A FRACTIONAL MATHEMATICAL MODEL ON TRANSMISSION AND CONTROL OF THE SPREAD OF MYIASIS IN HUMAN SYSTEM USING INSECT REPELLENTS

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CERTIFICATION

This work is carried out by **Eze, Hyginus Ejike, PG/M.Sc./17/00063** under the supervision of Prof. G.C.E. Mbah both of the Department of Mathematics, University of Nigeria, Nsukka. It is original work and has not been submitted in part or full for any other degree to this university or any other university.

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DECLARATION

I declare that the contents of this thesis are original except where due reference has been made. It has not been submitted before for any degree to any other institution.

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DEDICATION

This work is dedicated to Almighty God for his divine wisdom and direction all through the duration of this work.

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I am eternally indebted to the Almighty God who never leaves or forsakes those who diligently trust in Him no matter what happens. My deepest appreciation also goes to my Supervisor, Prof. G.C.E. Mbah, who was more like a father to me. His patience, tolerance is indeed deeply appreciated. I thank him for reading through this work and for his useful comments. I wouldn't have finished this work without him. Thanks a million times! My sincere appreciation goes to all the lecturers in the Department of Mathematics in University of Nigeria, Nsukka. Dr. G Akuchu, Dr. D. Agbebaku, Mr. Onah Sunday to mention but a few. I want to thank in a special way Mr. Nnaemeka Stanley Aguegboh who always listened to me and helped me out. God bless him. I am also grateful to my course mates, Mr. Nnaji Daniel (Prof. of the class), Mr. Ezema Godwin (class rep.), Mr. Onuche Samuel, Mr. Eze Osita, Mr. Iyoke Paul (one-line) to mention but a few. You guys are wonderful people. I am so happy to have worked with all of them. I also wish to show my indebtedness to my elder brother, Mr. Eze Francis Chimezie who through his financial effort and show of love encouraged me all along. And also my siblings, Mr. Eze Jude, Mr. Eze Martins, Mr. Eze Stanly, Mr. Eze Livinus and Mrs. Eze Victoria. Thank God for creating a common womb for all of us to come through.

ABSTRACT

In this project, we proposed a fractional Susceptible, Expose, Infected, Treated and Recovered order model to study the transmission dynamics of Myiasis. We showed the existence of the equilibrium states. The basic reproduction number of the model was evaluated in terms of parameters in the model using the next generation matrix approach. We provided the conditions for the stability of the disease-free and the endemic equilibrium points. A detailed stability analysis of the model was carried out. Numerical simulations of the model were carried out using Adams-type predictor-corrector method which showed in detail the population dynamics of the disease. From the simulations, the level of impacts of the parameters of the model were demonstrated.

Table of Contents

CERTIFICATION ii

DECLARATION iii

DEDICATION iv

ACKNOWLEDGEMENT v

ABSTRACT vi

CHAPTER ONE 1

1.1 INTRODUCTION 1

1.2 SIGNIFICANCE OF THE STUDY 2

1.3 AIM AND OBJECTIVES OF THE STUDY 3

1.4 SCOPE OF THE STUDY 3

1.5 LIMITATIONS OF THE STUDY 3

CHAPTER TWO 4

2.1 LITERATURE REVIEW 4

2.2 THE PRESENT WORK 7

CHAPTER THREE 8

3.1 INTRODUCTION 8

3.1.1HISTORY 8

3.1.2 DEFINITION 8

3.2 FORMS OF MYIASIS 9

3.2.1 CUTANEOUS MYIASIS 9

3.2.2 BODY CAVITY MYIASIS 9

3.2.3 ACCIDENTAL MYIASIS 10

3.3 SIGNS AND SYMPTOMS 10

3.4 PREDISPOSING FACTORS 11

3.5 PREVELANCES 11

3.6 MODE OF TRANSMISSION 11

3.7 AGENT OF MYIASIS 12

3.7.1 HUMAN BOTFLY (DERMATOBIA HOMINIS) 12

3.7.2 CORDYLOBIA ANTHROPOPHAGA (TUMBU FLY): 12

3.8 VECTORS AND RESERVOIRS 12
3.9 INCUBATION PERIOD 13
3.10 CONTROL MEASURE 14
3.11 TREATMENT 14
3.11.1 SUFFOCATION APPROACHES 14
3.11.2 SURGICAL DEBRIDGEMENT 14
3.11.3 LIDOCAINE INJECTION APPROACH 15
3.12 PRELIMINARY CONCEPTS 15
3.12.1 FRACTIONAL CALCULUS 15
3.12.2 DEFINITIONS 16
CHAPTER FOUR 17
4.1 MODEL ANALYSIS 17
4.1.1 MODEL ASSUMPTIONS 17
4.1.2 MODEL VARIABLES AND PARAMETERS 18
4.1.3 MODEL DESCRIPTION 19
4.1.4 THE MODEL EQUATIONS 20
4.1.5 THE SEITR MODEL WITH CONTROL MEASURE 21
4.1.6 THE FRACTIONAL SEITR MODEL WITH CONTROL MEASURE 21
4.2 INVARIANT REGION 22
4.3 POSITIVITY OF SOLUTION 24
4.4 THE BASIC REPRODUCTION NUMBER, $\boldsymbol{R0}$. 25
4.5 EQUILIBRIUM STATE OF THE MODEL. 29
4.6 LOCAL STABILITY OF THE DISEASE FREE EQUILIBRIUM (DFE) OF THE MODEL 29
4.7 GLOBAL STABILITY OF THE DISEASE FREE EQUILIBRIUM (DFE) OF THE MODEL444 32
4.8 ENDEMIC EQUILIBRIUM POINT 35
4.9 ANALYSIS OF STABILITY OF THE ENDEMIC EQUILIBRIUM POINT 37
4.10 MODEL SOLUTION USING LAPLACE ADOMIAN DECOMPOSITION METHOD 39
4.11 NUMERICAL SIMULATIONS AND RESULTS 49
4.11.1 THE PREDICTOR-CORRECTOR MATLAB CODES FDE12.M FOR SOLVING FRACTIONAL DIFFERENTIAL EQUATIONS USING MATLAB. 49
CHAPTER FIVE 59

SUMMARY, CONCLUSION AND RECOMMENDATION 59
5.1 SUMMARY 59
5.2 CONCLUSION 59
5.3 RECOMMENDATION 60
REFERENCES 61

CHAPTER ONE

1.1 INTRODUCTION

Mathematics as a discipline, originated as a problem solving tool in the Babylonian era, where problems of flooding, storage and building were effectively tackled with the proper development of mathematical techniques. In this research, an attempt was made to study the dynamics of myiasis infection. By so doing, we developed its mathematical model through the knowledge of the study. The threshold parameters of the disease were estimated under the studied population.

Myiasis is an invasion of human and vertebrate animals by two wings flies' larvae, which for sometimes feed on the living host or swallowed food (Zumpt, 1965). It could be defined as a symbiotic invasion of tissues and organs in the vertebrates by the larvae of the dipterous flies, Demire et al. (2014).

The disease is mostly seen in the tropics and subtropics of Africa and the Americas. It can still be experienced in other parts of the earth but not as much as African and American region, (Hakeem & Bhattacharyya, 2009). In Egypt, the climatic conditions and presence of all sorts of breeding places favour the occurrence of myiasis in different districts of the country. The invasion is mostly and usually subdermal which will later create swellings on skin of the host. It could also occur on the existing wounds and on the natural body orifice, (Derraik et al., 2010).

Myiasis is a prevalent invasion on mammals (human and other vertebrate animals). In a community, individual experiences the challenge mostly when they are more close and have direct contact with other vertebrates, Ahmad et al, (2011). The occurrence of the invasion starts when the female dipteran fly deposits eggs, which at times will produce a clinical manifestation that are equivalent to the body site involved, Khan et al, (2011). According to otolaryngologists, the disease may affect the nose, ear, mouth, the skull and the neck region. Low socioeconomic status, mental retardation, poor personal hygiene, chronic suppurative otitis media are possible predisposing factors, Lane & Crosskey, (1993).

Majorly, myiasis is classified according to aspects concerned to the case in question. For this reason, the clinical explanatory of this infection is based on the affected body part of the host.

Instances are cutaneous (dermal and sub dermal), body cavity (nasopharyngeal, auricular, ophthalmic or ocular, oral), intestinal (gastric, rectal) and urogenital myiasis.

On the other hand, is the aspect of how the hosts are related to the parasite which provides the knowledge into the biology of the fly species causing the myiasis and its likely effect. Based on this relationship, it is further classified as Obligatory and Facultative, incidental or accidental. Obligatory is when the fly eggs cannot complete its lifecycle without getting in contact with the free living body hosts, while facultative is where such living body host is not essential to the life cycle of the fly eggs, (Gabriel J & John, 2009). Obligatory may be specific (primary), semi specific (secondary), or opportunistic (accidental).

1.2 SIGNIFICANCE OF THE STUDY

Since myiasis is the condition where larva of certain fly species feed on the living hosts for growth and development, the result of a retrospective study on this incidence of myiasis among human will lead to considerable reduction of the live traits and increased health disorderliness among the individuals, which results to a huge loss as it may cause imbalances of certain neurotransmitters in the brain and altered mental states, (Clarke, 2013).

The essence of the study is a matter of great concern among the medical field for human health. According to Victoria et al (2019), which they have conducted a systematic literature research from 1997 to 2017 and investigated an average of 464 international case reports in 79 countries which were calculated that the investigated cases were caused by 41 various species of flies. Therefore, it is of a great important to improve on this study for a healthy life in the society.

1.3 AIM AND OBJECTIVES OF THE STUDY

The aim of this work is to develop a mathematical model that will help in the management and control of the spread of myiasis parasites by understanding the mode of transmission, causes, preventive measure, control measure, treatment measure. The specific objectives of this work are to:

1. Build fractional order differential equation model that describes the control of flies using insect repellent.
2. Carry out mathematical detailed study on myiasis and its control by insect repellent.
3. To find out the conditions that promotes the spread of myiasis.
4. Study the control mechanisms and preventive measures of myiasis.
5. Graphical Interpretation of the result of the analysis of the model.

1.4 SCOPE OF THE STUDY

Fractional order SEITR mathematical model as a tool for the prevention of myiasis parasites was presented. The control measure was the insect repellent containing diethyltoluamide. A detailed analysis of the equilibrium points was obtained after which the graphical solution of the fractional order model equations were presented and the results were interpreted.

1.5 LIMITATIONS OF THE STUDY

The fractional-order SEITRP model with control measure and of order $\alpha \in (0,1]$ in the sense of Caputo are presented as a system of differential equations. Each compartment of the transmission stages is represented with an equation so that in all, we have a system of equations which will be studied as a representation of disease transmission model. We use the Laplace transform approach to prove the positivity of the invariant region with respect to the fractional model. Only the equilibrium and the stability analysis are carried out on the modelled equations to understand the behavior of the transmitted disease over time.

CHAPTER TWO

2.1 LITERATURE REVIEW

In this chapter, we shall look into various research work and other literature that are related to this study. A look into these reveals that there are a good number of experts who have contributed to the understanding of the dynamics of myiasis infection and its mode of transmission worldwide.

Charles & Augustus, (2004), have studied a case of a 70 years old woman who resided in Abia State, precisely Aba, South East of Nigeria. She had a seven days of "multiple itchy, painful boils" on the breast whom was later transferred to the surgery clinic as the pain and swelling of the right breast increased. Her history showed that she always went to her village where tumbu fly is endemic, spread her washed cloths on the ground and does not iron them before putting on. After diagnosing her, she was discovered with a severe painful distress, numerous discharging sinuses of which each had a whitish object at the opening and concluded of cutaneous myiasis of the breast. On treatment, fourteen larvae were extracted from the affected part and those larvae were identified as the larvae of *Cordylobia anthropophagy* by the experts on the field. To get rid of the wound, application of ampicillin was used on the affected part while tetanus toxoid injection, analgesics and daily wound dressing with eusol were also adopted to ease the wound healing.

Joachim et al. (2007), studied a case of an old man delivered from the challenge of myiasis infection who has been experiencing a painful furunculous swelling on his scalp for three days after a two weeks' journey to Costa Rica. On diagnosing, the patient's past experiences showed that it was human myiasis caused by botfly. Treatment was not so easy because of the nature of the affected part (scalp) as it was not easy to squeeze and extract the larvae. But at this stage, a sonography examination was adopted and the larvae were extracted then the wound got healed.

Henok (2009), had conducted a cross sectional study of canine cutaneous myiasis in five randomly selected kebeles of Dire Dawa Administrative council from December 2009 up to April 2010 to determine the prevalence of canine cutaneous myiasis and to assess factors that determine the occurrence of the disease specifically in dog. Active questionnaire survey among 60 households was used for which 384 dogs were sampled. From a total of 384 dogs, 162 (42%)

were living with the disease, cutaneous myiasis, of which 120 (74%) among these were harboring whereas the remaining 42(26%) observed dogs were found infested with the third and second instar larvae of Cordylobia anthropophagy. The larvae were identified in Dire Dawa regional diagnostic veterinary parasitological laboratory.

Ojemudia et al (2010), carried out a study to show the impact of animals in causing myiasis infection to human in Jos South Local Government Area, Plateau State. They started by extracting larvae at different occasions from six different myiasis patients from their difference affected parts of body. Those six patients are of different age and gender. Similar approach was used to investigate animals, dogs to be precise of which a total of five hundred and ten dogs were examined for routine checkup. At the end of the investigation, the larvae extracted from the six patients and the five hundred and ten dogs were identified as cordylobia anthropophagy. Therefore, since those larvae from both human and the animal are the same, it implies that animals have impact in increasing the rate of human myiasis.

Dibyendu et al (2014), considered a simple mathematical model to analyze the disease dynamics of Cutaneous Leishmaniasis consisting susceptible and infected populations of human and vector. In this article, their focus was to reduce the vector population so that the disease may be controlled. Here, they try to explore the effect of insecticide for controlling the vector population through impulsive mode. They established the efficiency of the insecticide spraying, which contributes a greater impact on the dynamics to move the system towards disease free situation.

Ogbalu et al (2016), studied a case of human myiasis due to lack of interest on its risk factors by Elders of the Niger Delta and South-East of Nigeria. They investigated the average of ten elderlies' patients having various cases of myiasis at their old age (92 years). After the investigation, they extracted maggots from their different parts of the body. majority of these myiasis causing agents discovered from their affected parts were tumbu fly, lucilia sericata, Musca domestica. As a result, the old villagers could not take adequate care of themselves due to their age bracket which exposed them to the health challenge. This showed that identifying predisposing factors is an essential tool in getting rid of the spread of myiasis infection.

Leonardo et al (2016), presented an epidemiological model that describes the behavior of parasite and host populations. The model was validated with real data of nestlings of the bird community

present in a thirty hectares' area in Santa Fe, Argentina. It consists of two weakly coupled population models, one for the larval population and the other for the nestling population. It takes into account, among other things, the importance of age structure for both populations, the immune response rate on the host and larval survivor rate, the incidence of larval load on host death rate, along with others. The work presented a simple and intuitive way to represent the behavior of this complex biological system and it is a good starting point for future studies.

How & Nik, (2017), reported a case of a young white lady brought to her doctor after a week complain of two thigh lumps and another on her left flank before her holiday in the Gambia. Although her doctor had treated her of having multiple skin pain, thereafter still gave her antibiotic therapy for the latest development. After two days, she started experiencing a wrinkle movement on around her affected part and was transferred to the hospital where a live maggot was extracted when pressure was applied to the lesion. The larva was identified as African tumbu fly by the parasitologist in the hospital, where she was also recommended to cover the wound with Vaseline and occlusive dressing to suffocate any further maggots on the wound.

