one and how the distribution of the infectious period affects the outbreak. They answered these questions by using generating functions and percolation theory. They observed that the early stages of an epidemic outbreak can be approximated by a branching process, also this approximation is possible until approximately the \sqrt{nth} infection in a population that consists of n individuals. They also show that an epidemic outbreak is possible when the transmission probability is above the epidemic threshold. They concluded that the transmission of the disease depend on the infectious periods, if the infectious periods are fixed for all individuals the transmission is independent and identically distributed whereas if the infectious periods are random this is

not the case. If the infectious periods are random the extinction probability is smaller when we assume that the transmission is independent than when we do not.

Weiss (2013) published the SIR model and the Foundations of Public Health. In their work, they introduced and analysed the most fundamental transmission model for a directly transmitted infectious disease. The model consists of a system of three coupled non-linear ordinary differential equations which does not hold a formula solution. Down the way they illustrated how the model helps to lay a theoretical basis for public health interventions and how several cornerstones of public health required such a model to illuminate. They apply their compartment models to study disease transmission like the number of laboratory confirmed flu cases in the US during 2009-2010 H1N1 pandemic. They stated that if all the assumptions of the SIR model held for the outbreak, it would imply there would be only one peak. They also observed that the US experienced only one peak every year expect during the pandemic years 1918, 1968, 2009, where is experienced two or more waves. They also used comparatively extensions of the SIR model to reveal five plausible mechanisms, each of which could have generated the two peaks during 2009, both quantitatively and qualitatively. The first two mechanisms capture changes in virus transmission and behavioural changes. The third mechanism involves population heterogeneity where each wave spreads through one sub-population. The fourth mechanism is virus mutation which causes delayed susceptibility of individuals, and the fifth mechanism is waning immunity. They used the models to inspect the effects of border control at the beginning of the epidemic and the timing of any amount of available vaccinations. They also use the models to try to understand why China had only one peak and the US had two peaks.

Almost all the aforementioned works, they indicate that fractional differential

equations plays an important and increasing role in the application of modelling real life problem by non-integer order.

In this work, we formulate a fractional SIR model, stability analysis and provide a general approximate and analytical approach to the solution of the fractional differential equations.

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Chapter 3

Methodology

3.1 Basic Concepts and Definitions

In this section we recall some definitions and basic results related in solving fractional differential equation.

3.1.1 The Gamma Function

This function is basically tied to fractional calculus by definition. It's explanation is simply the generality of the factorial for all real numbers.

The definition of the gamma function is given by

$$\Gamma(z) = \int_0^{\infty} e^{-u} u^{z-1} du, \text{ for all } z \in R$$

This function is only one of its kind in that the value for any quantity is, by consequence of the form of the integral, equivalent to that quantity z minus one times the gamma of the quantity minus one.

$\Gamma(z+1) = z\Gamma(z)$, also when $z \in N_+$, then $\gamma(z) = (z-1)!$

This can be shown through a simple integration by parts. Using the gamma functions we define the function $\Phi(t)$, which is useful for proving alternate forms of the fractional integral $\Phi(t)$, is given by $\Phi_{\alpha}(t) = \frac{t_+^{\alpha-1}}{\Gamma(\alpha)}$ (Loverro, 2004).

3.1.2 Beta Function

Beta Function, furthermore known as Euler Integral of the first kind, is an important link in fractional calculus, its solution is defined through the use of multiple Gamma Functions, but also share a form that is typically similar to the Fractional

Integral/ Derivative of many functions, mostly polynomials of the form t^α and the Mittag - Leffler Function.

The equation below demonstrates the Beta Integral and its solution in terms of the Gamma function

$$B(p, q) = \int_0^1 (1-u)^{p-1} u^{q-1} du = \frac{\Gamma(p)\Gamma(q)}{\Gamma(p+q)} = B(q, p) \text{ where } p, q \in R \text{ (Loverro, 2004).}$$

3.1.3 Laplace Transformation and Convolution

The Laplace Transform is a function transformation usually used in the solution of complex differential equation. With the Laplace transform it is often possible to avoid working with equations of difference differential order directly by translating the problem into a domain where the solution presents itself algebraically. The formal definition of the Laplace transform is given by

$$\mathcal{L}\{f(t)\} = \int_0^\infty e^{-st} f(t) dt = \tilde{f}(s)$$

Also commonly used is the Laplace convolution, given by

$$f(t) * g(t) = \int_0^b f(t-\tau)g(\tau)d(\tau) = g(t) * f(t)$$

The convolution of two function in the domain of t is sometime complex to resolve, however in the Laplace domain(s), the convolution results in the simple function multiplication as shown below.

$$\mathcal{L}\{f(t) * g(t)\} = \tilde{f}(s)\tilde{g}(s)$$

The final vital property of the Laplace transform that should be addressed is the Laplace transform of a derivative of integer order n of the function f(t), given by

$$\mathcal{L}\{f^n(t)\} = s^n \tilde{f}(s) - \sum_{k=0}^{n-1} s^{n-k-1} f^{(k)}(0) = s^n \tilde{f}(s) - \sum_{k=0}^{n-1} s^k f^{n-k-1}(0)$$

(Loverro, 2004).

3.1.4 The Mittag-Leffler Function

This is an important function that finds prevalent use in the world of fractional calculus. This function plays an equivalent role in the solution of non-integer order differential equations. The standard definition of the Mittag-Leffler is given by,

$$E_{\alpha}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + 1)}, \alpha > 0$$

The exponential function correspond to $\alpha = 1$.

It is also common to represent the Mittag-Leffler function in two arguments, α and β such that

$$E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}, \alpha > 0, \beta > 0$$

This is the more generalized form of the function (Loverro, 2004).

3.2 Definitions and Derivations

3.2.1 The Fractional Integral

Riemann-Liouville demonstrates the formula regularly attributed to Cauchy for evaluating the n^{th} integration of the function $f(t)$

$$\int \dots \int_0^t f(\tau) d\tau = \frac{1}{(n-1)!} \int_0^t (t-\tau)^{n-1} f(\tau) d\tau$$

For the shortened illustration of this formula, Liouville introduce the operator J^n such that

$$J^n f(t) := f_n(t) = \frac{1}{(n-1)!} \int_0^t (t-\tau)^{n-1} f(\tau) d\tau$$

Often one will also used another operator, D^{-n} instead of J^n

Hence by replacing the factorial expression for its gamma function equivalent, then the equation is generalized for all $\alpha \in \mathbb{R}_+$, as

$$J_{\alpha} f(t) := f_{\alpha}(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} f(\tau) d\tau$$

First, Liouville considered integrations of order $\alpha = 0$ to be an identity operator, that is

$$J^0 f(t) = f(t)$$

Also, given the nature of the integral's description and based on the rule from which it came (Cauchy Repeated Integral Equation), it can be seen that just as

$$J^n J^m = J^{m+n} = J^m J^n, m, n \in \mathbb{N}$$

so to,

$$J^\alpha J^\beta = J^{\alpha+\beta} = J^\beta J^\alpha, \alpha, \beta \in \mathbb{R}$$

The one presupposed condition placed upon a function $f(t)$ that needs to be content for these and other similar properties to remain true, is that $f(t)$ be a fundamental function, that is, that it is vanishing for $t \leq 0$

The effect is such that $f(0) = f_n(0) = f_\alpha(0) \equiv 0$

An extra property of the Riemann-Liouville integral appears after the introduction of the function Φ as shown below,

$$\Phi_\alpha = \frac{t^{\alpha-1}}{\Gamma(\alpha)} \Rightarrow \Phi_\alpha(t) * f(t) = \int_0^t \frac{(t-\tau)^{\alpha-1}}{\Gamma(\alpha)} f(\tau) d\tau$$

t_+ denotes the function vanishes for $t \geq 0$ and hence the equation Φ_α is a fundamental function. From the definition of the Laplace convolution, it follows that

$$J^\alpha f(t) = \Phi_\alpha(t) * f(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} f(\tau) d\tau$$

Here it was obtained by finding the Laplace Transform of the Riemann-Liouville fractional integral.

It is shown in Φ_α that the fractional integral could be expressed as the convolution of two terms, thus, Φ_α and $f(t)$. The Laplace transform of $t^{\alpha-1}$ is given by

$$\mathcal{L}\{t^{\alpha-1}\} = \Gamma(\alpha) S^{-\alpha}$$

Thus, given the convolution relation to the fractional integral through Φ_α and the Laplace transform of the convolution, then the Laplace transform of the fractional integral is found to be

$$\mathcal{L}\{J^\alpha\} = S^{-\alpha} \bar{f}(s) \text{ (Loverro, 2004).}$$

3.2.2 Fractional Derivative

Left Hand Definition vs Right Hand Definition

Consider a differentiation of order $\alpha = \frac{1}{2}$, $\alpha \in \mathbb{R}_+$. Now, choose an integer m such that $m - 1 < \alpha < m$. Given these numbers, there are two likely ways to define the derivative. The first method, which will be call the Left Hand Definition is demonstrated graphically

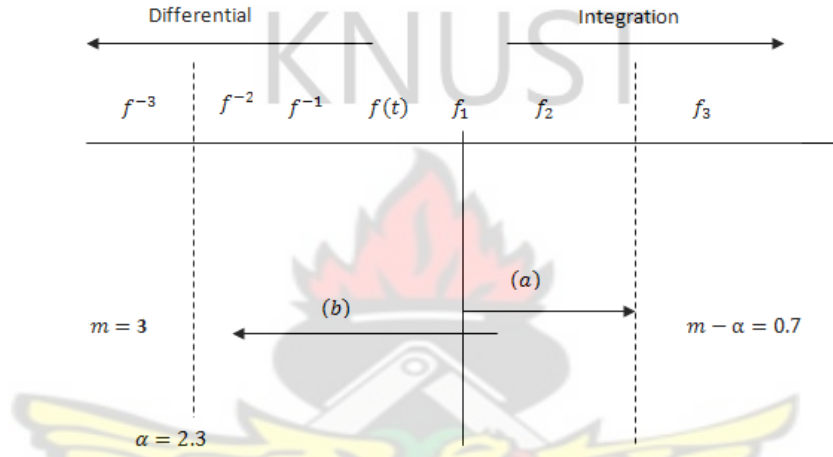


Figure 3.1: Graphical Representation of left hand definition method

The explanation for this method is simple. Having found the integer m , the first step of the procedure is complete operation (a), that is, integrate the function $f(t)$ by order $m - \alpha = 0.7$ where for the figure, $\alpha = 2.3$. Second, differentiate the resulting function, $f_{0.7}(t)$ by order $m = 3$ (operation(b)), thereby achieving a resulting differentiation of order α .

