ADDIS ABAB UNIVERSITY

COLLEGE OF NATURAL AND COMPUTATIONAL SCIENCES

DEPARTMENT OF MATHEMATICS

ENDEMIC MALARIA DYNAMICS WITH VARIABLE HUMAN AND MOSQUITO POPULATIONS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE MASTERS PROGRAM AT ADDIS ABABA UNIVERSITY

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ADDIS ABABA, ETHIOPIA

AUGUST, 2024

The undersigned hereby certify that they have read and recommend to the College of Natural and Computational Sciences for acceptance of the preliminary thesis entitled

"**ENDEMIC MALARIA DYNAMICS WITH VARIABLE HUMAN AND MOSQUITO POPULATIONS**"

By Talelign Tatek Geremew in partial fulfilment of the requirements for second degree.

Dated: August 2024

External Examiner: _____

Research supervisor: _____

Examine committee: _____

_____

Date: August, 2024

Author: **Talelign Tatek**

Title: A Mathematical Model for Endemic Malaria with Variable Human and Mosquito . . Populations

Department: Mathematics

Degree: **Masters** Convocation: August Year: 2024

ABSTRACT

A malaria disease transmission dynamics analysis is done using a deterministic differential equation model for endemic malaria dynamics that incorporates a variable human and mosquito population. Disease free and endemic equilibrium points are calculated and their stability is discussed. For this analysis, a threshold parameter $\tilde{R}_o$ is calculated. The disease can continue only if $\tilde{R}_o$ is greater than 1and the disease-free equilibrium point is globally stable when $\tilde{R}_o$ is less than 1.We have also shown that there exists an endemic equilibrium point for the model. Numerical stimulation is used to demonstrate the population dynamics and its dependence in the threshold parameter.

Key Words: Malaria disease model, threshold parameter, stability, numerical simulations

Acknowledgement

I would like to extend my deepest gratitude to all those who have contributed to the successful completion of this project on Endemic Malaria Dynamics with variable Human and Mosquito Populations. Firstly, I am profoundly grateful to my advisor, [**Dr. KASSAHUN WORKALEMAHU]**, for their invariable guidance, support, and encouragement through this research. This expertise and insights haven been instrumental in shaping the direction and outcomes of the study. I would also like to thank my colleges and team members, [Addis Ababa University]

For their collaborative efforts, dedication and hard work. Their contributions have been crucial in the data collection, analysis and interpretation phases of this project. Special thanks to my families for the providing the necessary resources and facilities to conduct this research. Lastly, I am indebted to my family and friends for their unwavering support and understanding during this research.

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CHAPTER ONE

1. INTRODUCTION

Malaria is a parasite-borne illness that is widespread throughout the world. Currently, malaria affects at least 300 million people globally, and it causes one to 1.5 million fatalities per year [1].

Malaria is becoming more common in many urban places across the world, and nearly all high endemicity areas are found in developing nations where poor drainage creates vast pools of stagnant water that serve as perfect breeding grounds for disease-carrying insects like Anopheles mosquitoes [2–7].

Every time an infected Anopheles mosquito feeds on blood, a female mosquito spreads the malaria parasite from one person to another. Fever episodes and anemia are signs of malaria fever in an infected individual. For humans, the incubation period lasts roughly 12 days, but for mosquitoes, it lasts roughly 10 days. Depending on the parasite strain, the duration may extend.

There is a wealth of research on mathematical models of communicable diseases. Take a look at the Hothcote et al. survey, for instance [9].It is common practice to assume a constant total population in the majority of these models. Since Ross [10] was the first to predict the dynamics of malaria transmission [11–13], mathematical modeling of malaria has developed. Who developed Ross' findings and proposed the super infection theory?

Using data from the Garic project [14], many studies have been carried out on the epidemiology of malaria, and one of the most outstanding the mathematical model proposed by Dietz et al [15], which Nedleman[16] has analyzed in detail. Further works on the subject include [17-19] and the review by Nedleman[20].Today, we are faced with the need to predict the dynamics and transmission of indirectly diseases with greater accuracy over long period of time , and more often with limited empirical data.

Under the presumption that immunity lasts for an extended period of time regardless of infection exposure, the dynamics of numerous communicable illnesses have been thoroughly examined

Nonetheless, it seems that ongoing exposure maintained the immunity to malaria. Therefore, immunity may offer protection against serious disease without completely eradicating persistent, minor illnesses, making the traditional concept of immunity as complete refractoriness to infection

potentially overly restrictive. Asymptomatic immunological carriers could therefore be contagious. It is well recognized that inadequate immunity allows malaria to spread, which makes disease control tactics more difficult because both symptomatic and asymptomatic infected people are now part of the infection reservoir.

It is evident that when dealing with endemic diseases like malaria, which have a high death rate, the assumption of constant population size in epidemiological models—which is generally valid when researching diseases of short duration with modest effects on mortality—may no longer be applicable. In actuality, a considerable degree of fluctuation in mosquito populations is always introduced by the interaction between the human population and mosquito populations, hence it is invalid to assume that the population is constant.

In mathematically tractable assessments of mathematical models for malaria transmission with varying population densities, the pseudo equilibrium hypothesis is frequently used to exchange information about infected mosquitoes for information about all insects and affected individuals. In this paper, Nedelma demonstrates how the inoculation rate in Dietz, Molineaux, and Thomas' malaria model [15] is dependent on a pseudo-equilibrium rate approximation to the differential equation that characterizes the dynamics of mosquitoes.

This estimate may be fairly restricted when we take into account that, with the exception of blood transfusion instances, the dynamics of the vector population play a major role in the transmission of the malaria parasite from person to person. Thus, when considering the dynamics of the mosquito population, it is imperative to investigate the dynamics and transmission of the malaria parasite

In this instance, we create and examine a model with mosquito population compartments. We include a class of individuals in our model who may be infectious but who have a partial immunity to the malarial sickness, in keeping with the concept put forth by Aron [26–27]. By modifying the logistic equation to account for disease-related mortality, we suppose that both the human and vector populations have density-dependent death rates, causing the overall populations to fluctuate over time. For instance, Nisbet and Gurney [29] employed the density-dependent mortality rates in a population assumption, which was initially proposed by Verhulst [28], in their investigation of predator-prey models.

1.1. Aims of the thesis

The primary objective of this thesis is to understand and predict the behavior of endemic malaria dynamics with in variable human and mosquito populations.

1.1.1 The specific objectives of this thesis are:

- To analyze the effects of variations in human and mosquito populations on the dynamics of malaria disease transmission.
- To study the impact of seasonal fluctuation.
- To assess the impact of variable populations on the incidence, prevalence and prevention of malaria disease.

1.2 Significance of the thesis

- The outcome of this thesis can be used to predict the behavior of the dynamics in the interaction between the mosquito and host populations.
- The thesis will be used to indicate future conditions and more control techniques in a way that is suitable for policy makers.