Yeon et al (2018) studied a case of dermal and subdermal myiasis infection in Korea Republic. They attempted to explain here the situation of furunculous cutaneous myiasis caused by Cordylobia anthropophagy larvae in a Korean traveler returned from Central Africa. On the same way, a patient, 55-year-old man, had traveled to Equatorial Guinea, in Central Africa for a month and just returned to Korea. Physical examinations showed two tender erythematous nodules with small central ulceration on the left buttock and thigh. During skin examination, then two larvae came out from the affected area. The larvae were C. anthropophagy which was identified by paired mouth hooks and two posterior spiracles, which lack a distinct chitins rim. Although rarely described in Korea until now, cutaneous myiasis may be encountered more frequently with increasing international travel and exchange workers to tropical areas.

Wall et al (1993), have developed a simulation model of the dynamics of populations of the sheep blowfly, Lucilia sericata, based on a detailed knowledge of what happened on dynamics of the temperature to the rates of development of component stages of the L. sericata life-cycle. In the model the first cohort of adults emerges on day 1, nominally 1 May. On each subsequent day during the simulation, representative average daily temperatures are calculated from a third

order polynomial, fitted to daily temperatures measurements made in the west of England in 1991.

As a result, the model was used to consider effects of pre-adult mortality, such as might be produced by the application of insecticide to kill eggs or feeding larvae. A range of mortality levels were added to the simulation to remove a fixed proportion of the pre-adult individuals each life-cycle loop. Initially, these mortality levels were sustained throughout the blowfly season to simulate the effects of continuous control. The results of the simulations showed that mortality levels of at least 90% were required to halt population growth and that sustained mortality of greater than 90% each generation was required to affect a decline in the blowfly population.

2.2 THE PRESENT WORK

Most researchers in the past hardly discussed transmission dynamics of myiasis with control measure by applying fractional calculus. Inspired by this, in the present work we considered the fractional order SEITR model by using a system of fractional order differential equation in the sense of Caputo.

CHAPTER THREE

3.1 INTRODUCTION

3.1.1HISTORY

"Myia" is a Greek word meaning "fly" which was first found by (Hope FW, 1840) to conveyed the meaning of the diseases of humans caused by dipteran fly as an alternative to those that are being caused by insect larvae in a broad sense.

According to Zumpt, (1965), it is an invasion of human and vertebrate animals with two wings flies' larvae, which for sometimes feed on the living host or swallowed food.

3.1.2 DEFINITION

Myiasis is an invasion of human and vertebrate animals by two wings flies' larvae, which for sometimes feed on the living host or swallowed food (Zumpt, 1965). It can be defined as an exploitative invasion of tissues and organs in the vertebrates by the larvae of the dipterous flies, (Demire et al., 2014).

The disease is existing in human and animal bodies which are caused by different species of the Dipteran flies, known as two wing-flies such as Dermatobia Hominis (Human botfly), Cordylobia anthropophagy (Tumbu fly), Musca domestica (house fly) etc. Majorly, myiasis that emanate as a result tumbu flies and botflies infestation are popular in all species of the dipteran larvae. Although many other species that infect animal can as well do the same in human. Myiasis is an ephemeral and controllable disease. The disease is not usually fatal except if one develops negligence over it by avoiding the necessary precautions and a lack of the knowledge of its risk factors. The disease can be treated using various approaches, that will be listed on course of this work.

3.2 FORMS OF MYIASIS

Generally, the two approaches in classifying the various forms of myiasis are:

1. The classification that are based on the body part involved which is known as Clinical form.
2. The classification that are based on the connection between the host and the parasites which is known as parasitological form.

Here, we discuss the clinical form of myiasis as they have been classified based on the affected part of the host which includes cutaneous, body cavity and accidental.

3.2.1 CUTANEOUS MYIASIS

Dermal and subdermal myiasis commonly known as cutaneous is a disorderliness of the skin tissues altered by larvae of dipteran flies which as a result cause burrows or boils on the dermal layers, invade and increase the existing wounds or may form wounds themselves.

Cutaneous myiasis is a clinical form of myiasis which are subdivided into subcutaneous, migratory swellings and furunculous myiasis. Cutaneous myiasis is a short term exploitative infestation of the skin of human and other vertebrates by fly larvae, commonly known as botfly and tumbu fly larvae. In a nutshell, cutaneous myiasis is mainly caused by the larvae of Dermatobia hominis in the central and south America while the larvae of Cordylobia anthropophagy mainly caused cutaneous myiasis in Africa, (McGraw & Turiansky, 2008). Myiasis is called traumatic when an open wound is involved while it is termed furuncular when it involves boil-like lesion.

3.2.2 BODY CAVITY MYIASIS

Body cavity myiasis are the myiasis that occurs on the natural orifice such as aural, nasal, opthalmomyiasis and oral myiasis. In cavitory myiasis, the problem begins by destroying the tissue so that those larvae may gain access to the brain, where they can cause meningitis eventually leading to death. The larvae of the human botfly, the Old World and New World screwworms (*C. bezziana* and *C. hominivorax*) may invade to cause the myiasis of the eye, orbit, nose, or ear canal.

3.2.3 ACCIDENTAL MYIASIS

As the name implies, accidental myiasis is when the larvae or the egg of the dipteran flies found itself in the genitourinary environment either by having a contaminated meal which might have come in contact with the eggs or by accidentally ingesting larvae. Accidental myiasis is responsible for intestinal myiasis and urogenital myiasis. According to (Zumpt, 1965), the term "pseudomyiasis" occur when the third group of species can cause accidental myiasis as the larvae of those species got swallowed accidentally.

In parasitological terms, we talk about the parasitological form of myiasis. By this approach, it means the etymology of the spread of myiasis infection which are of two groups, namely:

1. Those that required living host for their proper growth and development termed "Obligatory parasites" and
2. Those that mainly required decaying organic matter such as carrion, feace sand, rotting vegetable, but occasionally deposit their eggs or larvae on live hosts. It is termed "Facultative parasites"

But from the facultative, their ability to initiate this infection depends on the stages those parasites found themselves. The stages are primary, secondary and tertiary. Primary stage can only initiate myiasis but secondary and tertiary stages take over from the primary stage, (Zumpt, 1965).

It should be noted that adult dipteran flies do not cause myiasis infection. In other words, the adults are generally harmless. On its life-cycle, it is at the larvae stage that is linked to myiasis since the egg stage has a very brief duration or might be even absent. Then on this larvae stage, the second instar to mature third-stage larva produce myiasis infection.

3.3 SIGNS AND SYMPTOMS

Signs exist before or during exposed period of the myiasis infection while symptoms comes during manifestation or infectious period. Therefore, the symptoms of myiasis infection includes the following:

1. A painful boils, sores and prolong wounds on the skin.
2. Nose congestion, nose blockages, nose irritation, fever and facial edema.
3. Hearing disturbances, crawling sensation in the region and buzzing noises.

4. Severe irritation in the eye and swelling of the eye.

Generally, the signs of myiasis includes boils, vision abnormalities diarrhea and vomiting.

3.4 PREDISPOSING FACTORS

Having known that myiasis infection is an ephemeral and self-limiting infection, it will be of great important to abide by some of the risk factors that may exposes individuals to the disease such as poor hygienic conditions, closeness of vicinity and domestic animals, neglecting open wounds, alcoholics and so on.

In addition to this, proper caring and visitation should be paid to those of advanced age and psychiatric disorders patients as abandoning them may lead or create an avenue for the spread of myiasis infection. Awareness campaign on this infection draws the society to the saver side since ignorance can manipulate people's mind set which may lead them to correlate such ignorance to their superstitious beliefs.

3.5 PREVELANCES

Myiasis exists in whole Africa and American region, more especially in the warm and humid environments. According to Ahmad et al (2011), it is always seen especially in the hamlet regions as the villagers correlate with domestic animals.

3.6 MODE OF TRANSMISSION

It has been stated that the spread of myiasis infection emanates from the larvae of dipteran flies. These fly larvae had also been categorized based on the affected body part and the relationship between them and host involved. In the same manner, the mode of transmission depends on the category of the fly larvae involved. For instance, there are some larvae that need a living host for their growth, as a result of that some flies attach their eggs to mosquitoes or any other flies that harbor larvae and be waiting until the mosquito bite person then the larvae will enter the bite and continue its life cycle from there, some enters human skin through people's bare feet when they step on the soil that has been mixed with these larvae or burrow into skin when one puts on cloths that were hung on the grass after washing and one may also get the infection by accidental

ingesting of the larvae either by having an open meal which might have been contaminated by larvae.

There are some larvae also that can survive on both living and dead hosts and its transmission can occur either by having a neglected open wound or sores where by flies deposit their eggs on the dead tissues around the wound and some can be transmitted to people through pet that has been infected already.

3.7 AGENT OF MYIASIS

3.7.1 HUMAN BOTFLY (DERMATOBIA HOMINIS)

In America, larvae of human botfly are the major cause of subcutaneous myiasis like furuncular myiasis although there are other forms of myiasis that could be caused by the same larvae. Its mode of transmission is very technical such the flies attach their eggs to mosquito or any other flies that harbor these eggs like ticks and wait until the mosquito bite human or the tick bite animals, then the larvae will enter the bites. In this case, mosquito and the ticks are called the mechanical vectors. According to Goddard & Edman (1997), human botfly is one of the member of Oestridae family and it is measured 1.5cm in length, yellowish in color.

3.7.2 CORDYLOBIA ANTHROPOPHAGA (TUMBU FLY):

Cordylobia anthropophagy is another species of dipteran flies that commonly found in tropical Africa. They belong to the family of Calliphoridae (Adisa & Mbanaso, 2004). They are responsible for cutaneous myiasis and their mode of transmission is by depositing their larvae on the soiled cloths or blanket that has been spread out on the ground to dry so when human come in contact with them, then the eggs will incubate and hatch. On the hatching process, they cause burrow into the human skin and eventually develop to the infection.

3.8 VECTORS AND RESERVOIRS

Before discussing why and how larvae of the dipteran flies are transmitted to their various hosts, it will be essential to know whether those larvae really in need of the hosts or not. At this point, we shall be talking obligatory, facultative or accidental parasites of human or animals.

In the dynamics of myiasis infection, various species of flies have their respective mode of transmission. For instance, it is known that botflies convey its eggs to the host by blood sucking arthropods known as mosquito. In this case, mosquito is the vector since it is not affected by the disease. In the same way, to other species of flies that transmit its larvae to the host by any means as far as it does not affect that particular means, then that means is a vector. One may consider soiled cloth or blankets to be form of mechanical vector for transmission of the eggs to the hosts.

Reservoir, as the name implies, is the storage house of the myiasis infection. In this case, the disease could affect the host as they harbor the larvae but not usually. For instance, the reservoir of tumbu fly larvae is rats.

3.9 INCUBATION PERIOD

It has been stated that in America, larvae of human botfly are the major cause of subcutaneous myiasis like furuncular myiasis although there are other forms of myiasis that could be caused by the same larvae. Its mode of transmission is very technical such the flies attach their eggs to mosquito or any other flies that harbor these eggs like ticks and wait until the mosquito bite human or the tick bite animals, then the larvae will enter the bites.

The incubation period is 8-12 days after which the larvae then develop into the second and third instars, (McGraw & Turiansky, 2008). After that, a painful lesion will start forming where the larvae will remain for up 5 to 12 weeks (90 days) in the tissues of their human or animal host then will come out from the host and to the ground to pupate, (Dawood, 2002).

As discussed earlier, Cordylobia anthropophagy is another species of dipterian flies that commonly found in tropical Africa. They belong to the family of Calliphoridae, (Adisa & Mbanaso, 2004). They are responsible for cutaneous myiasis. The incubation period of the Larvae of C. anthropophagy is varying from 8-12 days, Jelinek et al (1995). Their maturity is between 10-20 days, emerged from the host's tissues.

3.10 CONTROL MEASURE

Control in general is the ability or power to influence the behavior of something that is observed to occur or to exist (phenomenon). Therefore, control measure of the spread of myiasis infection could be categorized based on the following strategies:

1. Eradication of fly population through the reduction of their breeding environment as a result of insecticides application.
2. Abstinence from its risk factors through the improvement on environmental sanitation and hygiene to avoid the infection.
3. Application of insect repellent so as to repel mosquitoes and any other vector that will expose the host to the infection.
4. Treatment: - This will be adopted as one of the control measure once the disease starts manifesting on body of the host.

3.11 TREATMENT

Medically, treatment is an attempt to proffer remedy to a health challenge. Concerning the treatment of myiasis infection, it has been stratified into various approaches as follows:

3.11.1 SUFFOCATION APPROACHES

As the name implies, this method will block the larvae respiratory track and as a result would force this organism to the surface in search of air then allow its removal with the help of forceps to be easy, when glue and pork fat are being applied on the surface of the lesion, (Brewer & Wilson, 1993). This aim could also be achieved by application of mineral oils and petroleum jelly on the surface of the lesion (Boggild et al. 2002). Notably, care must be taken during the operation as incomplete removal may lead to granuloma formation, (Dawood, 2002).

3.11.2 SURGICAL DEBRIDGEMENT

This is another approach of treating myiasis infection. This method is not suitable for all myiasis infection treatment. The idea of surgical extraction is only required when it has been confirmed that the organismal larvae are still between second and third stages of their cycle formation (Manson, 1996). With this, it will be assured that the larvae are completely removed.

3.11.3 LIDOCAINE INJECTION APPROACH

This is another alternative method to surgical and suffocation. On this treatment, the technique of lidocaine injection is applied at the base of the tissue cavity where the larvae inhabit, therefore creating sufficient fluid pressure to extract the larvae out of the lesion where it could be easily removed (Adam et al. 2019). The limitation on this approach is that it cannot be applied in a case involving multiple larvae as the required dose of lidocaine for the multiple larvae could be toxic to the body system.

3.12 PRELIMINARY CONCEPTS

3.12.1 FRACTIONAL CALCULUS

Fractional calculus is just a name kept for history reasons and for the theory of integrals and derivatives of any order and it is far older than integers calculus. This branch of mathematics had given the edge of taking the power of real or complex number of the differential operator. The history of this fractional calculus had been in existence for past decades but as of then, it has not been popular due to lack of its knowledge among science and other field of study coupled with its complexity until few decades ago when ideas started flowing in such a way that it could be used to explain some basic nature in a better way. Although plenty of research works has carried out on real life phenomena especially on disease epidemiology with the conventional calculus. But since the development of the fractional calculus, majority of works that have done with this conventional calculus have been gone to next level by modifying them to fractional order due to what nature understand better.

Having it on ground that most of epidemiological models have been carried out by ordinary derivatives coupled with the reason that integer calculus is a subset of fractional calculus, we decided to adopt the fractional approach on this research work.

From the literature, numerous definitions were enumerated from different researchers. Among them, Riemann Liouville, Grunwald and Caputo contributed heavily but they are not generally equivalent to one another on their research (Podlubny, 1999). From the study of the three definitions above, it is found out that Caputo's definition has more edge on this study than others reason being that '' the initial conditions of fractional order differential equation with Caputo's

derivatives is the same with integer order derivatives''. Knowing this, we restrict our attention to the Caputo derivative of order $\alpha > 0$, which is rather to the real life.

For the purpose of this research work, we now list some well-known definitions and results from the literature.

