MATHEMATICAL MODEL ON THE DYNAMICS OF MAIZE LETHAL NECROSIS DISEASE (MLND) UNDER THE DEPLOYMENT OF A BIOLOGICAL ENEMY

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MSC/MTH/17/0339

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BY

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A THESIS SUBMITTED TO THE DEPERTMENT OF MATHEMATICS, SCHOOL OF PHYSICAL SCIENCES, IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF SCIENCE IN MATHEMATICS OF THE MODDIBO ADAMA UNIVERSITY OF TECHNOLOGY, YOLA

DECLARATION

I hereby declare that this thesis was written by me and it is a record of my own research work. It has not been presented before in any previous application for a higher degree. All references cited have been duly acknowledged.

_____
Musa Manga

_____
Date

DEDICATION

This project is dedicated to my Late Father Muhammad Manga, my mother Khadija Musa, my wife Hafsat Adam Yanga and my children Maryam, Hassan and Khadija.

APPROVAL PAGE

This thesis entitled 'Mathematical model on the dynamics of maize lethal necrosis disease (MLND) under the deployment of a biological enemy' meets the regulations governing the award of Masters of the Modibbo Adama University of Technology, Yola and is approved for its contribution to knowledge and literary presentation.

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ABSTRACT

In this thesis, we modified the mathematical model due to Williams, Dmitry ad Livingstone (2018), by incorporating biological control using cucumeris (predator) that preys on vector (thrips) to control vector population toward eradication of maize lethal necrosis disease (MLND). The modified model has been used to study the impact of control strategies on the transmission dynamics of MLND in maize population. The existence of the disease free equilibrium state has been established. Further, the basic reproduction number R_0 was obtained using the approach of next generation matrix. The local and global stability analyses carried out and the results revealed that the disease free equilibrium is locally and globally asymptotically stable when Ro < 1, furthermore, numerical simulation of the modified model was carried out using ode45 solver in MATLAB. The results show that the MLND can be eradicated when biological control measure is implemented.

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CHAPTER ONE

INTRODUCTION

1.1 Background of the Study

Maize Lethal Necrosis disease (MLND) is caused by double infection with Maize chlorotic mottle virus (MCMV) and any of the cereal viruses in potyviridae group; Sugarcane mosaic virus (SCMV), Maize dwarf mosaic virus (MDMV) or Wheat streak mosaic virus (WSMV). Highly affected areas may experience a massive yield loss of over

95% and this will affect total maize yield produced in the region. Maize plants are susceptible to MLN at all stages in their growth, from seedling to maturity. As with all viral diseases in plants, a carrier known as a "vector" transmits the MLN causing viruses from plant to plant and field to field. MCMV is carried by thrips and beetles. A plant disease is any abnormal condition that alters the appearance or function of a plant. It is a physiological process that affects some or all plant function. Disease may also reduce yield and quality of harvested product (Nault, Styer, Coffey, Gordon, Negi, & Niblett, 1978).

In Africa, the disease was first reported in Kenya, in September 2011 although its extent at that point suggested that the disease has been present for some time. According to the Kenyan Ministry of Agriculture, two percent of the maize harvest was affected in 2012. MLND has also spread rapidly into Tanzania, Uganda, Rwanda and South Sudan in the meantime. There is a preliminary report of the disease in Uganda. In Ethiopia the disease was first observed in the Rift Valley (Mahuku *et al.*,2015).

Mathematical model for the spread of the viruses causing MLN within and between growing seasons was studied due to Frank *et al.* (2019), their model allows for transmission via vectors, soil and seed, as well as the exogenous source of infection. By teir model parameterization, they predict how management affects disease prevalence and crop performance over multiple seasons. They said that crop rotation is often required to affect full control.

Ecoepidemiological model and analysis of Maize Streak Virus transmission dynamics in maize plant was dtdied by Hailevesus, Oluwole and David (2019), where they proposed and used stability theory of differential equation. The basic reproduction number with respect to the MSV free equilibrium obtained using next generation matrix approach. They also established the condition for local asymptotic stability of MSV and endemic equilibrium. Their model exhibit forward bifurcation and the sensitivity indices of various embedded parameters with respect to MSV eradication or spreading are determined.

A mathematical model for the dynamics of MLND was developed by William, Dmitry and Livingstone (2018), where they studied and analyzed the dynamics of MLND in the maize crop population. In their research they did not account for any control measure that will help to reduce the MLND transmission. In this research we wish to employ one of the control methods (Biological control) that will control the vector population so as to eradicate or reduce the magnitude of disease transmission.

1.2 Statement of the Problem

Maize Lethal Necrosis disease (MLND) is a viral disease that can cause fatal damage to maize plant. This is very common in Africa. William, Dmitry and Livingstone (2018) developed a SEI mathematical model that studied and analysed the dynamics of the MLND in maize crop plant. Thus, in their work, they do not consider any control measure on the effect of MLND in maize crop. However, this research modified the existing model by incorporating biological control using Cucumeris (as a predator) that preys on vector (thrips) in order to control the vector population toward eradicating or reducing the intensity of the disease transmission in maize plants.

1.3 Aim and Objectives of the Study

The aim of this research is to develop a mathematical model on the dynamics of MLND incorporating biological control using Cucumeris (as a predator) that feeds on thrips (prey) into the model developed by Williams, Dmitry and Livinstone (2018) to control the vector population towards eradication of the Maize lethal necrosis disease.

Objectives of this research are to:

i. drive existence and uniqueness of the solution, invariant region of the modified model and positivity of the solution of the model.
ii. obtain the basic reproduction number (R_0) using next generation matrix.
iii. analyse local and global stability of the disease free equilibrium states of the proposed model.
iv. carry out numerical experiments on the proposed model.

1.4 Significance of Study

Sub optimal yield of most of the maize crop, is partly due to attack of MLND at different stages. Therefore, this research would be useful in finding environmentally friendly means of controlling the MLND transmission or rather eradicate it. This study could ensure farmers increased maize yield which will in turn improve on food security.

1.5 Scope of the Study

The study incorporate biological control agent (Cucumeris) to modify the existing model by William, Dmitry and Livingstone (2018), which consist system of non-linear

ordinary differential equations and analysis the stability of he disease free equilibrium state and perform numeral experiments on the proposed model.

1.6 Operational Definitions of Terms

1.6.1 ***Symptoms:*** Is the visible effects of disease on plant. Any detectable change in coour, shape, and/or function of the plant in response to pathogens or disease causing agents.

1.6.2 ***Vector:*** Are living organism that can transmit infectious plant disease from insects to plant or.

1.6.3 ***Pathogen:*** This is a biological agent that causes disease or illness to its host.

1.6.4 ***Environment:*** This is natural unit that provide living things with every things that is necessary to survive such as nutrients in soil, water and sunlight.

1.6.5 ***Lethal:*** Able to cause plant death or extremely dangerous.

1.6.6 ***Necrosis:*** Is a form of ell injury which result into premature death o plant cell.

1.6.7 ***Motttle:*** This is a pattern of irregular marks, spots or different shape on a plant

1.6.8 ***Mosaic virus:*** This is a virus that causes the leaves to have a speckled appearance.

1.7.9 ***Virus:*** This is a very small particle that is capable of infecting a cell and potentially causing disease.

CHAPTER TWO

LITERATURE REVIEW

2.1 Mathematical Modeling

Benyah (2005) defined mathematical modeling as the process of creating a mathematical representation of some phenomenon. Furthermore, modeling ha become an important scientific technique of analysis for quite some time and is becoming more and more powerful tool to solve problems arising from science, engineering and industries. Squires, Hazel and Tappenden (2011), described mathematical model as a representation f the real world, characterized by the use of mathematics to represent the parts of the real world problems that are of interest and relationship between those parts. They are of the opinion that model can be use to inform policy decision by synthesing a diverse rnge of evidence within coherence and explicit frame work. They see model itself as an assumption, abstraction and simplification.

2.2 MLND Transmission

Cabanas, Watanabe, Higashi, and Bressan (2013) found that species of corn thrips (Scientifically known as Frankliniella williamsi) was found to transmit MCMV semi-persistently .The thrips transmit MCMV after acquisition periods of 3hours, with no evidence for latent periods; both larvae and adults retained the ability to transmit the virus for a period of 6 days after acquisition. The range of vectors for MCMV in Africa is not known, although thrips have been observed in all fields where maize is grown, including in MLN and MCMV- affected farms. It is possible that thrips and other vectors could be playing a major role in MCMV movement within and between fields in the affected countries in Africa.

Mahuku *et al.* (2015) stated that transmission from infected maize seed is usually very low (0.04%). However, even at low rate, seed transmission is epidemiologically significant as maize is only propagated through seed and it leads to introduction of virus into new areas through seed. Also, in conjunction with secondary spread by insect vectors, low rates of seed transmission can translate into high numbers of infected plants, resulting in epidemics. It is also possible for MCMV to be transmitted at a very small rate through infected soil, as the virus can survive in corn residue..

2.3 Disease Control

Leff, *et al.* (2004) said that effective control should target the three components required for disease to occur which are, the virus, the vector and a susceptible host which come together in a suitable environment. The most effective management of MLN would be achieved through the use of integrated approaches combining cultural practices, chemical and biological control of insect vectors, and use of resistant cultivars. In Hawaii, MCMV is managed through the integration of cultural practices with suitable insecticides and host tolerance.

McMurtry and Croft (1997) found that Cucumeris is the preferred predator for thrips control. They are tan coloured mites found on the underside of leaves along the views or inside mature plant. These mites are most effective at preventing thrips build up when applied early in the growing season at the first sign of the thrips. Establishment of Ccumeris required 3 – 4 weeks, so they should be applied before thrips problems develop. Because Cucumeris feed on immature thrips stages a decrease in adult thrip population will not occur for about 3-weeks. Neoseiulus cucumeris (Oudemans), first described by Oudemans in 1930, is a generalist foliar predator known worldwide for its biocontrol potential against a spectrum of pests (whiteflies, thrips, mites, aphids, and psyllids) of horticultural importance. Its ability to survive on plant pollen in the absence of prey and commercialization make this mite one of the most easily adaptable and readily available natural enemies for greenhouse, nursery, or interiorscape production systems.

LeBeck and Leppla (2010) investigate that cucumeris was the first phytoseiid mite found to feed on thrips, but until 1980, its use in thrips management was little explored. However, in the past three decades, due to growing concerns over risks (resistance, environmental, health) associated with chemical control, the use of alternate pest management strategies has received considerable attention. In that context, the use of generalist predators that can perform as a broad spectrum insecticide has been greatly encouraged. Currently in the US, Neoseiulus cucumeris is commercially available by the member companies of the Association of Natural Biocontrol Producers (ANBP) including Tip Top Bio-Control.

Avery, Kumar, Wekesa, Powell, McKenzie, and Osborne (2014) said that in recent years, various formulations (delivery systems) of Neoseiulus cucumeris have been developed to improve their survival and dispersal in commercial crop production systems.

Buckets (100,000 mites) or bottled (50,000 mites) containers are available for direct release in field, greenhouse, and nursery operations. Both systems consist of predatory mites and bran mites (Tyrophagus putrescentiae Schrank, a temporary food source of the predatory mite) mixed with bran or vermiculite. Mite application rates can vary depending upon the crop, climatic conditions, pest species and density, resulting in the recommended application rate to range between 50–100 mites per m^2. A slow release delivery system (controlled release method) is also available, where mites along with the dispersal medium and temporary food are contained in sachets (1000 mites/ sachet) that can be hung on the host plants. Tiny pores within the sachets allow gradual movement of the mite nymphs onto the host plants in subsequent weeks following their application. Although, the predatory mites feed aggressively on several pest species, time spent in prey searching, reproduction, and their dispersion to cover the affected area can slow the pest management. Due to this shortcoming, Neoseiulus cucumeris is generally used as a preventive control tool and can provide efficient control of a pest in its incipient stage of infestation.

Nyamwamu, Mwangi, Gathu and Miano (2015), in their work transmission mechanisms of Maize lethal necrosis disease (MLND) in Kenya said that, maize production is threatened by maize lethal necrosis disease (MLND) which reduced yield by 90 – 100%. It is caused by double infection of maize chlorotic mottle virus (MCMV) and sugarcane mossaic virus (SCMV). MCMV is vectored by thrips and beetles while SCMV by aphics .They further discovered that, the disease induced mottle, leaf and plant death, leaf necrosis and distorted small ears. All maize are susceptible to the disease. They said that the rate of disease spread has been extremely rapid, raising the question of the role of pollen on MLND epidemiology. The study was aims at understanding the transmission mechanisms and the viruses characteristics of viruses causing MLND and also determined the role of pollen in MLND spread including the role of *Frankliniella Williams* and F. *Occidentallis* in pollen mediated transmission.

Fatma, Tileye and Patrick (2015) investigated in their study Insights of maize lethal necrotic disease (MLND) and discovered that MLND is a new disease in East Africa, first reported in Kenya in 2011 and then spread to Tanzania, Uganda and Rwanda. The disease is caused by Maize Chlorotic Mottle Virus (MCMV) in combination with viruses of genus Potyvirus, mostly Sugarcane Mosaic Virus (SCMV). Their finding revealed that co-infection is the one that results in intensive to complete yield loss. Diagnosis of MLND based on symptoms is reported ineffective because symptoms like stunting and chlorosis

resembles nutrient deficiencies or maize mosaic. Detection and characterization of MLND causing viruses have been done by techniques such as enzyme-linked immune-sorbent assay (ELISA), polymerase chain reaction (PCR) and next generation sequencing. Relatively little work has been done to characterize MLND causing viruses in Tanzania prior to those techniques. Their result show that the disease can be managed through the use of certified seeds, sanitation, quarantine, crop rotation, the use of resistant/tolerant maize varieties and other cultural practices. Finally they conclude that use of resistant maize varieties is considered the most reliable, eco-friendly, effective and economical way of managing MLND.

Chelang *et at.* (2019) worked on Genetic architecture of maize chlorotic mottle virus and maize lethal necrosis through GWAS, linkage analysis and genomic prediction in tropical maize germplasm. Their finding reveled that, Maize lethal necrosis (MLN) is a serious threat to the food security of maize-growing smallholders in sub-Saharan Africa. The objective of their study was to gain insights and validate the genetic architecture of resistance to MCMV and MLN in maize. They applied linkage mapping to three doubled-haploid populations and a genome-wide association study (GWAS) on 380 diverse maize lines. For all the populations, phenotypic variation for MCMV and MLN was significant, and heritability was moderate to high. Linkage mapping revealed 13 quantitative trait loci (QTLs) for MCMV resistance and 12 QTLs conferring MLN resistance. One major-effect QTL, qMCMV3-108/qMLN3-108, was consistent across populations for both MCMV and MLN resistance. Joint linkage association mapping (JLAM) revealed 18 and 21 main-effect QTLs for MCMV and MLN resistance, respectively. Another major-effect QTL, MCMV6-17/qMLN617, was detected for both MCMV and MLN resistance. The GWAS revealed a total of 54 SNPs (MCMV-13 and MLN-41) significantly associated ($P \leq 5.60 \times 10{-}05$) with MCMV and MLN resistance. Most of the GWAS-identified SNPs were within or adjacent to the QTLs detected through linkage mapping. The prediction accuracy for within populations as well as the combined populations is promising; however, the accuracy was low across populations. Overall, MCMV resistance is controlled by a few major and many minor-effect loci and seems more complex than the genetic architecture for MLN resistance.