CHAPTER TWO
2. THE BASIC MODEL

We define the following contact parameters below as follows in order to provide motivation for the creation of our mathematical model.

Symbol	Explanation
c_{hv},$\tilde{c}_{hv}$	Respective infectivity of an infectious no immune and a partially immune human. Defined as the probability that a bite by susceptible mosquito on an infected human will transfer the infection to the mosquito.
$\mathbf{c_{vh}}$	The infectivity of the mosquito. Defined as the probability that a bite by an infected. Mosquito on a susceptible human will transfer the infection to the human.
a_v	The man biting-rate of the mosquitoes. Defined as the average number of bites given to humans by each mosquito per unit time.

Table 1.1 Parameters with their explanations

The following characteristics are used to classify the human and vector populations into states or categories, these are: - susceptible, Incubating, Infectious and Immune individuals.

At time **t**, there are $\mathbf{S_h}$=susceptible humans

$\mathbf{E_h}$ =Incubating humans

$\mathbf{I_h}$ =infectious humans,

$\mathbf{R_h}$ = partially immune humans,

$\mathbf{S_v}$ = susceptible mosquitoes

$\mathbf{E_v}$ = incubating mosquitoes and

$\mathbf{I_v}$ =infectious mosquitoes.

Note that the mosquito population does not have an immune class, since their infective period ends with their death.

The total human and vector populations at time t, given by:

$\mathbf{N_h = S_h + E_h + I_h + R_h}$ and

$\mathbf{N_v = S_v + E_v + I_v}$ are respectively,

The model assumes all newborns are susceptible in both populations (no vertical transmission) and a uniform birth rate. The birth rates for both human and mosquitoes are

$\boldsymbol{\lambda_h > 0}$ and $\boldsymbol{\lambda_v > 0}$, respectively.

Immune human individuals lose their immunity at a rate of $\boldsymbol{\beta_h > 0}$, and incubating individuals in both populations become infectious with rates of $\mathbf{v_h > 0}$ and $\boldsymbol{v_v > 0}$.

Infectious human individuals either recover with a rate $\mathbf{r_h}$ (without any substantial gain in immunity) to join the susceptible category or with a rate $\mathbf{a_h}$ into the immune room, where they stay for a period of their immunity before joining the susceptible class.

Every individuals in both human and mosquito populations undergoes death rates of $\mathbf{f_h(N_h)}$, $\mathbf{f_v(N_v)}$, respectively, where for the consequence, $\mathbf{f_h}$ and $\mathbf{f_v}$ are imitative to be strictly monotone increasing continuously differentiable functions of their arguments statements.

Indeed infected human individual's free-from the disease at the extra rate $\boldsymbol{\gamma_h > 0}$.

The efficient striking rates between the two populations, which may defined as the mean number of striking per day that will guide to the infectious of one individual if the other individual was infectious, depends on a number of reasons. These are:

the biting rate of the mosquitoes, the transmission probabilities between the species and the amount of individuals in both populations.

Shortly, from the above definitions and factors, we have the following:

There are $\mathbf{a_v N_v / N_h}$ bites per human per time if $\mathbf{a_v}$ is the man –biting rate of the mosquito.

Since there are (S_h) susceptible humans and the proportion of the total number of bites that are potentially infectious to humans is $\frac{I_v}{N_v}$,

The number of potentially infectious bites given to susceptible humans is $a_v I_v S_h/N_h$ bites per time.

In all cases, only a fraction of these bites, namely (c_{vh}), successfully infect humans. Hence we have the model given below:

$$\textbf{Human exposure rates} = \left(\frac{c_{hv}a_v I_v}{N_h}\right) S_h \qquad (1)$$

Similarly, if the classes I_h and R_h are included in the reservoir of potentially infectious human material, we have

$$\textbf{Mosquitoes rate of exposure} = \left(\frac{c_{hv}a_v I_h}{N_h}\right) S_v + \left(\frac{\tilde{c}_{hv}\acute{a}_v R_h}{N_h}\right) S_v. \qquad (2)$$

Now, we use fundamental mass action concepts to build the equations that characterize the transmission of the disease.

$$\frac{dS_h}{dt} = \lambda_h N_h + \beta_h R_h + r_h I_h - f_h(N_h)\, S_h - \left(\frac{c_{vh}a_v I_v}{N_h}\right) S_h,$$

$$\frac{dE_h}{dt} = \left(\frac{c_{vh}a_v I_v}{N_h}\right) S_h - (v_h + f_h(N_h))E_h, \qquad (3)$$

$$\frac{dI_h}{dt} = v_h E_h - (r_h + a_h + r_h(N_h))I_h,$$

$$\frac{dR_h}{dt} = a_h I_h - (\beta_h + f_h(N_h)) \quad I_h,$$

$$\frac{dS_v}{dt} = \lambda_v N_v - f_v(N_v)\, S_v - \left(\frac{c_{hv}a_v I_h}{N_h}\right) S_v - \left(\frac{c\acute{}_{hv}\, a_v R_h}{N_h}\right) S_v,$$

$$\frac{dE_v}{dt} = \left(\frac{c_{hv}a_v I_h}{N_h}\right) S_v + \left(\frac{c\acute{}_{hv}a_v R_h}{N_h}\right) S_v - (v_v + f_v(N_v))\, E_v,$$

$$\frac{dI_v}{dt} = v_v E_v - f_v(N_v)\, I_v,$$

And

$$\frac{dN_h}{dt} = \lambda_h N_h - f_h(N_h)N_h - r_h I_h, \text{ and}$$

$\frac{dN_v}{dt} = \lambda_v N_v - f_v(N_v)N_v$, **(4)**

When all model parameters are taken to be positive then the formula for:

$N_h = S_h + E_h + I_h + R_h$ and

$N_v = S_v + E_v + I_v$ are acquired by summing the pertinent equations in [3].

These formulas hold true for: $N_h > 0$ with $0 \leq S_h/N_h, I_h/N_h\ R_h/N_h \leq 1$.

When $N_h = 0$, we interpret the quantities involving division by N_h as zero.cf [32]

It should be noted that the formulation above differs from that found in [33], where Esteva and Vargas create a similar model for gangue fever a disease carried by a mosquito, Aedes aegypti.

There, they use a term $[\lambda_h\ S_h\ I_v]/N_v$ to represent the rate of recruitment into the infectious human compartment. This requires that λ_h have quasi-dimensional units of per time.

The parameter λ_h represents the rate of requirement at which human are bitten by mosquitoes over time. It has the following

Mosquito biting rate: This is the average rate of times on a mosquito bites a human with in a given period.

Percentage of rate that results in infection: This accounts for the probability that a mosquito bite leads to infection.