3.12.2 DEFINITIONS

By Caputo, the fractional derivative of order $\alpha \in (n-1, n), n \in N$ of a function $f(t)$ is defined by :

$$D_t^{\alpha} f(t) = \frac{1}{\Gamma(\alpha - n)} \int_a^t \frac{f^{(n)}(\vartheta) d\vartheta}{(t-\vartheta)^{\alpha+1-n}}, \qquad n-1 < \alpha < n \tag{3.12.1}$$

Definition 3.12

Mittag-Leffler function of two parameter type is defined by the series expansion below:

$$E_{\alpha,\beta}(Z) = \sum_{k=0}^{\infty} \frac{Z^k}{\Gamma(\alpha k + \beta)}, \quad \alpha > 0, \beta > 0 \tag{3.12.2}$$

Definition 3.13

The Laplace transform of the Caputo derivative is defined as follow:

$$\mathrm{L}\{D_t^{\alpha} f(t)\} = s^{\alpha} f(s) - \sum_{k=0}^{n-1} s^{\alpha-k-1} f^{(k)}(0), \ n-1 < \alpha < n, \ n \in N \tag{3.12.3}$$

Here and elsewhere, Γ denotes the Gamma function.

CHAPTER FOUR

4.1 MODEL ANALYSIS

Despite many notable achievements made in the control of infectious diseases, human and vertebrates animals are still facing the challenges of diseases caused by various transmissible pathogens. This has shown us how the ecological and evolutionary dynamics of these infections has grown into a wide range of interconnected temporal, organizational and spatial scales which span hours to months, cells to ecosystems and local to global spread.

Undoubtedly, the rapid spread of these infections could be that some pathogens are transmitted directly from individuals to another, some circulate among multiple hosts, some require mechanical vector for their transmission while some could still survive in their environmental reservoirs.

Consequently, all these would pose enormous threat to human health. Therefore, to spare human life, many factors are required to be initiated for prevention and control on these diseases such like emphasis on the increment of antimicrobial resistance, increased human connectivity and changeable human behavior as a matter of fact, from local to global level.

Mathematical models offer valuable tools used for information breakdown to understand epidemiological patterns and for developing quantitative evidence for decision making in global health.

4.1.1 MODEL ASSUMPTIONS

In developing this model, the following assumptions have been considered:

1. The disease cannot kill once an individual is treated.
2. Individuals become exposed once they interact with the pathogen.
3. The mechanical vector (mosquito) and the domestic animal (reservoir host) are in the pathogen class.
4. One can recover if treated and may die due to the disease if not treated.
5. The study is carried out under rural setting.

4.1.2 MODEL VARIABLES AND PARAMETERS

Table 4.1: Model variable and parameters with their interpretation for the myiasis infection.

Variables / Parameters	Interpretations
$S(t)$	Number of individuals on the susceptible class at time (t)
$E(t)$	Number of individuals on the exposed class at time (t)
$I(t)$	Number of individuals on the infected class at time (t)
$T(t)$	Number of individuals on the treated class at time (t)
$R(t)$	Number of individuals on the recovered class at time (t)
$P(t)$	Number of organisms on the pathogen class at time (t)
$N(t)$	Total of human population size at time (t)
Λ_h	Recruitment rate of human
β	The rate at which the susceptible individuals interact with the pathogen
μ	Natural death rate for human
μ_1	Natural death rate of pathogen
δ	Death rate as a result of the disease
τ	The rate at which the exposed individuals become infected
γ	The rate at which the diseased population recovered without treatment
ρ	The rate at which the recovered individuals become susceptible
φ	The rate at which the infected individuals go for treatment
σ	The rate at which the predator feed on the pathogen
Λ_p	The rate at which female flies migrate into human population
c	Rate of adherent to the use of insect repellent

4.1.3 MODEL DESCRIPTION

The transmission dynamics of myiasis infection among the measured population is simplified to six differential equations. These six equations represent five different groups of people and the disease causing agent (pathogen) according to their epidemiological state namely: the Susceptible $S(t)$, the Exposed $E(t)$, the Infected $I(t)$, the Treated $T(t)$, the Recovered $R(t)$ and the Pathogen $P(t)$. The susceptible $S(t)$ increases due to births into the population at the rate $\wedge_h$. When there is an interaction of a susceptible individual with the pathogens, transmission occurs at a rate, β which will be controlled at $\beta(1-c)$ and altered the rate of susceptible joining the Exposed class, $E(t)$. Exposed class in this case is the incubation period of the eggs at human skin temperature. After this stage, individuals will progress to the infected class, $I(t)$, at the rate τ, and the infected class are those having the disease by showing its symptoms and signs. Some will recover without therapy at a rate γ, while some after treatment (therapy) will progress to treatment class, $T(t)$ at the rate, φ. The treated individuals will join the recovered class, $R(t)$ at the proportion, $(1-\gamma)$ since some infected individuals may die if not treated due to the disease and the disease induce death rate will be δ. The Recovered class, $R(t)$ consists of those that have recovered from the disease and they can become susceptible again at the rate ρ. In addition, individuals could die by nature at a rate μ in each compartment.

In the pathogen class, $P(t)$, it increases due to number of adult female flies migrating into the human population environment at the rate $\wedge_{p,}$ where predators feed on the pathogen at the rate σ, and the pathogen can as well die naturally at the rate μ_1.

Taking into account of this, we formulate model. The models can be described by a flow chart as shown below:

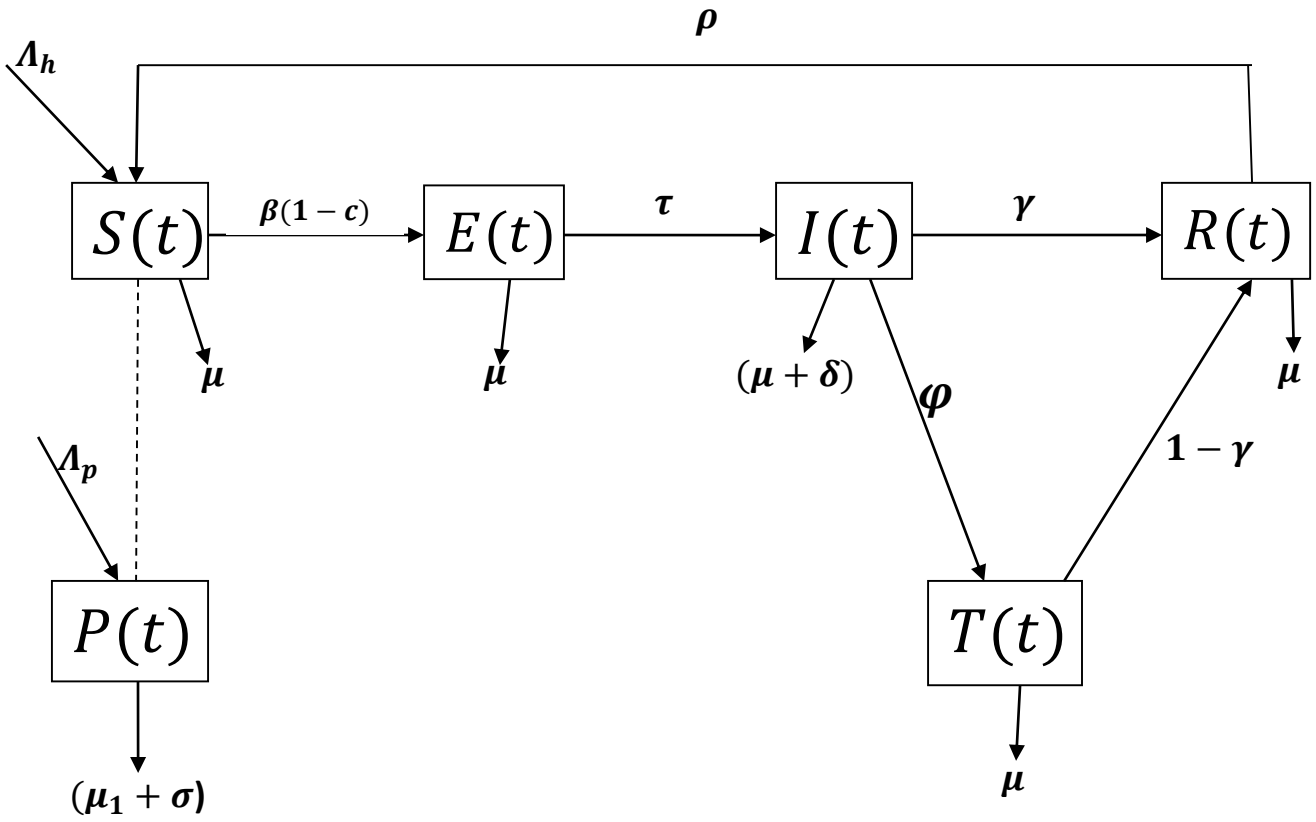

FIGURE 4.1 FLOW DIAGRAM OF THE MODEL

4.1.4 THE MODEL EQUATIONS

With the application of above listed assumptions and model description, transmission dynamics of myiasis can be described by the following differential equations:

4.1.5 THE SEITR MODEL WITH CONTROL MEASURE

$$\begin{aligned}
\frac{dS}{dt} &= \wedge_h + \rho R - \beta(1-c)SP - \mu S \\
\frac{dE}{dt} &= \beta(1-c)SP - E(\mu+\tau) \\
\frac{dI}{dt} &= \tau E - I(\gamma+\varphi+\mu+\delta) \\
\frac{dT}{dt} &= \varphi I - T[\mu+(1-\gamma)] \\
\frac{dR}{dt} &= \gamma I + (1-\gamma)T - R(\mu+\rho) \\
\frac{dP}{dt} &= \wedge_P - P(\mu_1+\sigma)
\end{aligned} \tag{4.1}$$

In this research work, we focused our attention on fractional order , α of above equation (4.1) using Caputo definition. Therefore, having this in mind we give the fractional order model with control measure.

4.1.6 THE FRACTIONAL SEITR MODEL WITH CONTROL MEASURE

Having considered the accuracy of the order of integer and fractional derivatives equivalent, we then replace the integer order with fractional order,$\alpha \in (0,1]$ in Caputo sense. The fractional-order SEITRP model with control measure is given as follows:

$$\begin{aligned}
f_1 &= D_t^{\alpha} S(t) = \wedge_h + \rho R - \beta(1-c)SP - \mu S \\
f_2 &= D_t^{\alpha} E(t) = \beta(1-c)SP - E(\mu+\tau) \\
f_3 &= D_t^{\alpha} I(t) = \tau E - I(\gamma+\varphi+\mu+\delta) \\
f_4 &= D_t^{\alpha} T(t) = \varphi I - T[\mu+(1-\gamma)] \\
f_5 &= D_t^{\alpha} R(t) = \gamma I + (1-\gamma)T - R(\mu+\rho) \\
f_6 &= D_t^{\alpha} P(t) = \wedge_p - P(\mu_1+\sigma)
\end{aligned} \tag{4.2}$$

By setting $\alpha = 1$, the system of equation above can be reduced to integer order system.

With the non-negative initial condition:

$S(0) = S_0\,, E(0) = E_0, I(0) = I_0, T(0) = I_{T0}, R(0) = R_0, P(0) = P_0.$

$N = S + E + I + T + R + P, (S, E, I, I_T, R, P) \in \mathcal{R}_+^6$

$\dot{\mathrm{N}}_h = \dot{\mathrm{S}} + \dot{E} + \dot{I} + \dot{\mathrm{T}} + \dot{\mathrm{R}}$ (4.2a)

By adding equation (4.2a), we have:

$D_t^\alpha N_h = \wedge_h - \mu N_h - \delta I$ (4.2b)

But at DFE, no disease hence $\delta = 0$, so equation (4.2b) become

$D_t^\alpha N_h = \wedge_h - \mu N_h$ (4.2c)

So that $\wedge_h - \mu N_h = 0$ or $N_h = \frac{\wedge_h}{\mu}$, where $N_h = population\ of\ human.$

4.2 INVARIANT REGION

Lemma 4.1

Given a closed set $\Omega = \{(S, E, I, T, R) \in R_+^5 : S + E + I + T + R = N_h\}$. By the model (4.2), all solutions of the model with initial conditions in Ω remain in Ω.

Proof:

The fractional derivative of the total human population, obtained by adding all the human equations of model (4.2), is given by system (4.2c).

The Laplace transform of (4.2c) gives:

$s^\alpha N_h(s) - s^{\alpha-1} N_h(0) = \frac{\wedge}{S} - \mu N_h(s)$ (4.2d)

$N_h(s) = \frac{\Lambda_h}{S(S^\alpha + \mu)} + \frac{S^{\alpha-1}}{S^\alpha + \mu} N_h(0)$ (4.2e)

Taking the inverse Laplace transform of (4.2e), we have:

$$N(t) = N(0)E_{\alpha,1}(-\mu t^{\alpha}) + \wedge t^{\alpha}E_{\alpha,\alpha+1}(-\mu t^{\alpha}) \quad (4.2f)$$

where $E_{\propto,\beta}$ could written as function in Mittag-leffler form. But since the Mittag-Leffler functions has an asymptotic behavior, it follows that:

$$E_{\alpha,1}N(t) = \sum_{k=0}^{\infty} \frac{N^k(t)}{\Gamma(\alpha k+1)}, \ \alpha > 0, \quad (4.2g)$$

$$E_{\alpha,\alpha+1}N(t) = \sum_{k=0}^{\infty} \frac{N^k(t)}{\Gamma(\alpha k+\alpha+1)}, \ \alpha > 0, \quad (4.2i)$$

Expanding (4.2g), we have

$$E_{\alpha,1}N(t) = \frac{1}{\Gamma(1)} + \frac{N(t)}{\Gamma(\alpha+1)} + \frac{N^2(t)}{\Gamma(2\alpha+1)} + \cdots$$

Expanding (4.2i), we have

$$E_{\alpha,\alpha+1}N(t) = \frac{1}{\Gamma(\alpha+1)} + \frac{N(t)}{\Gamma(2\alpha+1)} + \frac{N^2(t)}{\Gamma(3\alpha+1)} + \cdots$$

Since Mittag-Leffler function has an asymptotic property, we have

$$N(t) = 1 + O(N)$$

Taking limit as $k \to \infty$, we have

$$N(t) \approx 1$$

Then, it is clear that Ω is a positive invariant set. Therefore, all solutions of the model with initial conditions in Ω remain in Ω for all $t > 0$. ■

4.3 POSITIVITY OF SOLUTION

Theorem 4.1

The solution of the human equation of the model (4.2), are feasible at $t > 0$ if they enter the invariant region Ω.