This method is represented in its mathematical form as,

$$D^\alpha f(t) = \begin{cases} \frac{d^m}{dt^m} \left[\frac{1}{\Gamma(m-\alpha)} \int_0^t \frac{f(\tau)}{(t-\tau)^{\alpha+1-m}} d\tau \right], & m-1 < \alpha < m \\ \frac{d^m}{dt^m} f(t), & \alpha = m \end{cases}$$

The second method, referred to here as the Right Hand Definition, is similarly shown graphically

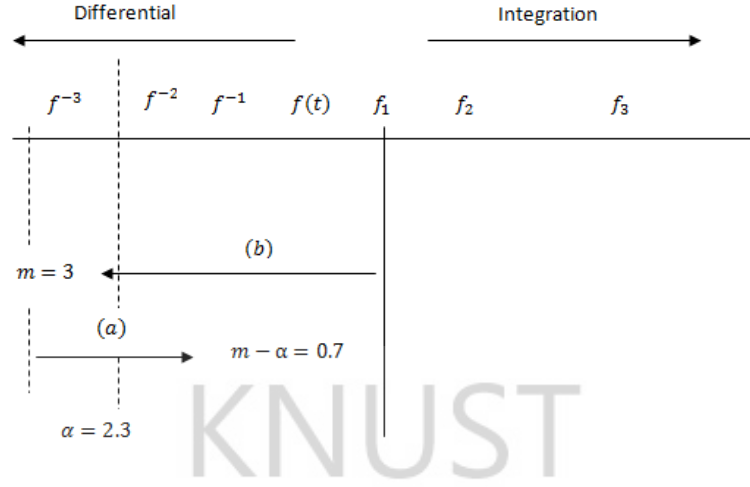


Figure 3.2: Graphical representation of Right - Hand Definition Method

The Right- Hand Definition attempts to appear at the same result using the same operations (a), (b) but in the overturn order. The mathematical result of this is shown below

$$D^\alpha f(t) = \begin{cases} \frac{d^m}{\Gamma(m-\alpha)} \int_0^t \frac{f^{(m)}(\tau)}{(t-\tau)^{\alpha+1-m}} d\tau, & m-1 < \alpha < m \\ \frac{d^m}{dt^m} f(t), & \alpha = m \end{cases}$$

The second definition, although referred to here as the ‘Right Hand’ Definition was initially formulated by Caputo, and is therefore, usually called the Caputo Fractional Derivative.

However, for the Right Hand definition, because one differentiates the function $f(t)$ by order m , it must be well-known that not only must $f(0) = 0$, but also $f^{(1)} = f^{(2)} = \dots = f^{(m)} = 0$.

The Right Hand Definition is necessary when solving non-integer order differential equation.

Also, the fractional derivative of a constant using the LHD is not zero (0), and in-fact is equal to

$$D^\alpha c = \frac{ct^{-\alpha}}{\Gamma(1-\alpha)}$$

From the above explanation, it can be observe that representing the practicality of the RHD over the LHD is suitably simple.

Finding the Laplace transform of the LHD fractional derivative, this may be written in the form $D^\alpha f(t) = g^{(m)}(t)$, where $g(t) = J^{m-\alpha} f(t)$, $m-1 \leq \alpha \leq m$

Using the Laplace transform of a derivative of integer order n of function f(t) and the definition of the fractional Integral- Laplace transform, Caputo found that

$$\mathcal{L}\{D^\alpha f(t)\} = s^m \tilde{g}(s) - \sum_{k=0}^{m-1} s^k g^{(m-k-1)}(0) = s^\alpha \tilde{f}(s) - \sum_{k=0}^{m-1} s^k D^{(\alpha-k-1)} f(0)$$

The requisite initial conditions are for all k to n-1 terms, fractional order derivative of f(t).

For the LHD the derivative is written in the form

$$D^\alpha * f(t) = J^{m-\alpha} g(t), \text{ where } g(t) = f^{(m)}(t), m-1 \leq \alpha < m$$

The Laplace transform is then given as

$$\mathcal{L}\{D^\alpha * f(t)\} = s^{-(m-\alpha)} \tilde{g}(s) = s^\alpha \tilde{f}(s) - \sum_{k=0}^{m-1} s^{\alpha-k-1} f^{(k)}(0) \text{ (Loverro, 2004).}$$

For fractional order differentiation, we will make use of Caputo's definition owing to its convenience for initial conditions of the differential equations. It also help us to find a connection between what is likely and what is realistic.

Definition: The fractional integral of order $\sigma > 0$ for a function $f : \mathbb{R}^+ \rightarrow \mathbb{R}$ is defined by

$$I^\sigma f(t) = \frac{1}{\Gamma(\sigma)} \int_0^t (t-\tau)^{\sigma-1} f(\tau) d\tau$$

and the Caputo fractional derivative of order $\sigma \in (n-1, n)$ of f(t) is defined by

$$D^\sigma f(t) = I^{n-\sigma} D^n f(t)$$

with n-1 being the integer part of σ and $D = \frac{d}{dt}$. Also, Γ denotes the Gamma Function.

3.3 Model Derivation

The model we study in this thesis is a fractional order SIR epidemic model with vaccination and treatment. The total population $N(t)$ is partitioned into three (3) compartments which are Susceptible, Infectious and recovered with sizes denoted by $S(t)$, $I(t)$ and $R(t)$ in that order. Let b denote the birth (recruitment) rate of the population, β denote the disease transmission rate which is assumed to take place with direct contact between infectious and susceptible hosts. We assume that the natural death rate $d(N)$ depends on the size of the population. For convenience, d is assumed to be a continuous and non-decreasing function on \mathbb{R}^+ , which does not disagree with natural phenomena. We assume α to be disease-induced death rate. Also, we assume there exists μ_1 and μ_2 which respectively denotes proportion of the susceptible that is vaccinated per unit time and proportion of the infective that is treated per unit time.

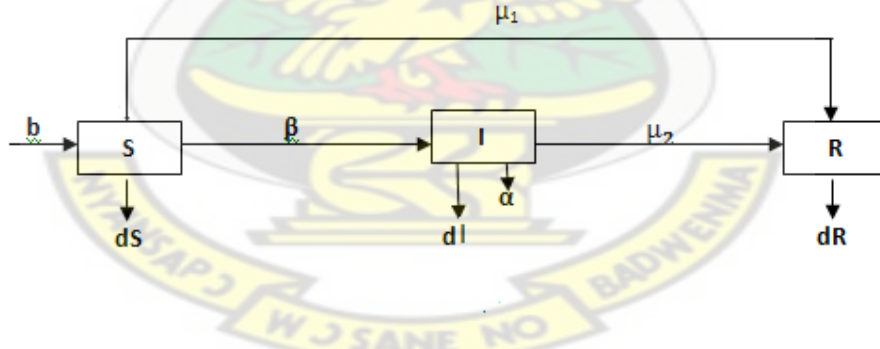


Figure 3.3: Flowchart showing the compartment model for SIR

Table 3.1: Variables used and their meaning

Variable	Meaning
$S(t)$	The number of Susceptible individuals at time, t
$I(t)$	The number of Infectious individuals at time, t
$R(t)$	The number of recovered individuals at time, t

Table 3.2: Parameters used and their meaning

Parameter	Meaning
b	birth/recruitment rate
β	diseases transmission rate between infections and susceptible
d	natural dearth rate
α	disease-induced dearth rate
μ_1	the proportion of susceptible that is vaccinated per unit time
μ_2	the proportion of infectious that is treated per unit time

The assumptions of the model leads to the following system of differential equations

$$\frac{dS}{dt} = b - \beta SI - dS - \mu_1 S$$

$$\frac{dI}{dt} = \beta SI - \mu_2 I - dI - \alpha I$$

$$\frac{dR}{dt} = \mu_1 S + \mu_2 I - dR$$

By fractionalizing the system above, where $D^\sigma S$, $D^\sigma I$ and $D^\sigma R$ are the derivatives of $S(t)$, $I(t)$, and $R(t)$ respectively, of arbitrary order σ in the sense of Caputo and $0 < \sigma < 1$, then the system leads to fractional differential equations given by,

$$D^\sigma S = b - \beta SI - dS - \mu_1 S \quad (1)$$

$$D^\sigma I = \beta SI - \mu_2 I - dI - \alpha I \quad (2)$$

$$D^\sigma R = \mu_1 S + \mu_2 I - dR \quad (3)$$

where $S(0) = S_0$, $I(0) = I_0$, $R(0) = R_0$

The reason for considering a fractional order system instead of its integer order matching part is that fractional order differential equations are generalization of integer order differential equations. Also, using fractional order differential equations can help us to decrease the errors arising from the neglected parameters in modeling real life phenomena.

Note that the system of fractional differential equations above can be reduced to an integer order system by setting $\sigma = 1$

But the equation of the population can be obtain by adding (1), (2) and (3)

$$D^\sigma(S + I + R) = b - \beta SI - dS - \mu_1 S + \beta SI - \mu_2 I - dI - \alpha I + \mu_1 S + \mu_2 I - dR$$

But $N = S + I + R$, therefore by substitute, we have

$$D^\sigma N = b - Nd - \alpha I$$

Then we have,

$$D^\sigma N = b - Nd - \alpha I \quad (4)$$

This means that the population size is not constant.

This implies that $R(t)$ can always be obtained by the equation

$$R(t) = N(t) - S(t) - I(t)$$

We now consider our system of equations as

$$D^\sigma S = b - \beta SI - dS - \mu_1 S \quad (5)$$

$$D^\sigma I = \beta SI - \mu_2 I - dI - \alpha I \quad (6)$$

$$D^\sigma N = b - Nd - \alpha I \quad (7)$$

$$S(0) = S_0, I(0) = I_0, N(0) = N_0, \text{ where } 0 < \sigma < 1$$

3.3.1 Non-Negative Solutions

Given the equation of the population as, $D^\sigma N = b - Nd - \alpha I$, and from the dynamics described by equations (1)-(4), then the region

$$\mathbb{R}_+^3 = (x \in \mathbb{R}^3 : x \geq 0) \text{ and } x(t) = (S(t), I(t), R(t))^T$$

is positively invariant (non-negative solutions)

Methods of Solution

From (7) we have, $D^\sigma N = b - Nd - \alpha I$

By grouping or rearranging the variable of interest , we have

$$D^\sigma N + dN = b - \alpha I$$

Now assume that $f(t) = b - \alpha I$, which is a constant function of time

then we have $D^\sigma N + dN = f(t)$

Taking Laplace transform throughout, we have

$$\mathcal{L}\{D^\sigma N\} + \mathcal{L}\{dN\} = \mathcal{L}\{f(t)\}$$

but $\mathcal{L}\{D^\sigma N\} = s^\sigma \tilde{N} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+)$, where $\sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+)$ is the initial condition

$$\mathcal{L}\{dN\} = d\mathcal{L}\{N\} = ds^2 \tilde{N} \text{ and } \mathcal{L}\{f(t)\} = F(s)$$

This implies that,

$$s^\sigma \tilde{N} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) + ds^2 \tilde{N} = F(s)$$

$$\text{Thus, } (s^\sigma + ds^2) \tilde{N} = \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) + F(s)$$