Obiora and Kevin (2016) in their work discovered that Pathogens and insects can have important negative effects on yields of crops cultivated by humans. These effects can be important for the food security or financial well-being of individuals. In particular,

maize is a very important staple crop worldwide and is vulnerable to diseases. They developed a mathematical model that evaluates the impacts of foliar diseases on the population dynamics of maize plants. Qualitative analyses of the important mathematical features of the model are carried out. The study show how the methodology can be extended to reducing the spread of foliar diseases through effective control measures with minimum costs.

Olga and Tibor (2015) investigated Maize pathogen interaction: .They reported that maize *(Zea mays*) is a host to numerous pathogenic species that impose serious diseases to its ear and foliage, negatively affecting the yield and the quality of the maize crop. They summarize interactions of maize with its agriculturally important pathogens that were assessed at the proteome level. Also employed differential analyses, such as the comparison of pathogen-resistant and susceptible maize varieties, as well as changes in maize proteomes after pathogen challenge, numerous proteins were identified as possible candidates in maize resistance. They describe findings of various research groups that used mainly mass spectrometry-based, high through-put proteomic tools to investigate maize interactions with fungal pathogens Aspergillums avus Fusarium spp., and Curvularia lunata , and viral agents Rice Black-streaked Dwarf Virus and Sugarcane Mosaic Virus.

Kannan, Ismail and Bunewan (2018) wrote that, maize dwarf mosaic virus (MDMV) is a serious maize pathogen, epidemic worldwide and one of the most common virus diseases for monocotyledonous plants, causing up to 70% loss in corn yield globally since 1960. MDMV belongs to the genus Potyvirus (Potyviridae). MDMV is a single stranded positive sense RNA virus and is transmitted in a non-persistent manner by several aphid species. MDMV is amongst the most important virus diseases in maize worldwide.

Yanxuan and Zhi-Qiang (2000) reported that, the predatory (Amblyseius cucumeris), was studied in the laboratory to evaluate its potential as a biocontrol agent against the spider mite and thrips. A. cucumeris started to lay eggs at the age of 3 days with a daily rate of 1-4 eggs (average of 2.2) over a period of 7-18 days and a total fecundity of 14-47 eggs. The number of prey consumed by predators increased with prey density and the number of eggs produced was directly correlated with the number of prey consumed. Female predators consumed twice as many female spider mites as did male. A cucumeris females were unable to invade intact webnests of S. nanjingensis but were able to invade and liked to stay and lay eggs in broken nests with existing openings or holes.

Herbert, Chair, George and Thomas (2013) discovered that, thrips are major agriculturalpests throughout much of the United States. More information is needed about sampling methodmanagementpractices,and insecticide susceptibility to hel p better control this pest.Two year survey was conducted to determine the species present i n southeast Virginia and the population characteristics of those species. Thrips were monitored using yellow sticky traps. In general thrips populations began to build up beginning in April, peaked in August,1012 and then started to decline. Differences in this trend were observed between species. Also study was conducted in seedling soybean to evaluate the within -plant location of thrips, whether a plant subsample could be used for thrips monitoring , and to determine the thrips species complex present.

Olaniyan (2015) wrote that Maize (*Zea mays l*) is always preferred to the crops and it is fast belonging an industrial crop in Sub-Saharan African countries. Nigeria has been divided into low, medium, medium high and high maize production potential groups. Traditionally, maize was mostly grown in forest ecology in Nigeria but large scale production has move to the Savannah zone, especially the northern guinea where yield potential is uch higher. Maize yield in Nigeria is still very low due to biotic, abiotic agronomic factors like insect, disease soil infertility and, unavailability of improved germplasms, weeds, and uncertain access to markets etc.

CHAPTER THREE

RESEARCH METHODOLOGY

3.1 Assumptions of the Existing Model

William, Dmitry and Livingstone (2018) developed their model with the following assumptions:

i. Pathogen from infected maize plant reaches vegetative environment by vector and through shedding which is due to wind, humans, rain and birds.
ii. Transmission of infection from vegetative environment to susceptible maize is through vector and shedding, which is due to wind, humans, rain and bds.
iii. Once the vectors become the carriers of the disease, it is for the whole of their life.
iv. The population is heterogeneous. This is to say, the individuals which composed the population can be grouped into different classes in accordance to their epidemiological state.
v. Each susceptible individual in the class has equal chance of getting infected by infectious individuals with adequate contact.
vi. The only pathway of entering into the population is through sowing seeds and the only way of exit is though death from MLND related cause. Migration and other causes of death are ignored.

3.2 Description of the Existing Model

William, Dmitry and Livingstone (2018) presents an SI-SEI-type model of host and vector populations that incorporates: The maize host population, which is categorized as Susceptible Sub-population (S_m), Exposed maize sub-population (E_m) and Infected maize sub-population (I_m). The model includes the vector population, which is categorized as Susceptible vector sub-population (S_v) and infected vector sub-population (I_v). It also includes: virus in the environment (P_o). In their model, there is no recruitment rate since it describes a single season and no plantation of maize plants. The class S_m declines constantly at the rate (η), which is due to force of infection between virus in the environment (P_o), direct contact of maize plant to maize plant (I_m) and vector from infected maize plants (I_v). The infected maize plants increases the number of exposed plants (E_m) at the same constant rate (η). However, some susceptible maize plants remain in the exposed

state while the remaining ones immediately progress to infected maize class, (I_m) at a constant rate (α_m). Then the infected maize has disease-induced constant death rate σ_m. They considered that there is no natural death in the host plants population for all three classes as maize plants never naturally die till harvested at the end of season. The infected maize and the environment transmit the MNLD virus to the susceptible vector, S_v at constant rates (β_{mv}) and (β_o) respectively. The susceptible vectors are constantly recruited at the rate Λ, constantly progress to infected vector class with mass action $\lambda(P_o,I_m)$, with proportionality constant rate λ and die naturally at a constant rate μ_v. The infected vectors also naturally die at the same constant rate μ_v. The MNLD virus in the environment are recruited at the rate proportional to (θP_o) and from the infected maize plants at a constant rate (σ), and then die naturally at the rate(μ).

Table 3.1: Variables of the existing model and their descriptions

Variables	**Descriptions**
N_m	Total number of the maize plant population
N_v	Total number of vector population
S_m	Susceptible population maze plant
S_v	Susceptible vector population
E_m	Exposed maize plant with latent incubation period
I_m	Infected population maize plant
I_v	Infected vector population for maize plant
P_o	Other plants carrier of pathogens (vegetative environment)

Table 3.2: Parameters of the exiting model and their descriptions

Parameters	Descriptions
β_m	Contact rate from infected maize to susceptible vector population
μ	Natural death rate of the vector within environment
β_o	Contact rate of other plant (not maize) carrying pathogen with maize
β_{ov}	Contact rate of vectors in the environment to the susceptible vectors
β_{vm}	Contact rate from vector carrying virus to susceptible maize population
β_{mm}	Contact rate of contagious maize to susceptible maize population
λ_{vo}	Contact rate from vector carrying virus to the vegetative environment
λ_{mo}	Contact rate from infected maize plant to vegetative environment
$\wedge$	Recruitment rate into susceptible vectors
η	Force of infection rate of masses of action due to vectors I_m and virus in P_0
λ	Force of infection rate of combined masses of action due to infected maize and virus in the environment
μ_v	Natural death rate of vector population
σ	Contribution of infected maize to the growth of virus
θ	Contribution of infected plant to the growth of virus in the environment
α_m	Progression rate of exposed maize plant to be infected

3.3 Diagram of the Existing Model

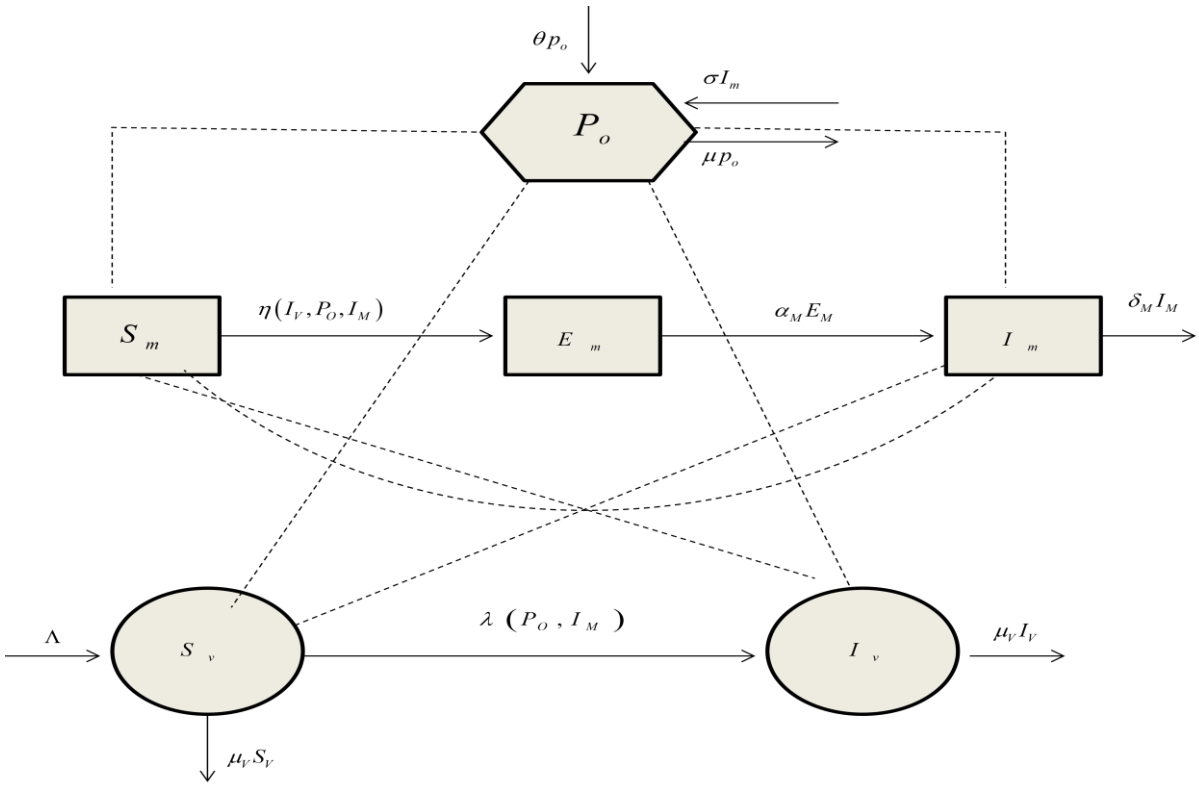

Figure 3.1: Model diagram of maize host and vector populations

3.3 Existing Model Equations of Maize Host and Vector Population

$$\frac{dS_m}{dt} = -(\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}) S_m \tag{3.1}$$

$$\frac{dE_m}{dt} = (\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}) S_m - \alpha_m E_m \tag{3.2}$$

$$\frac{dI_m}{dt} = \alpha_m E_m - \sigma_m I_m \tag{3.3}$$

$$\frac{dS_v}{dt} = \Lambda - (\beta_{ov} \frac{P_o}{k + P_o} + \beta_{mv} + \frac{I_m}{N_m}) S_v - \mu_v S_v \tag{3.4}$$

$$\frac{dI_v}{dt} = (\beta_o \frac{P_o}{k + P_o} + \beta_{mv} \frac{I_m}{N_m}) S_v - \mu_v I_v \tag{3.5}$$

$$\frac{dP_o}{dt} = \lambda_{mo} \frac{I_m}{N_m} + \lambda_{ov} \frac{I_v}{N_v} - \mu_o P_o \tag{3.6}$$

3.4 New Assumptions

In addition to the assumption of the existing model the following has been made:

i. The vectors (thrips) can only transmit the MLND causing virus at their larvae and adult stages for the period of 6 days. After that they return to susceptible class.

ii. preys population will grows exponentially when the predator is absence.

iii. the predator population will multiply when prey is presence.

iv. the predator population will decrease exponentially when the preys is absence.

3.5 Description of the Modified Model

The modified model for the MLND is partition into host and vector population that incorporate the maize host population, which is subdivided into Susceptible maize population (S_m), Exposed maize population (E_m) and Infected maize population (I_m). The vector population whch is subdivided into susceptible vector population (S_v) and infected vector population (I_v). It also includes virus in the environment (P_o) and natural enemy of

the vector (C). The class S_m decline at constant rate (η), due to the force of infection between virus in the environment (P_o), direct contact of susceptible maize plant (S_m) to infected maize plant (I_m) and vector carrying the virus (I_v). The infected maize plant increase the number of Expose class (E_m) at a constant rate (η). However, some susceptible maize plant remain in the exposed state throughout the season while soe progress to Infected class at constant rate (α_m). Maize plant die naturally at constant rate (μ_m). Both the infected maize plant (I_m) and virus in the environment (P_o) transmit MLND causing virus to Susceptible vector (S_v) at constant rate (β_{mv}) and (β_{0v}) respectively. The Susceptible vector recruited at constant rate (∧), constantly progress to Infected vector class (I_v) with mass action ϕ (P_o, I_m), with proportionality constant rate (ϕ). Infected vector (I_v) return to Susceptible vector (S_v) at constant rate (π_m). Both the Susceptible vector (S_v) and Infected vector (I_v) die naturally at constant rate (μ_v) and due to capture by predator at constant rate (μ_c) respectively. The MLND in the environment are recruited at the rate proportional to (θP_o) and die naturally at rate (μ_o). The predator is recruited at rate (γ), population of predator increase at rate (ε_c) as a result of interaction between predator and vector and die naturally at constant rate (μ).

Table 3.3: Variables of the modified model and their descriptions

Variables	**Descriptions**
N_m	Total number of the maize plant population
N_v	Total number of vector population
S_m	Susceptible population maze plant
E_m	Exposed maize plant with latent incubation period
I_m	Infected population maize plant
S_v	Susceptible vector population
I_v	Infected vector population for maize plant
P_o	Other plants carrier of pathogens (vegetative environment)
C	Total number of predator population

Table 3.4: Parameters of the modified model and their descriptions

Parameters	Descriptions
β_{mv}	Contact rate from infected maize to susceptible vector population
μ_0	Natural death rate of he vector within environment
β_0	Contact rate of other plant (not maize) carrying pathogen with maize
β_{0v}	Contact rate of virus in the environment to the susceptible vectors
β_{mv}	Contact rate from vector carrying virus to susceptible maize population
β_{mm}	Contact rate of contagious maize to susceptible maize population
ϕ_{0v}	Contact rate from vector carrying virus to the vegetative environment
ϕ_{mo}	Contact rate from infected maize plant to vegetative environment
$\wedge$	Recruitment rate into susceptible vectors
η	Force of infection rate of 3 masses of action due to vectors I_m and virus in P_0
λ	Force of infection rate of combined masses of action due to infected maize and virus in the environment
μ_v	Natural death rate of vector population
θ	Contribution of infected plant to the growth of virus in the environment
α_m	Progression rate of exposed maize plant to be infected
μ_m	Natural death rate of maize plant.
π_m	Return of infected vector when becomes susceptible.
μ_c	Capture efficiency of the predator.
μ	Natural death rate of enemy
γ	Recruitment rate into predator class.
ε_c	Conversion efficiency of the predator.