Vector to human population ratio: The proportion of mosquitoes relative to humans. A higher ratio means more mosquitoes per human, increasing the likelihood of a person being bitten.

Nevertheless, contact rates are sometimes taken to be directly proportional to the overall population in classical endemic models involving population of the same species as in [22], constant as in [35], or in some intermediate form as in [36].By applying the conventional methods outlined in [37],it can demonstrated that if beginning conditions are given for every state variable in the system [3] at time t=0 with $S_h(0) + E_h(0) + I_h(0) + R_h\ (0) = N_h\ (0)$,

then there exists a unique solution satisfying these initial conditions for all $t \geq 0$ with

$S_h(t) + E_h(t) + I_h(t) + R_h(t) = N_h\ (t)$ for all $t \geq 0$. It can also be verified that $N_h(0)>0$, then

N_h(t) > 0 for all t, where as if $N_h(0)$ =0, then $N_h(t)$ = 0 for all t.

Similarly, the vector equations with matching expressions are of course subject to identical considerations. As a result, both mathematically and physically, system [3] is well posed. According to [4], there are at least two steady state solutions to the equation that describes how the entire vector population behaves: $N_v{}^*$=0 and $N_v{}^*$=$f_v{}^{-1}(\lambda_v)$.Since, by hypothesis, f_v is a strictly monotonic increasing function of N_v, $f_v{}^{-1}$ exists and is unique.

A linearization about $N_v{}^*$=0 yieads the linear approximation $N_v = (\lambda_v - f_v(0))\, N_v$, where the dot represents differentiation with respect to t. Hence, $f_v(0)$ is the linear death rate the mosquito population and we assume that $\lambda_v \geq f_v(0) \geq 0$.This further implies that $f_v{}^{-1}(\lambda_v)$ exists and is nonnegative.Similar considerations on the total human populations also shows that we require $\lambda_v \geq f_h(0) \geq 0$. An analysis of sire model with generalized density dependent death rates may be found in {32}.

Here, for positive constants μ_h, μ_v μ_{2h} and μ_{2v}, we consider the forms

$f_v(N_v)= \mu_v+\mu_{2v}N_v$

$f_h(N_v)= \mu_h+\mu_{2h}N_h$, for f_v andf_h, **(5)**

So in the absence of the disease, both the populations are modeled by the logistic growth model with carrying capacities

$\frac{\lambda_v-\mu_h}{\mu_{2h}}$ and $\frac{\lambda_v-\mu_v}{\mu_{2v}}$ for the human and vector populations, respectively.

These carrying capacities exist and are nonnegative if $\lambda_h \geq \mu_h$ and $\lambda_v \geq \mu_v$.

We make the modification of variable because it is simpler to analyze the model in terms of proportions of susceptible, incubating, infected, and immune individuals.

$\mathcal{U} = \frac{S_h}{N_h}\ \mathcal{R}$ $R = \frac{R_h}{N_h}$

$\mathcal{V} = \frac{\mathrm{E_h}}{\mathrm{N_h}}$ $x = \frac{S_v}{N_v}$

$\mathcal{W}=\frac{I_h}{N_h}$ $y = \frac{E_v}{N_v}$ and $\mathcal{Z}=\frac{I_v}{N_v}$ **(6)**

So $\mathcal{U} + \mathcal{V} + \mathcal{W} + \mathcal{R} = 1 \Rightarrow \mathcal{V} = 1 - \mathcal{U} - \mathcal{W} - \mathcal{R}$, $\mathcal{X} + \mathcal{Y} + \mathcal{Z} = 1 \Rightarrow \mathcal{Y} = 1 - \mathcal{X} - y$ **(7)**

Additionally, by assigning, $r = \mu_o t$ the total populations by their respective carrying capacities, we arbitrary scale time t with the quantity$\frac{1}{\mu_v}$ setting by:

$$N_h=\left(\frac{(\lambda_v-\mu_h)}{\mu_{2h}}\right) N_h{}^*, \qquad N_v = \left(\frac{(\lambda_v-\mu_v)}{\mu_{2v}}\right) N_v{}^*$$

Hence, we introduce the following dimensionless parameters after dropping asterisks.

$r = \mu_o t$ $\lambda = \frac{\lambda_h}{\mu_v}$ $\beta = \frac{\beta_h}{\mu_v}$ $\gamma = \frac{\gamma_h}{\mu_v}$ $v = \frac{v_h}{\mu_v}$

$\tau = \frac{\tau_h}{\mu_v}$ $\alpha = \frac{\alpha_h}{\mu_v}$ $e = \frac{e_h}{\mu_v}$ $a = \frac{a_h}{\mu_v}$ $b = \frac{c_{hv}a_v}{\mu_v}$ **(8)**

$\lambda = \frac{\tilde{c}_{hv}a_v}{\mu_v}$ $e = \frac{\mu_h}{\mu_v}$ $e = \frac{v_v}{\mu_v}$ $\xi(N_h, N_v) = \frac{c_{vh}a_{2h}(\lambda_v-\mu_v)N_v}{\mu_{2v}\mu_v(\lambda_h-\mu_h)}$

For parameters pertaining to the human population, we have utilized Greek letters in addition to the parameter. System 3 turns into

$$\frac{du}{dr} = -\lambda(1-u) + \beta R + rw + \gamma wu - \xi uz$$

$$\frac{dw}{dr} = v(1-v-R) + \gamma w^2 - (r + \alpha + \gamma + \lambda + v)w \qquad \textbf{(9)}$$

$$\frac{dR}{dr} = \alpha w + \gamma wR - (\beta + \lambda)R$$

$$\frac{dx}{dr} = \alpha(1-x) - (\beta + \lambda)R$$

$$\frac{dz}{dr} = e(1-x) - (a+e)z$$

In this case, we have removed v and y from the system by using (7), and we have expressed ξ in a way that best highlights the contact rates' density dependency. The whole population equation, which actually dictates behavior, now has the form of:

$$\frac{dN_h}{dr} = (\lambda - \epsilon)(1 - N_h)N_h - \gamma N_h w$$

$$\frac{dN_v}{dr} = (a - 1)(1 - N_v)N_v \qquad \textbf{(10)}$$

Where r represents another independent variable

$\lambda, \epsilon, \gamma, and$ are parameters that govern the growth and interaction between the populations.

W represents another variable that interacts with the human populations related to the vector population.

N_h and N_v represents human and vector populations. The first term $(\lambda - \epsilon)(1 - N_h)N_h$ represents the logistic growth model for the human population. The factor $1 - N_h$ ensures that population growth slowdown as N_h approaches to 1.

The second term $-\gamma N_h w$ represents a reduction in human population.

From the vector population growth model the factor $\alpha - 1$ controls the growth rate of the vector population, and (1−Nv) ensues that growth slows as N_v approaches its maximum.