Proof

Total population of human is obtained by adding all the human equations of model (4.2), given as:

$$D_t^{\alpha} N_h = \Lambda_h - \mu N_h$$

Taking $\alpha = 1$,

$$\frac{dN_h}{dt} = \Lambda_h - \mu N_h$$

$$\frac{dN_h}{dt} + \mu N_h = \Lambda_h \quad the\ integrating\ factor\ (I.F) = e^{\mu t}$$

$\Rightarrow$

$$e^{\mu t}\left(\frac{dN_h}{dt} + \mu N_h\right) = \Lambda_h e^{\mu t}$$

$$\frac{d}{dt}(N_h e^{\mu t}) = \Lambda_h e^{\mu t}$$

$$N_h e^{\mu t} = \int \Lambda_h e^{\mu t} dt$$

$$N_h e^{\mu t} = \frac{\Lambda_h}{\mu} e^{\mu t} + e^{C} \qquad e^{C} = const.of\ int$$

$\Rightarrow$

$$N_h(t) = \frac{\Lambda_h}{\mu} + e^{C-\mu t}$$

At $t = 0$,

$N_h(0) \Rightarrow$

$\frac{\Lambda_h}{\mu} + e^C = 0$

$\Rightarrow$

$e^C = -\frac{\Lambda_h}{\mu}$

$\therefore \quad N_h(t) = \frac{\Lambda_h}{\mu} - \frac{\Lambda_h}{\mu} e^{-\mu t}$

$N_h(t) = \frac{\Lambda_h}{\mu}(1 - e^{-\mu t})$

But, $(1 - e^{-\mu t}) = 1 - \frac{1}{e^{\mu t}} \geq 0 \; as \; t \to \infty$

$\therefore$

$N_h(t) \geq 0$

It implies that the set $\Omega = [S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, T(0) \geq 0, R(0) \geq 0]$

Therefore, the solution of the human equation of the model (4.2), are mathematically well posed

■

4.4 THE BASIC REPRODUCTION NUMBER, R_0.

The basic reproduction number, R_O is defined as the expected number of secondary infections produced by an index case in a completely susceptible population, (Anderson & May, 1991). This number is a measure of the potential for disease spread within a population. The basic reproductive ratio, R_O, is also defined as the expected number of secondary infections arising from a single individual during his or her entire infectious period, in a population of susceptible, Heffernan et al (2005). This concept is fundamental to the study of epidemiology and within-host pathogen dynamics. Most importantly, R_0 often serves as a threshold parameter that predicts whether an infection will spread. Related parameters which share this threshold behavior, however, may or may not give the true value of R_0. A paper by Hurford et al (2009), provided a

method for calculating R_0, which is the formation of the Next Generation Matrix. It is comprised of two parts: F and V^{-1}, where,

$$F = \left|\frac{\partial f_i(x_o)}{\partial x_j}\right| , is\ non\ negative\ Matrix$$

$$V = \left|\frac{\partial v_i(x_o)}{\partial x_j}\right| , is\ non\ singular\ Matrix$$

The f_i are the new infections, while the v_i are transfer of infection from one compartment to another. x_o is the disease free equilibrium point. R_0 is the spectral radius of the next generation matrix, which is the dominant Eigenvalue of the same matrix. To calculate this, we consider the Exposed compartments, infected compartment, treated compartment and Pathogen compartment as follow:

$$\left.\begin{aligned} D_t^\alpha E(t) &= \beta(1-c)SP - E(\mu+\tau) \\ D_t^\alpha I(t) &= \tau E - I(\gamma+\varphi+\mu+\delta) \\ D_t^\alpha T(t) &= \varphi I - T[\mu+(1-\gamma)] \\ D_t^\alpha P(t) &= \Lambda_p - P(\mu_1+\sigma) \end{aligned}\right\} \qquad 4.7.1$$

Define:

$$f_i = \begin{pmatrix} \beta(1-c)SP \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$V_i = \begin{pmatrix} \mu E + \tau E \\ I(\gamma+\varphi+\mu+\delta) - \tau E \\ T[\mu+(1-\gamma)] - \varphi I \\ P(\mu_1+\sigma) - \Lambda_P \end{pmatrix}$$

The Jacobian of f_i and V_i with respect to E, I, T, and P are:

$$F = \begin{pmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial P} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial P} \\ \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial P} \\ \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial P} \end{pmatrix}, \quad V = \begin{pmatrix} \frac{\partial V_1}{\partial E} & \frac{\partial V_1}{\partial I} & \frac{\partial V_1}{\partial T} & \frac{\partial V_1}{\partial P} \\ \frac{\partial V_2}{\partial E} & \frac{\partial V_2}{\partial I} & \frac{\partial V_2}{\partial T} & \frac{\partial V_2}{\partial P} \\ \frac{\partial V_3}{\partial E} & \frac{\partial V_3}{\partial I} & \frac{\partial V_3}{\partial T} & \frac{\partial V_3}{\partial P} \\ \frac{\partial V_4}{\partial E} & \frac{\partial V_4}{\partial I} & \frac{\partial V_4}{\partial T} & \frac{\partial V_4}{\partial P} \end{pmatrix}$$

Where $i = 1, 2, 3$ & 4 for each class

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta(1-c)S \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \qquad V = \begin{pmatrix} \mu+\tau & 0 & 0 & 0 \\ -\tau & \gamma+\varphi+\mu+\delta & 0 & 0 \\ 0 & -\varphi & \mu+1-\gamma & 0 \\ 0 & 0 & 0 & \mu_1+\sigma \end{pmatrix}$$

Where,

$V^{-1} = \frac{adjoint\ of\ V}{det.of\ V}$

$for\ adjoint\ of\ V = \ transpose\ of\ cofactor\ of\ V.$

$$|V| = \begin{vmatrix} \mu+\tau & 0 & 0 & 0 \\ -\tau & \gamma+\varphi+\mu+\delta & 0 & 0 \\ 0 & -\varphi & \mu+1-\gamma & 0 \\ 0 & 0 & 0 & \mu_1+\sigma \end{vmatrix}$$

$$V^{-1} = \frac{1}{abcd}\begin{pmatrix} bcd & 0 & 0 & 0 \\ \tau cd & acd & 0 & 0 \\ \tau\varphi d & a\varphi d & abd & 0 \\ 0 & 0 & 0 & abc \end{pmatrix} \qquad V^{-1} = \begin{pmatrix} \frac{1}{a} & 0 & 0 & 0 \\ \frac{\tau}{ab} & \frac{1}{b} & 0 & 0 \\ \frac{\tau\varphi}{abc} & \frac{\varphi}{bc} & \frac{1}{c} & 0 \\ 0 & 0 & 0 & \frac{1}{d} \end{pmatrix}$$

Where

$a = \mu + \tau, \ \ b = \gamma + \varphi + \mu + \delta, \qquad c = \mu + (1-\gamma), \ \ d = \mu_1 + \sigma.$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \beta(1-c)S \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{a} & 0 & 0 & 0 \\ \frac{\tau}{ab} & \frac{1}{b} & 0 & 0 \\ \frac{\tau\varphi}{abc} & \frac{\varphi}{bc} & \frac{1}{c} & 0 \\ 0 & 0 & 0 & \frac{1}{d} \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta(1-c)S}{d} \end{pmatrix}$$

$$= \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta(1-c)S}{\mu_1+\sigma} \end{pmatrix}$$

The dominant eigenvalue of FV^{-1} is obtained from the characteristic equation, $|FV^{-1} - \lambda I| = 0$, where λ= eigenvalues and I = 4 by 4 identity matrix. Thus, we have

$$\begin{vmatrix} -\lambda & 0 & 0 & 0 \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & \frac{\beta(1-c)S}{\mu_1+\sigma} - \lambda \end{vmatrix} = 0$$

$$-\lambda^3\left(\frac{\beta(1-c)S}{\mu_1+\sigma} - \lambda\right) = 0$$

$$\frac{\beta(1-c)S}{\mu_1+\sigma} - \lambda = 0$$

$$\lambda = \frac{\beta(1-c)S}{\mu_1+\sigma} = the\ dominant\ eigenvalue$$

$$R_0 = \frac{\beta(1-c)S}{\mu_1+\sigma}$$

Evaluating above at DFE gives

$$R_0 = \frac{\beta(1-c)\Lambda_h}{\mu(\mu_1+\sigma)}$$

Remark 4.1

Consider the threshold value, $R_0 = \frac{\beta(1-c)\wedge_h}{\mu(\mu_1+\sigma)}$, which show the basic reproduction number of the system (4.2). The biological interpretation of R_0 is that the disease will invade the population if it exceeds 1, and will die out if R_0 is less than 1.

4.5 EQUILIBRIUM STATE OF THE MODEL.

An equilibrium point is a point at which variables of a system remain unchanged over time. The equilibrium point of system (4.2) satisfies the following system:

$$D^{\propto}S(t) = D^{\propto}E(t) = D^{\propto}I(t) = D^{\propto}T(t) = D^{\propto}R(t) = D^{\propto}P(t) = 0 \qquad (4.8.1)$$

Such that the system (4.2) becomes

$$\left.\begin{aligned} 0 &= \wedge_h + \rho R^0 - \beta S^0 P^0(1-c) - \mu S \\ 0 &= \beta S^0 P^0(1-c) - E^0(\mu+\tau) \\ 0 &= \tau E^0 - I^0(\gamma+\varphi+\mu+\delta) \\ 0 &= \varphi I^0 - T^0[\mu+(1-\gamma)] \\ 0 &= \gamma I^0 + (1-\gamma)T^0 - R^0(\mu+\rho) \\ 0 &= \wedge_p - P^0(\mu_1+\sigma) \end{aligned}\right\} \qquad (4.8.2)$$

4.6 LOCAL STABILITY OF THE DISEASE FREE EQUILIBRIUM (DFE) OF THE MODEL

The disease free equilibrium is the scenario at which the population remains in the absence of myiasis, hence nobody is infected or recovered, and this implies that $E^0 = I^0 = T^0 = R^0 = 0$.

The disease free equilibrium (DFE) of the disease is obtained by setting:

$$D^{\propto}S^0(t) = D^{\propto}E^0(t) = D^{\propto}I^0(t) = D^{\propto}T^0(t) = D^{\propto}R^0(t) = D^{\propto}P^0(t) = 0$$

On solving the right hand side of equation (4.8.2), we get the disease free equilibrium state of the model.

$$0 = \Lambda_h + \rho R - \beta S^0 P^0 (1 - c) - \mu S^0$$

$$\mu S^0 = \Lambda_h$$

$$\Rightarrow S^0 = \frac{\Lambda_h}{\mu}$$

Thus, the DFE is given by:

$$(S^0, E^0, I^0, T^O, R^0, P^0) = \left(\frac{\Lambda_h}{\mu}, 0,0,0,0,0\right) \quad (4.9.1)$$

To evaluate the local stability of the disease free equilibrium (DFE) of the model. We form the Jacobian matrix of the model (4.2) given by:

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial P} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial P} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial P} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial P} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial P} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial T} & \frac{\partial f_6}{\partial R} & \frac{\partial f_6}{\partial P} \end{pmatrix}$$

$$J = \begin{pmatrix} -\beta P(1-c) - \mu & 0 & 0 & 0 & \rho & -\beta P(1-c) \\ \beta P(1-c) & -(\mu + \tau) & 0 & 0 & 0 & \beta S(1-c) \\ 0 & \tau & -(\gamma + \varphi + \mu + \delta) & 0 & 0 & 0 \\ 0 & 0 & \varphi & -[\mu + (1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu + \rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1 + \sigma) \end{pmatrix}$$

At disease free equilibrium (DFE), we have

$$J^0 = \begin{pmatrix} -\mu & 0 & 0 & 0 & \rho & 0 \\ 0 & -(\mu+\tau) & 0 & 0 & 0 & \beta S(1-c) \\ 0 & \tau & -(\gamma+\varphi+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \varphi & -[\mu+(1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) \end{pmatrix}$$

$|J^0 - \lambda I| = 0$

$\Rightarrow$

$$\begin{vmatrix} -\mu-\lambda & 0 & 0 & 0 & \rho & 0 \\ 0 & -(\mu+\tau)-\lambda & 0 & 0 & 0 & \beta S(1-c) \\ 0 & \tau & -(\gamma+\varphi+\mu+\delta)-\lambda & 0 & 0 & 0 \\ 0 & 0 & \varphi & -[\mu+(1-\gamma)]-\lambda & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho)-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma)-\lambda \end{vmatrix}$$

$= 0$

$\Rightarrow$

$$\begin{vmatrix} -a-\lambda & 0 & 0 & 0 & \rho & 0 \\ 0 & -b-\lambda & 0 & 0 & 0 & \beta S(1-c) \\ 0 & \tau & -c-\lambda & 0 & 0 & 0 \\ 0 & 0 & \varphi & -d-\lambda & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -h-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -f-\lambda \end{vmatrix} = 0 \qquad (4.9.2)$$

Where $a = \mu,\ b = \mu + \tau,\ c = (\gamma + \varphi + \mu + \delta),\ d = [\mu + (1-\gamma)],\ h = \mu + \rho\ \ and$

$f = \mu_1 + \sigma.$

The characteristics equation of the above matrix (4.6c) becomes:

$(-a-\lambda)(-b-\lambda)(-c-\lambda)(-d-\lambda)(-h-\lambda)(-f-\lambda) = 0$

$\Rightarrow$

$\lambda_1 = -a = -\mu$

$\lambda_2 = -b = -(\mu + \tau)$

$\lambda_3 = -c = -(\gamma + \emptyset + \mu + \delta)$

$\lambda_4 = -d = -[\mu + (1 - \gamma)]$

$\lambda_5 = -h = -(\mu + \rho)$

$\lambda_6 = -f = -(\mu_1 + \sigma)$

Since all the above eigenvalue are less than zero, then the equilibrium point $\left(\frac{\Lambda_h}{\mu}, 0,0,0,0,0\right)$ is locally asymptotically stable.

4.7 GLOBAL STABILITY OF THE DISEASE FREE EQUILIBRIUM (DFE) OF THE MODEL444

We have our Reproduction number as:

$$R_0 = \frac{\beta \Lambda_h (1 - c)}{\mu(\mu_1 + \sigma)} \qquad (4.7.1)$$

To construct a Lyapunov function for the model, we use the following steps:

Let $V = A_1 E + A_2 I + A_3 T, \ \ where\ A_1, A_2, A_3 > 0$ (4.7.2)

The function (4.7.2) is continuous and has its unique minimum at equilibrium point. Then

$\dot{V} = A_1 \dot{E} + A_2 \dot{I} + A_3 \dot{T}$

$$\dot{V} = \frac{\partial V}{\partial E}\frac{dE}{dt} + \frac{\partial V}{\partial I}\frac{dI}{dt} + \frac{\partial V}{\partial T}\frac{dT}{dt}$$

$$= A_1[\beta(1 - C)SP - E(\mu + \tau)] + A_2[\tau E - I(\gamma + \varphi + \mu + \delta)] + A_3[\varphi I - T(\mu + 1 - \gamma)] \qquad (4.7.3)$$

Let $\lambda = \beta P(1 - c)$ be the force of infection

$$\therefore\ \dot{V} = A_1[\lambda S - E(\mu + \tau)] + A_2[\tau E - I(\gamma + \varphi + \mu + \delta)] + A_3[\varphi I - T(\mu + 1 - \gamma)] \qquad (4.7.4)$$

Then, we rearrange (4.7.4) to factor out the variables:

$$\dot{V} = S[A_1\lambda] - E[A_1(\mu+\tau) - A_2\tau] - I[(\gamma+\varphi+\mu+\delta)A_2 - A_3\emptyset] - T[A_3(\mu+1-\gamma)] \quad (4.7.5)$$

Then,

1. Equating the coefficient of force of infection to the numerator of R_0 (neglecting the contact rate).
2. Equate the coefficient of "E" to zero.
3. Equate the coefficient of I and T to the denominator of R_0. That is,

$$A_1 = \frac{\Lambda_h}{S} \quad (3i)$$

$$A_2(\gamma+\varphi+\mu+\delta) - A_3\varphi = \mu(\mu_1+\sigma) \quad (3ii)$$

$$A_3(\mu+1-\gamma) = \mu(\mu_1+\sigma) \quad (3iii)$$

$$A_1(\mu+\tau) - A_2\tau = 0 \quad (3iv)$$

From $(3i)$,

$$A_1 = \frac{\Lambda_h}{S}$$

From $(3iii)$,

$$A_3 = \frac{\mu(\mu_1+\sigma)}{\mu+1-\gamma}$$

From $(3ii)$,

$$A_2 = \frac{A_3\varphi + \mu(\mu_1+\sigma)}{\gamma+\varphi+\mu+\delta}$$

$$= \frac{\frac{\varphi\mu(\mu_1+\sigma)}{\mu+1-\gamma} + \mu(\mu_1+\sigma)}{\gamma+\varphi+\mu+\delta}$$

$$= \mu(\mu_1+\sigma)\left(\frac{\varphi+(\mu+1-\gamma)}{(\mu+1-\gamma)(\gamma+\varphi+\mu+\delta)}\right)$$