3.6 Diagram of the Modified Model

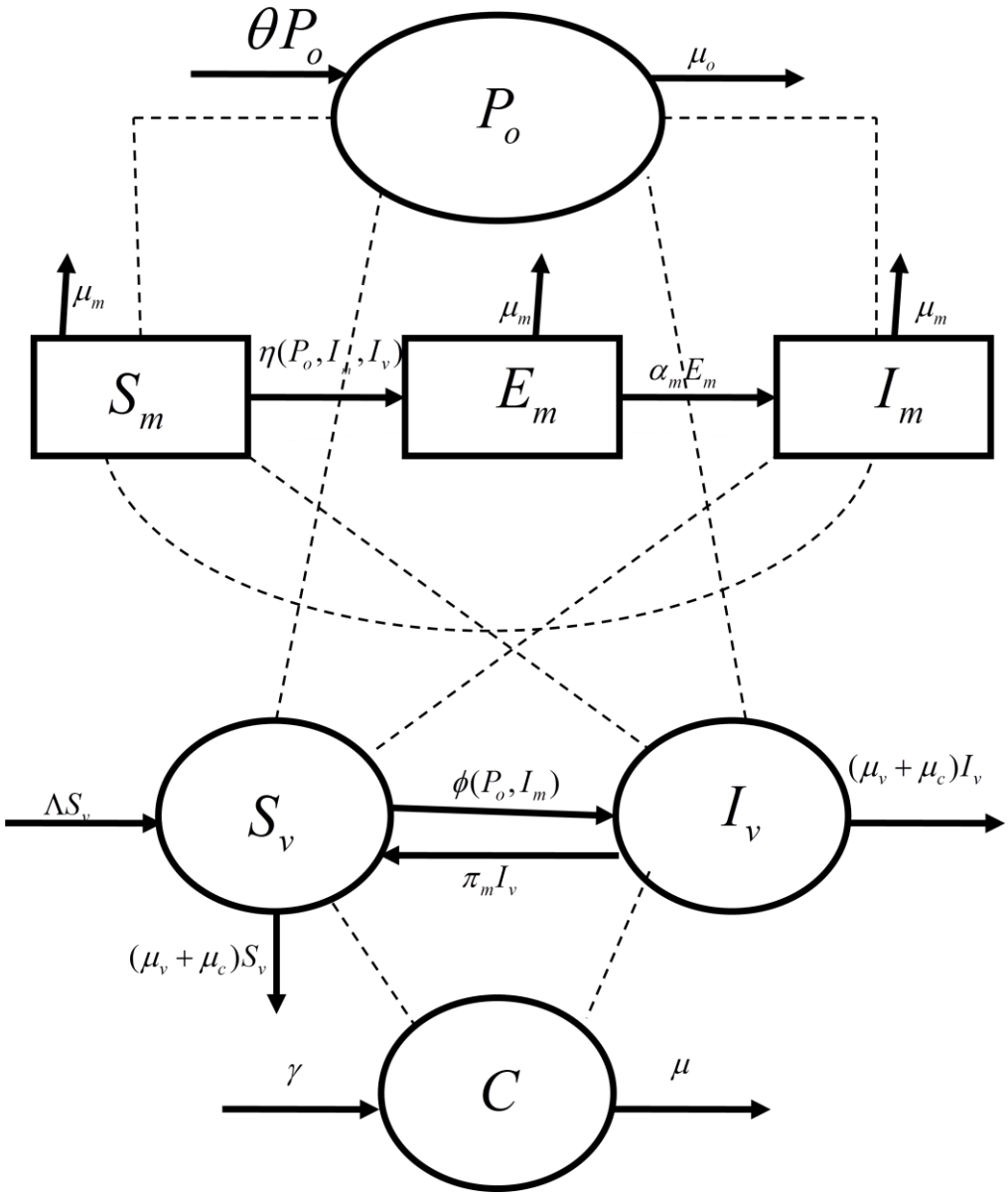

Figure 3.2: Model diagram of maize host and vector

3.7 Modified Equations of the Model

$$\left.\begin{aligned}
\frac{dS_m}{dt} &= -(\beta_o \frac{P_o}{k+P_o} + \beta_{vm}\frac{I_v}{N_v} + \beta_{mm}\frac{I_m}{N_m} + \mu_m)S_m \\
\frac{dE_m}{dt} &= (\beta_o \frac{P_o}{k+P_o} + \beta_{vm}\frac{I_v}{N_v} + \beta_{mm}\frac{I_m}{N_m})S_m - (\alpha_m + \mu_m)E_m \\
\frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m \\
\frac{dS_v}{dt} &= \Lambda - (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m} + \mu_v + \mu_c C)S_v + \pi_m I_v \\
\frac{dI_v}{dt} &= (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m})S_v - (\mu_v + \mu_c C + \pi_m)I_v \\
\frac{dP_o}{dt} &= \phi_{mo}\frac{I_m}{N_m} + \phi_{ov}\frac{I_v}{N_v} + (\theta - \mu_o)P_o \\
\frac{dC}{dt} &= \gamma C + \varepsilon_c(S_v + I_v)C - \mu C
\end{aligned}\right\} \quad (3.7)$$

3.8 Method of Analysis

3.8.1 Disease free equilibrium (DFE) state

Disease free equilibrium is a steady state solution when there is no disease spreading in the population. We shall thus obtain DFE of the system of equations (3.7) when all the variables and parameters relation to disease transmission are zero.

3.8.2 Stability of DFE state

We shall use the method of linearization to establish the local stability of the DFE of the proposed model by obtaining Jacobean matrix of the equation in proportion and evaluate the Jacobean matrix at the DFE state. The *Jacobean* matrix is defined by

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial x_1}\frac{\partial f_1}{\partial x_2} & \cdots & \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1}\frac{\partial f_2}{\partial x_2} & \cdots & \frac{\partial f_2}{\partial x_n} \\ \vdots & & \vdots \\ \frac{\partial f_n}{\partial x_1}\frac{\partial f_n}{\partial x_2} & \cdots & \frac{\partial f_n}{\partial x_n} \end{pmatrix}$$

Where J is called the Jacobean matrix associated with the general model equation

$$\dot{x} = f(x)$$

$$Thus, J(S_m, E_m, I_m, S_v, I_v, P_0, C) = \begin{pmatrix} \frac{\partial f_1}{\partial S_m} & \frac{\partial f_1}{\partial E_m} & \frac{\partial f_1}{\partial I_m} & \frac{\partial f_1}{\partial S_v} & \frac{\partial f_1}{\partial I_v} & \frac{\partial f_1}{\partial P_o} & \frac{\partial f_1}{\partial C} \\ \frac{\partial f_2}{\partial S_m} & \frac{\partial f_2}{\partial E_m} & \frac{\partial f_2}{\partial I_m} & \frac{\partial f_2}{\partial S_v} & \frac{\partial f_2}{\partial I_v} & \frac{\partial f_2}{\partial P_o} & \frac{\partial f_2}{\partial C} \\ \frac{\partial f_3}{\partial S_m} & \frac{\partial f_3}{\partial E_m} & \frac{\partial f_3}{\partial I_m} & \frac{\partial f_3}{\partial S_v} & \frac{\partial f_3}{\partial I_v} & \frac{\partial f_3}{\partial P_o} & \frac{\partial f_3}{\partial C} \\ \frac{\partial f_4}{\partial S_m} & \frac{\partial f_4}{\partial E_m} & \frac{\partial f_4}{\partial I_m} & \frac{\partial f_4}{\partial S_v} & \frac{\partial f_4}{\partial I_v} & \frac{\partial f_4}{\partial P_o} & \frac{\partial f_4}{\partial C} \\ \frac{\partial f_5}{\partial S_m} & \frac{\partial f_5}{\partial E_m} & \frac{\partial f_5}{\partial I_m} & \frac{\partial f_5}{\partial S_v} & \frac{\partial f_5}{\partial I_v} & \frac{\partial f_5}{\partial P_o} & \frac{\partial f_5}{\partial C} \\ \frac{\partial f_6}{\partial S_m} & \frac{\partial f_6}{\partial E_m} & \frac{\partial f_6}{\partial I_m} & \frac{\partial f_6}{\partial S_v} & \frac{\partial f_6}{\partial I_v} & \frac{\partial f_6}{\partial P_o} & \frac{\partial f_6}{\partial C} \\ \frac{\partial f_7}{\partial S_m} & \frac{\partial f_7}{\partial E_m} & \frac{\partial f_7}{\partial I_m} & \frac{\partial f_7}{\partial S_v} & \frac{\partial f_7}{\partial I_v} & \frac{\partial f 7}{\partial P_o} & \frac{\partial f_7}{\partial C} \end{pmatrix}$$

Then the local stability of DFE will be determine base on the eigen values of the Jacobean matrix.

3.8.3 Basic reproduction number

The next generation matrix techniques by Diekman and Heesterbeek (2009) was used for the computation of the basic reproduction number R_0.

The next generation matrix is use to derive the basic reproduction number, for a compartment model of the spread of infectious diseases. To calculate the basic reproduction number by using next generation matrix, the whole population is divided into n-compartments in which there are $m < n$ infected compartments. let $x_i = 1, 2, 3, \ldots, m$ be the number of infected individuals n i^{th} infected compartments at time t. Now the model is

$$\frac{\partial x_i}{\partial t} = F(x) - V(x)$$

Where $V(x) = [V_i^-(x) - V_i^+(x)]$

Let x_0 be the disease free equilibrium. The values of the Jacobean matrices $F(x)$ and $V(x)$ are $\mathrm{F}(x_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}$ and $V(x_0) = \begin{pmatrix} V & 0 \\ f_3 & J_4 \end{pmatrix}$ respectively. Here F and V are matrices, defined as

$$F = [\frac{\partial f_i}{\partial x_j}(x_0)] \quad \text{and}$$

$$V = [\frac{\partial v_i}{\partial x_j}(x_0)] \quad \text{with } 1 \leq i, j \leq m$$

Now, the matrix FV^{-1} is known as next generation matrix. The largest eigen value or spectral radius of FV^{-1} is the basic reproduction number of the model.

3.8.4 Numerical scheme

In this research, MATLAB R2015a was used for the numerical experiments of the model

3.8.5 Source of data

The study adopted the theoretical data from specified literature in an event where real data are not tenable.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Introduction

In this chapter, we present the analytical study of our work; we established the existence and uniqueness of the solution, positivity of the solution, existence of disease free equilibrium (DFE) point and obtained the basic reproduction number of the disease. We also analyzed both local and global stability of the disease free equilibrium point of the model and carry out numerical simulation using MATLAB R2015a.

4.1.1 The Existence and the uniqueness of the solution

The validity and authenticity of any mathematical model depends on whether the given system of equations has a solution and if the solution exists is unique. We shall use the Lipchitz condition to verify the existence and uniqueness of the system of equations (3.7).

4.1.1.1 Statement of theorem

Consider the system of equation below

$$\left.\begin{aligned} x_1^1 &= f_1(t, x_1, x_2, \ldots x_n), x_1(t_0) = x_{10} \\ x_2^1 &= f_1(t, x_1, x_2, \ldots x_n), x_2(t_0) = x_{20} \\ &\cdot \quad \cdot \quad \cdot \\ &\cdot \quad \cdot \quad \cdot \\ &\cdot \quad \cdot \quad \cdot \\ x_n^1 &= f_n(t, x_1, x_2, \ldots x_n), x_n(t_0) = x_{n0} \end{aligned}\right\} \tag{4.1}$$

We may write equation (4.1) in compact form as

$$x^1 = f_1(t, x), x_1(t_0) = x_0 \tag{4.2}$$

Theorem:4.1.1

Let D denote the region

$$\left|t - t_o\right| \le a, \left\|x - x_o\right\| \le b,\ x = (x_1, x_2, \ldots, x_n),\ x_o = (x_{10}, x_{20}, \ldots, x_{n0}) \tag{4.3}$$

And suppose that $F(t, x)$ satisfies the Lipchitz condition

$$\left\| f(t, x_1) - f(t, x_2 \right\| \leq k \left\| x_1 - x_2 \right\| \tag{4.4}$$

Whenever the pairs (t, x_1) and (t, x_2) belong to D^1, where K is a positive constant. Then there is a constant $\delta > 0$ such that there exist a unique continues vector solution of $\underline{X}(t)$ of system (4.2) in the interval $|t - t_o| \leq \delta$. It is important to note that the condition (4.4) is satisfied by the requirement that $\frac{\partial f_i}{\partial x_j}, i, j = 1, 2, \ldots$ be continues and bounded in D^1.

Theorem 4.1.2

Let D denote the region defined in $1 \leq \varepsilon \leq R$, such that $1 \leq \varepsilon \leq R$, and $0 < R < \infty$, hold then there exist a unique solution of system equations (3.7) which is bounded in the region D.

Then there exists a unique solution of the system of equations (3.7) which is bounded in D.

Proof:

Let

$$\lambda_m = \beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}, \lambda_v = \beta_{ov} \frac{P_o}{k + P_o} + \beta_{mv} \frac{I_m}{N_m} + \mu_c C, \lambda_c = \varepsilon_c (S_v + I_v), \sigma = \mu_c C,$$

$\phi_{mo} = \lambda_{mo}$ and $\phi_{ov} = \lambda_{ov}$ also

Let $S_m = x_1, E_m = x_2, I_m = x_3, S_v = x_4, I_v = x_5, P_o = x_6$ and $C = x_7$

Then system of equations (3.7) becomes

$$\left.\begin{aligned}
F_1 &= -(\lambda_m + \mu_m) x_1 \\
F_2 &= \lambda_m x_1 - (\alpha_m + \mu_m) x_2 \\
F_3 &= \alpha_m x_2 - \mu_m x_3 \\
F_4 &= \Lambda - (\lambda_v + \mu_v) x_4 + \pi_m x_5 \\
F_5 &= \lambda_v x_4 - (\mu_v + \varepsilon_c + \pi_m) x_5 \\
F_6 &= \phi_{mo} \frac{x_3}{N_m} + \phi_{ov} \frac{x_5}{N_v} + (\theta - \mu_o) x_6 \\
F_7 &= (\gamma + \lambda_c - \mu) x_7
\end{aligned}\right\} \tag{4.5}$$

It is sufficient to show that $\frac{\partial f_i}{\partial x_i}, i = 1, 2, 3, 4, 5, 6, 7$ are continuos and bounded in D.