Assumption on stability

We assume that $\lambda - \epsilon - \gamma > 0$, which clearly guarantees the existence of nonnegative steady state solution for $N_h(t)$ as t→ ∞ . This means that under this condition the human population reaches a stable equilibrium where the growth and loss terms balance each other

Now the model in terms of populations (9) is defined in the subset $\boldsymbol{\Omega} \times [\mathbf{0}, \infty)$ of $\mathbb{R}^6_+$, where

$$\Omega = \{(u, w, \mathcal{R}, \mathcal{X}, \mathcal{Z}): 0 \leq u, w, \mathcal{R}, \mathcal{X}, \mathcal{Z} \leq 1, 0 \leq u + w + R \leq 1, 0 \leq x + z \leq 1\} \qquad \textbf{(11)}$$

This define the feasible region for the variables in our model. All variables are constrained between 0 and 1, and some are subject to additional conditions that ensure their sum does not exceed certain limits.

Initial conditions

The population can yield the initial values by earlier equation (6-8), meaning that the system s initial conditions can be derived or computed based on previous relationships that describe the population dynamics.

CHAPTER THREE

3. ANALYSIS OF THE MODEL

3.1. EXISTENCE OF THE STEADY STATE SOLUTIONS

In chapter three we analysis the steady state solutions for the mathematical model introduced earlier by investigating the existence of equilibrium solutions where the system of differential equations reaches a stable state. This involves using threshold parameter $\tilde{R}_O$, likely analogous to a basic reproduction number in epidemiological models. Which determines whether the populations will grow or decline over time.

Threshold parameter $\tilde{R}_O$ plays a crucial role in determining the system dynamics: That is

If $\tilde{R}_0 \geq 1$, the disease (vector population) tends to grow, leading to a potential unstable or endemic state.

If $\tilde{R}_0 \leq 1$, the disease (vector population) declines, leading to a disease free equilibrium or trivial equilibrium.

To establish the existence of the steady state solution, we introduce the following proposition.

Proposition 3.1: **Existence of Endemic equilibrium**

There is at least one equilibrium in the proportionately calculated model, when $\tilde{R}_0 \geq 1$.This equilibrium is characterized by non-negativities steady state values $\mathcal{U}^*, \mathcal{W}^*, \mathcal{R}^*, \mathcal{X}^*, \mathcal{Z}^*$ for the variables u, w, R, x, z representing different population or densities in the model.

Endemic Equilibrium E, the equilibrium point E is given by:

E = $(\mathcal{U}, \mathcal{W}, \mathcal{R}, \mathcal{X}, \mathcal{Z}) = (\mathcal{U}^*, \mathcal{W}^*, \mathcal{R}^*, \mathcal{X}^*, \mathcal{Z}^*)$ with $\mathcal{U}^*, \mathcal{W}^*, \mathcal{R}^*, \mathcal{X}^*, \mathcal{Z}^*$ all nonnegative, whose existence and properties are determined by

T**he threshold parameter $\tilde{R}_0$,** where

$$\tilde{R}_0 = \frac{\xi e \nu (\alpha c + b(\beta + \lambda))}{a(a+e)(\beta+\lambda)(\lambda+\nu)(a+r+\gamma+\lambda)} \qquad \textbf{(12)}$$

This parameter determines whether the system has an endemic equilibrium $(R_0 \geq 1)$or a trivial equilibrium $(\tilde{R}_0 \leq 1)$.

Proof: Let $(u^*, w^*, R^*, x^*, z^*)$ be a constant solution of model (9). We express as $\mathcal{U}^*, \mathcal{W}^*, \mathcal{X}^*, \mathcal{Z}^*$ in terms of $\mathcal{R}^*$ as follows:

$$w^*(R^*) = \frac{(\beta+\lambda)R^*}{a+\gamma R^*}$$

$$u^*(R^*) = 1 - R^* + \frac{(\beta+\lambda)R^*\left(\gamma(\beta+\lambda)R^* - M(\alpha*\gamma R^*)\right)}{(\alpha+\gamma R^*)^2 \upsilon} \quad \textbf{(13)}$$

$$x^*(R^*) = \frac{a(\alpha+\gamma R^*)}{a\alpha+(\alpha c+b(\beta+\lambda)+a\gamma)R^*+c\gamma R^{*2}}$$

$$z^*(R^*) = \left(\frac{e}{a+e}\right)\frac{(\alpha c+b(\beta+\lambda)+c\gamma R^*)R^*}{a\gamma+(\alpha c+b(\beta+\lambda)+\alpha\gamma)R^*+c\gamma R^{*2}}$$

Sixth-order polynomial:

By substituting these expressions into the first of (9) of the system and setting it to zero we obtain the following sixth order polynomial in R^*. .

$$R^*(A_5 R^{*5} + A_4 R^{*4} + A_3 R^{*3} + A_2 R^{*2} + A_1 R^{*1} + A_0 R^{*0}) = 0 \text{ , . } \quad \textbf{(14)}$$

This equation provides the possible values for R^* , where $R^* = 0$ is always a solution, representing the disease free state. Where,

$$A_5 = \text{acD}\tilde{R}_O\, \gamma^4\, v^2 \xi,$$

$$A_4 = \gamma^3 v(Ac(\beta+\lambda)(-B + a\beta(\beta+\lambda) + a\alpha v) + AD)\left(c + a\tilde{R}_O\right)$$

$$+cD\tilde{R}_O(B - a(\beta+\lambda)^2 + a(2\alpha-\gamma)v)\xi),$$

$$A_3 = \gamma^2\Big(A(\beta+\lambda)\Big(A(-B + a\beta(\beta+\lambda) + a\alpha v) + v\big(-\big(B(c\alpha + a\gamma)\big) + a(\beta+\lambda)(\alpha\beta\gamma -$$

$$c\alpha\lambda) + a\alpha(c\alpha+\alpha\gamma)v)\Big)\Big) + D\Big(A^2 + A\tilde{R}_O(B - a(\beta+\lambda)^2) + A\Big(2c\alpha + a\big(2\tilde{R}_O\alpha + \gamma -$$

$$\tilde{R}_O\gamma\big)\Big)v + c\alpha\tilde{R}_O v\big(2B - a(\beta+\lambda)^2 + \alpha(\alpha - 3\gamma v)\big)\xi\Big)\Big),$$

$A_2 = \alpha\gamma\left(A(\beta+\lambda)\left(-(A(\beta+\alpha\lambda)(\beta+\lambda)-a\alpha v)\right)+a\gamma v\left(2B+\alpha(\beta^2-\lambda^2+2\alpha v)\right)\right)+$