Taking the initial condition to be zero, thus $\sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) = 0$, then we have

$$\tilde{N} = \frac{F(s)}{s^\sigma + ds^2}, \text{ which involves two functions}$$

We now find the Laplace inverse transform of \tilde{N} to get $N(t)$, then let $\tilde{g}(s) = \frac{1}{s^\sigma + ds^2}$

and $\tilde{k}(s) = F(s)$

$$\text{Now, } \tilde{g}(s) = \frac{1}{s^\sigma + ds^2} = \frac{1}{s^2} \left(\frac{1}{s^{\sigma-2} + d} \right) = \frac{1}{d} \frac{s^{-2}}{\left(\frac{s^{\sigma-2}}{d} + 1 \right)}$$

$$\text{Then } \tilde{g}(s) = \frac{1}{d} \left(\frac{s^{-2}}{1 - \left(-\frac{s^{\sigma-2}}{d} \right)} \right) = \frac{1}{d} \sum_{k=0}^{\infty} (-1)^k s^{-2} \left(\frac{s^{-(\sigma-2)}}{d} \right)^k$$

$$\tilde{g}(s) = \frac{1}{d} \sum_{k=0}^{\infty} (-1)^k \left(\frac{s^{-k(\sigma-2)-2}}{d^k} \right)$$

$$\tilde{g}(s) = \frac{1}{d} \sum_{k=0}^{\infty} (-1)^k \left(\frac{s^{-[k(\sigma-2)+2]}}{d^k} \right)$$

but

$$\mathcal{L}^{-1}\{s^{-p}\} = \frac{t^{p-1}}{\Gamma(p)}, \text{ where } \Gamma \text{ is a Gamma function}$$

Now we let $p = k(\sigma - 2) + 2$, and by substitute into the above formula we have,

$$\mathcal{L}^{-1}\{s^{-[k(\sigma-2)+2]}\} = \frac{t^{k[\sigma-2]+2-1}}{\Gamma(k(\sigma-2)+2)}$$

$$\Rightarrow g(t) = \frac{1}{d} \sum_{k=0}^{\infty} \left(\frac{-1}{d}\right)^k \frac{t^{k[\sigma-2]+2-1}}{\Gamma(k(\sigma-2)+2)}, \quad d > 0$$

$$\text{Thus, } g(t) = \frac{1}{d} t \sum_{k=0}^{\infty} \frac{\left(-\frac{t^{\sigma-2}}{d}\right)^k}{\Gamma(k(\sigma-2)+2)}, \quad d > 0$$

Now assume $z = \frac{-t^{\sigma-2}}{d}$, $u = \sigma - 2$, $v = 2$, we have

$$g(t) = \frac{1}{d} t \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(uk + v)}, \text{ then by Mittag-Leffler function, we can let}$$

$$E_{u,v}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(uk + v)}$$

$$\Rightarrow g(t) = \frac{1}{d} t E_{u,v}(z) \text{ and } k(t) = F(t)$$

Hence we have, $N(t) = \frac{1}{d} t E_{u,v}(z) F(t)$, where, $u > 0$, $v > 0$ and $d > 0$

Also by convolution, we can deduce that

$$N(t) = \frac{1}{d} \int_0^t t E_{u,v}(z) F(t) dt, \quad u > 0, v > 0,$$

thus

$$N(t) = g(t) * k(t) = \frac{1}{d} \int_0^t (t - t') E_{\sigma-2,2}\left(\frac{-(t-t')^{\sigma-2}}{d}\right)^k F(t') dt'$$

Therefore we show that,

$$N(t) = \frac{1}{d} \int_0^t (t - t') E_{\sigma-2,2}\left(\frac{-(t-t')^{\sigma-2}}{d}\right)^k F(t') dt' \geq 0, \text{ since } d > 0,$$

Also if $k \rightarrow \infty$, $t \rightarrow \infty$, then $N(t) \rightarrow \frac{b}{d} \geq 0$, hence proved that the solution is positive invariant

$$\text{From equation (5),} \quad D^\sigma S = b - \beta SI - dS - \mu_1 S$$

by grouping like terms, we have $D^\sigma S + \beta SI + dS + \mu_1 S = b$

Taking Laplace transform throughout we have,

$$\mathcal{L}(D^\sigma S) + \mathcal{L}(\beta SI) + \mathcal{L}(dS) + \mathcal{L}(\mu_1 S) = \mathcal{L}(b)$$

taking constant out we have

$$\mathcal{L}(D^\sigma S) + \beta I \mathcal{L}(S) + d \mathcal{L}(S) + \mu_1 \mathcal{L}(S) = b \mathcal{L}(1)$$

where

$$\mathcal{L}(D^\sigma S) = s^\sigma \tilde{S} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+)$$

$$\beta I \mathcal{L}(S) = \beta I s^2 \tilde{S}$$

$$d \mathcal{L}(S) = d s^2 \tilde{S},$$

$$\mu_1 \mathcal{L}(S) = \mu_1 s^2 \tilde{S}$$

$$b \mathcal{L}(1) = b s^{-1} \text{ by substitution, we have, } s^\sigma \tilde{S} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) + \beta I s^2 \tilde{S} + d s^2 \tilde{S} + \mu_1 s^2 \tilde{S} = b s^{-1}$$

Taking the initial condition to be zero, then we have

$$s^\sigma \tilde{S} + \beta I s^2 \tilde{S} + d s^2 \tilde{S} + \mu_1 s^2 \tilde{S} = b s^{-1}$$

$$\text{Now by factorizing } \tilde{S} \text{ leads to, } \tilde{S}(s^\sigma + \beta I s^2 + d s^2 + \mu_1 s^2) = b s^{-1}$$

$$\text{Making } \tilde{S} \text{ the subject, thus, } \tilde{S} = \frac{b s^{-1}}{s^\sigma + \beta I s^2 + d s^2 + \mu_1 s^2}$$

$$\tilde{S} = \frac{b s^{-1}}{s^2(s^{\sigma-2} + \beta I + d + \mu_1)}, \text{ where death rate (d) is positive}$$

Assume that $\beta I + d + \mu_1 = c$, then we have

$$\tilde{S} = \frac{b s^{-1}}{s^2(s^{\sigma-2} + c)} = \frac{b s^{-3}}{s^{\sigma-2} + c} = \frac{b s^{-3}}{c(\frac{s^{\sigma-2}}{c} + 1)}$$

We now have to find the Laplace inverse transform of \tilde{S} to get $S(t)$

$$\tilde{S} = \frac{b s^{-3}}{c(1 + \frac{s^{\sigma-2}}{c})} = \frac{b s^{-3}}{c(1 - (-\frac{s^{\sigma-2}}{c}))} = \frac{b}{c} \frac{s^{-3}}{(1 - (-\frac{s^{\sigma-2}}{c}))}$$

We now have, $\tilde{S} = \frac{b}{c} \sum_{k=0}^{\infty} (-1)^k s^{-3} \left(\frac{s^{-(\sigma-2)}}{c} \right)^k$

$$\Rightarrow \tilde{S} = \frac{b}{c} \sum_{k=0}^{\infty} (-1)^k \left(\frac{s^{-k(\sigma-2)-3}}{c^k} \right)$$

$$\text{Thus } \tilde{S} = \frac{b}{c} \sum_{k=0}^{\infty} (-1)^k \left(\frac{s^{-[k(\sigma-2)+3]}}{c^k} \right)$$

but $\mathcal{L}^{-1}\{s^{-p}\} = \frac{t^{p-1}}{\Gamma(p)}$, where Γ is a Gamma function.

Now we let $p = k(\sigma - 2) + 3$, so by substitute we have,

$$\mathcal{L}^{-1}\{s^{-[k(\sigma-2)+3]}\} = \frac{t^{k(\sigma-2)+3-1}}{\Gamma(k(\sigma-2)+3)} = \frac{t^{k(\sigma-2)+2}}{\Gamma(k(\sigma-2)+3)}$$

$$\text{This implies that, } S(t) = \frac{b}{c} \sum_{k=0}^{\infty} \left(\frac{-1}{c} \right)^k \frac{t^{k(\sigma-2)+2}}{\Gamma(k(\sigma-2)+3)} = \frac{b}{c} \sum_{k=0}^{\infty} t^2 \frac{\left(\frac{-t^{\sigma-2}}{c} \right)^k}{\Gamma(k(\sigma-2)+3)}$$

If we let $z = \frac{-t^{\sigma-2}}{c}$, $u = \sigma - 2$ and $v = 3$ then we have

$$S(t) = \frac{b}{c} t^2 \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(uk + v)}, \text{ therefore by Mittag-Leffler function application, we can let}$$

$$E_{u,v}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(uk + v)}, \Rightarrow S(t) = \frac{b}{c} t^2 E_{u,v}(z), u > 0, v > 0$$

Therefore, $S(t) = \frac{b}{c} t^2 E_{u,v}(z) \geq 0$, since $d > 0$ and if $t \rightarrow \infty$. Also, from the above solution we assume that $u = \sigma - 2$, $v = 3$, $z = \frac{-t^{\sigma-2}}{c}$, and $c = \beta I + d + \mu_1$

$$\text{From equation (6), } D^\sigma I = \beta SI - \mu_2 I - dI - \alpha I$$

By grouping variable of interest, we have $D^\sigma I - \beta SI + \mu_2 I + dI + \alpha I = 0$

Taking Laplace transform throughout, thus,

$$\mathcal{L}(D^\sigma I) - \mathcal{L}(\beta SI) + \mathcal{L}(\mu_2 I) + \mathcal{L}(dI) + \mathcal{L}(\alpha I) = \mathcal{L}(0)$$

where,

$$\mathcal{L}(D^\sigma I) = s^\sigma \tilde{I} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+)$$

$$\mathcal{L}(\beta SI) = \beta S \mathcal{L}(I) = \beta S \tilde{I} s^2$$

$$\mathcal{L}(\mu_2 I) = \mu_2 \mathcal{L}(I) = \mu_2 \tilde{I} s^2$$

$$\mathcal{L}(dI) = d \mathcal{L}(I) = d \tilde{I} s^2$$

$$\mathcal{L}(\alpha I) = \alpha \mathcal{L}(I) = \alpha \tilde{I} s^2$$

$$\mathcal{L}(0) = 0$$

By substituting into the Laplace transform of the equation above, thus,

$$s^\sigma \tilde{I} - \sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) - \beta S \tilde{I} s^2 + \mu_2 \tilde{I} s^2 + d \tilde{I} s^2 + \alpha \tilde{I} s^2 = 0$$

Taking the initial condition, $\sum_{n=0}^{n-1} s^{\sigma-n-1} N(0_+) = 0$, then the equation becomes

$$s^\sigma \tilde{I} - \beta S \tilde{I} s^2 + \mu_2 \tilde{I} s^2 + d \tilde{I} s^2 + \alpha \tilde{I} s^2 = 0, \quad d > 0$$

Now by factorizing \tilde{I} , we have, $\tilde{I}(s^\sigma - \beta S s^2 + \mu_2 s^2 + d s^2 + \alpha s^2) = 0$

Making \tilde{I} the subject, thus, $\tilde{I} = \frac{0}{s^\sigma - \beta S s^2 + \mu_2 s^2 + d s^2 + \alpha s^2} = 0$

We now take the Laplace inverse of \tilde{I} to get $I(t)$, but $\tilde{I} = 0$

Therefore, $\mathcal{L}^{-1}\{\tilde{I}\} = \mathcal{L}^{-1}\{0\} = 0 \Rightarrow I(t) = 0$

3.3.2 Basic Reproductive Number (R_0) of the Model

The basic reproduction number R_0 is defined as the average number of Secondary cases generated by a typical infective (patience) within a population with

no immunity to the disease, in the absence of interventions produced by a single infected individual introduced into a population of N susceptible. It is denoted by R_0 (Kermack and McKendrick, 1927). If $R_0 < 0$, then there is no epidemics, that is the disease dies out. If $R_0 > 0$, then it implies that the disease spreads in the susceptible population. But when $R_0 = 0$, then the disease becomes endemic, meaning the disease remains in the population at a constant rate, as one infected individual transmit the disease to one susceptible.