From first equation in system of equations (4.5) we have

$$\left|\frac{\partial F_1}{\partial x_1}\right| = \left|-(\lambda_m + \mu_m)\right| < \infty$$

$$\left|\frac{\partial F_1}{\partial x_2}\right| = \left|\frac{\partial F_1}{\partial x_3}\right| = \left|\frac{\partial F_1}{\partial x_4}\right| = \left|\frac{\partial F_1}{\partial x_5}\right| = \left|\frac{\partial F_1}{\partial x_6}\right| = \left|\frac{\partial F_1}{\partial x_7}\right| = 0 < \infty$$

From second equation in system of equations (4.5) we have

$$\left|\frac{\partial F_2}{\partial x_1}\right| = \left|\lambda_m\right| < \infty$$

$$\left|\frac{\partial F_2}{\partial x_2}\right| = \left|-(\alpha_m + \mu_m)\right| < \infty$$

$$\left|\frac{\partial F_2}{\partial x_3}\right| = \left|\frac{\partial F_2}{\partial x_4}\right| = \left|\frac{\partial F_2}{\partial x_5}\right| = \left|\frac{\partial F_2}{\partial x_6}\right| = \left|\frac{\partial F_2}{\partial x_7}\right| = 0 < \infty$$

From third equation in system of equations (4.5) we have

$$\left|\frac{\partial F_3}{\partial x_2}\right| = \left|\alpha_m\right| < \infty$$

$$\left|\frac{\partial F_3}{\partial x_3}\right| = \left|-\mu_m\right| < \infty$$

$$\left|\frac{\partial F_3}{\partial x_1}\right| = \left|\frac{\partial F_3}{\partial x_4}\right| = \left|\frac{\partial F_3}{\partial x_5}\right| = \left|\frac{\partial F_3}{\partial x_6}\right| = \left|\frac{\partial F_3}{\partial x_7}\right| = 0 < \infty$$

From fourth equation in system of equations (4.5) w have

$$\left|\frac{\partial F_4}{\partial x_4}\right| = \left|-(\lambda_v + \mu_v)\right| < \infty$$

$$\left|\frac{\partial F_4}{\partial x_5}\right| = \left|\pi_m\right| < \infty$$

$$\left|\frac{\partial F_4}{\partial x_1}\right| = \left|\frac{\partial F_4}{\partial x_2}\right| = \left|\frac{\partial F_4}{\partial x_3}\right| = \left|\frac{\partial F_4}{\partial x_6}\right| = \left|\frac{\partial F_4}{\partial x_7}\right| = 0 < \infty$$

From firth equation in system of equations (4.5) w have

$$\left|\frac{\partial F_5}{\partial x_4}\right| = \left|\lambda_v\right| < \infty$$

$$\left|\frac{\partial F_5}{\partial x_5}\right| = \left|-(\mu_v + \sigma + \pi_m)\right| < \infty$$

$$\left|\frac{\partial F_5}{\partial x_1}\right| = \left|\frac{\partial F_5}{\partial x_2}\right| = \left|\frac{\partial F_5}{\partial x_3}\right| = \left|\frac{\partial F_5}{\partial x_6}\right| = \left|\frac{\partial F_5}{\partial x_7}\right| = 0 < \infty$$

From sixth equation in system of equations (4.5) we have

$$\left|\frac{\partial F_6}{\partial x_3}\right| = \left|\frac{\phi_{mo}}{N_m}\right| < \infty \;,\; \left|\frac{\partial F_6}{\partial x_5}\right| = \left|\frac{\phi_{ov}}{N_v}\right| < \infty \;,\; \left|\frac{\partial F_6}{\partial x_6}\right| = \left|-(\mu_o - \theta)\right| < \infty$$

$$\left|\frac{\partial F_6}{\partial x_1}\right| = \left|\frac{\partial F_6}{\partial x_2}\right| = \left|\frac{\partial F_6}{\partial x_4}\right| = \left|\frac{\partial F_6}{\partial x_7}\right| = 0 < \infty$$

From seventh equation in system of equations (4.5) we have

$$\left|\frac{\partial F_7}{\partial x_7}\right| = \left|(\gamma + \lambda_c - \mu)\right| < \infty$$

$$\left|\frac{\partial F_7}{\partial x_1}\right| = \left|\frac{\partial F_7}{\partial x_2}\right| = \left|\frac{\partial F_7}{\partial x_3}\right| = \left|\frac{\partial F_7}{\partial x_4}\right| = \left|\frac{\partial F_7}{\partial x_5}\right| = \left|\frac{\partial F_7}{\partial x_6}\right| = 0 < \infty$$

Clearly all these partial derivatives are continuos and bounded. Hence by theorem 2, there exist a unique solution for he system of equations (4.5) in the region D.

4.1.2 Invariant region

Lemma 4.1.2

Let $(S_m, E_m, I_m, S_v, I_v, P_0, C)$ be the solution of the system of equations (4.5) with the initial conditions and biological feasible region given by the set $\Omega = \Omega_m + \Omega_v$ where

$$\Omega_m = \left\{(S_m, E_m, I_m) \in R^3 : N_m \leq -\mu_m N_m\right\}$$

$$\Omega_v = \left\{(S_v, I_v) \in R^2 : N_v \leq \frac{\Lambda}{(\mu_v + \mu_c)}\right\}$$

Proof

$$N_m = S_m + E_m + I_m$$

$$N_m(t) = S_m(t) + E_m(t) + I_m(t)$$

$$\frac{dN_m}{dt} = \frac{dS_m}{dt} + \frac{dE_m}{dt} + \frac{dI_m}{dt}$$

$$= -(\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m} + \mu_m)S_m + (\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m})S_m - (\alpha_m + \mu_m)E_m$$

$$+\alpha_m E_m - \mu_m I_m$$

$$= -\mu_m S_m - \mu_m E_m - \mu_m I_m$$

$$\frac{dN_m}{dt} = -\mu_m(S_m + E_m + I_m)$$

$$\frac{dN_m}{dt} = -\mu_m N_m$$

$$\Rightarrow \frac{dN_m}{dt} \leq -\mu_m N_m$$

By separation of variables, we have

$$\frac{dN_m}{N_m} \leq -\mu_m dt$$

Integrating both sides, we have

$$\int \frac{1}{N_m} dN_m \leq \int -\mu_m dt$$

$$InN_m \leq -\mu_m t + C$$

Taking exponential of both sides, we have

$$N_m \leq e^{-\mu_m t + C}$$

$$N_m(t) \leq e^{-\mu_m t}.e^c$$

Let $C = e^c$

$$N_m(t) \leq C e^{-\mu_m t}$$

as t = 0

$$N_m(0) = C$$

$$N_m(t) = N_m(0)e^{-\mu_m t}$$

$$as\ t \to \infty$$

$$N_m(t) = N_m(0)$$

Similarly,

$$N_v = S_v + I_v$$

$$N_v(t) = S_v(t) + I_v(t)$$

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dI_v}{dt}$$

$$= \Lambda - (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m} + \mu_v + \mu_c C)S_v + \pi_m I_v + (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m})S_v - (\mu_v + \mu_c C + \pi_m)I_v$$

$$= \Lambda - (\mu_v + \mu_c C)(S_v + I_v)$$

$$\frac{dN_v}{dt} = \Lambda - (\mu_v + \mu_c C)N_v$$

$$\frac{dN_v}{dt} + (\mu_v + \mu_c C)N_v = \Lambda$$

By integrating factor we have

$$IF = e^{\int p\,dt} = e^{\int (\mu_v + \mu_c C)t + c_1}$$

$$(\mu_v + \mu_c C)e^{(\mu_v + \mu_c C)} = \Lambda \int e^{(\mu_v + \mu_c C) + c_1}$$

$$= \frac{\Lambda e^{(\mu_v + \mu_c C)}}{(\mu_v + \mu_c C)} + C_1$$

$$= \left(\frac{\Lambda e^{(\mu_v + \mu_c C)}}{(\mu_v + \mu_c C)} + C_1\right) e^{(\mu_v + \mu_c C)t}$$

$$= \frac{\Lambda}{(\mu_v + \mu_c C)} + C_1 e^{(\mu_v + \mu_c C)t}$$

$as\ t \to 0$, we have

$$N_v(0) = \frac{\Lambda}{(\mu_v + \mu_c C)} + C_1$$

$$C = N_v(0) - \frac{\Lambda}{(\mu_v + \mu_c C)}$$

as $t \to \infty$, we have

$$N_v(t) = \frac{\Lambda}{(\mu_v + \mu_c C)}$$

Therefore, the system of equations (4.5) is well posed epidemiologically and mathematically. Hence it is sufficient to study the dynamics of the system of equations (4.5).

4.1.3 Positivity of the solution

Since the system of equations (4.5) given, considered plants and vector populations, it is appropriate to show that all the state variables of the system are non negative.

Theorem 4.1.3:

For non negative initial conditions of the system of equations given by (4.5), the solution $(S_m, E_m, I_m, S_v, I_v, P_0, C)$ of the system of equations (4.5) are all non negative for all time $t > 0$

Proof:

From the first equation of system of equations (4.5)

ie $\frac{dS_m}{dt} = -(\lambda_m + \mu_m)S_m$

We have

$$\frac{dS_m}{dt} \geq -(\lambda_m + \mu_m)S_m$$

By separation of variables, we have

$$\frac{dS_m}{S_m} \geq -(\lambda_m + \mu_m)dt$$

Integrating both side

$$\int \frac{1}{S_m} dS_m \geq \int -(\lambda_m + \mu_m)tdt$$

$$InS_m \geq \int -(\lambda_m + \mu_m)tdt + c$$

Taking exponential of both sides, we have

$$S_m(t) \geq e^{-\int(\lambda_m + \mu_m)tdt}.e^c > 0$$

$$S_m \geq C_1 e^{-\int(\lambda_m + \mu_m)tdt}$$

Applying the initial condition t = 0

$$S_m(0) \geq C_1$$

$$S_m \geq S_m(0)e^{-\int(\lambda_m+\mu_m)tdt} > 0$$

$$S_m(t) > 0, \forall\, t > 0$$

Since $S_m(0) > 0$, then $S_m(t) > 0$ for $t > 0$.

From the second equation of system of equation (4.5)

i.e $\dfrac{dE_m}{dt} = \lambda_m S_m - (\alpha_m + \mu_m)E_m$

W have

$$\frac{dE_m}{dt} \geq -(\alpha_m + \mu_m)E_m$$

By separation of variables, we have

$$\frac{dE_m}{E_m} \geq -(\alpha_m + \mu_m)dt$$

Integrating both sides, we have

$$\int \frac{1}{E_m} dE_m \geq \int -(\alpha_m + \mu_m)dt$$

$$InE_m \geq -(\alpha_m + \mu_m)t + C$$

Taking exponential of both sides

$$E_m(t) \geq e^{-(\alpha_m+\mu_m)t+C}$$

$$E_m(t) \geq e^{-(\alpha_m+\mu_m)t}.e^{C}$$

Let $e^{C} = C_2$

$$E_m(t) \geq C_1 e^{-(\alpha_m+\mu_m)t}$$

Applying initial condition $t = 0$

$$E_m(0) \geq C_1 e^{0}$$

$$\Rightarrow E_m(0) = C_2$$

Put $C_2 = E_m(0)$, we have

$$E_m(t) > E_m(0)e^{-(\alpha_m+\mu_m)t} > 0$$

$$E_m > 0 \; for \; t > 0$$

Since $E_m(0) > 0$, then $E_m(t) > 0$ for $t > 0$.

From the third equation of system of equations (4.5)

i.e $$\frac{dI_m}{dt} = \alpha_{E_m} E_m + \mu_m I_m$$

We have

$$\frac{dI_m}{dt} \geq -\mu_m I_m$$

By separation of variable, we have

$$\frac{dI_m}{I_m} \geq -\mu_m dt$$

Integrating both sides, we have

$$\int \frac{1}{I_m} dI_m \geq \int -\mu_m dt$$

$$InI_m \geq -\mu_m t + c$$

Taking exponential of both sides, we have

$$I_m \geq e^{-\mu_m t + c}$$

$$I_m \geq e^{-\mu_m} t.e^c$$

$$let \; C_3 = e^c$$

$$I_m(t) \geq C_3 e^{-\mu_m t}$$

Applying initial condition $t = 0$, we have

$$I_m(0) \geq C_3 e^0$$

$$\Rightarrow I_m(0) = C_3$$

Put $C_3 = I_m(0)$, we have

$$I_m(t) \geq I_m(0)e^{-\mu_m t} > 0$$

Since $I_m(0) > 0$, then $I_m(t) > 0, \quad for\ t > 0.$

From the fourth equation of system of equation (4.5)

i.e $\dfrac{dS_v}{dt} = \Lambda - (\lambda_v + \mu_v)S_v + \pi_m I_v$

We have

$$\frac{dS_v}{dt} \geq \Lambda - (\lambda_v + \mu_v)S_v + \pi_m I_v$$

By separation of variable, we have

$$\frac{dS_v}{S_v} \geq (\lambda_v + \mu_v)dt$$

Integrating both sides, we have

$$\int \frac{1}{S_v} dS_v \geq \int -(\lambda_v + \mu_v)dt$$

$$InS_v \geq \int -(\lambda_v + \mu_v)tdt + c$$

Taking exponential of both sides, we have

$$S_v \geq e^{\int -(\lambda_m + \mu_v)tdt}.e^c > 0$$

$$S_v(t) \geq C_4 e^{\int -(\lambda_m + \mu_v)tdt}$$

Applying initial condition $t = o$, we have

$$S_v(0) \geq C_4$$

$$\Rightarrow S_v(t) \geq S(0)e^{\int -(\lambda_m + \mu_v)tdt} > 0$$

Since $S_v(0) > 0$, then $S_v(t) > 0, \quad for\ t > 0.$

From the fifth equation of system of equations (4.1)

i.e $\dfrac{dI_v}{dt} = \lambda_v S_v - (\mu_v + \sigma + \pi_m)I_v$

We have

$$\frac{dI_v}{dt} \geq -(\mu_v + \sigma + \pi_m)I_v$$

By separation of variables, we have

$$\frac{dI_v}{I_v} \geq -(\mu_v + \sigma + \pi_m)dt$$

Integrating both sides, we have

$$\int \frac{1_v}{I_v} dI \geq \int -(\mu_v + \sigma + \pi_m)dt$$

$$InI_v \geq -(\mu_v + \sigma + \pi_m)t + c$$

Taking exponential of both side

$$I_v(t) \geq e^{-(\mu_v+\sigma+\pi_m)t+c}$$

$$I_v(t) \geq e^{-(\mu_v+\sigma+\pi_m)t} + e^{c}$$

Let $C_5 = e^{c}$

$$I_v(t) \geq C_5 e^{-(\mu_v+\sigma+\pi_m)t}$$

Apply initial condition $t = 0$ we have

$$I_v(0) \geq C_5 e^{0}$$

$$\Rightarrow I_v(0) \geq C_5$$

Let $C_5 = I_v(0)$, we have

$$I_v(t) \geq I_v(0) C_5 e^{-(\mu_v+\sigma+\pi_m)t} > 0$$

From the sixth equation of system of equation (4.5)

$$\text{ie} \quad \frac{dP_o}{dt} = \phi_{mo} \frac{I_m}{N_m} + \phi_{ov} \frac{I_v}{N_v} + (\theta - \mu_o) P_o$$