$D\left(2A^2+\quad A\tilde{R}_o(2B-\alpha(\beta+\lambda)^2)+A\left(\left(c+a\tilde{R}_o\right)a-3a\left(-1+\tilde{R}_o\right)\gamma\right)V+c\tilde{R}_o\alpha v(\beta-\right.$

$\left.3\alpha\gamma v)\right)\xi,$

$A_1 = \quad \alpha^2\left(-\left(aA\gamma(\beta+\lambda)v(\beta+a\lambda(\beta+\lambda)-a\alpha v)\right)+D\left(A^2+AB\tilde{R}_o-3aA\left(-1+\tilde{R}_o\right)\gamma v-\right.\right.$

$\left.\left.ac\tilde{R}_{o\alpha\gamma v^2}\right)\xi\right)$

$A_0=\alpha AD\alpha^3 v\left(1-\tilde{R}_o\right)\xi$

And

$A=v\left(\alpha c+b(\beta+\lambda)\right)$

$B=a\left(\alpha v+M(\beta+\lambda)\right)$ **(15)**

$D=\frac{a}{\xi}(\beta+\lambda)(v(\alpha+\gamma+r)+\lambda M)$

$M=\alpha+r+\gamma+v+\lambda$

This implies,$R^*=0$ is a solution. Note that A_5 is positive while the sign of A_o coincides with that $\left(1-\tilde{R}_o\right)$, so that if $\tilde{R}_o$ >1, A_0 <0, in which case, we have at least one sign change in the sequence of coefficient $\{A_5,..,A_0\}$.

Descates Rule of signs,

By **Descartes Rule of signs**, we can infer the existence of positive real root of the polynomial. Since here exists A_5 is positive and the sign of A_0 depends on $1-\tilde{R}_o$, if $\tilde{R}_o > 1$, then $A_0 < 0$. This implies that there is a t least one sign change in the sequence of coefficients $\{A_5,..,A_0\}$ meaning three is at least one positive real root for the R^* in addition to $R^*=0$. Hence, by **Descartes Rule s Of Signs** there exists at least one positive real root for (14) aside from the root R^*=0, whenever $\tilde{R}_o$>1.■

Remark: From an **epidemiologically realistic** point of view, all we require is that there be a solution $R^* \in [0,1]$ satisfying (14). When such a solution exists, we call it realistic and values for the other steady states are given by (13).When $R^*=0$, the steady state postulated by proposition

(3.1) is the solution $E_0 = (u, v. R, x, z) = (1,0,0,1,0)$ as a constant solution, called the disease free equilibrium **DFE**.

$u = 1,$: The entire population is uninfected

$w = 0,$ There are no infected populations

$R = 0,$ No proportion of the population is infected or in contact with the disease.

$x = 1,$ Full recovery or absence of infection in the population.

$z = 0,$ No presence of the vector or diseases agents in the environment. This equilibrium describes a healthy population with no disease transmission, often referred to as the diseases free equilibrium (**DFE**).

Preposition 3.2 If Nv =0 or any of $\upsilon, e\ or\ \xi$ is zero, then the only realistic solution R^* of (14) is the solution $R^* = 0$ and the model formulated in terms of preposition has only the disease free equilibrium **DFE** E_0: $(u, , w, R, x, z)$=$(1,0,0,1,0)$ as a constant solution.

Proof:-

If $N_v = 0$, then from (8),ξ =0 and the first of (9) shows the only possible nonnegative constant solution for the system is the solution E_0 . The rest of the preposition follows immediately upon substitution of these values of $\xi, \upsilon, or\ e$ in the coefficient of (14).This preposition 3.2 gives some conditions such that the parameter $\tilde{R}_o$ can vanish. Since the parameter $\xi$is a group of many parameters including the total vector and host population the condition $\xi = 0,$ interpreted in different ways. For instance,$\xi = 0,$ mean the transmission probability is zero or the total vector population is zero,$\xi \to 0$ as$N_v \to 0$.The form (14) shows the DFE always exist. When$\tilde{R}_o > 1,$ a second equilibrium different from the DFE is established. This give the following result.

Proposition 3.3 Let $\tilde{R}_0$ >1, then the condition

$$\alpha\lambda - \beta\gamma \geq 0 \qquad (16)$$

is a sufficient condition to guarantee the existence at least one value $R^* \in (0,1)$ that solves equation (14). In this case, when $R^* > 1,$ there is at least one **feasible equilibrium** solution in the proposition-based model that differs from the disease-free equilibrium and is referred to as the

endemic equilibrium. Moreover, when $\gamma = 0$, this endemic equilibrium, $E_\gamma = 0$ is unique and can be expressible in terms of $\tilde{R}_o$.

Proof. Consider the function g: $\mathbb{R} \to \mathbb{R}$ defined by

$g(R^*) = A_5R^{*5} + A_4R^{*4} + A_3R^{*3} + A_2R^{*2} + A_1R^* + A_0,$

Where the coefficients A_i, i =0, 1… 5 are those of (14). We can confirm that

Step 1. Evaluate $g(0) = A_0 = \frac{e\alpha v A^2\alpha^3}{a+e}\left(\frac{1-\tilde{R}_o}{\tilde{R}_o}\right)$. Hence, $g(0) < 0$, when $\tilde{R}_o > 1$

Several algebraic operations demonstrate that

Step 2. $g(1) = (\beta+\lambda)\left(aM\ \tilde{R}_o(a+\gamma)\lambda(\beta+\lambda)c(a+\gamma) + b(\beta+\lambda)\right)\left(M(a+\gamma) - \gamma(\beta+\gamma)\right)$

$+ \left(M(a+\gamma) - \gamma(\beta+\lambda)\right)\left((-a\beta + \lambda\alpha\lambda)ca + b(\beta+\lambda)\right)\left(c(\alpha+\gamma) + b(\beta+\lambda)\right)$

$+ c\,(a+\gamma)\left(c\beta\left(\tilde{R}_o a(r+a) + \left(r\ \tilde{R}_o + \left(-1+2\ \tilde{R}_o\right)a\right)\gamma + \tilde{R}_o\gamma^2\right)\right)$

$+ c\left(a\left(a + \tilde{R}_o(r+a)\right) + \tilde{R}_o(r+2a)\gamma + \left(\tilde{R}_o\gamma^2\right)\lambda + b(\beta+\lambda)\left(\tilde{R}_o\right)\beta\right)\right)$

$+ \left(-1+\tilde{R}_o\right)\beta\gamma + \left(a + \tilde{R}_o(r+\alpha+\gamma)\right)\Big)v$

$+ (\alpha+\gamma)^2(r+a+\gamma)\left(ca + b(\beta+\lambda)\right)\left((a+c)(\alpha+\gamma) + b(\beta+\lambda)\right)v^2$

We can see that when condition (16) holds, g(1)≥0 when $\tilde{R}_0 > 1$, the existence of the root $R^*\epsilon(0,1)$ follows from the intermediate value theorem. Now, when $\gamma = 0$, (14) reduces to the first degree polynomial in R^* and using the parameter grouping in (15), we establish the following solution.