$\therefore$

$$A_2 = \mu(\mu_1 + \sigma)\left(\frac{\varphi + \mu + 1 - \gamma}{(\mu + 1 - \gamma)(\gamma + \varphi + \mu + \delta)}\right)$$

Then we substitute the value of $A_1, A_2\ and\ A_3$ into (4.7.5), and then we have

$$\dot{V} = S[A_1\lambda] - I[(\gamma + \varphi + \mu + \delta)A_2 - A_3\varphi] - T[A_3(\mu + 1 - \gamma)] \qquad (4.7.6)$$

Since $E[A_1(\mu + \tau) - A_2\tau] = 0$

From (4.7.6), we have

$$\dot{V} = S[A_1\lambda] - I\left(\frac{\mu(\mu_1 + \sigma)(\varphi + \mu + 1 - \gamma)}{\mu + 1 - \gamma} - \frac{\mu\varphi(\mu_1 + \sigma)}{\mu + 1 - \gamma}\right) - T\left(\frac{\mu(\mu_1 + \sigma)(\varphi + \mu + 1 - \gamma)}{\gamma + \varphi + \mu + \delta}\right)$$

$$\dot{V} = S[A_1\lambda] - I\left(\frac{\mu(\mu_1 + \sigma)}{\mu + 1 - \gamma}\right)(\varphi + \mu + 1 - \gamma - \varphi) - T\left(\frac{\mu(\mu_1 + \sigma)(\varphi + \mu + 1 - \gamma)}{\gamma + \varphi + \mu + \delta}\right)$$

$$\dot{V} = S[A_1\lambda] - I\left(\frac{\mu(\mu_1 + \sigma)}{\mu + 1 - \gamma}\right)(\mu + 1 - \gamma) - T\left(\frac{\mu(\mu_1 + \sigma)(\varphi + \mu + 1 - \gamma)}{\gamma + \varphi + \mu + \delta}\right)$$

$$\dot{V} = S\left[\lambda\frac{\Lambda_h}{S}\right] - I(\mu(\mu_1 + \sigma)) - T(\mu(\mu_1 + \sigma))\left(\frac{\varphi + \mu + 1 - \gamma}{\gamma + \varphi + \mu + \delta}\right)$$

$$\dot{V} = S\left[\lambda\frac{\Lambda_h}{S}\right] - \mu(\mu_1 + \sigma)\left(I + T\frac{\varphi + \mu + 1 - \gamma}{\gamma + \varphi + \mu + \delta}\right)$$

Recall $\lambda = P\beta(1 - c)$

$$Let\ \frac{\varphi + \mu + 1 - \gamma}{\gamma + \varphi + \mu + \delta} = K_1$$

$$\dot{V} = P\beta\Lambda_h(1 - c) - \mu(\mu_1 + \sigma)(I + TK_1)$$

$$\dot{V} = \mu(\mu_1 + \sigma)(I + TK_1)\left(\frac{P\beta\ \wedge_h (1 - c)}{(\mu(\mu_1 + \sigma))(I + TK_1)} - 1\right)$$

$$\dot{V} = \mu(\mu_1 + \sigma)(I + TK_1)\left(\frac{P}{I + TK_1} * \frac{\beta \wedge_h (1-c)}{\mu(\mu_1 + \sigma)} - 1\right)$$

$$\dot{V} = \mu(\mu_1 + \sigma)(I + TK_1)\left(\frac{P}{I + TK_1} * R_O - 1\right)$$

If $P \leq I + TK_1$, and

- If $R_O < 1 \Rightarrow \dot{V} < 0$ *Implies its Global stable*
- If $R_O > 1 \Rightarrow \dot{V} > 0$ *Implies its Global unstable*.

4.8 ENDEMIC EQUILIBRIUM POINT

The endemic equilibrium point is the point where the disease cannot be totally eradicated but remains in the population. Here, it is assumed that there is myiasis in the population. And for myiasis to persist in the population, the classes must not be zero at equilibrium state. This implies that $S(t) \neq 0, E(t) \neq 0, I(t) \neq 0,\ T(t) \neq 0, R(t) \neq 0, and\ P(t) \neq 0$. in other words, if $E_+ = (S^*, E^*, I^*, T^*, R^*, P^*)$ is the endemic equilibrium point, then $(S^*, E^*, I^*, T^*, R^*, P^*) \neq (0,0,0,0,0,0)$.

In order to obtain the endemic equilibrium point, we solve the system of equations (4.8.2) simultaneously. From the system (4.8.2), at equilibrium, we have:

$$0 = \Lambda_h + \rho R^* - \beta S^* P^* (1-c) - \mu S^* - -(i)$$

$$0 = \beta S^* P^* (1-c) - E^*(\mu + \tau) - - - -(ii)$$

$$0 = \tau E^* - I^*(\gamma + \varphi + \mu + \delta) - - - -(iii)$$

$$0 = \varphi I^* - T^*[\mu + (1-\gamma)] - - - - - (iv)$$

$$0 = \gamma I^* + (1-\gamma)T^* - R^*(\mu + \rho) - - - (v)$$

$$0 = \Lambda_p - P^*(\mu_1 + \sigma) - - - - - - - (vi)$$

$From\ (vi),$

$$P^* = \frac{\Lambda_p}{\mu_1 + \sigma} = K_1$$

$From\ (iii),$

$$E = \frac{I^*(\gamma + \varphi + \mu + \delta)}{\tau} = I^* K_2 \qquad where\ \ K_2 = \frac{(\gamma + \varphi + \mu + \delta)}{\tau}$$

$From(iv),$

$$T^* = \frac{\varphi I^*}{\mu + (1-\gamma)} = I^* K_3, \quad where\ \ K_3 = \frac{\varphi}{\mu + (1-\gamma)}$$

$From\ (v),$

$$R^* = \frac{\gamma I^*}{\mu + \rho} + \frac{(1-\gamma)}{\mu + \rho} T^* = \frac{\gamma I}{\mu + \rho} + \frac{(1-\gamma)}{\mu + \rho} * \frac{\varphi I}{\mu + (1-\gamma)}$$

$\Rightarrow$

$$R^* = \frac{I}{\mu + \rho}\left(\gamma + \frac{\varphi(1-\gamma)}{\mu + (1-\gamma)}\right) = I^* K_4, where\ K_4 = \frac{1}{\mu + \rho}\left(\gamma + \frac{\varphi(1-\gamma)}{\mu + (1-\gamma)}\right)$$

$From\ (ii),$

$$\frac{\beta S^* \Lambda_p (1-c)}{\mu_1 + \sigma} = E^*(\mu + \tau)$$

$$S^* = \frac{E^*(\mu + \tau)(\mu_1 + \sigma)}{\beta \Lambda_p (1-c)}$$

$\Rightarrow$

$$S^* = \frac{I(\gamma + \varphi + \mu + \delta)(\mu + \tau)(\mu_1 + \sigma)}{\tau \beta \Lambda_p (1-c)} = I^* K_5, \ \ wher\ K_5$$

$$= \frac{(\gamma + \varphi + \mu + \delta)(\mu + \tau)(\mu_1 + \sigma)}{\tau \beta \Lambda_p (1-c)}$$

Therefore,

$$E_+ = (I^* K_5, I^* K_2, I^* K_3, I^* K_4, K_1)$$

4.9 ANALYSIS OF STABILITY OF THE ENDEMIC EQUILIBRIUM POINT

Definition 4.1

A square matrix $'A' = (a_{ij})$ is called a $'Z'$ matrix or said to have a Z-sign pattern if $a_{ij} > 0, \forall\, i = j$ and $a_{ij} \leq 0, \forall\, i \neq j$.

Definition 4.2

Let $A = a_{ij}$ be a Z matrix. Then, Z is called an M matrix if all the leading principal minors of Z are positive.

Lemma 4.2

Let A be a square matrix. Then, A is non-singular if it is strictly diagonally dominant.

Lemma 4.3

In a Z matrix $A = (a_{ij})$, $if\ \ \sum_{j=1}^{5} a_{ij} > 0,\ \forall\, i,$ then the leading principal minors are all positive.

Lemma 4.4

If a matrix $'A' = (a_{ij})$ is an M- matrix, then all its eigenvalues have positive real parts.

The Jacobian matrix of the model is given by:

$$J = \begin{pmatrix} -\beta P(1-c) - \mu & 0 & 0 & 0 & \rho & -\beta P(1-c) \\ \beta P(1-c) & -(\mu+\tau) & 0 & 0 & 0 & \beta S(1-c) \\ 0 & \tau & -(\gamma + \emptyset + \mu + \delta) & 0 & 0 & 0 \\ 0 & 0 & \emptyset & -[\mu + (1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1 + \sigma) \end{pmatrix}$$

The Jacobian matrix$J(E_+)$, evaluated at the endemic equilibrium point is given by

$J(E_+)$

$$= \begin{pmatrix} -\beta K_1(1-c)-\mu & 0 & 0 & 0 & \rho & -\beta K_1(1-c) \\ \beta K_1(1-c) & -(\mu+\tau) & 0 & 0 & 0 & \beta I K_5(1-c) \\ 0 & \tau & -(\gamma+\emptyset+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \emptyset & -[\mu+(1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) \end{pmatrix}$$

Where

$$K_1 = P = \frac{\wedge_p}{\mu_1+\sigma}$$

$$IK_5 = S = \frac{I(\gamma+\emptyset+\mu+\delta)(\mu+\tau)(\mu_1+\sigma)}{\tau\beta\wedge_p(1-c)}.$$

The eigenvalues are obtained from the characteristics equation: $|J(E_+) - \lambda I| = 0$

Thus,

$$\begin{vmatrix} -\beta K_1(1-c)-\mu & 0 & 0 & 0 & \rho & -\beta K_1(1-c) \\ \beta K_1(1-c) & -(\mu+\tau) & 0 & 0 & 0 & \beta I K_5(1-c) \\ 0 & \tau & -(\gamma+\emptyset+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \emptyset & -[\mu+(1-\gamma)] & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -(\mu+\rho) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_1+\sigma) \end{vmatrix}$$

$= 0$

$\Rightarrow$

$$\begin{vmatrix} -a-\lambda & 0 & 0 & 0 & \rho & -\beta K_1(1-c) \\ \beta K_1(1-c) & -b-\lambda & 0 & 0 & 0 & \beta I K_5(1-c) \\ 0 & \tau & -c-\lambda & 0 & 0 & 0 \\ 0 & 0 & \emptyset & -d-\lambda & 0 & 0 \\ 0 & 0 & \gamma & 1-\gamma & -f-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -h-\lambda \end{vmatrix} = 0 - - - (4.12.1)$$

Where

$$a = \beta K_1(1-c)+\mu,\ \ b = \mu+\tau,\ c = (\gamma+\emptyset+\mu+\delta),\ \ d = [\mu+(1-\gamma)],$$

$$h = \mu+\rho, and\ f = \mu_1+\sigma.$$

$Determinant\ Matrix\ (4.12.1)\ implies,$

$$(-h-\lambda)\begin{vmatrix} -a-\lambda & 0 & 0 & 0 & \rho \\ \beta K_1(1-c) & -b-\lambda & 0 & 0 & 0 \\ 0 & \tau & -c-\lambda & 0 & 0 \\ 0 & 0 & \emptyset & -d-\lambda & 0 \\ 0 & 0 & \gamma & 1-\gamma & -f-\lambda \end{vmatrix} = 0 - - - - - - - - (4.12.2)$$

$Multiply\ (4.12.2)\ through\ by\ negative, we\ have$

$$(-h-\lambda)\begin{vmatrix} a+\lambda & 0 & 0 & 0 & -\rho \\ -\beta K_1(1-c) & b+\lambda & 0 & 0 & 0 \\ 0 & -\tau & c+\lambda & 0 & 0 \\ 0 & 0 & -\emptyset & d+\lambda & 0 \\ 0 & 0 & -\gamma & -(1-\gamma) & f+\lambda \end{vmatrix} = 0 - - - - - - - - - (4.12.3)$$

Now, consider the matrices of (4.12.3). We observed that its entries satisfy $a_{ij} > 0, \forall\ i = j$ and $a_{ij} \leq 0, \forall\ i \neq j$. Consequently, all the leading principal minors are positive, hence lemma (4.3) holds. Therefore, all eigenvalues of the M- matrix have positive real parts. It follows that all the eigenvalues of (4.12.3) have negative real parts. This shows that the endemic equilibrium point E_+, is locally and asymptotically stable.

4.10 MODEL SOLUTION USING LAPLACE ADOMIAN DECOMPOSITION METHOD

We decide to obtain the analytical solution of the model (4.2) using Laplace Adomian Decomposition Method. Though there are other workable method but using this approach will yield a better approximation to the exact solution only in a few iterations. The method is as follow:

From the model (4.2), we have

$$D_t^{\alpha} S(t) = \Lambda_h + \rho R - \beta(1-c)SP - \mu S - - - - - - - - - \ (i)$$

$$D_t^{\alpha} E(t) = \ \beta(1-c)SP - E(\mu+\tau) - - - - - - - - - - - \ (ii)$$

$$D_t^{\alpha} I(t) = \ \tau E - I(\gamma+\varphi+\mu+\delta) - - - - - - - - - - - - (iii)$$

$$D_t^{\alpha} T(t) = \ \varphi I - T[\mu+(1-\gamma)] - - - - - - - - - - - - - (iv)$$

$$D_t^{\alpha} R(t) = \gamma I + (1-\gamma)T - R(\mu+\rho) - - - - - - - - - - - - (v)$$

$$D_t^{\alpha}P(t) = \Lambda_p - P(\mu_1 + \sigma) - - - - - - - - - - - - - - - \; (vi)$$

From (i)we have

$$D_t^{\alpha}S(t) = \Lambda_h + \rho R - \beta(1-c)SP - \mu S$$

$Letting\ S = F, we\ have$

$$D_t^{\alpha}F(t) = \Lambda_h + \rho R - \beta(1-c)FP - \mu F$$

Taking Laplace Transform with respect to F, we have

$$\mathbf{L}\{D_t^{\alpha}F(t)\} = \boldsymbol{L}\{\Lambda_h + \rho R - \beta(1-c)FP - \mu F\}$$

$\Rightarrow$

$$s^{\alpha}\boldsymbol{F}(s) - \sum_{k}^{n-1} s^{\alpha-k-1}F^{(k)}(0) = \boldsymbol{L}\{\wedge_h\} + \rho\boldsymbol{L}\{R(t)\} - \mu\boldsymbol{F}(s) - \beta(1-c)\boldsymbol{L}\{FP\}, 0 < \alpha < 1$$

$$s^{\alpha}\boldsymbol{F}(s) - \left(s^{\alpha-1}F(0)\right) = \frac{\wedge_h}{s} + \rho s^{-\mathbf{2}} - \mu\boldsymbol{F}(s) - \beta(1-c)\boldsymbol{L}\{FP\} \;\; for\ initial\ condition,$$

$$F(0) = n_1$$

$$\boldsymbol{F}(s)(s^{\alpha} + \mu) = \wedge_h s^{-1} + \rho s^{-\mathbf{2}} + s^{\alpha-1}n_1 - \beta(1-c)\boldsymbol{L}\{FP\}$$

$$\boldsymbol{F}(s) = \frac{\wedge_h s^{-1}}{(s^{\alpha}+\mu)} + \frac{\rho s^{-2}}{(s^{\alpha}+\mu)} + \frac{s^{\alpha-1}n_1}{(s^{\alpha}+\mu)} - \frac{1}{(s^{\alpha}+\mu)}\omega\boldsymbol{L}\{FP\} \;\; , where\ \omega = \beta(1-c)$$