We have

$$\frac{dP_o}{dt} \geq -(\mu_o - \theta) P_o$$

By separation of variables, we have

$$\frac{dP_o}{P_o} \geq -(\mu_o - \theta)dt$$

Integrating both sides, we have

$$\int \frac{1}{P_o} dP_o \geq \int -(\mu_o - \theta)dt$$

$$InP_o \geq -(\mu_o - \theta)t + c$$

Taking exponential of both side, we have

$$P_o(t) \geq e^{-(\mu_o - \theta)t + c}$$

$$P_o(t) \geq e^{-(\mu_o - \theta)t}.e^{c}$$

Let $C_6 = e^{c}$ we have

$$P_o(t) \geq C_6 e^{-(\mu_o - \theta)t}$$

Applying initial condition t= 0, we have

$$P_o(0) \geq C_6 e^{0}$$

$$\Rightarrow P_o(0) = C_6$$

Put $P_o(0) = C_6$, we have

$$P_o(t) \geq P_o(0)e^{-(\mu_o - \theta)t} > 0$$

From the last equation of system equations (4.5)

i.e $$\frac{dC}{dt} = (\gamma + \lambda_c - \mu)C$$

We have

$$\frac{dC}{dt} \geq (\gamma + \lambda_c - \mu)C$$

By separation of variables, we have

$$\frac{dC}{C} \geq (\gamma + \lambda_c - \mu)dt$$

Integrating both sides, we have

$$\int \frac{1}{C} dC \geq \int (\gamma + \lambda_c - \mu)dt$$

$$InC \geq (\gamma + \lambda_c - \mu)t + c$$

Taking exponential of both sides, we have

$$C(t) \geq e^{(\gamma+\lambda_c-\mu)t+c}$$

$$C(t) \geq e^{(\gamma+\lambda_c-\mu)t}.e^{c}$$

Let $C_7 = e^{c}$,

$$C(t) \geq C_7 e^{(\gamma+\lambda_c-\mu)t}$$

Applying initial condition t = 0, we have

$$C(0) = C_7 e^{0}$$

$$\Rightarrow c_7 = C(0)$$

Put C_7, we have

$$C(t) \geq C(0)e^{(\gamma+\lambda_c-\mu)t} > 0$$

We have shown that all the variables are positive $for\ t > 0$, hence the proof.

4.1.4 Existence of Disease free equilibrium (DFE) point

In order to find the equilibrium points, we set the RHS of system of equations (3.7) to zero.

$$\text{i.e} \quad \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = \frac{dP_o}{dt} = \frac{dC}{dt} = 0 \tag{4.6}$$

$$-\left(\beta_o \frac{P_o^*}{k+P_o^*} + \beta_{vm}\frac{I_v^*}{N_v^*} + \beta_{mm}\frac{I_m^*}{N_m^*} + \mu_m\right)S_m^* = 0 \tag{4.7}$$

$$\left(\beta_o \frac{P_o^*}{k+P_o^*} + \beta_{vm}\frac{I_v^*}{N_v^*} + \beta_{mm}\frac{I_m^*}{N_m^*}\right)S_m^* - (\alpha_m+\mu_m)E_m^* = 0 \tag{4.8}$$

$$\alpha_m E_m^* - \mu_m I_m^* = 0 \tag{4.9}$$

$$\Lambda - \left(\beta_{ov}\frac{P_o^*}{k+P_o^*} + \beta_{mv}\frac{I_m^*}{N_m^*} + \mu_v + \mu_c C^*\right)S_v^* + \pi_m I_v^* = 0 \tag{4.10}$$

$$\left(\beta_{ov}\frac{P_o^*}{k+P_o^*} + \beta_{mv}\frac{I_m^*}{N_m^*}\right)S_v^* - (\mu_v + \mu_c C^* + \pi_m)I_v^* = 0 \tag{4.11}$$

$$\phi_{mo}\frac{I_m^*}{N_m^*} + \phi_{ov}\frac{I_v^*}{N_v^*} + (\theta - \mu_o)P_o^* = 0 \tag{4.12}$$

$$(\gamma C + \varepsilon_c (S_v + I_v) C - \mu C^* = 0 \tag{4.13}$$

At disease free equilibrium

$$E_m^* = I_m^* = I_v^* = P_o^* = C^* = 0 \tag{4.14}$$

Substituting equation (4.14) into equations (4.7) we have

$$-\mu_m S_m^* = 0$$

But

$$S_m^* = N_m^* - \mu_m S_m^*$$

$$S_m (1 + \mu_m) = N_m$$

$$S_m^* = \frac{N_m^*}{(1 + \mu_m)} \tag{4.15}$$

Similarly, putting equation (4.14) into equation (4.10) we have

$$\Lambda - \mu_v S_v^* = 0$$

$$S_v^* = \frac{\Lambda}{\mu_v} \tag{4.16}$$

Therefore, from equations (4.14), (4.15) and (4.16), the disease free equilibrium point is given by E_0.

$$\text{i.e} \quad E_0 = (S_m^*, E_m^*, I_m^*, S_v^*, I_v^*, P_o^*, C^*) = \left(\frac{N_m^*}{1 + \mu_m}, 0, 0, \frac{\Lambda}{\mu_v}, 0, 0, 0 \right) \tag{4.17}$$

4.1.5 Basic reproduction number of maize lethal necrosis disease

The basic reproduction number is the number of secondary infection produced as a result of contagious maize plants and a vector in total susceptible maize population. In order to obtain basic reproduction R_0, using next generating matrix, we use the approach by Diekman and Heesterbeek (2009).

Firstly, we arrange the system to get group of infection s classes only that is (E_m, I_m, I_v, P_o). We assume $f_i(x)$ be the rate of appearance of new infections (transmission) in compartment i, $V_i^+(x)$ be the transmission after new infections (transmission rate by all other means) and $V_i^-(x)$ be the rate of transfer of maize plants out of compartment i.

Consider the equation of the infected population in system of equations (4.1) given by

$$\left.\begin{aligned}
\frac{dE_m}{dt} &= (\beta_o \frac{P_o}{k+P_o} + \beta_{vm}\frac{I_v}{N_v} + \beta_{mm}\frac{I_m}{N_m})S_m - (\alpha_m + \mu_m)E_m \\
\frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m \\
\frac{dI_v}{dt} &= (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m})S_v - (\mu_v + \mu_c C + \pi_m)I_v \\
\frac{dP_o}{dt} &= \phi_{mo}\frac{I_m}{N_m} + \phi_{ov}\frac{I_v}{N_v} + (\theta - \mu_o)P_o
\end{aligned}\right\} \quad (4.18)$$

From equation (4.18) we have

$$f_i = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{bmatrix} = \begin{bmatrix} (\beta_o \frac{P_o}{k+P_o} + \beta_{vm}\frac{I_v}{N_v} + \beta_{mm}\frac{I_m}{N_m})S_m \\ 0 \\ (\beta_{ov}\frac{P_o}{k+P_o} + \beta_{mv}\frac{I_m}{N_m})S_v \\ 0 \end{bmatrix} \quad (4.19)$$

Similarly,

$$v_i = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{bmatrix} = \begin{bmatrix} (\alpha_m + \mu_m)E_m \\ -\alpha_m E_m - \mu_m I_m \\ (\mu_v + \mu_c C + \pi_m)I_v \\ -\phi_{mo}\frac{I_m}{N_m} - \phi_{ov}\frac{I_v}{N_v} - (\theta - \mu_o)P_o \end{bmatrix} \quad (4.20)$$

$$\frac{\partial f_i}{\partial x_i} = \begin{bmatrix} \frac{\partial F_1}{\partial E_m} & \frac{\partial F_1}{\partial I_m} & \frac{\partial F_1}{\partial I_v} & \frac{\partial F_1}{\partial P_o} \\ \frac{\partial F_2}{\partial E_m} & \frac{\partial F_2}{\partial I_m} & \frac{\partial F_2}{\partial I_v} & \frac{\partial F_2}{\partial P_o} \\ \frac{\partial F_3}{\partial E_m} & \frac{\partial F_3}{\partial \mathrm{I}_m} & \frac{\partial F_3}{\partial I_v} & \frac{\partial F_3}{\partial P_o} \\ \frac{\partial F_4}{\partial E_m} & \frac{\partial F_4}{\partial I_m} & \frac{\partial F_4}{\partial I_v} & \frac{\partial F_4}{\partial P_o} \end{bmatrix} \tag{4.21}$$

Substituting equation (4.19) into equation (4.22) we have

$$F = \begin{bmatrix} 0 & \beta_{mm}\frac{S_m}{N_m} & \beta_{vm}\frac{S_m}{N_v} & \beta_o\frac{kS_m}{(k+P_o)^2} \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{mv}\frac{S_v}{N_m} & 0 & \beta_{ov}\frac{kS_v}{(k+P_o)^2} \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{4.22}$$

Therefore, by evaluating F at disease free equilibrium, we have

$$F = \frac{\partial f_i(E_0)}{\partial x_i} = \begin{bmatrix} 0 & \frac{\beta_{mm}}{(1+\mu_m)} & \frac{\beta_{vm}N_m^*}{N_v(1+\mu_m)} & \frac{\beta_o N_m^*}{k(1+\mu_m)} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{mv}\Lambda}{\mu_v N_m^*} & 0 & \frac{\beta_{ovv}}{k\mu_v} \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{4.23}$$

Similarly,

$$\frac{\partial v_i}{\partial x_i} = \begin{bmatrix} \frac{\partial V_1}{\partial E_m} & \frac{\partial V_1}{\partial I_m} & \frac{\partial V_1}{\partial I_v} & \frac{\partial V_1}{\partial P_o} \\ \frac{\partial V_2}{\partial E_m} & \frac{\partial V_2}{\partial I_m} & \frac{\partial V_2}{\partial I_v} & \frac{\partial V_2}{\partial P_o} \\ \frac{\partial V_3}{\partial E_m} & \frac{\partial V_3}{\partial \mathrm{I}_m} & \frac{\partial V_3}{\partial I_v} & \frac{\partial V_3}{\partial P_o} \\ \frac{\partial V_4}{\partial E_m} & \frac{\partial V_4}{\partial I_m} & \frac{\partial V_4}{\partial I_v} & \frac{\partial V_4}{\partial P_o} \end{bmatrix} \tag{4.24}$$

Substituting equation (4.20) into equation (4.25) we have

$$V = \begin{bmatrix} (\alpha_m + \mu_m) & 0 & 0 & 0 \\ -\alpha_m & \mu_m & 0 & 0 \\ 0 & 0 & (\mu_v + \pi_m) & 0 \\ 0 & \frac{-\phi_{mo}}{N_m^*} & \frac{-\phi_{ov}}{N_v^*} & (\theta - \mu_o) \end{bmatrix} \quad (4.25)$$

From equation (4.26), let $a_1 = (\alpha_m + \mu_m)$, $a_2 = \mu_m$, $a_3 = (\mu_v + \pi_m)$ and $a_4 = (\theta - \mu_o)$. Thus

$$V = \begin{bmatrix} a_1 & 0 & 0 & 0 \\ -\alpha_m & a_2 & 0 & 0 \\ 0 & 0 & a_3 & 0 \\ 0 & \frac{-\phi_{mo}}{N_m^*} & \frac{-\phi_{ov}}{N_m^*} & a_4 \end{bmatrix} \quad (4.26)$$

Now, from equation (4.27), we find the determinant of V $(\det V)$, as

$$\det V = \begin{bmatrix} a_1 & 0 & 0 & 0 \\ -\alpha_m & a_2 & 0 & 0 \\ 0 & 0 & a_3 & 0 \\ 0 & \frac{-\phi_{mo}}{N_m^*} & \frac{-\phi_{ov}}{N_v^*} & a_4 \end{bmatrix}, \Rightarrow \det V = a_1 a_2 a_3 a_4$$

Then, Co-factor of V

$$co-factor\ of\ V = \begin{bmatrix} a_2 a_3 a_4 & -\alpha a_3 a_4 & 0 & \frac{-\phi_{mo}\alpha_m a_3}{N_m^*} \\ 0 & a_1 a_2 a_4 & 0 & \frac{\phi_{mo} a_1 a_2}{N_m^*} \\ 0 & 0 & a_1 a_2 a_4 & \frac{-\phi_{ov} a_1 a_2}{N_v^*} \\ 0 & 0 & 0 & a_1 a_2 a_3 \end{bmatrix}$$

$$Adjoint\ of\ V = \begin{bmatrix} a_2 a_3 a_4 & 0 & 0 & 0 \\ \alpha a_3 a_4 & a_1 a_2 a_4 & 0 & 0 \\ 0 & 0 & a_1 a_2 a_4 & 0 \\ \frac{\phi_{mo}\alpha_m a_3}{N_m^*} & \frac{\phi_{mo} a_1 a_2}{N_m^*} & \frac{\phi_{ov} a_1 a_2}{N_v^*} & a_1 a_2 a_3 \end{bmatrix}$$

$$\frac{1}{a_1 a_2 a_3 a_4}\begin{bmatrix} a_2 a_3 a_4 & 0 & 0 & 0 \\ \alpha_m a_3 a_4 & a_1 a_3 a_4 & 0 & 0 \\ 0 & 0 & a_{11} a_{22} a_{44} & 0 \\ \dfrac{a_3 \lambda_{mv} \alpha_m}{N_m^*} & \dfrac{a_1 a_3 \phi_{mo}}{N_m^*} & \dfrac{a_1 a_2 \phi_{ov}}{N_m^*} & a_1 a_2 a_3 \end{bmatrix}$$

$$\text{Thus, } V^{-1} = \begin{bmatrix} \dfrac{1}{a_1} & 0 & 0 & 0 \\ \dfrac{\alpha_m}{a_1 a_2} & \dfrac{1}{a_2} & 0 & 0 \\ 0 & 0 & \dfrac{1}{a_3} & 0 \\ \dfrac{\phi_{mo}\alpha_m}{a_1 a_2 a_4 N_m^*} & \dfrac{\phi_{mo}}{a_2 a_4 N_m^*} & \dfrac{\phi_{ov}}{a_3 a_4 N_v^*} & \dfrac{1}{a_4} \end{bmatrix} \qquad (4.27)$$