$$u^* = \frac{A+B}{A+B\tilde{R}_0} \qquad w^* = \frac{\alpha v(\beta+\lambda)(\tilde{R}_0-1)}{A+B\tilde{R}_0} \qquad z^* = \frac{D(\tilde{R}_0-1)}{A+B}$$

$$R^* = \frac{v\alpha a(\tilde{R}_0-1)}{A+B\tilde{R}_0} \qquad x^* = \frac{A+B}{(A+B)\tilde{R}_0} \qquad \textbf{(17)}$$

This is realistic only when $\tilde{R}_0 > 1$ with $\tilde{R}_0 = 1$ giving the **DFE**. ■

N.B. Form the above remark the constant solution postulated by preposition 3.1 are realistic if and only if each of them lies in the compact interval [0, 1]. The calculation show that $R^* = 1$ is not realistic because u^* is negative. So, we shall assume that $0 \leq R^* < 1$. Similarly,$w^* = 1$ is not realistic and we require $0 \leq w^* < 1$.

Bounding of R^*

From equation (13), we have

$0 \leq w^* < 1 \Longrightarrow 0 \leq \frac{(\beta+\lambda)R^*}{\alpha+\gamma R^*} < 1$, which leads

$$0 \leq R^* \leq \frac{\alpha}{\beta+\lambda-\gamma} < 1. \qquad \textbf{(18)}$$

The last inequality in (18) is strictly less than 1 because $\frac{\alpha}{\beta+\lambda-\gamma} = 1$, then $w^* = 1$, which is not allowed. This leads to demonstrate that when the parameters are chosen so that (16) and (18) are satisfied, then u^* as defined by (13) lies in the compact interval [0,1].

Steady-state solution for Total populations

The corresponding steady state values for the total vector and human populations are computed from equation (10). The steady state solution for which the total vector and human populations are both zero is **not realistic** in the sense that we have nothing to prove (both populations are extinct).

From (10), when $\lambda < 1$ (Respectively, $\alpha < 1$), the total human (respectively, vector) population tends to zero as t$\rightarrow \infty$.Here,we consider only the case $\lambda > \ell$ respectively, $(\alpha > 1)$, from which we see that, in the absence of the disease ,there is exponential growth of both populations near $N_h = 0$(respectively, $N_v = 0$) with saturation near $N_h = 1$(respectively, $N_v = 1$).In the presence of the disease ,when $\gamma = 0$,the endemic equillibria for both the vector and human populations are (N_h, N_v)=(1.1).

As disease related deaths set in, a new equilibrium the total human population, whose size is determined by the relative magnitude of γ, is established. This equilibrium is obtained by substituting the steady state value w^* into the appropriate equation from (10).This gives

$$N^*_h = 1 - \frac{\gamma}{\lambda-\epsilon} w^* \qquad \textbf{(19)}$$

If the disease related death rate is serve enough (i.e. γ is enough large), this equilibrium can ceases to exist.

Using w^* (13), we have $0 \leq N_h^* \leq 1 \Rightarrow 0 \leq R^* \leq \frac{(\lambda-\epsilon)\alpha}{\gamma(\beta+\epsilon)}$

Hence, the endemic equilibrium R^* when it exists must be a root of polynomial (14) and, in addition, should satisfy

$$0 \leq R^* < min\left\{1, \frac{(\lambda-\epsilon)\alpha}{\gamma(\beta+\epsilon)}, \frac{\alpha}{\beta+\lambda-\gamma}\right\}. \quad \textbf{(20)}$$

This minimum will exist whenever $\lambda > \epsilon$ since$\beta + \lambda > \gamma$.

There are two different approaches to thinking about a disease being contained in a population of different sizes. A milder demand would be that the proportions w, z, and possibly R, trend to zero with increasing time; cf[38,39]. The tougher approach would be to demand that the total number of infective that the reservoir of infection here

I_h,I_v and possibly $R_h \rightarrow 0$ with increasing time. Thus we seek condition for the stability of the endemic proportional state $(u^*, w^*, R^*, x^* Z^*)$ with $W^* > 0, R^* > 0$ and $z^* > 0$ and for the stability of the **DFE** $(u^*, w^*, R^*, x^* Z^*) = (1,0,0,1,0)$..The parameter $\tilde{R}_0$ is the basic reproduction ratio. It is usually defined as the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual during its entire period of infectiousness, and mathematically as the dominant eigenvalue of a positive operator [40].

Based on our definition,

- ξ is dependent on the likelihood of transmission,
- The frequency at which mosquitoes bite humans,
- The environment's ability to support both species, and
- The proportion of vector to human populations.

As a result, the proportions' behavior is highly dependent on the behavior of both the overall vector and human populations. Our representation has significance since the entire population is observable and quantifiable within a certain demographic. In this case, $\tilde{R}_o$ rises as the number of vectors grows.

CHAPTER FOUR

4. STABILITY OF THE EQUILLIBRIA

To analyze the local stability of the equilibrium solutions $(u^*, w^*, R^*, x^*, z^*)$ of the system, we need to examine the eigenvalues of the Jacoian matrix evaluated at the equilibrium.

By lingering system (9) about the equilibrium solution the local stability of the equilibrium solutions can be investigated as. The Jacobian matrix is so given

$$\begin{pmatrix} \gamma w^* - \lambda = \xi u^* & r + \gamma u^* & \beta & 0 & -\xi u^* \\ -v & 2\gamma R^* - M & -v & 0 & 0 \\ 0 & \alpha + \gamma R^* & \gamma w^* - \beta - \lambda & 0 & 0 \\ 0 & -bx^* & -cx^* & -\frac{a}{x^*} & 0 \\ 0 & 0 & 0 & -e & -(a+e) \end{pmatrix} \quad \textbf{(21)}$$

Where we have used the fact that $\alpha(1 - x^*) - bx^*w^* - cx^*R^* = 0$ to replace $\alpha + bw^* + cR^*$ by $\frac{a}{x^*}$.