Now, the system of equations is,

$$D^\sigma S = b - \beta SI - dS - \mu_1 S \quad (8)$$

$$D^\sigma I = \beta SI - \mu_2 I - dI - \alpha I \quad (9)$$

$$D^\sigma N = b - Nd - \alpha I \quad (10)$$

with initial conditions, $S(0) = S_0, I(0) = I_0, N(0) = N_0$, where $0 < \sigma < 1$

We determine (R_0) by using the Next Generation Matrix Approach or by simply imposing the non-negativity condition on the infected Compartment I .

The Next Generation Matrix is given by $K = FV^{-1}$.

The basic reproduction number is the eigenvalue of largest magnitude or spectra radius of the next generation matrix.

We re-order the above equations to get

$$D^\sigma S = b - \beta SI - dS - \mu_1 S \dots\dots\dots f_2(I, S, N)$$

$$D^\sigma I = \beta SI - \mu_2 I - dI - \alpha I \dots\dots\dots f_1(I, S, N)$$

$$D^\sigma N = b - Nd - \alpha I \dots\dots\dots f_3(I, S, N)$$

where $0 < \sigma < 1$

Linearization of the above model gives the Generation matrix (G) evaluated at the disease free equilibrium (DFE),

$$G = \begin{bmatrix} f_1 I & f_1 S & f_1 N \\ f_2 I & f_2 S & f_2 N \\ f_3 I & f_3 S & f_3 N \end{bmatrix}$$

Since f_1 and f_2 form a subsystem describing the generation and transition of infectious, the Jacobian matrix associated with the linearized subsystem at Dis-

ease Free Equilibrium (DFE) is given by,

$$J(I, S) = \begin{bmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial S} \\ \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial S} \end{bmatrix}$$

$$\text{Thus, } J_{DFE}(I, S) = \begin{bmatrix} \beta S_0 - (\mu_2 + d + \alpha) & 0 \\ -\beta S_0 & -(d + \mu_1) \end{bmatrix}$$

J_{DFE} is decomposed as $F - V$, where F is the transition matrix describing the changes in individual states such as removal by death or recovery. Knowing matrices F and V , R_0 can be simply obtained by calculating the spectra radius of NGM $K = FV^{-1}$. The DFE is locally stable if $R_0 < 1$, whereas it is unstable if $R_0 > 1$ (Diekmann and Heesterbeek, 2000).

$$J_{DFE} = F - V = \begin{bmatrix} \beta S_0 & 0 \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \mu_2 + d + \alpha & 0 \\ \beta S_0 & d + \mu_1 \end{bmatrix},$$

$$\text{Thus, } F = \begin{bmatrix} \beta S_0 & 0 \\ 0 & 0 \end{bmatrix} \text{ and } v = \begin{bmatrix} \mu_2 + d + \alpha & 0 \\ \beta S_0 & d + \mu_1 \end{bmatrix}$$

$$V^{-1} = \frac{1}{(d + \mu_1)(\mu_2 + d + \alpha)} \begin{bmatrix} d + \mu_1 \alpha & 0 \\ -\beta S_0 & \mu_2 + d + \alpha \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \beta S_0 & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu_2 + d + \alpha} & 0 \\ \frac{-\beta S_0}{(d + \mu_1)(\mu_2 + d + \alpha)} & \frac{1}{d + \mu_1} \end{bmatrix}$$

$$K = FV^{-1} = \begin{bmatrix} \frac{\beta S_0}{\mu_2 + d + \alpha} & 0 \\ 0 & 0 \end{bmatrix}$$

At Disease Free Equilibrium (DFE), $S_0 = \frac{b}{d + \mu_1}$, so by substitute into the above

matrix, the Next Generation Matrix (NGM) becomes

$$K = FV^{-1} = \begin{bmatrix} \frac{b\beta}{(d + \mu_1)(\mu_2 + d + \alpha)} & 0 \\ 0 & 0 \end{bmatrix}$$

Since R_0 is the most dominant eigenvalue of the NGM, then

$$R_0 = \frac{b\beta}{(d + \mu_1)(\mu_2 + d + \alpha)}$$

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3.3.3 Equilibrium point and Stability

To determine the stability analysis of the model, we first evaluate the equilibrium point(s) or steady states of the system of fractional differential equations (8), (9), and (10).

The equilibrium points involved determine the disease-free (where $I = 0$) and endemic (where $I \neq 0$).

Consider the initial value problem (8)-(10) with $0 < \sigma < 1$. To evaluate the equilibrium points of (8)-(10), let

$$D^\sigma S = 0$$

$$D^\sigma I = 0$$

$$D^\sigma N = 0$$

3.3.4 The Disease-Free Equilibrium Solution

At the disease-free equilibrium, we consider the case where there is no infection.

We now consider the equations below and solve for the values of S and N, since at this point there is no infection, thus $I = 0$

$$b - \beta SI - dS - \mu_1 S = 0 \quad (11)$$

$$\beta SI - \mu_2 I - dI - \alpha I = 0 \quad (12)$$

$$b - Nd - \alpha I = 0 \quad (13)$$

From equation (12)

$$I(\beta S - \mu_2 - d - \alpha) = 0$$

$$I = 0$$

substitute $I = 0$, into equation (11), we have

$$Sb - dS - \mu_1 S = 0, \text{ this implies that, } S = \frac{b}{d+\mu_1}$$

Now substituting $I = 0$, into equation (13) yields

$$b - Nd = 0, \text{ implies } N = \frac{b}{d}.$$

At diseases - free equilibrium solution,

$$(S^*, I^*, N^*) = (\frac{b}{d+\mu_1}, 0, \frac{b}{d})$$

3.3.5 An Endemic Equilibrium Solution

For this stage, thus an endemic equilibrium solution, we consider the case where there is infection.

From equation (12)

$$S = \frac{\mu_2 + d + \alpha}{\beta}$$

Substitute S into equation (11) to find I, then we have

$$b - \beta(\frac{\mu_2 + d + \alpha}{\beta})I - d(\frac{\mu_2 + d + \alpha}{\beta}) - \mu_1(\frac{\mu_2 + d + \alpha}{\beta}) = 0$$

$$b - (\mu_2 + d + \alpha)I = d(\frac{\mu_2 + d + \alpha}{\beta}) + \mu_1(\frac{\mu_2 + d + \alpha}{\beta})$$

$$I = \frac{b\beta - d(\mu_2 + d + \alpha) - \mu_1(\mu_2 + d + \alpha)}{\beta(\mu_2 + d + \alpha)}$$

$$I = \frac{b\beta - (\mu_1 + d)(\mu_2 + d + \alpha)}{\beta(\mu_2 + d + \alpha)} = (R_0 - 1) \frac{\mu_1 + d}{\beta}$$

Now substitute I into equation (13) to get N,

$$Nd = b - \alpha I$$

$$Nd = \frac{b\beta(\mu_2 + d + \alpha) - \alpha(b\beta - (\mu_1 + d)(\mu_2 + d + \alpha))}{\beta(\mu_2 + d + \alpha)}$$

$$Nd = \frac{b\beta\mu_2 + b\beta d + \alpha b\beta - \alpha b\beta + \alpha(\mu_1 + d)(\mu_2 + d + \alpha)}{\beta(\mu_2 + d + \alpha)}$$

$$N = \frac{b\beta(\mu_2 + d) + \alpha(\mu_1 + d)(\mu_2 + d + \alpha)}{d\beta(\mu_2 + d + \alpha)}$$

At endemic equilibrium we have the point,

$$(S^*, I^*, N^*) = \left(\frac{\mu_2 + d + \alpha}{\beta}, \frac{b\beta - (\mu_1 + d)(\mu_2 + d + \alpha)}{\beta(\mu_2 + d + \alpha)}, \frac{b\beta(\mu_2 + d) + \alpha(\mu_1 + d)(\mu_2 + d + \alpha)}{d\beta(\mu_2 + d + \alpha)} \right)$$

3.3.6 Stability Analysis of Disease-Free Equilibrium Point

Theorem 2: The disease-free equilibrium point of the system is asymptotically stable if $R_0 < 1$

Proof: To determine the stability of the system at disease-free equilibrium, we consider the linearized form of the system of the equations below about the equilibrium point.

$$D^s S = b - \beta SI - dS - \mu_1 S$$

$$D^s I = \beta SI - \mu_2 I - dI - \alpha I$$

$$D^s N = b - Nd - \alpha I$$

The Jacobian matrix J of the system is

$$J = \begin{bmatrix} -\beta I - d - \mu_1 & -\beta S & 0 \\ \beta I & \beta S - \mu_2 - d - \alpha & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

At disease-free equilibrium, $S = \frac{b}{d+\mu_1}$, $I = 0$ and $N = \frac{b}{d}$, therefore, by substitute into J, the Jacobian matrix becomes,

$$J_{DFE} = \begin{bmatrix} -d - \mu_1 & \frac{-b\beta}{d+\mu_1} & 0 \\ 0 & \frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

Now by finding the characteristic equation of the matrix above, we set the determinant of $J_{DFE} - \lambda$ to zero,

Thus,

$$\det(J_{DFE} - \lambda I) = \begin{vmatrix} -d - \mu_1 - \lambda & \frac{-b\beta}{d+\mu_1} & 0 \\ 0 & \frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda & 0 \\ 0 & -\alpha & -d - \lambda \end{vmatrix} = 0$$

We divide the above matrix into three 2×2 matrices and find their determinants.