Taking the Laplace inverse Transform, we have

$$F(t) = 1 - E_{\alpha}(-\mu t^{\alpha}) + \rho E_{\alpha,1}(-\mu t^{\alpha}) + n_1 t^{\alpha} E_{\alpha,\alpha+1}(-\mu t^{\alpha}) - \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{FP\}\right\} - -(ai)$$

It is assumed that the solution of $F(t)$ is in the form of an infinite series as follow:

$$F(t) = \sum_{n=0}^{\infty} F_n(t) = F_0(t) + F_1(t) + F_2(t) + \cdots - - - - - - - - - - - -(bi)$$

The Nonlinear term involved is $F(t)P(t)$ decomposed by Adomian polynomial as:

$$F(t)P(t) = \sum_{n=0}^{\infty} A_n -(ci)$$

Where

$$A_n = \frac{1}{n!}\frac{d^n}{dt^n}[\sum_{k=0}^{n}\lambda^k F_k(t), \sum_{k=0}^{n}\lambda^k P_k(t)]_{\lambda=0} - - - - - - - - - - - - - (di)$$

$\therefore$

$F(t) \Rightarrow$

$$\left(\frac{1}{n!}\frac{d^n}{dt^n}\right)\sum_{k=0}^{n}\lambda^k F_k(t) = \left(\frac{1}{n!}\frac{d^n}{dt^n}\right)(\lambda^0 F_0(t) + \lambda F(t) + \lambda^2 F_2(t) + \lambda^3 F_3(t) + \cdots + \lambda^n F_n(t))$$

$F(t) \Rightarrow$

$$\left(\frac{1}{n!}\frac{d^n}{dt^n}\right)F_0(t) = A_n, \qquad since\ \lambda = 0$$

For $n = 0$,

$$F_0(t) = A_0 - (ei)$$

From (ci), it implies that

$$F_0(t)P_0(t) = A_0 - (fi)$$

But from(bi) , we have

$$F_0(t) = F(t) = \boldsymbol{L}^{-1}\{\boldsymbol{F}(s)\}$$

$\Rightarrow$

$$\boldsymbol{L}\{F_0(t)\} = \boldsymbol{F}(s) = \frac{\wedge_h s^{-1}}{(s^\alpha + \mu)} + \frac{\rho s^{-2}}{(s^\alpha + \mu)} + \frac{s^{\alpha-1}n_1}{(s^\alpha + \mu)}$$

$$\boldsymbol{L}\{F_0(t)\} = \frac{\wedge_h s^{-1}}{(s^\alpha + \mu)} + \frac{\rho s^{-2}}{(s^\alpha + \mu)} + \frac{s^{\alpha-1}n_1}{(s^\alpha + \mu)}$$

$$F_0(t) = \boldsymbol{L}^{-1}\left\{\frac{\wedge_h s^{-1}}{(s^\alpha + \mu)} + \frac{\rho s^{-2}}{(s^\alpha + \mu)} + \frac{s^{\alpha-1}n_1}{(s^\alpha + \mu)}\right\}$$

$$F_0(t) = 1 - E_\alpha(-\mu t^\alpha) + \rho E_{\alpha,1}(-\mu t^\alpha) + n_1 t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha) - - - - - \ (4.13.1)$$

From (ai), Laplace Transform with respect to $F_1(t)$ is taken to become

$$\boldsymbol{L}\{F_1(t)\} = -\frac{\omega}{(s^\alpha + \mu)}\boldsymbol{L}\{A_0\} - (gi)$$

⋮

$$\boldsymbol{L}\{F_{n+1}(t)\} = -\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{A_n\}$$

To obtain $A_1, A_2, \dots, A_n$

Now, from(fi), we have

$$F_0(t)P_0(t) = A_0$$

Following the pattern, A_1 is obtained as follow

$$A_1 = \frac{1}{1!}\frac{d}{dt}[\lambda^0 F_0(t)\lambda^0 P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[\lambda^0 F_0(t)\lambda^0 P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[F_0(t)P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[F_0(t)P_0(t)], \qquad Since\ \lambda = 0$$

$$A_1 = F_0(t)P_0(t) + F_1(t)P_1(t) - (fii)$$

∴

From (gi), we have

$$\boldsymbol{L}\{F_1(t)\} = -\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{A_0\}$$

Taking the inverse Laplace Transform, we have

$$F_1(t) = -\boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{A_0\}\right\} - - - - - - - - - - - - - - - - - - (\mathbf{4.13.2})$$

$$F_2(t) = -\boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{A_1\}\right\} - - - - - - - - - - - - - - - - - - (\mathbf{4.13.3})$$

⋮

$$F_{n+1}(t) = \boldsymbol{L}^{-1}\left\{\frac{\Lambda_h s^{-1}}{(s^{\alpha}+\mu)} + \frac{\rho s^{-2}}{(s^{\alpha}+\mu)} + \frac{s^{\alpha-1}n_1}{(s^{\alpha}+\mu)}\right\} - \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\mu)}\boldsymbol{L}\{A_n\}\right\}, \qquad n \geq 0$$

Hence all terms of $F(t)$ are calculated and the general solution obtained according to Adomian Decomposition Method as:

$$F(t) = \sum_{n=0}^{\infty} F_n(t) = F_0(t) + F_1(t) + F_2(t) + \cdots$$

And this proved for the convergence of the series

From (ii)we have

$$D_t^{\alpha} E(t) = \beta(1-c)SP - E(\mu+\tau)$$

$$Letting\ S = F, we\ have$$

$$D_t^{\alpha} E(t) = \beta(1-c)FP - E(\mu+\tau)$$

Taking Laplace Transform with respect to E, we have

$$\mathbf{L}\{D_t^{\alpha} E(t)\} = \boldsymbol{L}\{\beta(1-c)FP - E(\mu+\tau)\}$$

$$\Rightarrow$$

$$s^{\alpha}\boldsymbol{E}(s) - \sum_{k}^{n-1} s^{\alpha-k-1}E^{(k)}(0) = \beta(1-c)\boldsymbol{L}\{FP\} - \pi\boldsymbol{L}\{E\}, \qquad for\ (\mu+\tau) = \pi$$

$$s^{\alpha}\boldsymbol{E}(s) - \left(s^{\alpha-1}E(0)\right) = \beta(1-c)\boldsymbol{L}\{FP\} - \pi\boldsymbol{E}(s)\ \ for\ initial\ cond., E(0) = n_2$$

$$\boldsymbol{E}(s)(s^{\alpha}+\pi) = \beta(1-c)\boldsymbol{L}\{FP\} + s^{\alpha-1}n_2$$

$$\boldsymbol{E}(s) = \frac{s^{\alpha-1}n_2}{(s^{\alpha}+\pi)} + \frac{1}{(s^{\alpha}+\pi)}\omega\boldsymbol{L}\{FP\}\ , where\ \omega = \beta(1-c)$$

Taking the Laplace inverse Transform, we have

$$E(t) = n_2 t^{\alpha} E_{\alpha,\alpha+1}(-\pi t^{\alpha}) + \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{FP\}\right\} ---------(\theta i)$$

It is assumed that the solution of $E(t)$ is in the form of an infinite series as follow:

$$E(t) = \sum_{n=0}^{\infty} E_n(t) = E_0(t) + E_1(t) + E_2(t) + \cdots ----------(\theta ii)$$

The Nonlinear term involved is $F(t)P(t)$ decomposed by Adomian polynomial as:

$$F(t)P(t) = \sum_{n=0}^{\infty} A_n \; ---------- (\theta iii)$$

Where

$$A_n = \frac{1}{n!}\frac{d^n}{dt^n}[\sum_{k=0}^{n} \lambda^k F_k(t), \sum_{k=0}^{n} \lambda^k P_k(t)]_{\lambda=0} \; ---------- (\theta iv)$$

$\therefore$

$E(t) \Rightarrow$

$$\left(\frac{1}{n!}\frac{d^n}{dt^n}\right)\sum_{k=0}^{n} \lambda^k F_k(t) = \left(\frac{1}{n!}\frac{d^n}{dt^n}\right)(\lambda^0 F_0(t) + \lambda F(t) + \lambda^2 F_2(t) + \lambda^3 F_3(t) + \cdots + \lambda^n F_n(t))$$

$E(t) \Rightarrow$

$$\left(\frac{1}{n!}\frac{d^n}{dt^n}\right)F_0(t) = A_n, \qquad since\ \lambda = 0$$

For $n = 0$,

$$F_0(t) = A_0 \; ---------- (\theta v)$$

From (θiii), It implies that

$$F_0(t)P_0(t) = A_0 \; ---------- (\theta vi)$$

But from(θii) , we have

$$E_0(t) = E(t) = \boldsymbol{L}^{-1}\{\boldsymbol{E}(s)\}$$

$\Rightarrow$

$$\boldsymbol{L}\{E_0(t)\} = \boldsymbol{E}(s) = \frac{s^{\alpha-1}n_2}{(s^\alpha + \pi)}$$

$$\boldsymbol{L}\{E_0(t)\} = \frac{s^{\alpha-1}n_2}{(s^\alpha + \pi)}$$

$$E_0(t) = \boldsymbol{L}^{-1}\left\{\frac{s^{\alpha-1}n_2}{(s^\alpha + \pi)}\right\}$$

$$E_0(t) = n_2 t^\alpha E_{\alpha,\alpha+1}(-\pi t^\alpha) \; ----------(4.13.4)$$

From (θi), Laplace Transform with respect to $E_1(t)$ is taken to become

$$\boldsymbol{L}\{E_1(t)\} = \frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{A_0\} - (\theta vii)$$

$$\vdots$$

$$\boldsymbol{L}\{E_{n+1}(t)\} = \frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{A_n\}$$

To obtain $A_1, A_2, \dots, A_n$

Now, from(θvi), we have

$F_0(t)P_0(t) = A_0$

Following the pattern, A_1 is obtained as follow

$$A_1 = \frac{1}{1!}\frac{d}{dt}[\lambda^0 F_0(t)\lambda^0 P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[\lambda^0 F_0(t)\lambda^0 P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[F_0(t)P_0(t) + \lambda^1 F_1(t)\lambda^1 P_1(t) + \lambda^2 F_2(t)\lambda^2 P_2(t) + \cdots]_{\lambda=0}$$

$$A_1 = \frac{d}{dt}[F_0(t)P_0(t)], \qquad Since\ \lambda = 0$$

$$A_1 = F_0(t)P_0(t) + F_1(t)P_1(t) - (\theta viii)$$

$\therefore$

From (θvii)we have

$$\boldsymbol{L}\{E_1(t)\} = \frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{A_0\}$$

Taking the inverse Laplace Transform, we have

$$E_1(t) = \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{A_0\}\right\} - (\mathbf{4.13.5})$$

$$E_2(t) = \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^{\alpha}+\pi)}\boldsymbol{L}\{A_1\}\right\} - (\mathbf{4.13.6})$$

$$\vdots$$

$$E_{n+1}(t) = \boldsymbol{L}^{-1}\left\{\frac{s^{\alpha-1}n_2}{(s^\alpha+\pi)}\right\} + \boldsymbol{L}^{-1}\left\{\frac{\omega}{(s^\alpha+\pi)}\boldsymbol{L}\{A_n\}\right\}, \qquad n \geq 0$$

Hence all terms of $E(t)$ are calculated and the general solution obtained according to Adomian Decomposition Method as:

$$E(t) = \sum_{n=0}^{\infty} E_n(t) = E_0(t) + E_1(t) + E_2(t) + \cdots$$

And this proved for the convergence of the series

From (iii)we have

$$D_t^\alpha I(t) = \tau E - I(\gamma + \varphi + \mu + \delta) - (iii)$$

Taking Laplace Transform with respect I, we have

$$\mathbf{L}\{D_t^\alpha I(t)\} = \boldsymbol{L}\{\tau E\} - \boldsymbol{L}\{I(\gamma + \varphi + \mu + \delta)\}$$

$\Rightarrow$

$$s^\alpha \boldsymbol{I}(s) - \sum_{\pi}^{n-1} s^{\alpha-\pi-1} I^{(\pi)}(0) = \tau \boldsymbol{L}\{E(t)\} - \boldsymbol{I}(s)k, \;\; where\; k = (\gamma + \varphi + \mu + \delta)$$

$$s^\alpha \boldsymbol{I}(s) - \left(s^{\alpha-1} I(0)\right) = \tau \boldsymbol{L}\{E(t)\} - \boldsymbol{I}(s)k, \qquad for\; initial\; condition, I(0) = n_3$$

$$\boldsymbol{I}(s)(s^\alpha + k) = \tau \boldsymbol{L}\{E(t)\} + s^{\alpha-1} n_3$$

$$\boldsymbol{I}(s)(s^\alpha + k) = \tau s^{-2} + s^{\alpha-1} n_3$$

$$\boldsymbol{I}(s) = \frac{\tau s^{-2}}{(s^\alpha + k)} + \frac{s^{\alpha-1} n_3}{(s^\alpha + k)}$$

Taking the Laplace inverse Transform, we have

$$I(t) = \tau E_{\alpha,1}(-kt^\alpha) + n_3 t^\alpha E_{\alpha,\alpha+1}(-kt^\alpha)$$

$$E_{\alpha,1}(I) = (-\tau k)\sum_{i=0}^{\infty} \frac{I^i}{\Gamma(\alpha k + 1)} = -\tau k\left(1 + \frac{I(t)}{\alpha!} + \frac{I^2(t)}{2\alpha!} + \cdots\right), \;\&$$

$$E_{\alpha,\alpha+1}(T) = (-kn_3)\sum_{i=0}^{\infty}\frac{I^i}{\Gamma(\alpha k+\alpha+1)} = -kn_3\left(\frac{1}{\alpha!}+\frac{I(t)}{2\alpha!}+\frac{I^2(t)}{3\alpha!}+\cdots\right), \alpha>0, \beta>0$$

From (iv), we have

$$D_t^{\alpha}T(t) = \varphi I - T[\mu+(1-\gamma)]$$

Taking Laplace Transform with respect to T, we have

$$\mathbf{L}\{D_t^{\alpha}T(t)\} = \boldsymbol{L}\{\emptyset I\} - \boldsymbol{L}\{T(\mu+1-\gamma)\}$$

$\Rightarrow$

$$s^{\alpha}\boldsymbol{T}(s) - \sum_{k}^{n-1} s^{\alpha-k-1}T^{(k)}(0) = \boldsymbol{L}\{\emptyset I\} - \boldsymbol{T}(s)z, \;\; where\; z = \mu+1-\gamma$$

$$s^{\alpha}\boldsymbol{T}(s) - \left(s^{\alpha-1}T(0)\right) = \varphi\boldsymbol{L}\{I(t)\} - \boldsymbol{T}(s)z, \qquad for\; initial\; condition, T(0) = n_4$$

$$\boldsymbol{T}(s)(s^{\alpha}+z) = \varphi\boldsymbol{L}\{E(t)\} + s^{\alpha-1}n_4$$

$$\boldsymbol{T}(s)(s^{\alpha}+z) = \varphi s^{-2} + s^{\alpha-1}n_4$$

$$\boldsymbol{T}(s) = \frac{\varphi s^{-2}}{(s^{\alpha}+z)} + \frac{s^{\alpha-1}n_4}{(s^{\alpha}+z)}$$

Taking the Laplace inverse Transform, we have

$$T(t) = \varphi E_{\alpha,1}(-zt^{\alpha}) + n_4 t^{\alpha}E_{\alpha,\alpha+1}(-zt^{\alpha}),$$