Then,

$$FV^{-1} = \begin{bmatrix} 0 & \dfrac{\beta_{mm}}{(1+\mu_m)} & \dfrac{\beta_{vm} N_m^*}{N_v^*(1+\mu_m)} & \dfrac{\beta_o N_m^*}{k(1+\mu_m)} \\ 0 & 0 & 0 & 0 \\ 0 & \dfrac{\beta_{mv}\Lambda}{\mu_v N_m^*} & 0 & \dfrac{\beta_{ovv}}{k\mu_v} \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \dfrac{1}{a_1} & 0 & 0 & 0 \\ \dfrac{\alpha_m}{a_1 a_2} & \dfrac{1}{a_2} & 0 & 0 \\ 0 & 0 & \dfrac{1}{a_3} & 0 \\ \dfrac{\phi_{mo}\alpha_m}{a_1 a_2 a_4 N_m^*} & \dfrac{1}{a_2 a_4 N_m^*} & \dfrac{\phi_{ov}}{a_3 a_4 N_v^*} & \dfrac{1}{a_4} \end{bmatrix} \qquad (4.28)$$

$$\text{Then, } FV^{-1} = \begin{bmatrix} \dfrac{\beta_{mm}}{a_1 a_2(1+\mu_m)} + \dfrac{\beta_o \phi_{mo} \alpha_m}{k(1+\mu_m) a_1 a_2} & \dfrac{\beta_{mm}}{a_2(1+\mu_m)} + \dfrac{\beta_o N_m^*}{k(1+\mu_m) a_2 a_4 N_v^*} & \dfrac{\beta_{vm} N_m^*}{N_v^*(1+\mu_m) a_3} + \dfrac{\beta_o N_m^* \phi_{ov}}{k(1+\mu_m) a_3 a_4 N_v^*} & \dfrac{\beta_o N_m^*}{k(1+\mu_m) a_4} \\ 0 & 0 & 0 & 0 \\ \dfrac{\beta_{mv}\Lambda}{\mu_v N_m^* a_1 a_2} + \dfrac{\beta_{ov}\phi_m \alpha_m}{k\mu_v a_1 a_2 a_4 N_m^*} & \dfrac{\beta_{mv}\Lambda}{\mu_v N_m^* a_2} + \dfrac{\beta_{ov}}{k\mu_v a_2 a_4 N_m^*} & \dfrac{\beta_{ov}\phi_{ov}}{k\mu_v a_3 a_4 N_v^*} & \dfrac{\beta_{ovv}}{k\mu_v a_4} \\ 0 & 0 & 0 & 0 \end{bmatrix} \qquad (4.29)$$

Equation (4.30) becomes

$$FV^{-1} = \begin{bmatrix} A & B & C & D \\ 0 & 0 & 0 & 0 \\ E & F & G & H \\ 0 & 0 & 0 & 0 \end{bmatrix} \qquad (4.30)$$

Where $A = \frac{\beta_{mm}}{a_1 a_2 (1+\mu_m)} + \frac{\beta_o \phi_{mo} \alpha_m}{k(1+\mu_m) a_1 a_2 a_4}$, $B = \frac{\beta_{mm}}{a_2(1+\mu_m)} + \frac{\beta_o N_m^*}{k(1+\mu_m) a_2 a_4 N_v^*}$,

$C = \frac{\beta_{vm} N_m^*}{N_v^*(1+\mu_m) a_3} + \frac{\beta_o N_m^* \phi_{ov}}{k(1+\mu_m) a_3 a_4 N_v^*}$, $D = \frac{\beta_o N_m^*}{k(1+\mu_m) a_4}$,

$E = \frac{\beta_{mv} \Lambda}{\mu_v N_m^* a_1 a_2} + \frac{\beta_{ov} \phi_{mo} \alpha_m}{k \mu_v a_1 a_2 a_4 N_m^*}$, $F = \frac{\beta_{mv} \Lambda}{\mu_v N_m^* a_2} + \frac{\beta_{ov}}{k \mu_v a_2 a_4 N_m^*}$,

$G = \frac{\beta_{ov} \phi_{ov}}{k \mu_v a_3 a_4 N_m^*}$ and $H = \frac{\beta_{ovv}}{k \mu_v a_4}$.

$$R_0 = \rho\left(FV^{-1}\right) \tag{4.31}$$

Therefore, we evaluate the characteristics equation $\left|FV^{-1} - \lambda I\right| = 0$ of equation (4.31) to get

$$\begin{vmatrix} A-\lambda & B & C & D \\ 0 & 0-\lambda & 0 & 0 \\ E & F & G-\lambda & H \\ 0 & 0 & 0 & 0-\lambda \end{vmatrix} = 0 \tag{4.32}$$

Solve equation (4.33) for λ_i, i=1,2,3,4 , we have

$$-\lambda \begin{vmatrix} A-\lambda & B & B \\ 0 & -\lambda & 0 \\ E & F & G-\lambda \end{vmatrix} = 0$$

$$\lambda^2 \begin{vmatrix} A-\lambda & C \\ E & G-\lambda \end{vmatrix} = 0$$

$$\lambda_1 = \lambda_2 = 0$$

$$\begin{vmatrix} A-\lambda & C \\ E & G-\lambda \end{vmatrix} = 0$$

$$(A-\lambda)(G-\lambda) - CE = 0$$

$$AG - A\lambda - G\lambda + \lambda^2 - CE = 0$$

$$\lambda^2 - (A+G)\lambda + A - CE = 0$$

$$\lambda_{3,4} = \frac{A+G}{2} \pm \frac{1}{2}\sqrt{A^2 + G^2 + 2AG - 4AG + 4CE}$$

$$= \frac{A+G}{2} \pm \frac{1}{2}\sqrt{A^2 + G^2 - 2AG + 4CE}$$

$$= \frac{A+G}{2} \pm \frac{1}{2}\sqrt{A^2 + G^2 + 4CE - 2AG}$$

$$\lambda_3 = \frac{A+G}{2} - \frac{1}{2}\sqrt{A^2 + G^2 + 4CE - 2AG}$$

$$\lambda_4 = \frac{A+G}{2} + \frac{1}{2}\sqrt{A^2 + G^2 + 4CE - 2AG}$$

$$R_0 = \max\{\lambda_3, \lambda_4\}, hence\ R_0 = \lambda_4$$

$$R_0 = \frac{1}{2}\left[\frac{\beta_{mm}}{(\alpha_m + \mu_m)(1+\mu_m)\mu_m} + \frac{\beta_o \phi_{mo}\alpha_m}{k(1+\mu_m)\mu_m(\alpha_m+\mu_m)}\right] + \left[\frac{\beta_{ov}\phi_{ov}}{k\mu_v(\mu_v+\pi_m)(\theta-\mu_o)N_m^*}\right] +$$

$$\sqrt{\left[\frac{\beta_{mm}}{(\alpha_m + \mu_m)(1+\mu_m)\mu_m} + \frac{\beta_o \phi_{mo}\alpha_m}{k(1+\mu_m)\mu_m(\alpha_m+\mu_m)}\right]^2 + \left[\frac{\beta_{ov}\phi_{ov}}{k\mu_v(\mu_v+\pi_m)(\theta-\mu_o)N_m^*}\right]^2 +}$$

$$\frac{1}{2}\sqrt{4\left[\left(\frac{\beta_{vm}N_m^*}{N_v^*(1+\mu_m)a_3} + \frac{\beta_o N_m^* \phi_{ov}}{k(1+\mu_m)(\mu_v+\pi_m)(\theta-\mu_o)N_v^*}\right)\left(\frac{\beta_{mv}\Lambda}{\mu_v\mu_m N_m^*} + \frac{\beta_{ov}}{k\mu_v\mu_m(\theta-\mu_o)N_m^*}\right)\right] -}$$

$$2\left[\left(\frac{\beta_{mm}}{(\alpha_m+\mu_m)(1+\mu_m)\mu_m} + \frac{\beta_o\phi_{mo}\alpha_m}{k(1+\mu_m)\mu_m(\alpha_m+\mu_m)}\right)\left(\frac{\beta_{ov}\phi_{ov}}{k\mu_v(\mu_v+\pi_m)(\theta-\mu_o)N_m^*}\right)\right] < 1.$$

For R_0 to exist $k\mu_v(\mu_v+\pi_m)(\theta-\mu_o)N_m^* \neq 0$.

4.1.6 Local stability of the disease fee equilibrium

The local stability of the disease free equilibrium points can be established by showing that all the eigen values of the Jacobean matrix of linearized system evaluated at disease free equilibriumE_0 are negative and is based on the following theorem.

Theorem: 4.1.6

The disease free equilibrium of the system of equations (3.7) is locally asymptotically stable in Ω if $R_0 < 1$ and unstably, if $R_0 > 1$, where R_0 is the reproduction number.

Proof:

Thus, the Jacobean matrix J for the system of equation (3.7) is given by

$$J = \begin{bmatrix} \frac{\partial F_1}{\partial S_m} & \frac{\partial F_1}{\partial E_m} & \frac{\partial F_1}{\partial I_m} & \frac{\partial F_1}{\partial S_v} & \frac{\partial F_1}{\partial I_v} & \frac{\partial F_1}{\partial P_o} & \frac{\partial F_1}{\partial C} \\ \frac{\partial F_2}{\partial S_m} & \frac{\partial F_2}{\partial E_m} & \frac{\partial F_2}{\partial I_m} & \frac{\partial F_2}{\partial S_v} & \frac{\partial F_2}{\partial I_v} & \frac{\partial F_2}{\partial P_o} & \frac{\partial F_2}{\partial C} \\ \frac{\partial F_3}{\partial S_m} & \frac{\partial F_3}{\partial E_m} & \frac{\partial F_3}{\partial I_m} & \frac{\partial F_3}{\partial S_v} & \frac{\partial F_3}{\partial I_v} & \frac{\partial F_3}{\partial P_o} & \frac{\partial F_3}{\partial C} \\ \frac{\partial F_4}{\partial S_m} & \frac{\partial F_4}{\partial E_m} & \frac{\partial F_4}{\partial I_m} & \frac{\partial F_4}{\partial S_v} & \frac{\partial F_4}{\partial I_v} & \frac{\partial F_4}{\partial P_o} & \frac{\partial F_4}{\partial C} \\ \frac{\partial F_5}{\partial S_m} & \frac{\partial F_5}{\partial E_m} & \frac{\partial F_5}{\partial I_m} & \frac{\partial F_5}{\partial S_v} & \frac{\partial F_5}{\partial I_v} & \frac{\partial F_5}{\partial P_o} & \frac{\partial F_5}{\partial C} \\ \frac{\partial F_6}{\partial S_m} & \frac{\partial F_6}{\partial E_m} & \frac{\partial F_6}{\partial I_m} & \frac{\partial F_6}{\partial S_v} & \frac{\partial F_6}{\partial I_v} & \frac{\partial F_6}{\partial P_o} & \frac{\partial F_6}{\partial C} \\ \frac{\partial F_7}{\partial S_m} & \frac{\partial F_7}{\partial E_m} & \frac{\partial F_7}{\partial I_m} & \frac{\partial F_7}{\partial S_v} & \frac{\partial F_7}{\partial I_v} & \frac{\partial F_7}{\partial P_o} & \frac{\partial F_7}{\partial C} \end{bmatrix} \tag{4.33}$$

Evaluating the system of equations (3.7) into equation (4.33) we have

$$J = \begin{bmatrix} -(\beta_o \frac{P_o}{k+P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m} + \mu_m) & 0 & 0 & 0 & 0 & 0 & 0 \\ (\beta_o \frac{P_o}{k+P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}) & -(\alpha_m + \mu_m) & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_m & -\mu_m & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m} + \mu_v + \mu_c C) & \pi_m & 0 & 0 \\ 0 & 0 & 0 & (\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m}) & -(\mu_v + \mu_c C + \pi_m) & 0 & 0 \\ 0 & 0 & \frac{\lambda_{mo}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_o - \theta) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu - \gamma - \frac{\Lambda}{\mu_v}) \end{bmatrix} \tag{4.34}$$

Evaluating equation (4.34) at disease free equilibrium, we have

$$J = \begin{bmatrix} -\mu_m & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\alpha_m + \mu_m) & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_m & -\mu_m & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu_v & \pi_m & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\mu_v + \pi_m) & 0 & 0 \\ 0 & 0 & \frac{\lambda_{mo}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_o - \theta) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu - \gamma - \frac{\Lambda}{\mu_v}) \end{bmatrix} \tag{4.35}$$

Given that $\left| J(E_o) - \lambda I \right| = 0$ (4.36)

Substituting equation (4.35) into equation (4.36) we have

$$\left|J(E_o)-\lambda I\right|=\begin{vmatrix} -\mu_m-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\alpha_m+\mu_m)-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_m & -\mu_m-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu_v-\lambda & \pi_m & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\mu_v+\pi_m)-\lambda & 0 & 0 \\ 0 & 0 & \frac{\lambda_{mo}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_o-\theta)-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu-\gamma-\frac{\Lambda}{\mu_v})-\lambda \end{vmatrix}=0 \quad (4.37)$$

Solve equation (4.37) for $\lambda_i, i=1,2,3,4,5,6,7$, we have

$$(-\mu_m-\lambda)\begin{vmatrix} -(\alpha_m+\mu_m)-\lambda & 0 & 0 & 0 & 0 & 0 \\ \alpha_m & -\mu_m & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_v-\lambda & \pi_m & 0 & 0 \\ 0 & 0 & 0 & -(\mu_v+\pi_m)-\lambda & 0 & 0 \\ 0 & \frac{\lambda_{mv}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_O-\theta)-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu-\gamma-\frac{\Lambda}{\mu_v})-\lambda \end{vmatrix}=0$$

$$\Rightarrow \lambda_1=-\mu_m$$

$$-(\mu-\gamma-\frac{\Lambda}{\mu_v})-\lambda\begin{vmatrix} -(\alpha_m+\mu_m)-\lambda & 0 & 0 & 0 & 0 \\ \alpha_m & -\mu_m-\lambda & 0 & 0 & 0 \\ 0 & 0 & -\mu_v-\lambda & 0 & 0 \\ 0 & 0 & 0 & -(\mu_v+\pi_m)-\lambda & 0 \\ 0 & \frac{\lambda_{mv}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_o-\theta)-\lambda \end{vmatrix}=0$$

$$\Rightarrow \lambda_2=-(\mu-\gamma-\frac{\Lambda}{\mu_v})$$

$$-(\alpha_m+\mu_m)-\lambda\begin{vmatrix} -\mu_m-\lambda & 0 & 0 & 0 \\ 0 & -\mu_v-\lambda & 0 & 0 \\ 0 & 0 & -(\mu_v+\pi_m)-\lambda & 0 \\ \frac{\lambda_{mv}}{N_m} & 0 & \frac{\lambda_{ov}}{N_v} & -(\mu_o-\theta)-\lambda \end{vmatrix}=0$$

$$\Rightarrow \lambda_3=-(\alpha_m+\mu_m)$$

$$-\mu_m - \lambda \begin{vmatrix} -\mu_v - \lambda & \pi_m & 0 \\ 0 & -(\mu_v + \pi_m) - \lambda & 0 \\ 0 & \frac{\lambda_{mv}}{N_m} & -(\mu_o - \theta) - \lambda \end{vmatrix} = 0$$

$$\Rightarrow \lambda_4 = -\mu_m$$

$$-\mu_v - \lambda \begin{vmatrix} -(\mu_v + \pi_m) - \lambda & 0 \\ \frac{\lambda_{mv}}{N_m} & -(\mu_o - \theta) - \lambda \end{vmatrix} = 0$$

$$\Rightarrow \lambda_5 = -\mu_v$$

$$(-\mu_v - \pi_m - \lambda)(-\mu_o + \theta - \lambda) = 0$$
$$\Rightarrow \lambda_6 = -(\mu_o + \theta)$$

And

$$\lambda_7 = -(\mu_v + \pi_m)$$

Clearly, $\lambda_i < 0 \ \forall i = 1, 2, 3, 4, 5, 6, 7.$ Since the all the eigen values of the polynomial have negative real parts, we conclude that the disease free equilibrium (DFE) is locally asymptotically stable..