Let,

$$d_1 = \begin{bmatrix} \frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda & 0 \\ -\alpha & -d - \lambda \end{bmatrix}$$

$$d_2 = \begin{bmatrix} 0 & 0 \\ 0 & -d - \lambda \end{bmatrix}$$

$$d_3 = \begin{bmatrix} 0 & \frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda \\ 0 & -\alpha \end{bmatrix}$$

After finding d_1 , d_2 and d_3 we substitute the values into the formula below

$$\det(J_{DFE} - \lambda) = (-d - \mu_1 - \lambda) \times d_1 - \left(\frac{-b\beta}{d+\mu_1}\right) \times d_2 + 0 \times d_3$$

Now solving for d_1 , d_2 and d_3 we have

$$d_1 = \left(\frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda\right)(-d - \lambda) - 0(-\alpha) = 0$$

$$d_1 = \left(\frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda\right)(-d - \lambda)$$

$$d_2 = 0$$

$$d_3 = 0$$

By applying the formula and substituting the values of d_1 , d_2 and d_3 we have

$$\det(J_{DFE} - \lambda I) = (-d - \mu_1 - \lambda) \left(\frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda\right)(-d - \lambda) \times (-d - \lambda) = 0$$

This implies that,

$$(-d - \mu_1 - \lambda) \left(\frac{b\beta}{d+\mu_1} - \mu_2 - d - \alpha - \lambda\right)(-d - \lambda) \times (-d - \lambda) = 0$$

Therefore, $\lambda_1 = -(d + \mu_1)$, $\lambda_2 = \frac{b\beta}{d+\mu_1} - (\mu_2 + d + \alpha)$ and $\lambda_3 = -d$. This shows that the matrix J_{DFE} have eigenvalues,

$$\lambda_1 = -(d + \mu_1) < 0, \lambda_3 = -d < 0 \text{ but } \lambda_2 = \frac{b\beta}{d+\mu_1} - (\mu_2 + d + \alpha)$$

For asymptotic stability we require $\lambda_2 < 0$, thus, $\frac{b\beta}{d+\mu_1} - (\mu_2 + d + \alpha) < 0$, which is equivalent to $\frac{b\beta}{(d + \mu_1)(\mu_2 + d + \alpha)} = R_0 < 1$

Remarks: The case where $R_0 = 1$ is a critical threshold point because the

disease-free equilibrium loses its asymptotic stability and becomes (neutrally) stable. Furthermore, it becomes unstable right away $R_0 > 1$ and this will lead to the existence of a stable endemic equilibrium.

Notice that the case $R_0 = 1$ can literary be viewed as a transcritical bifurcation point where stability is exchanged between disease-free equilibrium and endemic equilibrium point.

3.3.7 Stability Analysis of Endemic Equilibrium Point

Theorem 3: The endemic equilibrium point is asymptotically stable if $R_0 > 1$.

Proof: The system has an endemic infection because of the introduction of those with secondary infection. To determine this, we linearized the Jacobian matrix J evaluated at the endemic equilibrium point.

The Jacobian matrix of the system is,

$$J = \begin{bmatrix} -\beta I - d - \mu_1 & -\beta S & 0 \\ \beta I & \beta S - \mu_2 - d - \alpha & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

At endemic equilibrium, $S^* = \frac{\mu_2 + d + \alpha}{\beta}$, $I^* = \frac{b\beta - (\mu_1 + d)(\mu_2 + d + \alpha)}{\beta(\mu_2 + d + \alpha)}$

and $N^* = \frac{b\beta(\mu_2 + d) + \alpha(\mu_1 + d)(\mu_2 + d + \alpha)}{d\beta(\mu_2 + d + \alpha)}$

Inserting S^* , I^* and N^* , into the Jacobian matrix gives

$$J = \begin{bmatrix} -\frac{(b\beta - (\mu_1 + d)(\mu_2 + d + \alpha))}{(\mu_2 + d + \alpha)} - d - \mu_1 & -(\mu_2 + d + \alpha) & 0 \\ \frac{(b\beta - (\mu_1 + d)(\mu_2 + d + \alpha))}{(\mu_2 + d + \alpha)} & (\mu_2 + d + \alpha) - \mu_2 - d - \alpha & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

$$J = \begin{bmatrix} -\frac{b\beta}{(\mu_2+d+\alpha)} & -(\mu_2+d+\alpha) & 0 \\ \frac{b\beta}{(\mu_2+d+\alpha)} - (\mu_1+d) & 0 & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

This can also be represented as,

$$J = \begin{bmatrix} -R_0(\mu_1+d) & -(\mu_2+d+\alpha) & 0 \\ (R_0-1)(\mu_1+d) & 0 & 0 \\ 0 & -\alpha & -d \end{bmatrix}$$

Where $R_0 = \frac{b\beta}{(\mu_1+d)(\mu_2+d+\alpha)}$ is the basic reproduction number.

We determine the eigenvalues of J by calculating the determinants of the matrix.

$$\text{Thus, } \det(J - \lambda I) = \begin{vmatrix} -R_0(\mu_1+d) - \lambda & -(\mu_2+d+\alpha) & 0 \\ (R_0-1)(\mu_1+d) & 0 - \lambda & 0 \\ 0 & -\alpha & -d - \lambda \end{vmatrix}$$

We divide the matrix into three 2×2 matrices to find the determinants.

Let,

$$d_1 = \begin{bmatrix} 0 - \lambda & 0 \\ -\alpha & -d - \lambda \end{bmatrix}, d_2 = \begin{bmatrix} (R_0-1)(\mu_1+d) & 0 \\ 0 & -d - \lambda \end{bmatrix} \text{ and}$$

$$d_3 = \begin{bmatrix} (R_0-1)(\mu_1+d) & 0 - \lambda \\ 0 & -\alpha \end{bmatrix}$$

We then apply the formula below after determine the value(s) of d_1, d_2 and d_3 .

$$\det(J - \lambda I) = (-R_0(\mu_1+d_1 - \lambda) \times d_1 - (-(\mu_2+d+\alpha)) \times d_2 + 0 \times d_3 = 0$$

$$\det(J - \lambda I) = (-R_0(\mu_1+d_1 - \lambda) \times d_1 + (\mu_2+d+\alpha) \times d_2$$

Now calculating the value(s) of d_1, d_2 and d_3 , we have

$$d_1 = (-\lambda)(-d - \lambda) + 0 \times (\alpha) \Rightarrow d_1 = (-\lambda)(-d - \lambda)$$

$$d_2 = (R_0 - 1)(\mu_1 + d)(-d - \lambda)$$

$$d_3 = (R_0 - 1)(\mu_1 + d)(-\alpha) - 0(0 - \lambda) \Rightarrow d_3 = (R_0 - 1)(\mu_1 + d)(-\alpha)$$

Inserting d_1, d_2 and d_3 into the formula gives,

$$\det(J - \lambda I) = (-R_0(\mu_1 + d) - \lambda) \times (-\lambda)(-d - \lambda) + (\mu_2 + d + \alpha) \times (R_0 - 1)(\mu_1 + d)(-d - \lambda) = 0$$

$$(-R_0(\mu_1 + d) - \lambda) \times (-\lambda)(-d - \lambda) + (\mu_2 + d + \alpha) \times (R_0 - 1)(\mu_1 + d)(-d - \lambda) = 0$$

$$(-d - \lambda)((-R_0(\mu_1 + d) - \lambda)(-\lambda) + (\mu_2 + d + \alpha)(R_0 - 1) \times (\mu_1 + d)) = 0$$

This implies that,

$$\lambda = -d \text{ and } \lambda^2 + R_0(\mu_1 + d)\lambda + (R_0 - 1)(\mu_1 + d)(\mu_2 + d + \alpha) = 0$$

Thus, $\lambda_1 = -d < 0$, but to find λ_2 and λ_3 from

$$\lambda^2 + R_0(\mu_1 + d)\lambda + (R_0 - 1)(\mu_1 + d)(\mu_2 + d + \alpha) = 0$$

Then by using the formula $\lambda_{2,3} = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$,

where $a = 1$, $b = R_0(\mu_1 + d)$ and $c = (R_0 - 1)(\mu_1 + d)(\mu_2 + d + \alpha)$

Inserting the value of a, b and c, we have

$$\lambda_{2,3} = \frac{-R_0(\mu_1 + d) \pm \sqrt{R^2(\mu_1 + d)^2 - 4(R_0 - 1)(\mu_1 + d)(\mu_2 + d + \alpha)}}{2}$$

This shows that if $R_0 > 1$, then $\lambda_2 < 0$ and $\lambda_3 < 0$, hence it becomes asymptotically stable.

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Chapter 4

Analysis

This chapter deals with the analysis and numerical simulation of the model. The estimation of the parameters, simulation analysis to illustrate our results on stability, as well as numerical simulation and graphical representation of the system of fractional differential equations.

4.1 Estimation of parameters

The parameter values and initial conditions used for the analysis and numerical solutions to the system of equations are shown in the table below,

Table 4.1: Parameters and values of the system of fractional differential equation

Parameters	Meaning	Values
b	birth/recruitment rate	0.03
β	disease transmission rate between infective and susceptible	0.75
d	natural dearth rate	0.02
α	disease -induced dearth rate	0.1
S_o	initial number of susceptible individuals	0.95×10^3
I_o	initial number of infective individuals	0.05×10^3
N_o	initial number of total population	1.0×10^3

Source: Yusuf and Benyah (2012)

4.1.1 Simulation

By using the Generalized Euler method (GEM), we obtained the numerical solution of the system of fractional differential equations.

For instance considering the initial value problem

$$D^\sigma y(t) = f(t, y(t)), \quad y(0) = y_0, \quad 0 < \sigma < 1, \quad t > 0$$

The general formula for the Fractional Euler's method of the above initial value problem is

$$y(t_{j+1}) = y(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} f(t_j, y(t_j))$$

for $j = 0, 1, \dots, k-1$. It is clear that if $\sigma = 1$, then the generalized Euler's method reduces to the classical Euler's method.

For this purposes we attempts to find numerical solution for a general class of fractional order SIR epidemic model of the disease.

Considering,

$$D^\sigma S(t) = b - \beta S(t)I(t) - dS(t) - \mu_1 S(t) \quad (14)$$

$$D^\sigma I(t) = \beta S(t)I(t) - \mu_2 I(t) - dI(t) - \alpha I(t) \quad (15)$$

$$D^\sigma N(t) = b - N(t)d - \alpha I(t) \quad (16)$$

$$S(0) = S_0 = 0.95 \times 10^3, \quad I(0) = I_0 = 0.05 \times 10^3, \quad N(0) = N_0 = 1.0 \times 10^3, \\ 0 < \sigma < 1, \quad t > 0$$

$$\text{From (14),} \quad D^\sigma S(t) = b - \beta S(t)I(t) - dS(t) - \mu_1 S(t)$$

$$\text{We let } f(t, S(t)) = b - \beta S(t)I(t) - dS(t) - \mu_1 S(t)$$

$$\Rightarrow D^\sigma S(t) = f(t, S(t)), \quad S(0) = S_0, \quad 0 < \sigma < 1, \quad t > 0$$

Let $[0, b]$ be the interval over which we want to find the solution of the problem.

We generate a set of points $(t_j, S(t_j))$ for our approximation.