Where

$$E_{\alpha,1}(T) = (-\varphi z)\sum_{k=0}^{\infty}\frac{T^k}{\Gamma(\alpha k+1)} = -\varphi z\left(1+\frac{T(t)}{\alpha!}+\frac{T^2(t)}{2\alpha!}+\cdots\right), \;\&$$

$$E_{\alpha,\alpha+1}(T) = (-zn_4)\sum_{k=0}^{\infty}\frac{T^k}{\Gamma(\alpha k+\alpha+1)} = -zn_4\left(\frac{1}{\alpha!}+\frac{T(t)}{2\alpha!}+\frac{T^2(t)}{3\alpha!}+\cdots\right), \alpha>0, \beta>0$$

From (v), we have

$$D_t^{\alpha}R(t) = \gamma I + (1-\gamma)T - R(\mu+\rho)$$

Taking Laplace Transform with respect to R, we have

$$\mathbf{L}\{D_t^{\alpha}R(t)\} = \boldsymbol{L}\{\gamma I\} + \boldsymbol{L}\{(1-\gamma)T\} - \boldsymbol{L}\{R(\mu+\rho)\}$$

$\Rightarrow$

$$s^{\alpha}\boldsymbol{R}(s) - \sum_{k}^{n-1} s^{\alpha-k-1}R^{(k)}(0) = \boldsymbol{L}\{\gamma I(t)\} + (1-\gamma)\boldsymbol{L}\{T(t)\} - \boldsymbol{R}(s)\xi, \;\; where\; \xi = \mu + \rho$$

$$s^{\alpha}\boldsymbol{R}(s) - \left(s^{\alpha-1}R(0)\right) = \gamma\boldsymbol{L}\{I(t)\} + (1-\gamma)\boldsymbol{L}\{T(t)\} - \boldsymbol{R}(s)\xi, for\; initial\; cond., R(0) = n_5$$

$$\boldsymbol{R}(s)(s^{\alpha} + \xi) = \gamma\boldsymbol{L}\{I(t)\} + (1-\gamma)\boldsymbol{L}\{T(t)\} + s^{\alpha-1}n_5$$

$$\boldsymbol{R}(s)(s^{\alpha} + \xi) = \gamma s^{-2} + (1-\gamma)s^{-2} + s^{\alpha-1}n_5$$

$$\boldsymbol{R}(s) = \frac{\gamma s^{-2}}{(s^{\alpha}+\xi)} + \frac{(1-\gamma)s^{-2}}{(s^{\alpha}+\xi)} + \frac{s^{\alpha-1}n_5}{(s^{\alpha}+\xi)}$$

Taking the Laplace inverse Transform, we have

$$R(t) = \gamma E_{\alpha,1}(-\xi t^{\alpha}) + (1-\gamma)E_{\alpha,1}(-\xi t^{\alpha}) + n_5 t^{\alpha}E_{\alpha,\alpha+1}(-\xi t^{\alpha})$$

$$R(t) = E_{\alpha,1}(-\xi t^{\alpha})\big(\gamma + (1-\gamma)\big) + n_5 t^{\alpha}E_{\alpha,\alpha+1}(-\xi t^{\alpha})$$

$$R(t) = E_{\alpha,1}(-\xi t^{\alpha}) + n_5 t^{\alpha}E_{\alpha,\alpha+1}(-\xi t^{\alpha})$$

Where,

$$E_{\alpha,1}(R) = (-\xi)\sum_{k=0}^{\infty}\frac{R^k}{\Gamma(\alpha k+1)} = -\xi\left(1 + \frac{R(t)}{\alpha!} + \frac{R^2(t)}{2\alpha!} + \cdots\right), \&$$

$$E_{\alpha,\alpha+1}(R) = (-\xi n_5)\sum_{k=0}^{\infty}\frac{T^k}{\Gamma(\alpha k+\alpha+1)} = -\xi n_5\left(\frac{1}{\alpha!} + \frac{R(t)}{2\alpha!} + \frac{R^2(t)}{3\alpha!} + \cdots\right), \alpha > 0, \beta > 0$$

From (vi)we have

$$D_t^{\alpha}P(t) = \wedge_p - P(\mu_1 + \sigma)$$

Taking Laplace Transform with respect to P, we have

$$\mathbf{L}\{D_t^{\alpha}P(t)\} = \boldsymbol{L}\{\wedge_p\} - \boldsymbol{L}\{P(\mu_1+\sigma)\}$$

$\Rightarrow$

$$s^{\alpha}\boldsymbol{P}(s) - \sum_{k}^{n-1} s^{\alpha-k-1}P^{(k)}(0) = \frac{\wedge_p}{s} - \boldsymbol{P}(s)m, \;\; where\; m = (\mu_1 + \sigma)$$

$$s^{\alpha}\boldsymbol{P}(s) - \left(s^{\alpha-1}P(0)\right) = \frac{\wedge_p}{s} - \boldsymbol{P}(s)m, \qquad for\ initial\ condition, P(0) = n_6$$

$$\boldsymbol{P}(s)(s^{\alpha} + m) = \frac{\wedge_p}{s} + s^{\alpha-1}n_6$$

$$\boldsymbol{P}(s)(s^{\alpha} + m) = \wedge_p s^{-1} + s^{\alpha-1}n_6$$

$$\boldsymbol{P}(s) = \frac{\wedge_p s^{-1}}{(s^{\alpha} + m)} + \frac{s^{\alpha-1}n_6}{(s^{\alpha} + m)}$$

Taking the Laplace inverse Transform, we have

$$P(t) = 1 - E_{\alpha}(-mt^{\alpha}) + n_6 t^{\alpha} E_{\alpha,\alpha+1}(-mt^{\alpha})$$

Where,

$$E_{\alpha,1}(P) = (-m)\sum_{k=0}^{\infty}\frac{P^k}{\Gamma(\alpha k + 1)} = -m\left(1 + \frac{P(t)}{\alpha!} + \frac{P^2(t)}{2\alpha!} + \cdots\right), \&$$

$$E_{\alpha,\alpha+1}(R) = (-mn_6)\sum_{k=0}^{\infty}\frac{P^k}{\Gamma(\alpha k + \alpha + 1)} = -mn_6\left(\frac{1}{\alpha!} + \frac{P(t)}{2\alpha!} + \frac{P^2(t)}{3\alpha!} + \cdots\right), \alpha, \beta > 0$$

4.11 NUMERICAL SIMULATIONS AND RESULTS

4.11.1 THE PREDICTOR-CORRECTOR MATLAB CODES FDE12.M FOR SOLVING FRACTIONAL DIFFERENTIAL EQUATIONS USING MATLAB.

In order to observe the effects that the parameter α has on the dynamics of the fractional model (4.2), we include several numerical simulations varying the value of the parameter. In simulating the fractional-order model (4.2), we used MATLAB to get the numerical solution of our model by applying Adams-type predictor-corrector method (Kai et al. 2002). This method is well known for numerical solutions of first-order problems (Kai et al. 2002).

Table 4.2: Estimated initial conditions and the parameters with values and their sources

Parameters	Value	source
Λ_h	20,000	Buneman et al (2009)
ρ	0.5115	Chitnis et al (2008)
β	0.4828	Chitnis et al (2008)
μ	1.9249	Chitnis et al (2008)
τ	0.5013	Chitnis et al (2008)
γ	0.4885	Chitnis et al (2008)
φ	1.4422	Chitnis et al (2008)
δ	0.1570	Chitnis et al (2008)
$1-\gamma$	0.5115	Chitnis et al (2008)
Λ_p	10,000	Estimated
μ_1	0.9407	Chitnis et al (2008)
σ	1.0345	Chitnis et al (2008)
c	$0 < c < 1$	Chitnis et al (2008)

Variables at Initial conditions	***Value***	***Source***
$S(0)$	500,000	Estimated
$E(0)$	150,000	Estimated
$I(0)$	100,000	Estimated
$T(0)$	70,000	Estimated
$R(0)$	120,000	Estimated
$P(0)$	600,000	Estimated

The simulation was run in days.

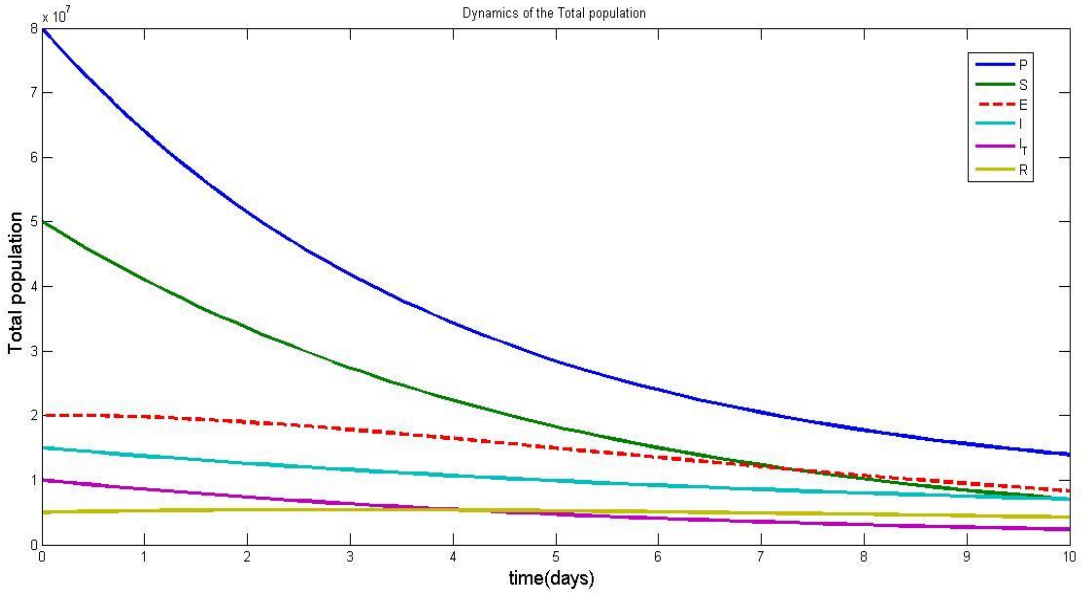

Figure 1: Dynamics of the Diseased Population.

From the figure above, the interaction will be high at the earlier stage due to the lack of awareness of its endemic, as a result of that led to increase in disease level which will drastically reduce the population to zero with time.

Therefore, the figure gave a decreasing graph which will collapses to zero with time.

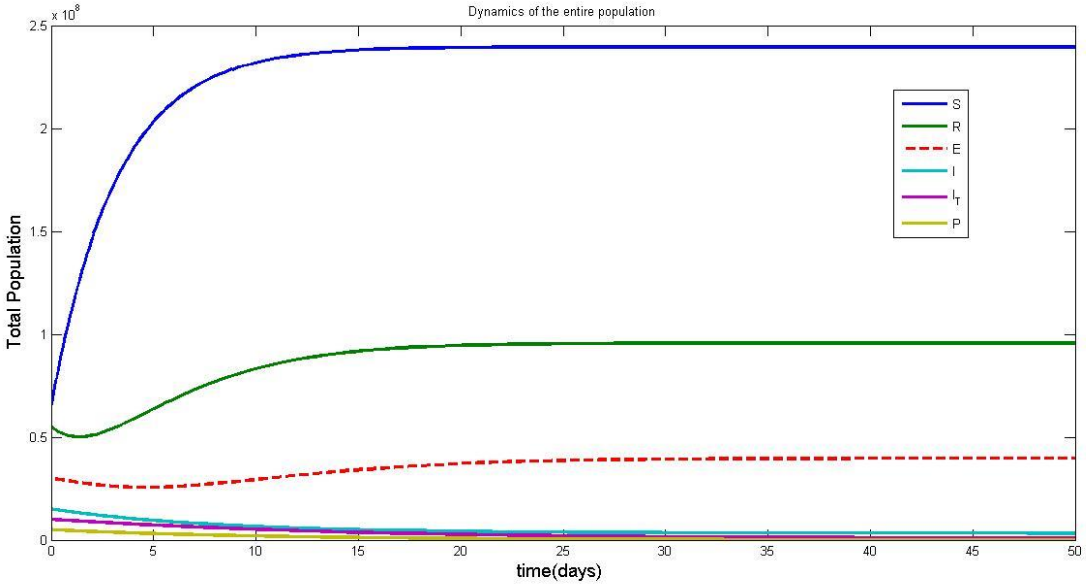

Figure 2: Effect of Insect Repellent on Diseased Population

From the Figure 2 above, the graph showed us that the disease infection started responding to the insect repellent containing diethyltoluamide once the population became aware of the disease endemic and adhered to the use of it.

Therefore, as time goes on, the use of insect repellent will become more effective, as so fizzle out the disease as the curve does not collapse to zero but will consequently increase the population class with time and hence gives us a locally asymptotical stability

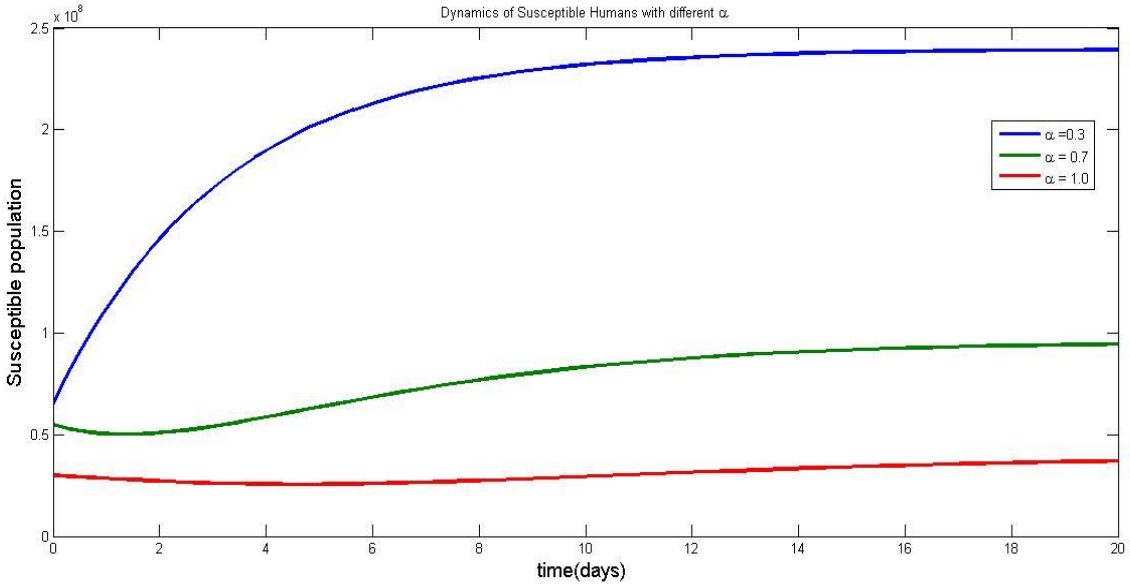

Figure 3: Variation of Susceptible Class with Different Alphas (α).

Figure 3 is from the controlled population. Here, we could be able to analyze the authenticity between fractional and the integer approach and we found out that the fractional approach is proved to be better than those at the integer case. As it is seen from the dialogue box, $\alpha = 1.0$ is undoubtedly a good one but as the population is under control, it has given the fractional approach a better result at $\alpha = 0.3$ since the susceptible class have gained more individuals. Therefore, the graph $\alpha = 0.3$ shows how the susceptible population increases with time and stabilizes at some point and that means that the system is locally asymptotically stable at that point.