The eigenvalues of J_E are the solutions of the fifth-order polynomial equation

$$\zeta^5 + a_1\zeta^4 + a_2\zeta^3 + a_3\zeta^2 + a_4\zeta + a_5 = 0 \quad \textbf{(22)}$$

Where

$a_1 = a + e + \frac{a}{x^*(R^*)} + B_1 + B_2 + B_3$,

$a_2 = \frac{a}{x^*(R^*)}(a + e) + (a + e + \frac{a}{x^*(R^*)})(B_1 + B_2 + B_3) + B_1(B_2 + B_3) + B_2B_3 + v(B_4 + B_5)$

$a_3 = \frac{a}{x^*(R^*)}(a + e)(B_1 + B_2 + B_3) + a + e + \frac{a}{x^*(R^*)})(B_1(B_2 + B_3) + (B_2B_3) + vB_2B_3 + v(B_4 + B_5)) + B_1(B_2B_3 + vB_4) + B_5B_3 + \beta B_4)) - \xi evu^*(R^*)b,$

$a_4 = \frac{a}{x^*(R^*)}(a + e)(B_1 + B_2 + B_3) + a + e + \frac{a}{x^*(R^*)})(B_1(B_2 + B_3) + (B_2B_3) + vB_2B_3 + v(B_4 + B_5)) + B_1(B_2B_3 + vB_4) + B_5B_3 + \beta B_4)) - \xi evu^*(R^*)b,$

$a_5 = \frac{a}{x^*(R^*)}(a + e)(B_1(B_2B_3 + vB_4) + vB_5B_3 + \beta B_4) - \xi evu^*(R^*)x^*(R^*)(cB_4 + \beta B_4),$

With B_i, i=1, 2, 3, 5 functions of the steady state solution R^* given by (13). Here,

$B_1=\lambda + \xi z^*(R^*) - \gamma w^*(R^*)$

B_2=M-$2\gamma w^*(R^*)$

$B_3= \beta + \lambda$-$2\gamma w^*(R^*)$ **(23)**

$B_4 =\alpha + \gamma(R^*)$

B_5=r+$\gamma u^*(R^*)$

Now from a stability point of view, all we wish to know is whether there exists a value ζ,that is a solution to (22) with $\Re(\zeta)>0$. If such a ζ exist, then the equilibrium solution is locally unstable to small perturbations, otherwise it is locally and symptomatically stable. Using the expressions in (13), we have

$B_1=\lambda + \xi z^*(R^*) - \gamma w^*(R^*) = \xi(R^*) + \frac{\lambda\alpha-\gamma\beta R^*}{\alpha+\gamma R^*} > 0,$

$B_2 =$ M-$2\gamma w^*(R^*) = (\alpha + V + r) + \frac{\alpha\lambda-\gamma\beta R^*}{\alpha+\gamma R^*} + \gamma(1 - w^*(R^*))>0,$

$B_3 = \beta + \lambda - \gamma w^*(R^*) = (\beta + \lambda)\left(1 - \frac{\gamma R^*}{\alpha+\gamma R^*}\right) > 0,$

Since $\lambda\alpha - \gamma\beta \geq 0$ from (16) and $0\leq w^*,\ R^*, x^* \leq 1$. Hence all groupings in the coefficients of (22) are positive. The coefficient a_1, s , a_2, a_3 are always positive; a_4 and a_5 could be negative.

Given equations (16) and (18) and the expression $\tilde{R}_0$ given by (12), we can show that whenever $\tilde{R}_0>1$, a_4 and a_5 are both negative. Hence, there are no positive real roots for polynomial (22).

The special case $\gamma = o$ is illuminating.

Now, we have the following results.

Proposition 4.1 Local and asymptotically stability of the Disease-Free Equilibrium (DFE)

The disease- free equilibrium is locally and asymptotically stable when $\tilde{R}_0 \leq 1$. Moreover, when $\gamma = 0$ and $\tilde{R}_0$>1, the unique endemic equilibrium $E_{\gamma=0}$, given by (3.3) is also locally and asymptotically stable.

Proof. The local stability of the DFE is determined by considering the value $R^* = 0$ in (22). Since the coefficients a_1, a_2 and a_3 are nonnegative, it is enough to show that a_4 and a_5 are also positive when $\tilde{R}_0 \leq 1$ and R^* = 0. This can be seen by making ξ, say the subject of equation (12) and substituting the resulting value of ξ together with R^* =0 in a_4 and a_5. When $\gamma = 0$, the nonzero positive value for R^* which exists for $\tilde{R}_0 > 1$ is given by (17). Again we verify that for this value R^*, a_4 and a_5 are positive $\tilde{R}_0 > 1$.The positivity of all the coefficients imply that there are no positive real roots for polynomial (22) satisfying the required conditions.

The necessary and sufficient condition for local asymptotic stability then follows from the **Routh-Hurwitz** conditions applied to polynomial (22).The straightforward but rather lengthy calculation are omitted.■

Preposition 4.2: The **DFE** is globally and asymptotically stable if $\tilde{R}_0 \leq 1$.

Proof: Consider the function $\boldsymbol{\ell}$: **Ω x [0,∞)**→ ℝ defined by

$$\ell = \frac{\lambda}{\lambda+v}\ (\mathrm{R} + \mathrm{w}) + \frac{v}{v+\lambda}\ (1\text{-u}) + \frac{a}{a+e}\,\mathrm{z} + \frac{e}{a+e}\,(1\text{-x}) \qquad \textbf{(24)}$$

Clearly ℓ>0, $\forall(u,w,R,x,z)\epsilon\Omega\backslash\{E_0\}$ where Ω is the region defined in (11) and $\{E_0\}$ is the singleton

{(1, 0, 0, 1, 0, 1)} Ω. Some calculation show that

$$\frac{d\ell}{dr} = \left(\frac{eb}{a+e} - (r+\lambda)\right)w + \left(\frac{ec}{a+e} - (\beta+\lambda)\right)R$$

$$+ \left(\frac{\xi v}{\lambda+v} - a\right)z - \gamma Ru - \frac{\gamma\lambda}{\lambda+v}ev - \left(\frac{\xi v}{\lambda+v} + \frac{ec}{a+e}\right)Rz$$

$$- \left(\frac{\xi v}{\lambda+v} + \frac{eb}{a+e}\right)wz - \frac{\xi v}{\lambda+v}vz - \left(\frac{eb}{a+e}w + \frac{ec}{a+e}R\right)y, \qquad \textbf{(25)}$$

From which we conclude that the original derivatives of ℓ in $\epsilon\Omega\backslash\{E_0\}$ is no positive whenever

$b \leq \frac{(r+\lambda)(a+e)}{e}, c \leq \frac{(\beta+\lambda)(a+e)}{e}\ \xi \leq \frac{a(\lambda+v)}{e}$ **(26)**

We verify that inequalities (26) put together imply that$\tilde{R}_0 \leq 1$. ℓ is a **Liapaunov function** for the system. The **Lapianov Lasalle theorem** [36] assumes as that all paths in $\epsilon\Omega\backslash\{E_0\}$ approach to the largest positively invariant subset $\widetilde{\Omega} \subset \Omega$. Where in

$$\frac{d\ell}{dr} = 0$$

$\widetilde{\boldsymbol{\Omega}}$ is the set $\{(w = o, R = 0, z = 0)\}$. Hence, $(w, R, z) \rightarrow (0,0,0)$ as $r \rightarrow \infty$.Hence on the u- and x-axes, we have the $\dot{u}=\lambda(1-u)$ and $\dot{x} = a(1-x)$, where the dot represents differentiation with respect to r. These shows that $\big(u(r), x(r)\big) \rightarrow$(1, 1) as r$\rightarrow \infty$. Hence E_0 is the only omega limit point of system (9) on the boundary of omega. And therefore cannot be the omega limit point of any other orbit starting in the interior of omega. ■

Generally, Preposition 4.1 and 4.2 state that there are two possible realistic equilibrium points:

One where the disease has died out proportionally,

If $\tilde{R}_0 > 1$, where there is a unique endemic equilibrium.