For convenience we subdivide the interval $[0, b]$ into k subintervals $[t_j, t_{j+1}]$ of equal width $h = \frac{b}{k}$ by using the nodes $t_j = jh$, for $j = 0, 1, \dots, k$.

Suppose that $S(t)$, $D^\sigma S(t)$, and $D^{2\sigma} S(t)$ are continuous on $[0, b]$ and by using the generalized Taylor's formula to expand $D^\sigma S(t) = f(t, S(t))$ about $t = t_0 = 0$. For

each value t there is a value c_1 so that

$$S(t) = S(t_0) + (D^\sigma S(t))(t_0) \frac{t^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} S(t))(c_1) \frac{t^{2\sigma}}{\Gamma(2\sigma + 1)}$$

Now when $(D^\sigma S(t))(t_0) = f(t, S(t))$ and $h = t_1$ are substitute into the above

equation, then the results is an expression for $S(t_1)$;

$$S(t_1) = S(t_0) + f(t_0, S(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} S(t_0))(c_1) \frac{h^{2\sigma}}{\Gamma(2\sigma + 1)}$$

If the step size h is chosen small enough, then we may neglect the higher order term (thus, $h^{2\sigma}$) and get

$$S(t_1) = S(t_0) + f(t_0, S(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)}$$

The process is repeated and generates a sequence of points that approximates the solution $S(t)$. Then, the general formula for fractional Euler's method of $S(t)$ is

$$t_{j+1} = t_j + h,$$

$$S(t_{j+1}) = S(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} f(t_j, S(t_j))$$

$$\text{but } f(t_j, S(t_j)) = b - \beta S(t_j)I(t_j) - dS(t_j) - \mu_1 S(t_j)$$

By inserting we have,

$$S(t_{j+1}) = S(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} (b - \beta S(t_j)I(t_j) - dS(t_j) - \mu_1 S(t_j))$$

for $j = 0, 1, \dots, k-1$.

$$\text{From (15), } D^\sigma I(t) = \beta S(t)I(t) - \mu_2 I(t) - dI(t) - \alpha I(t)$$

$$\text{Let } f(t, I(t)) = \beta S(t)I(t) - \mu_2 I(t) - dI(t) - \alpha I(t)$$

$$\Rightarrow D^\sigma I(t) = f(t, I(t)), \quad I(0) = I_0, \quad 0 < \sigma < 1, \quad t > 0$$

Let $[0, b]$ be the interval over which we want to find the solution of the problem.

We generate a set of points $(t_j, I(t_j))$ for our approximation.

For convenience we subdivide the interval $[0, b]$ into k subintervals $[t_j, t_{j+1}]$ of equal width $h = \frac{b}{k}$ by using the nodes $t_j = jh$, for $j = 0, 1, \dots, k$.

Suppose that $I(t)$, $D^\sigma I(t)$, and $D^{2\sigma} I(t)$ are continuous on $[0, b]$ and by using the generalized Taylor's formula to expand $D^\sigma I(t) = f(t, I(t))$ about $t = t_0 = 0$. For

each value t there is a value c_1 so that

$$I(t) = I(t_0) + (D^\sigma I(t))(t_0) \frac{t^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} I(t))(c_1) \frac{t^{2\sigma}}{\Gamma(2\sigma + 1)}$$

Now when $(D^\sigma I(t))(t_0) = f(t, I(t))$ and $h = t_1$ are substitute into the above

Taylor's expansion of $I(t)$, then the results is an expression for $I(t_1)$;

$$I(t_1) = I(t_0) + f(t_0, I(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} I(t_0))(c_1) \frac{h^{2\sigma}}{\Gamma(2\sigma + 1)}$$

Now if the step size h is chosen small enough, then we may neglect the higher order term (that is, $h^{2\sigma}$) and get

$$I(t_1) = I(t_0) + f(t_0, I(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)}$$

The process is repeated and generates a sequence of points that approximates the solution $I(t)$. Then, the general formula for fractional Euler's method of $I(t)$ is

$$t_{j+1} = t_j + h,$$

$$I(t_{j+1}) = I(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} f(t_j, I(t_j))$$

$$\text{but } f(t_j, I(t_j)) = \beta S(t_j)I(t_j) - \mu_2 I(t_j) - dI(t_j) - \alpha I(t_j)$$

By inserting we have,

$$I(t_{j+1}) = I(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} (\beta S(t_j)I(t_j) - \mu_2 I(t_j) - dI(t_j) - \alpha I(t_j))$$

for $j = 0, 1, \dots, k-1$.

$$\text{Similarly, from (16), } D^\sigma N(t) = b - dN(t) - \alpha I(t)$$

$$N(0) = N_0, \quad 0 < \sigma < 1, \quad t > 0$$

$$\text{Let } f(t, N(t)) = b - dN(t) - \alpha I(t)$$

$$\Rightarrow D^\sigma N(t) = f(t, N(t)), \quad N(0) = N_0, \quad 0 < \sigma < 1, \quad t > 0$$

Let $[0, b]$ be the interval over which we want to find the solution of the problem.

We generate a set of points $(t_j, N(t_j))$ for our approximation.

For convenience we subdivide the interval $[0, b]$ into k subintervals $[t_j, t_{j+1}]$ of equal width $h = \frac{b}{k}$ by using the nodes $t_j = jh$, for $j = 0, 1, \dots, k$.

Suppose that $N(t)$, $D^\sigma N(t)$, and $D^{2\sigma} N(t)$ are continuous on $[0, b]$ and by using the generalized Taylor's formula to expand $D^\sigma N(t) = f(t, N(t))$ about $t = t_0 = 0$.

For each value t there is a value c_1 so that

$$N(t) = N(t_0) + (D^\sigma N(t))(t_0) \frac{t^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} N(t))(c_1) \frac{t^{2\sigma}}{\Gamma(2\sigma + 1)}$$

Now when $(D^\sigma N(t))(t_0) = f(t, N(t))$ and $h = t_1$ are substitute into the above Taylor's expansion of $N(t)$, then the results is an expression for $N(t_1)$;

$$N(t_1) = N(t_0) + f(t_0, N(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)} + (D^{2\sigma} N(t_0))(c_1) \frac{h^{2\sigma}}{\Gamma(2\sigma + 1)}$$

Now if the step size h is chosen small enough, then we may neglect the higher order term (that is, $h^{2\sigma}$) and get

$$N(t_1) = N(t_0) + f(t_0, N(t_0)) \frac{h^\sigma}{\Gamma(\sigma + 1)}$$

This process is repeated and generates a sequence of points that approximates the solution $N(t)$. Then, the general formula for fractional Euler's method of

$$N(t) \text{ is } t_{j+1} = t_j + h, \\ N(t_{j+1}) = N(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} f(t_j, N(t_j))$$

but $f(t_j, N(t_j)) = b - dN(t_j) - \alpha I(t_j)$

By inserting we have,

$$N(t_{j+1}) = N(t_j) + \frac{h^\sigma}{\Gamma(\sigma + 1)} (b - dN(t_j) - \alpha I(t_j))$$

for $j = 0, 1, \dots, k - 1$.

4.1.2 Numerical Results

In this section, we will study the effect of vaccination(μ_1) and treatment(μ_2) on the disease described by the fractional SIR model using GEM.

From the parameter values given in Table 4.1, we estimate that $R_0 = \frac{0.0225}{(\mu_1 + 0.02)(\mu_2 + 0.12)}$

From the results in the presented figures, it is clear that varying the values of μ_1 and μ_2 will alter the number of susceptible and infected persons.

R_0 is known as the basic reproduction number. If $R_0 > 1$, the disease persists in the population but if $R_0 < 1$, the disease always die out.

For instance, if $\mu_1 = \mu_2 = 0$, then $R_0 = 9.3750 > 1$, the endemic equilibrium is locally asymptotically stable. Thus, the results show that if there are no vaccination (μ_1) and treatment (μ_2) then the disease persist, while the number of

susceptible decrease (see fig. 4.1) and the number of infectious increases (see fig. 4.2).

If $\mu_1 = \mu_2 = 1$, then $R_0 = 0.0197 < 1$, the disease free equilibrium is locally asymptotically stable. Thus, the disease will die out in the population whereas the size of the susceptible increase (see fig. 4.4) and that of the infectious decreases (see fig. 4.5).

It is obvious from the meaning of R_0 that R_0 decreases as the parameter μ_1 and μ_2 increases, hence R_0 can be low for high parametric value of μ_1 and μ_2 .

The obtained results are shown in Figure 4.1, 4.2, 4.3, 4.4, 4.5 where μ_1, μ_2 vary.

Figure 4.1: Size of the susceptible class over time for the system with no vaccination($\mu_1 = 0$) for different values of σ

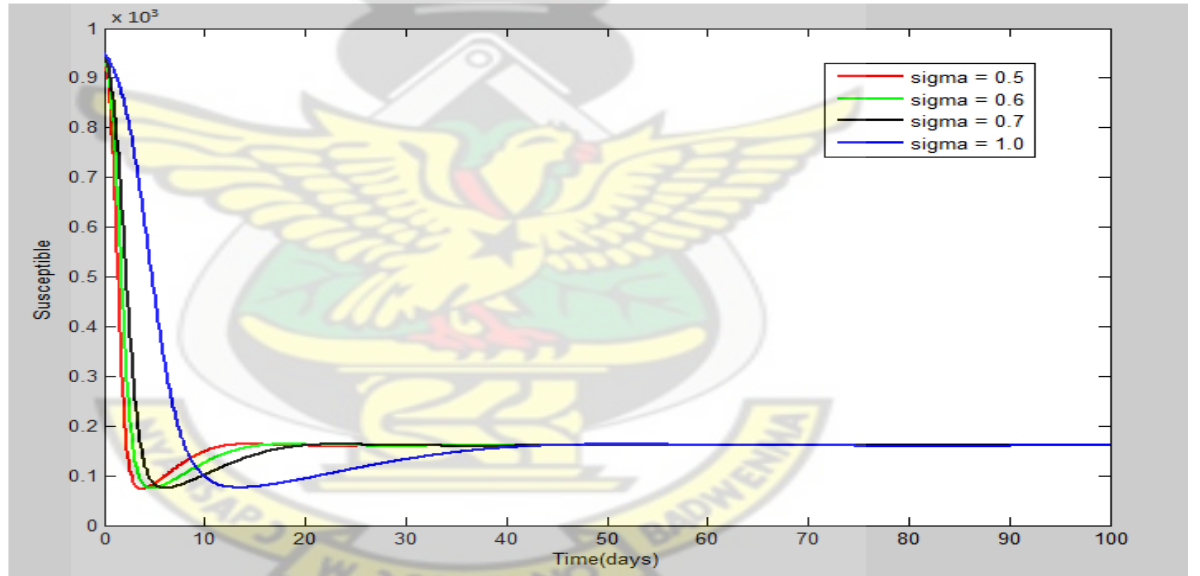


Figure 4.2: Size of the infectious class over time for the system with no treatment ($\mu_2 = 0$) for different values of σ