4.1.7 Global stability of the disease free equilibrium

To establish the global stability of disease free equilibrium, the two conditions (H_1 and H_2) as in Castillo-Chavez, Frerg and Huang (2002) must be satisfied for $R_0 < 1.$ we rewrite the system of equations (3.7) in the form

$$H_1: \ \frac{dX}{dt} F(X, Z)$$
$$H_2: \ \frac{dZ}{dt} G(X, Z); G(X, 0) = 0$$

Where $X = (S_m, S_v, C) \in R^3$ denoting the uninfected population and the component $Z \in R^4$ denoting the infected population. Thus, the disease free equilibrium is now denoted as $E^o = (X, 0), X = \left(\frac{N^*_m}{(1+\mu_m)}, \frac{\Lambda}{\mu_v}, 0 \right)$.

Now, for the first (H_1) condition, that s globally asymptotically stable of X, we have

$$\frac{dX}{dt} = F(X, 0) = \begin{bmatrix} N^*_m - (1+\mu_m) S^*_m \\ \Lambda - \mu_v S^*_m \\ 0 \end{bmatrix} \qquad (4.38)$$

From the first equation of the system of equations (3,7) we have

$$\frac{dS_m}{dt} = N^*_m - (1+\mu_m) S^*_m$$

$$\frac{dS_m}{dt} + (1+\mu_m) S^*_m = N^*_m$$

By using integrating factor

$$\frac{dS_m}{dt} + (1+\mu_m) S^*_m = N^*_m$$

$$\frac{dS_m}{dt} = N^*_m - (1+\mu_m) S^*_m$$

$$S_m IF = N_m \int IF\, dt$$

$$\therefore IF = e^{\int (1+\mu_m) dt} = e^{(1+\mu_m)t}$$

$$S^*_m e^{(1+\mu_m)t} = N^*_m \int e^{(1+\mu_m)t} dt$$

$$S^*_m e^{(1+\mu_m)t} = N^*_m \frac{e^{(1+\mu_m)t}}{(1+\mu_m)} + C$$

$$S^*_m(t) = \frac{N^*_m}{(1+\mu_m)} + C e^{-(1+\mu_m)t}$$

$$at\ t = o$$

$$S^*_m(0) = \frac{N^*_m}{(1+\mu_m)} + C$$

$$\Rightarrow C = S^*_m(0) - \frac{N^*_m}{(1+\mu_m)}$$

Substituting the value of C, we have

$$S^*_m(t) = \frac{N^*_m}{(1+\mu_m)} + \left(S^*_m(0) - \frac{N^*_m}{(1+\mu_m)} \right) e^{-(1+\mu_m)t}$$

$$as\ t \to \infty, S^*_m(t) \to \frac{N^*_m}{(1+\mu_m)}$$

From the fourth equation of the system of equations (3.7), we have

$$\frac{dS_v}{dt} = \Lambda - \mu_v S^*_v$$

$$\frac{dS_v}{dt} + \mu_v S^*_v = \Lambda$$

`By using integrating factor

$$S_v IF = \Lambda \int IF\, dt$$

$$IF = e^{\int \mu_v dt} = e^{\mu_v t}$$

$$S^*_v e^{\mu_v t} = \Lambda \int e^{\mu_v t dt}$$

$$S_v(t) = \frac{\Lambda}{\mu_v} + Ce^{\mu_v}$$

$$at\ t \to \infty \Rightarrow S^*_v(0) = \frac{\Lambda}{\mu_v} + C$$

$$\therefore C = S^*_v(0) - \frac{\Lambda}{\mu_v}$$

Substituting the value of C, we have

$$S^*_v(t) = \frac{\Lambda}{\mu_v} + \left(S^*_v(0) \frac{\Lambda}{\mu_v} \right) e^{\mu_v(0)}$$

$$as\ t \to \infty, S^*_v(t) \to \frac{\Lambda}{\mu_v}$$

$X^* = \left(\frac{N^*_m}{(1+\mu_m)}, \frac{\Lambda}{\mu_v}, 0 \right)$ is globally asymptotically stable.

Next or the second (H_2) condition

$$G(X.Z) = PZ - G(X,Z)$$

$$F(X,Z) = \begin{bmatrix} -(\beta_o \frac{P_o}{k+P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m} + \mu_m) S_m \\ \Lambda - (\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m} + \mu_v + \mu_c C) S_v + \pi_m I_v \\ \gamma C + \varepsilon_c (S_v + I_v) C - \mu C \end{bmatrix} \quad (4.39)$$

$$G(X,Z) = \begin{bmatrix} (\beta_o \frac{P_o}{k+P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}) S_m - (\alpha_m + \mu_m) E_m \\ \alpha_m E_m - \mu_m I_m \\ (\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m}) S_v - (\mu_v + \mu_c C + \pi_m) I_v \\ \phi_{mo} \frac{I_m}{N_m} + \phi_{ov} \frac{I_v}{N_v} + (\theta - \mu_o) P_o \end{bmatrix} \quad (4.40)$$

$$P = \begin{bmatrix} -(\alpha_m + \mu_m) & \beta_{mm} \frac{S_m}{N_m} & \beta_{vm} \frac{S_m}{N_v} & \beta_o \frac{k S_m}{(k+P_o)^2} \\ \alpha_m & -\mu_m & 0 & 0 \\ 0 & \beta_{mv} \frac{S_v}{N_m} & -(\mu_v + \mu_c C + \pi_m) & \beta_{ov} \frac{k S_v}{(k+P_o)^2} \\ 0 & \frac{\phi_{mo}}{N_m} & \frac{\phi_{ov}}{N_v} & -(\mu_o - \theta) \end{bmatrix} \quad (4.41)$$

Substituting equation (4.17) into equation (4.41) we have

$$P = \begin{bmatrix} -(\alpha_m + \mu_m) & \frac{\beta_{mm}}{(1+\mu_m)} & \frac{\beta_{vm} N^*_m}{N_v (1+\mu_m)} & \frac{\beta_o N^*_m}{k(1+\mu_m)} \\ \alpha_m & -\mu_m & 0 & 0 \\ 0 & \frac{\beta_{mv} \Lambda}{\mu_v N^*_m} & -(\mu_v + \pi_m) & \frac{\beta_{ovv} \Lambda}{k \mu_v} \\ 0 & \frac{\phi_{mo}}{N^*_m} & \frac{\phi_{ov}}{N^*_v} & -(\mu_o - \theta) \end{bmatrix}$$

$$Z = \begin{bmatrix} E_m \\ I_m \\ I_v \\ P_o \end{bmatrix}$$

$$PZ = \begin{bmatrix} -(\alpha_m + \mu_m) & \frac{\beta_{mm}}{(1+\mu_m)} & \frac{\beta_{vm} N^*_m}{N_v(1+\mu_m)} & \frac{\beta_o N^*_m}{k(1+\mu_m)} \\ \alpha_m & -\mu_m & 0 & 0 \\ 0 & \frac{\beta_{mv}\Lambda}{\mu_v N^*_m} & -(\mu_v + \pi_m) & \frac{\beta_{ovv}\Lambda}{k\mu_v} \\ 0 & \frac{\phi_{mo}}{N^*_m} & \frac{\phi_{ov}}{N^*_v} & -(\mu_o - \theta) \end{bmatrix} \begin{bmatrix} E_m \\ I_m \\ I_v \\ P_o \end{bmatrix} \quad (4.42)$$

$$\therefore \dot{G}(X, Z) = PZ - G(X, Z) \quad (4.44)$$

$$= \begin{bmatrix} -(\alpha_m + \mu_m)E_m + \frac{\beta_{mm} I_m}{(1+\mu_m)} + \frac{\beta_{vm} N_m I_v}{N_v(1+\mu_m)} + \frac{\beta_o N_m P_o}{(k+P_o)(1+\mu_m)} \\ \alpha_m E_m - \mu_m I_m \\ \frac{\beta_{mv}\Lambda I_m}{\mu_v N_m} - (\mu_v + \pi_m) I_v + \frac{\beta_{ovv}\Lambda P_o}{(k+P_o)\mu_v} \\ \frac{\phi_{mo} I_m}{N_m} + \frac{\phi_{ov} I_v}{N_v} - (\mu_o - \theta)P \end{bmatrix} - \begin{bmatrix} \frac{\beta_o N_m P_o}{(k+P_o)(1+\mu_m)} + \frac{\beta_{vm} N_m I_v}{N_v(1+\mu_m)} + \frac{\beta_{mm} I_m}{(1+\mu_m)} - (\alpha_m + \mu_m)E_m \\ \alpha_m E_m - \mu_m I_m \\ \frac{\beta_{ovv}\Lambda P_o}{(k+P_o)\mu_v} + \frac{\beta_{mv}\Lambda I_m}{\mu_v N_m} - (\mu_v + \pi_m) I_v \\ \frac{\phi_{mo} I_m}{N_m} + \frac{\phi_{ov} I_v}{N_v} - (\mu_o - \theta)P \end{bmatrix}$$

$$\therefore \dot{G}(X, Z) = PZ - G(X, Z) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$\therefore \dot{G}(X, Z) = PZ - G(X, Z) = [0, 0, 0, 0]^T$$

Hence the proof is complete.

But it is clear that $PZ - G(X, Z)$, then, $G(X, Z)$. This implies that, $G(X, Z) = 0$, hence the disease free equilibrium of the system is globally asymptotically stable.

4.1.8 Model simulation

We present the numerical simulation for the modified model and discussion of the simulation results using estimated parameters and adopt some from existing model and other related models.

We performed some numerical experiments using ode45 function from MATHLABR2015a to study the behaviuor of the system on the MLND under the deployment of natural enemy. The parameters values used in the simulation of this model are presented in table 4.1.

Table 4.1: Estimated and adopted parameter values and variables used in the simulation of the model.

Parameters/Variable	**Values**	**Source**
β_{mv}	0.0024	Williams, Dmitry and Livingstone (2018)
μ_0	0,0005	Williams, Dmitry and Livingstone (2018)
β_0	0.06	Williams, Dmitry and Livingstone (2018)
β_{0v}	0.06	Williams, Dmitry and Livingstone (2018)
β_{vm}	0.024	Williams, Dmitry and Livingstone (2018)
β_{mm}	0.28687	Nyamwamu, Mwangi, and Miano (2015)
ϕ_{0v}	0.07486	Nyamwamu, Mwangi, and Miano (2015)
ϕ_{mo}	0.010	Williams, Dmitry and Livingstone (2018)
$\wedge$	0.95	Williams, Dmitry and Livingstone (2018)
μ_v	0.05	Williams, Dmitry and Livingstone (2018)
θ	0.0018	Nyamwamu, Mwangi, and Miano (2015)
α_m	0.025	Nyamwamu, Mwangi, and Miano (2015)
μ_m	0.02.	Williams, Dmitry and Livingstone (2018)
π_m	0.03.	Nyamwamu, Mwangi, and Miano (2015)
μ_c	0.45	Assumed
μ	0.002	Assumed
γ	0.45	Assumed
ε_c	0.14	Assumed
S_m	30000	Williams, Dmitry and Livingstone (2018)
E_m	9000	Williams, Dmitry and Livingstone (2018)
I_m	5000	Williams, Dmitry and Livingstone (2018)
S_v	2000	Williams, Dmitry and Livingstone (2018)
I_v	800	Williams, Dmitry and Livingstone (2018)
P_0	20	Williams, Dmitry and Livingstone (2018)
C	500	Assumed

Table 4.1.

The results of the system of equations (3.7) with parameter values in table 4.1 are presented in the following figures

Experiment 1: We investigated the effects of vector population (thrips) on the maize plant, in absence of predators.

When the contact rate between the infected maize and susceptible maize and contact rate between infected vector and susceptible maize is low, the slower the spread of MLND. But

if the contact rate is high, MLND will spread within short period of time as illustrated in figure 4.1- 4.3.

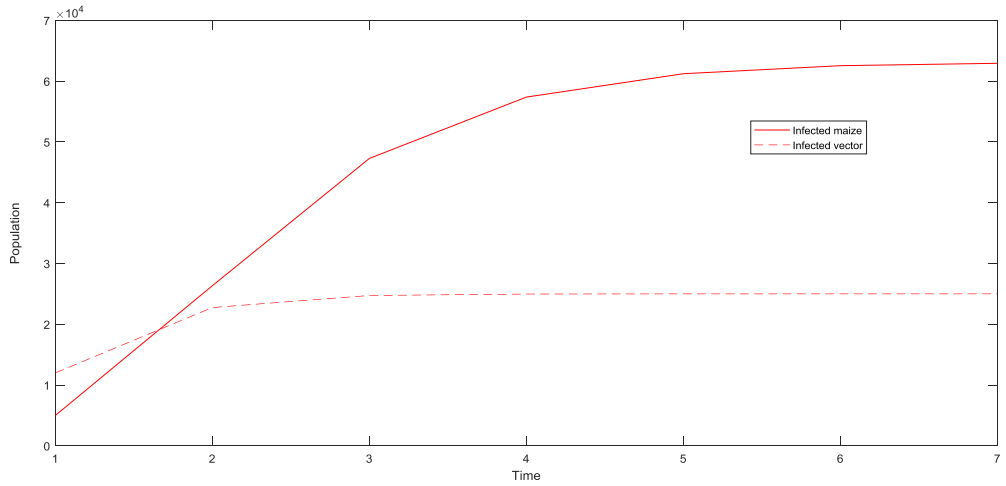

Figure 4.1: Show the effects of infected vector on the susceptible maize plant in the absence of predators. (β_{mv}) = 0.064, (β_{ov}) = 0.002, (μ_v) = 0.05, (π_m) = 0.003.

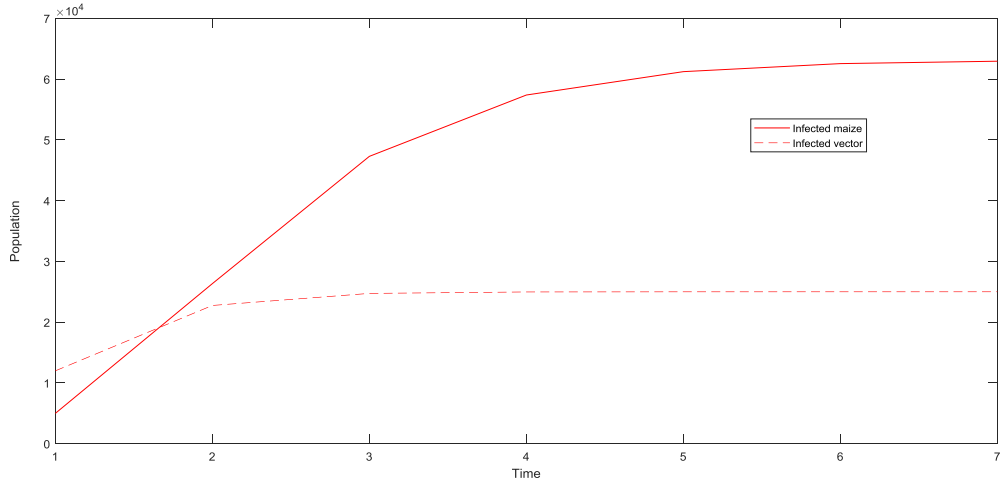

Figure 4.2: Illustrates the effects of infected vector on the susceptible maize plant in the absence of predators. (β_{mv}) = 0.084, (β_{ov}) = 0.03, (μ_v) = 0.03, (π_m) = 0.0003.