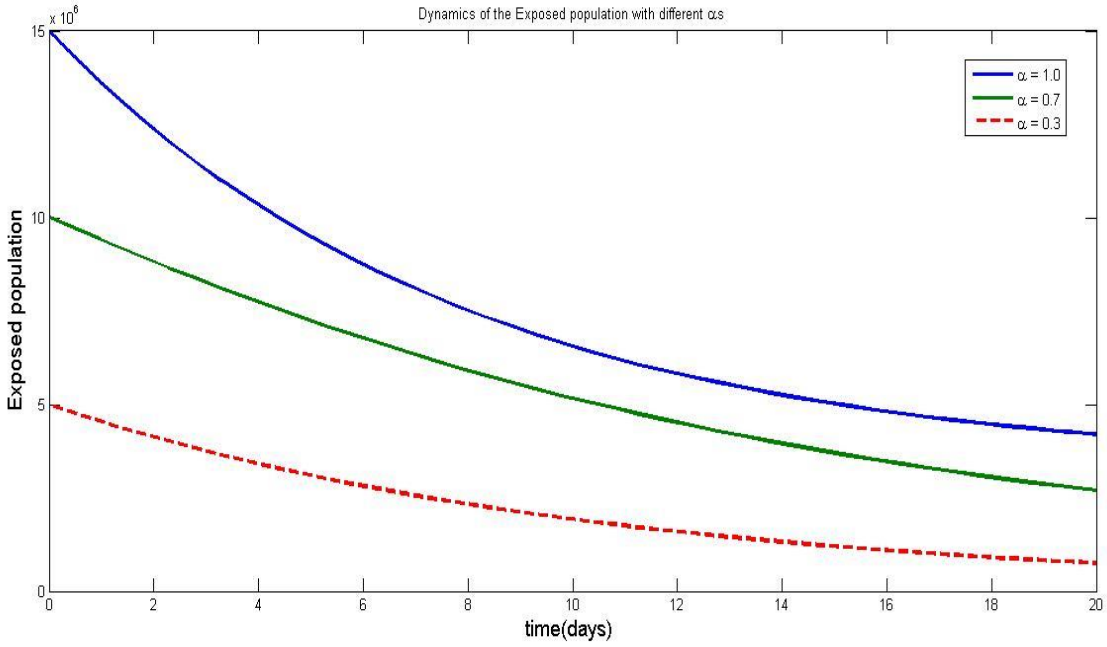

Figure 4: Variation of Exposed Class with Different Alphas (α).

From 4 captures a decrease in the exposed population with time and clearly shows that the exposed population will vanish at some point since the graph is from controlled population. Therefore, $\alpha = 0.3$ is a better result compared to $\alpha = 1.0$ and $\alpha = 0.7$

Therefore, since the graph captures a decrease in the exposed population with time and clearly shows that the exposed population will vanish at some point, it means that the model is LAS when $R_0 < 1$.

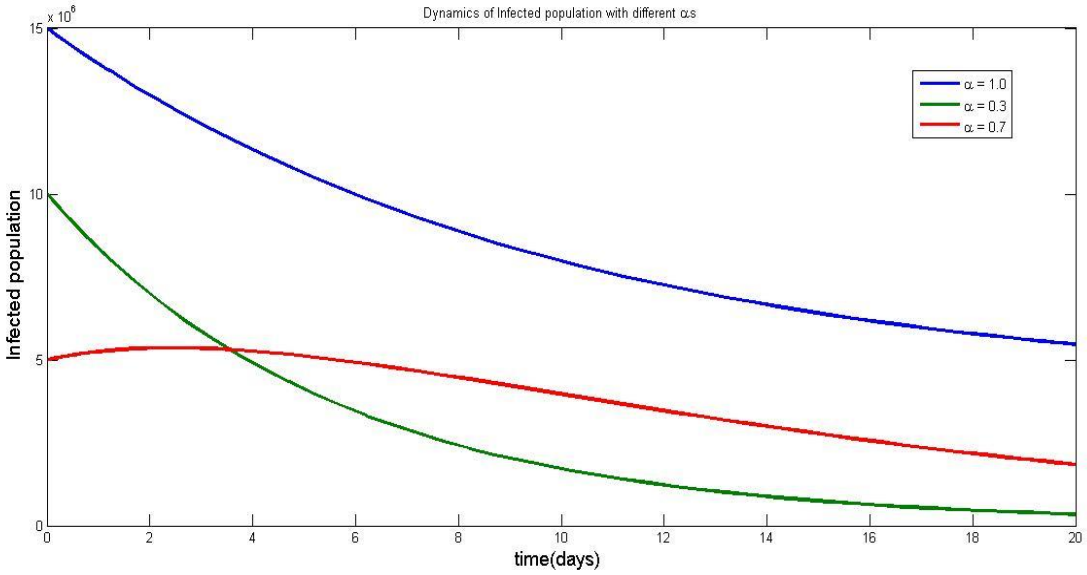

Figure 5: Variation of Infected Class with Different Alphas (α).

.Figure 5 captures a decrease in the infected population with time to an extent say$\alpha = 1.0$, but the interception between $\alpha = 0.3\ and\ \alpha = 0.7$ implies that infection does not vanish since it could not be controlled at this stage, therefore $\alpha = 0.3$ still give better result. That is, the rate of infection become low when alpha is low.

Figure 5 also says same but the infected class does not vanish since $R_0 > 1$, hence GAS is guaranteed.

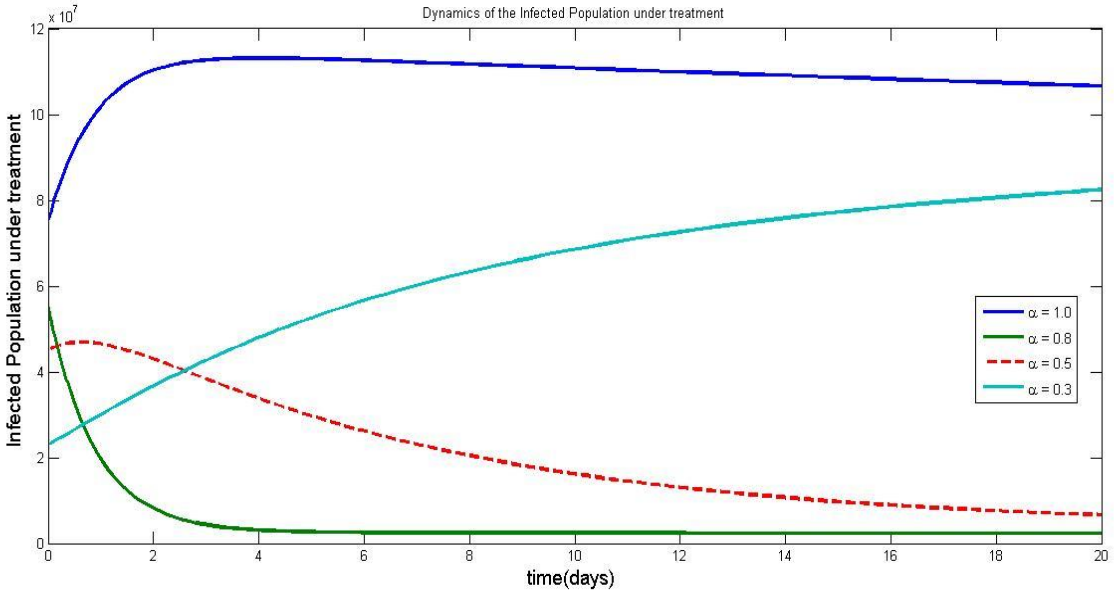

Figure 6: Variation of Infected Under Treatment Class with Different Alphas (α).

Figure 6 shows the variation of infected under treatment population with different alpha. Obviously at a lower value of alpha, the infected population under treatment decreases since it has been shown before at the rate of infection becomes low when alpha is low say (0.3) than when it is higher (1.0). Since the rate of infection has become low as shown earlier, the treatment class will decrease.

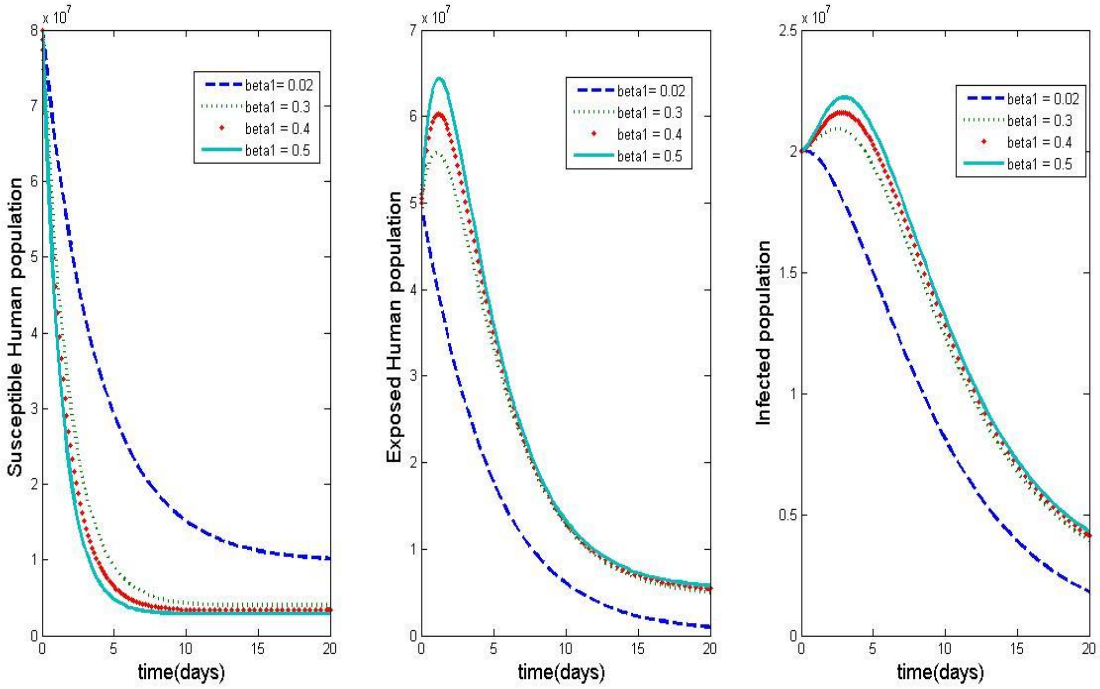

Figure 7: Dynamics of Sensitive Parameter On Diseased Population

From the susceptible graph, we observed that increasing the presence of parameter, β in the diseased population leads to decrease in the susceptible class. Then, the population keeps on reducing as the interaction level keep on increasing with time.

At the exposed graph, for the fact that the interaction rate has been increased on the susceptible class, then the exposed population will increase gradually with time.

At the infected graph, the infectiousness of the disease in the population will increase as well since the exposed class is increased.

In general, Figure 7 shows how a variation of the interaction level affects the susceptible, exposed and infected population. From susceptible, it gave a decreasing graph since the interaction increases in the absence of control on the population.

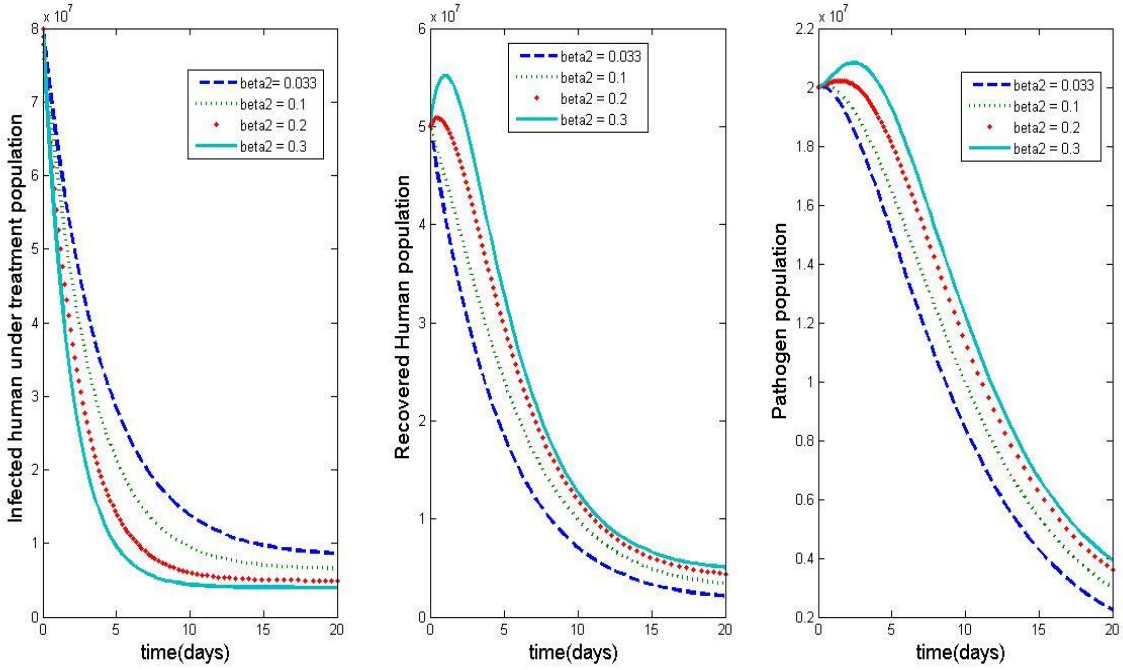

Figure 8: Dynamics of Sensitive Parameter On Diseased Population

From the treatment graph, since the infected population here have adopted treatment, then increasing more presence of the parameter, β will reduces the infectiousness of the disease in the population which increase the recovered population.

From the pathogens graph, off course increasing more presence of the interaction rate implies increased in the pathogens level.

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CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.1 SUMMARY

In this work, we consider a fractional mathematical model on the interaction between the disease causing agents (pathogens) and the susceptible population. The mathematical model has been derived which described the dynamics of the disease in the population from the flow diagram with strategies for control and treatment.

The model used in this work is a relatively simple ordinary differential equation-based model describing the interaction between the population and the disease causing agents. And the equilibrium, stability and the numerical analysis of this model paved the way in understanding of the interactions.

From the model, it was possible to investigate the impact of control measure on the diseased population. A stability analysis of the equilibrium of the mathematical model shows that under certain condition, it can be seen that the myiasis is almost completely eliminated by control and treatment.

5.2 CONCLUSION

We have presented fractional order model dynamics of myiasis infection with control measure and the effect of the control measure was felt as demonstrated on the graph. The model captures the causes, the predisposing factors of myiasis disease and a possible way of preventing the interactions. It can be seen in the model that the spread of the disease totally depends on the interaction between the pathogens and the people within the population. It is seen that if the proportion of population that is treated increases, then they will become susceptible again after recovery. The numerical simulations of the fractional order model with different values of α are performed by Caputo's derivative using the predictor corrector method of Adams-Bash forth Moulton type. The dynamics of the compartments have been shown in the graphs obtained. In addition, the results gave an insight that fractional order model is more suitable than its integer-order.

. In this work, it is observed that administration of therapy to the myiasis patient is also effective as they increased the recovered individuals with respect to time. In figure 8, at the recovered

class, the effect of treatment on the infected class has increased the recovered individuals with time even though the interaction increases more than 0.033. Individuals are free from disease in this class.

In conclusion, for the fact that one can recovered from myiasis infection without treatment does not implies that treatment is not necessary as it reduce the chances of secondary infection and uncomfortable about waiting for the required 8-12 weeks for the larvae to mature and exist on their own. It has also confirmed from the simulation result that insect repellent containing diethyltoluamide could be totally considered since its uses rescue the population from contacting the disease.

5.3 RECOMMENDATION

5.3.1 AWARENESS CAMPAIGN:

It is advisable to abstain from some of the factors that lead to myiasis infection. In this juncture, awareness campaign is adopted to emphasized the needful of the preventive and control measure and its benefit of not to neglect wounds by taking proper medical care; otherwise, myiasis may supervene.

5.3.2 PREVENTIVE AND CONTROL MEASURE

We recognize eco-system by introducing predators in the environment to feed on the disease causing agent thereby reduces the interaction level, it is advisable to recommend such in a society where the breeding is so high.

Alternatively, insect repellent is recommended to control the interactions which may not necessarily kill the pathogen for their economic important.

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