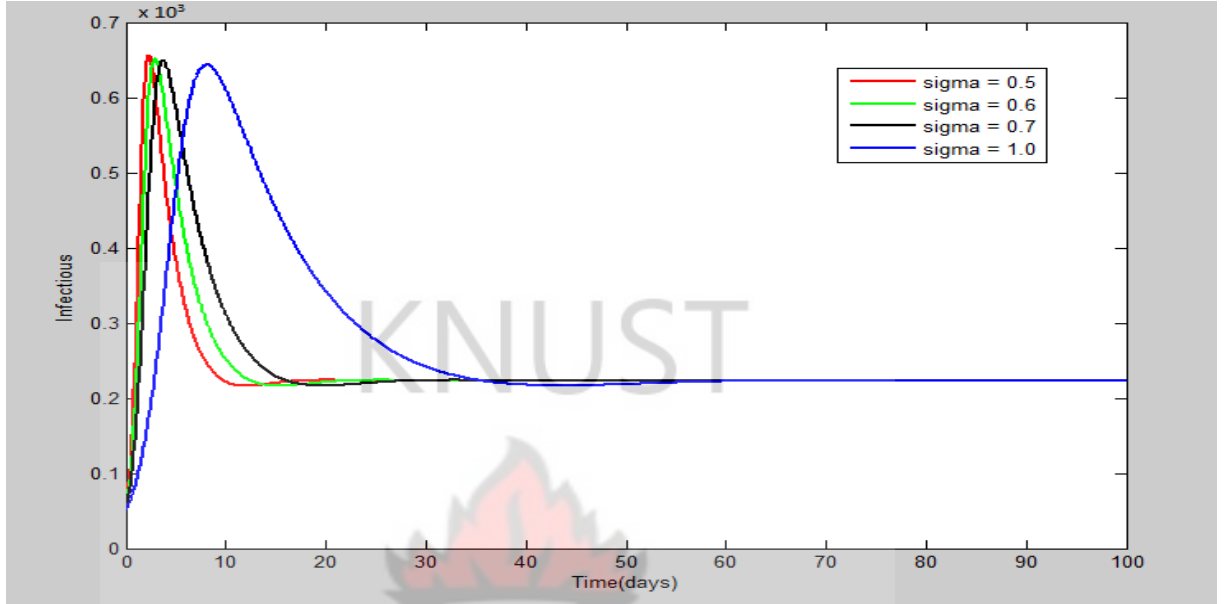


Figure 4.3: Size of the total population class over time for the system with different values of σ

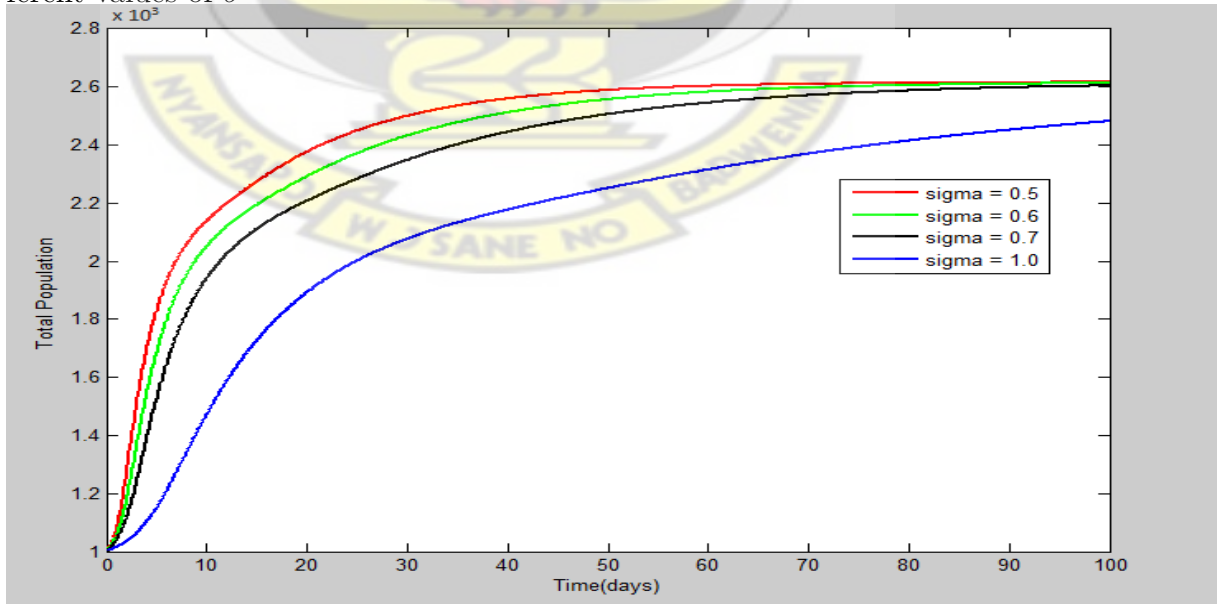


Figure 4.4: Size of the susceptible class over time for the system with vaccination($\mu_1 = 1$) for different values of σ

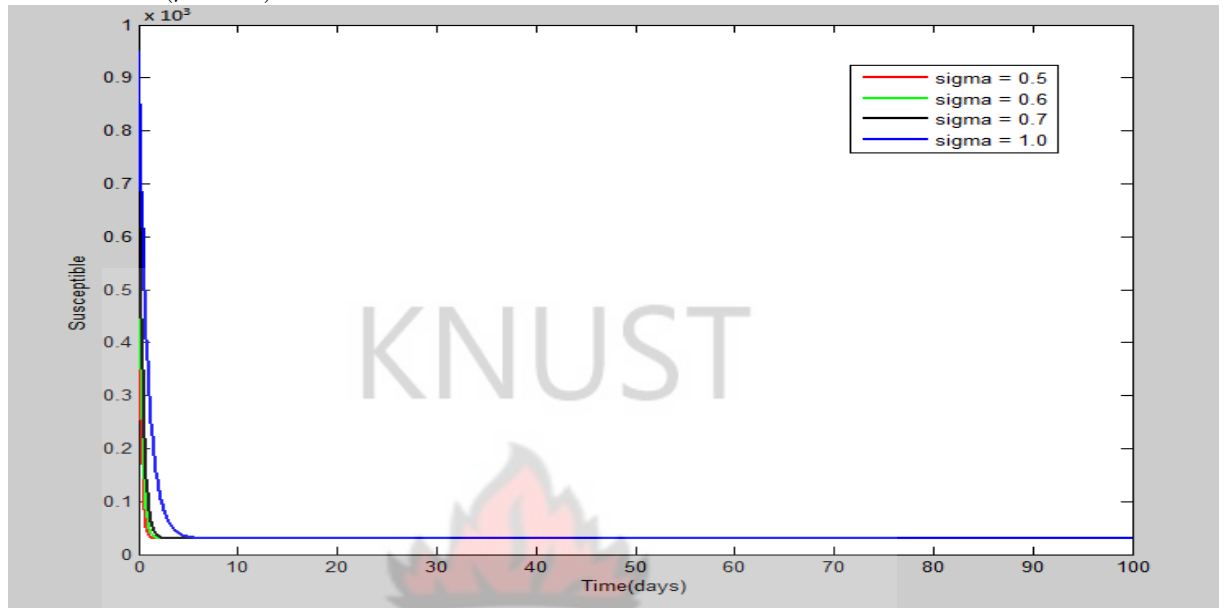
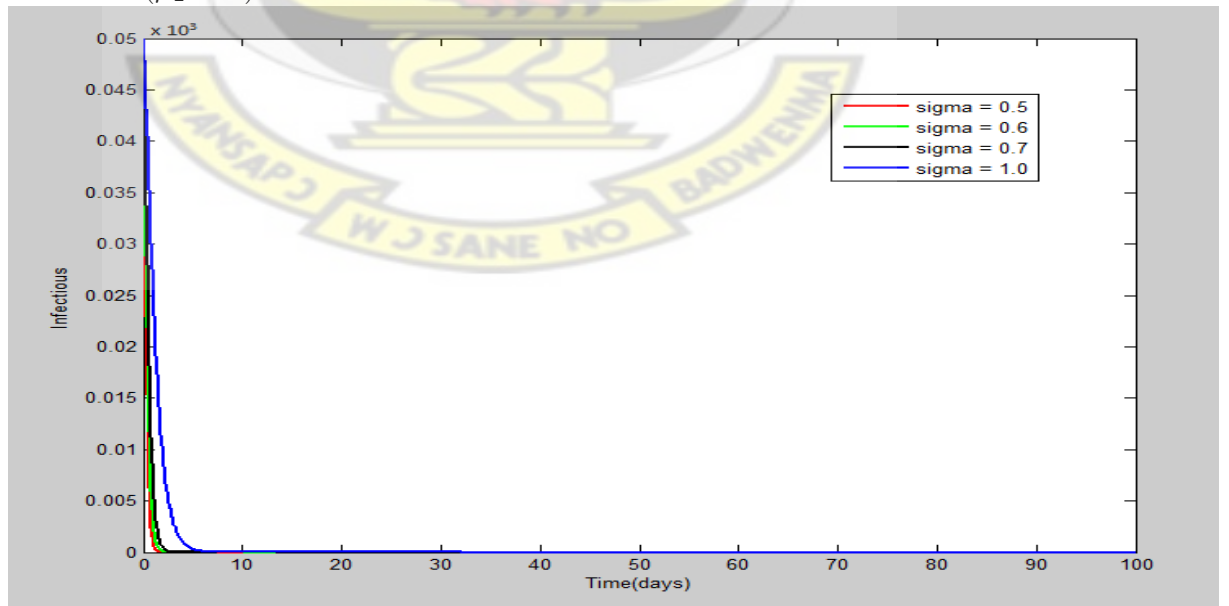


Figure 4.5: Size of the infectious class over time for the system with treatment($\mu_2 = 1$) for different values of σ



Chapter 5

Conclusion

In analyzing the mathematical behavior of the model we obtained the non-negative solutions of the model by Laplace transform. The fractional order model conformed to the assumption of the model.

The results confirmed that in the absence of vaccination and treatment the disease persists.

We have been able to extend the ODE to take care of all the properties and also the principle of the propose model possess memory. By comparing to the ordinary differential equation we observed that this provide more accurate results.

In the presented problem, the suspected group, the infected group and the total population group have been obtained, the results obtained indicate that when $\sigma = 1$ the solution of the fractional model, $D^\sigma S$, $D^\sigma I$ and $D^\sigma N$ reduce to the standard solution $S(t)$, $I(t)$ and $N(t)$, thus the model tends to ordinary differential equation.

The mathematical behavior indicate that the disease-free equilibrium point is locally asymptotically stable since $R_0 < 1$ and the endemic equilibrium point was also asymptotically stable since $R_0 > 1$. By using the stability analysis on a fractional order model, some sufficient conditions on the parameters for the local stability of equilibrium has been given.