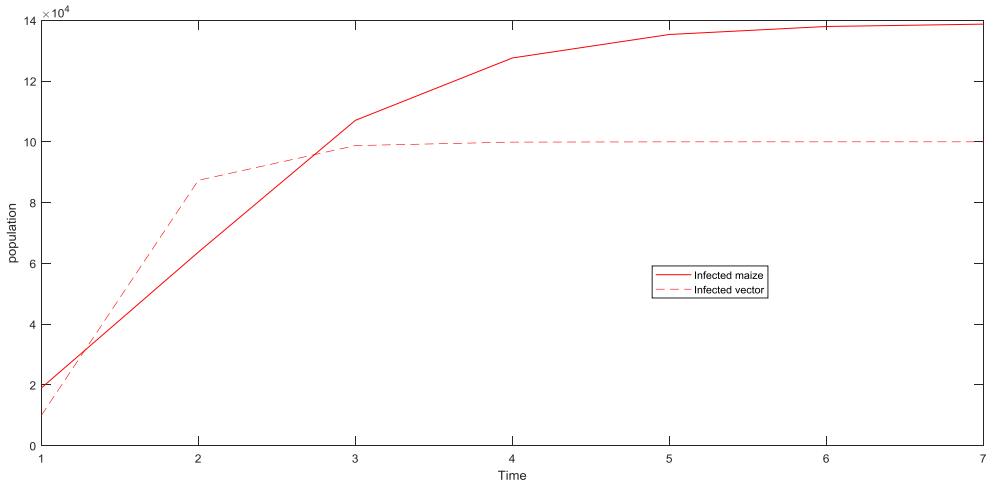

Figure 4.3: Illustrates the effects of infected vector on the susceptible maize plant in the absence of predators. (β_{mv}) = 0.086, (β_{ov}) = 0.02, (μ_v) = 0.003, (π_m) = 0.005.

Experiment 2: We investigates the impacts of ptrdatorsl on the vectors population (both susceptible and infected). When the capture rate for the vector by the predator is low, it will take long time for the vector to die. But if the capture rate is high, it will only take them short period of time to clear the vectors. Hence, this will eradicate MLND spread by vector as illustrated in Figure 4.4- 4.6.

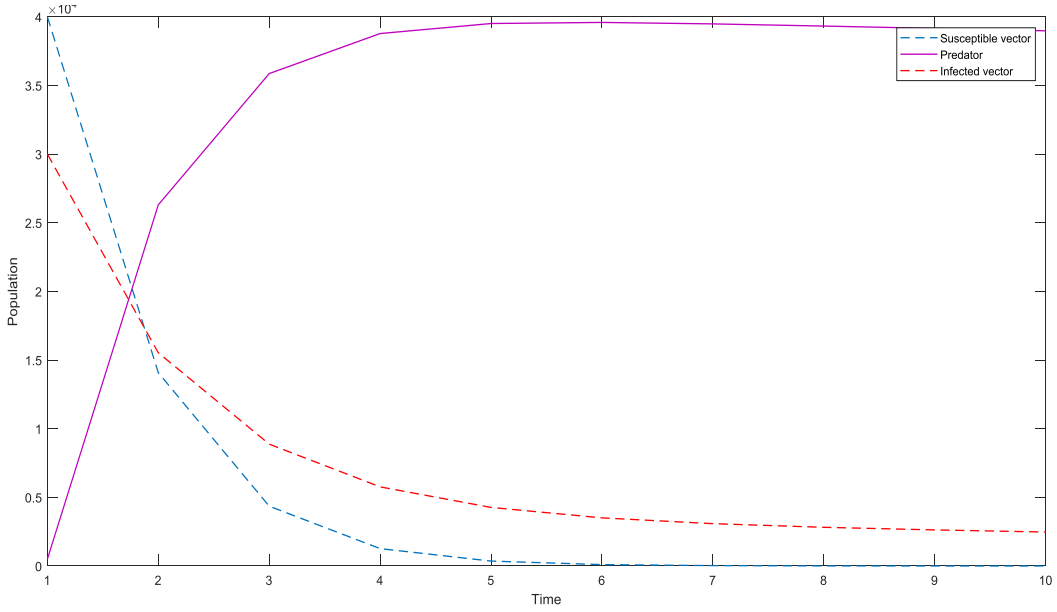

Figure 4.4: Illustrate the impacts of predators on the vector. γ =0.46, ε_c = 0.14, μ = 0.002, μ_c = 0.07.

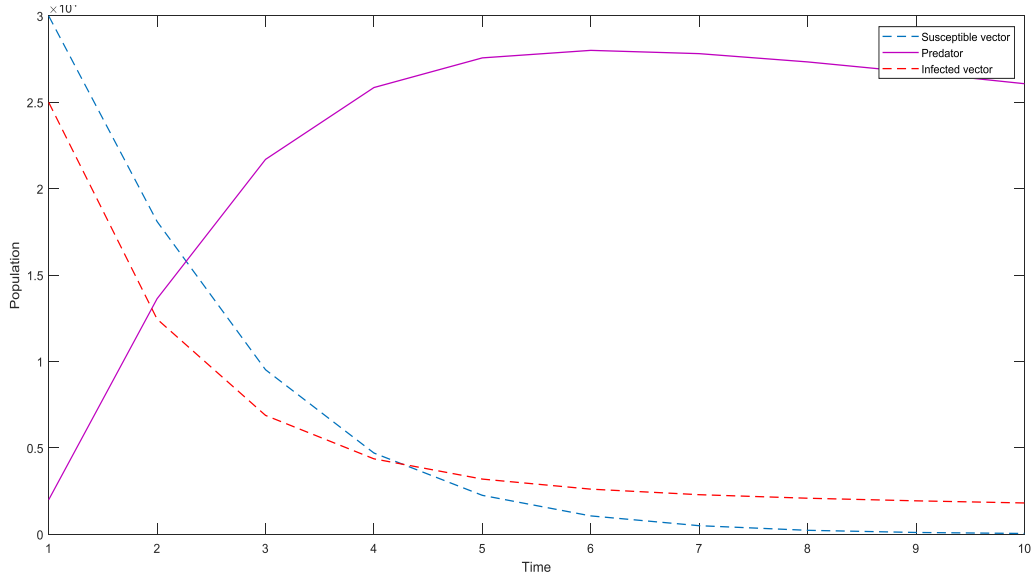

Figure 4.5: Illustrate the impacts of predators on the vector. γ =0.66, ε_c = 0.18, μ = 0.005, μ_c = 0.098.

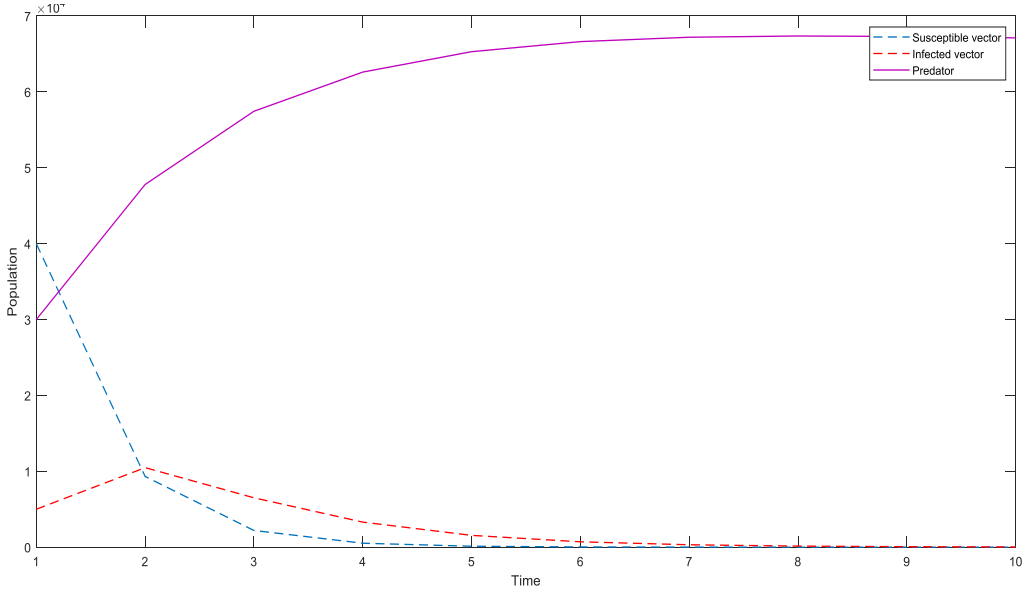

Figure 4.6: Illustrate the impacts of predators on the vector. γ =0.94, ε_c = 0.18, μ = 0.004, μ_c = 0.24.

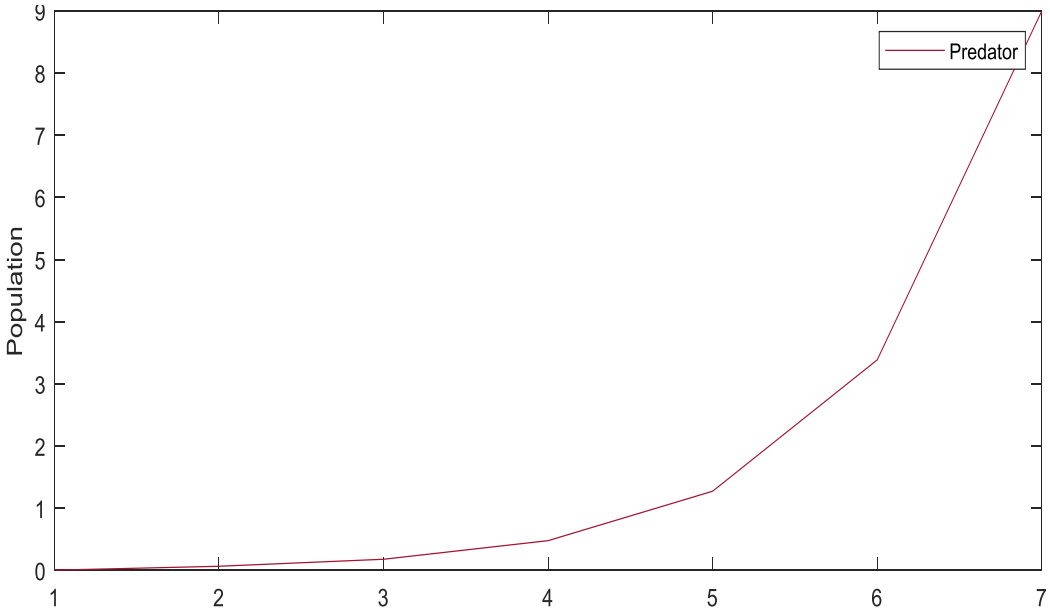

Figure 4.7: Illustrate how predator grows when vector is presence. γ =0.42, ε_c = 0.16, μ = 0.0002.

4.1.9 Discussion of results

Here, we discussed the results obtained in the analytical studies and the numerical simulations. The modified model consists of system of ordinary differential equations. The disease free equilibrium (DFE) was established which showed that in the absence of disease, susceptible maize population exits without recruitment and naturally dying of the maize. Other analytical results show that the system has unique solution in line with theorem (4.1.2). The basic reproduction number, $R_{0,}$ obtained indicate that there is an increasing in the disease transmission.

Finally, numerical solution established to illustrate the dynamic behavior of MLND in maize population and how some parameters affect the reproduction number R_0. It also illustrates the impact of predators on vector population.

Numerical simulation in experiment 1, shows effects of vector population in spreading MLND to maize population when there is no predator. In figure 4.1, we observed that the lesser vector population, the smaller number of infected maize while figure 4.3 illustrate that when the vector population is high, almost all the susceptible maize will become exposed and within short period of time will be infected.

Numerical simulations in experiment 2, Investigates the impacts of predators on the vectors population (both susceptible and infected). We have seen that when the capture rate for the vector by the predator is low, it will take long time for vector to be eradicated. But if the predator population is large, within short period of time, vectors population will decay through capture rate (μ_c) by predator and cause of natural death (μ_v). Hence, this will eradicate spread of MLND by vector as illustrated in Figure 4.4- 4.6.

Numerical simulation in figure 4.7, indicate that predator population grows exponentially when vector is presence. This occurred as a result of capture rate of vector by the predator (μ_c).

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMENDATION

5.0 Introduction

This chapter gives the summary of this research carried out on mathematical model on the dynamics of maize lethal necrosis disease under the deployment of a biological enemy and the conclusion made base on our findings.

5.1 Summary

In this research, we modified the mathematical model for the MLND dynamics and sensitivity analysis in a maize population by William, Dmitry and Livingstone (2018). We carried out analytical studies on the modified model where maize population was partition into three sub-populations; susceptible maize, exposed maize and infected maize. Similarly, the vector population was also partition into two sub–population; susceptible vector and infected vector. The model also consists of virus in the environment and predator (biocontrol) compartments in addition. We obtained existence and uniqueness of the solution, positivity of the solution, invariant region and we used next generation matrix approach in obtaining the basic reproduction number, R_0, which is the number of secondary infection. We also obtained disease free equilibrium (DFE) points and results showed that the disease free equilibrium point is locally asymptotically stable when $R_0<1$, indicating that is possible to end spread of MLND by vector in a maize population. We also obtained global stability of the disease free equilibrium points of the system and found is globally asymptotically stable.

5.2 Conclusion

This research, presents modified model of mathematical model on the dynamics of maize lethal necrosis disease under the deployment of a biological enemy. Analytical study was carried out using linearization method and the results shows that the disease free equilibrium (DFE) points are locally asymptotically stable (LAS) whenever $R_0 < 1$ and global asymptotically stable (GAS) whenever $R_0 <1$. The result from the numerical experiments carried out showed that there is a significant impact of predator on the vector population.

5.3 Recommendation

With this our findings, we recommended that government or organizations to further awareness on application of biocontrol in our farms. Since most of the insecticides have some effects either on the soil or harvested product.

5.4 Contribution to Knowledge

The modified model can be used to control vector population in maize farm that is suffering from MLND through vectors. Not only that, it will also add value to body of knowledge, and policy makers.

5.5 Objectives Achieved

i. The model which consists of system 6-equations of non-linear differential equations was extended to system of 7-equations of non-linear differential equation.
ii. The existence and uniqueness, invariant region and positivity of the solution of the modified model were established.
iii. The theoretical stability analysis of disease free equilibrium points was carried out.
iv. The modified model developed was numerically simulated.

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