We carried out numerical solution to confirm the analysis by applying GEM. Our studies on the use of GEM for solving the presented model prove that GEM is a good tool in solving the biological system. One of the advantages of GEM is its ability of preventing us with continuous solution, thus giving us a better understanding as well as detail over the time interval.

The numerical results confirmed that increasing the number of vaccination and

treatment will eradicate the disease completely.

Also, the basic reproductive number confirmed that in the absence of vaccination and treatment the disease persists whiles in the presence of vaccination and treatment the disease dies out.

In particular our work shows that FODE gives us a more realistic way of modeling any infectious disease since it helps to reduce errors arising from the neglected parameters.

Major Findings: Our major contribution to this work was the ability to prove the solution of the model by Laplace transformation.

5.1 Recommendations

We recommend that the public health sector can use the proposed model to understand the spread and control of measles.

Researchers and mathematical modelers should also use the equation proved, whenever they encounter such problem.

The global asymptotic behavior of the FODE of modeling the disease is still open.

We therefore recommend for further discussion on this question.

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Appendix A

CODES FOR FIGURE 4.1

Title: Size of the susceptible versus time with no vaccination for different values of order sigma

```
clear, clc;

b = 0.03;

beta = 0.75;

d = 0.02;

alpha = 0.1;

h = 0.1;

mu1 = 0;

mu2 = 0;

sigma = [0.5, 0.6, 0.7, 1.0];

t = 0 : h : 100; n = length(t);

m = length(sigma);

solS = []; solI = []; solN = [];

for k = 1 : m

    S = zeros(n, 1); I = S; N = S;

    S(1) = 0.95 × 103; I(1) = 0.05 × 103; N(1) = 1.0 × 103;

    for i = 1 : n - 1

        S(i + 1) = S(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - beta * S(i) * I(i) - d * S(i) - mu1 * S(i));

        I(i + 1) = I(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (beta * S(i) * I(i) - mu2 * I(i) - d * I(i) - alpha * I(i));

        N(i + 1) = N(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - d * N(i) + alpha * I(i));

    end

    solS(:, k) = S(:); solI(:, k) = I(:); solN(:, k) = N(:);

end
```

```

plot (t, solS(:, 1), 'r', t, solS(:, 2), 'g', t, solS(:, 3), 'k', t, solS(:, 4), 'b');
xlabel ('Time'), ylabel('Susceptible '), title('Size of the susceptible versus time
with no vaccination for different values of order sigma');
legend ('sigma = 0.5', 'sigma = 0.6', 'sigma = 0.7', 'sigma = 1.0')

```

CODES FOR FIGURE 4.2

Title: Size of the infectious versus time with no treatment for different values of order sigma

```

clear, clc;

b = 0.03;

beta = 0.75;

d = 0.02;

alpha = 0.1;

h = 0.1;

mu1 = 0;

mu2 = 0;

sigma = [0.5, 0.6, 0.7, 1.0];

t = 0 : h : 100; n = length(t);

m = length(sigma);

solS = []; solI = []; solN = [];

for k = 1 : m

S = zeros(n, 1); I = S; N = S;

S(1) = 0.95 × 103; I(1) = 0.05 × 103; N(1) = 1.0 × 103;

for i = 1 : n - 1

S(i + 1) = S(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - beta * S(i) * I(i) - d *
S(i) - mu1 * S(i));

I(i + 1) = I(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (beta * S(i) * I(i) - mu2 *
I(i) - d * I(i) - alpha * I(i));

```

```


$$N(i+1) = N(i) + (h^{\sigma(k)} / \text{gamma}(\sigma(k) + 1)) * (b - d * N(i) + \alpha * I(i));$$

end

solS(:, k) = S(:); solI(:, k) = I(:); solN(:, k) = N(:);

end

plot (t, solI(:, 1), 'r', t, solI(:, 2), 'g', t, solI(:, 3), 'k', t, solI(:, 4), 'b');

xlabel ('Time'), ylabel('Infectious '), title('Size of the infectious versus time with

no treatment for different values of order sigma ');

legend ('sigma = 0.5', 'sigma = 0.6', 'sigma = 0.7', 'sigma = 1.0')

```

CODES FOR FIGURE 4.3

Title: Size of the total population versus time for different values of order sigma

```

clear, clc;

b = 0.03;

beta = 0.75;

d = 0.02;

alpha = 0.1;

h = 0.1;

mu1 = 0;

mu2 = 0;

sigma = [0.5, 0.6, 0.7, 1.0];

t = 0 : h : 100; n = length(t);

m = length(sigma);

solS = []; solI = []; solN = [];

for k = 1 : m

S = zeros(n, 1); I = S; N = S;

S(1) = 0.95 × 103; I(1) = 0.05 × 103; N(1) = 1.0 × 103;

for i = 1 : n - 1

```

```


$$S(i+1) = S(i) + (h^{\sigma(k)} / \text{gamma}(\sigma(k) + 1)) * (b - \text{beta} * S(i) * I(i) - d * S(i) - \mu_1 * S(i));$$


$$I(i+1) = I(i) + (h^{\sigma(k)} / \text{gamma}(\sigma(k) + 1)) * (\text{beta} * S(i) * I(i) - \mu_2 * I(i) - d * I(i) - \alpha * I(i));$$


$$N(i+1) = N(i) + (h^{\sigma(k)} / \text{gamma}(\sigma(k) + 1)) * (b - d * N(i) + \alpha * I(i));$$

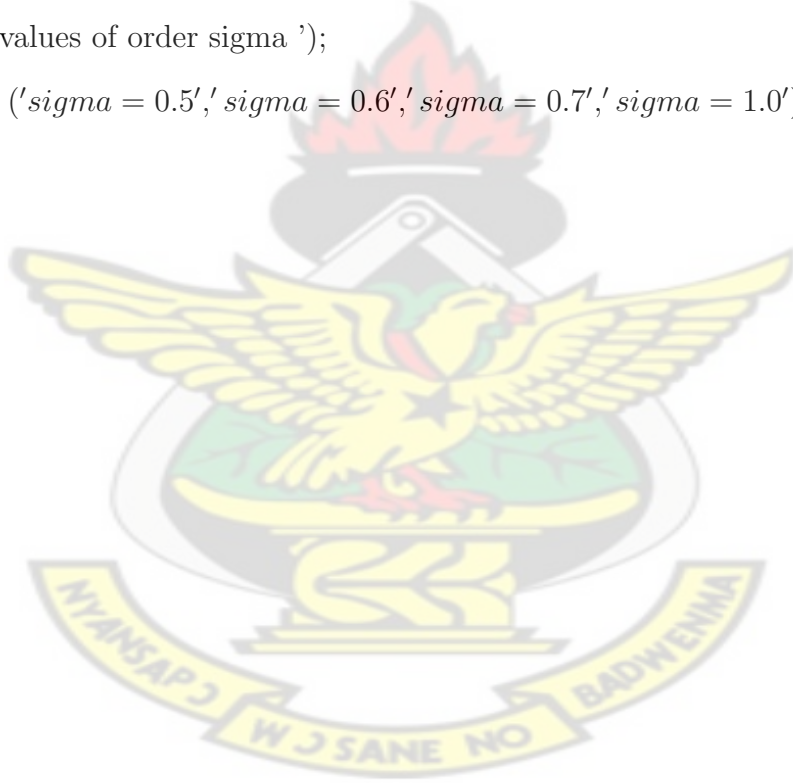
end

solS(:, k) = S(:); solI(:, k) = I(:); solN(:, k) = N(:);

end

plot(t, solN(:, 1), 'r', t, solN(:, 2), 'g', t, solN(:, 3), 'k', t, solN(:, 3), 'b'); xlabel('Time'),
ylabel('Total Population '), title('Size of the total population versus time for different values of order sigma ');
legend('sigma = 0.5', 'sigma = 0.6', 'sigma = 0.7', 'sigma = 1.0')

```



Appendix B

CODES FOR FIGURE 4.4

Title: Size of the susceptible versus time with vaccination for different values of order sigma

```
clear, clc;

b = 0.03;
beta = 0.75;
d = 0.02;
alpha = 0.1;
h = 0.1;
mu1 = 1;
mu2 = 1;
sigma = [0.5, 0.6, 0.7, 1.0];
t = 0 : h : 100; n = length(t);
m = length(sigma);
solS = []; solI = []; solN = [];
for k = 1 : m
    S = zeros(n, 1); I = S; N = S;
    S(1) = 0.95 × 103; I(1) = 0.05 × 103; N(1) = 1.0 × 103;
    for i = 1 : n - 1
        S(i + 1) = S(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - beta * S(i) * I(i) - d * S(i) - mu1 * S(i));
        I(i + 1) = I(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (beta * S(i) * I(i) - mu2 * I(i) - d * I(i) - alpha * I(i));
        N(i + 1) = N(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - d * N(i) + alpha * I(i));
    end
    solS(:, k) = S(:); solI(:, k) = I(:); solN(:, k) = N(:);
end
```

```

end

plot (t, solS(:, 1), 'r', t, solS(:, 2), 'g', t, solS(:, 3), 'k', t, solS(:, 4), 'b');

xlabel ('Time'), ylabel('Susceptible '), title('Size of the susceptible versus time
with no vaccination for different values of order sigma');

legend ('sigma = 0.5', 'sigma = 0.6', 'sigma = 0.7', 'sigma = 1.0')

```

CODES FOR FIGURE 4.5

Title: Size of the infectious versus time with treatment for different values of order sigma

```

clear, clc;

b = 0.03;

beta = 0.75;

d = 0.02;

alpha = 0.1;

h = 0.1;

mu1 = 1;

mu2 = 1;

sigma = [0.5, 0.6, 0.7, 1.0];

t = 0 : h : 100; n = length(t);

m = length(sigma);

solS = []; solI = []; solN = [];

for k = 1 : m

S = zeros(n, 1); I = S; N = S;

S(1) = 0.95 × 103; I(1) = 0.05 × 103; N(1) = 1.0 × 103;

for i = 1 : n - 1

S(i + 1) = S(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (b - beta * S(i) * I(i) - d *
S(i) - mu1 * S(i));

I(i + 1) = I(i) + (hsigma(k)/gamma(sigma(k) + 1)) * (beta * S(i) * I(i) - mu2 *

```

```

 $I(i) - d * I(i) - \alpha * I(i));$ 
 $N(i+1) = N(i) + (h^{\sigma(k)} / \gamma(\sigma(k) + 1)) * (b - d * N(i) + \alpha * I(i));$ 
end
solS(:, k) = S(:); solI(:, k) = I(:); solN(:, k) = N(:);
end
plot (t, solI(:, 1), 'r', t, solI(:, 2), 'g', t, solI(:, 3), 'k', t, solI(:, 4), 'b');
xlabel ('Time'), ylabel('Infectious '), title('Size of the infectious versus time with
no treatment for different values of order sigma ');
legend ('sigma = 0.5', 'sigma = 0.6', 'sigma = 0.7', 'sigma = 1.0')

```