$\tilde{R}_0$ is a unique threshold parameter which determines the behavior of the system.

Assuming that the endemic equilibrium's stability result is also global, and that at least one infectious mosquito (or human) exists at the beginning, we expect the disease to spread proportionately if $\tilde{R}_o > 1$, and to tend proportionately to the unique equilibrium if $\tilde{R}_o < 1$, thereby establishing itself in the community.

CHAPTER FIVE

5.1 STIMULATION

From the model formulated in terms of proportions above, we analyzed as showed the endemic equilibrium when it exists and locally asymptotical stable. In this unit we explain numerically formulated model in terms of proportions and the other, if $\tilde{R} > 1$ whenever the initial conditions are prescribed in the region Ω defined by [11]. To do this, a plan was written in **Fortran** to integrate equations [9], [10] and the result was generally verified using a clear result from a number of tries.

To examine the behavior of the system and to explain the globally stability of the endemic equilibrium, we choose numbers bearing in the non-dimensionalisation [8]. For a given numbers of sets the value ξ, depends on the values of the corresponding nonzero steady state values of N_v^* and N_h^*.

We note from (10) $(N_v^*, N_h^*) = (1,1)$, $\gamma = 0$

We choose $\boldsymbol{\lambda} = 0.00185$ which corresponding to approximately a per capita human birth rate of 28 births per thousand per year, $\boldsymbol{\beta} = 0.35$ which corresponding to a period of immunity 69 days as β increases, the period of immunity is reduced as β increases to infinity, the standard **SIES** model formed in epidemiology. We get results for $\gamma = 0.001$ which corresponding to a disease-induced death rate of approximately 1.53%. We set $\upsilon = 2.1$ which corresponding to an intrinsic incubation period of days. By assuming small number of humans recover from infectious without getting immunity and by setting $r = 0.3$ and $a = 0.4$.These corresponding to an infectious range of periods between 70 and 110 days for individuals for all classes. From the given approximation in [17, 18, 20] we choose b = 10.0 this value gives the transmission rate from human to vector about 0.4168. Even though we assume that immune human individuals perhaps still be infectious ,we assume that the infectiousness of members from this category is small compared to that of classes in the infectious compartment and arbitrary set c = b/10. Now we choose e =2.5 which corresponds to an extrinsic incubation period within 10 days. Here the assumed value of mosquito life span is about twenty days. The value μ_{2h}, μ_{2v} and μ_{ch} etc. are lumped into the constants ξ which again holds the ratio the vector and mosquito populations at the equilibrium. When the time t =0, then we have the initial conditions in the given proportions:

$\mu(0) = 0.85, w(0) = 0.05\ R(0) = 0.04\ , x(0) = 0.99\ z(0) = 0.0001.$

The plan was sets for different initial conditions and the qualitative form of the final finite amplitude steady-state solutions are similar. The simplest kind of initial data which considered all population to be essentially susceptible and gives one infectious mosquito or humans at a given time t = 0 was again tested or experimented. We have qualitatively the same result as the other cases. But, the general form of the solution is dictated by the way in which the quantity $\xi(N_h, N_v) = \xi(N^*, 1)$ and the relative growth of the human and mosquito populations. We put the general behavior of the given model in the figure below. The plotting form is the relative of the proportions to the total populations. The endemic steady state solution is unique, global and asymptotically stable.

Plotted on the same graph as the overall population to display the relative sizes of the proportions, Figures 1 and 2 depict, respectively, the initial and long-term behavior of the proportions in the human population. Conversely, Figure 3 depicts how the system behaves at lower values.

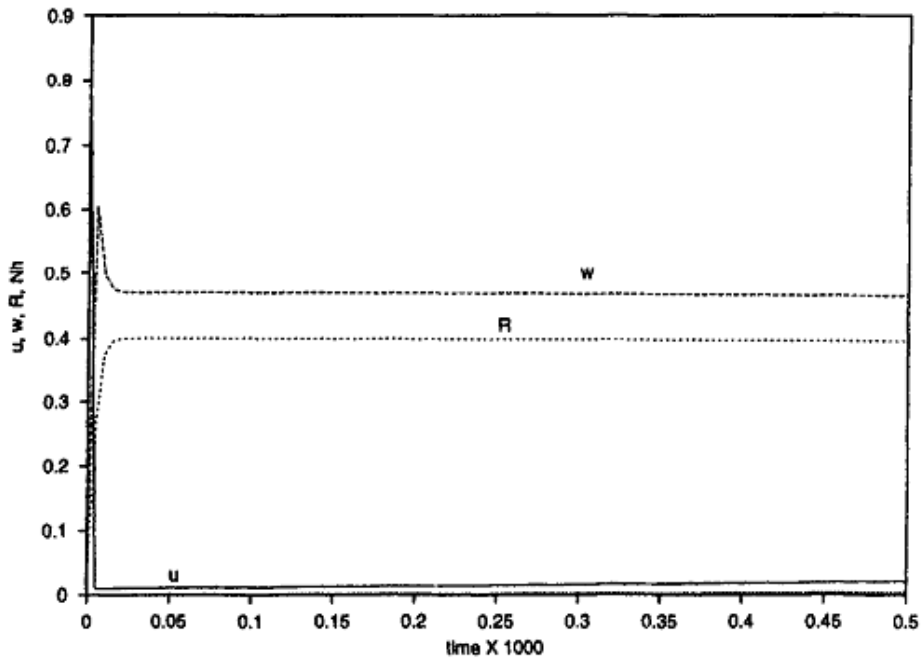

Figure 1**: Initial Behavior of Proportions and Total Human Population over Time**

Figure 1. Initial behavior of the proportions u, w, R, and the total human population N_h with time. The parameters are γ = 0.001, ξ= 4.066, α = 0.3, $\mathscr{r}$ = 0.2 β =0.35, ,λ = 0.00184, v = 2.0, b = 10, c = 1.0, λ-ϵ = 0.005, α = 1.002, e = 2.4. For these, R_o =

61.722. As time increases from zero, the proportion of susceptible drops while those of infectious and removed classes' increases. The proportion w peaks and drops again capturing the typical behavior of infectious individuals during an epidemic.

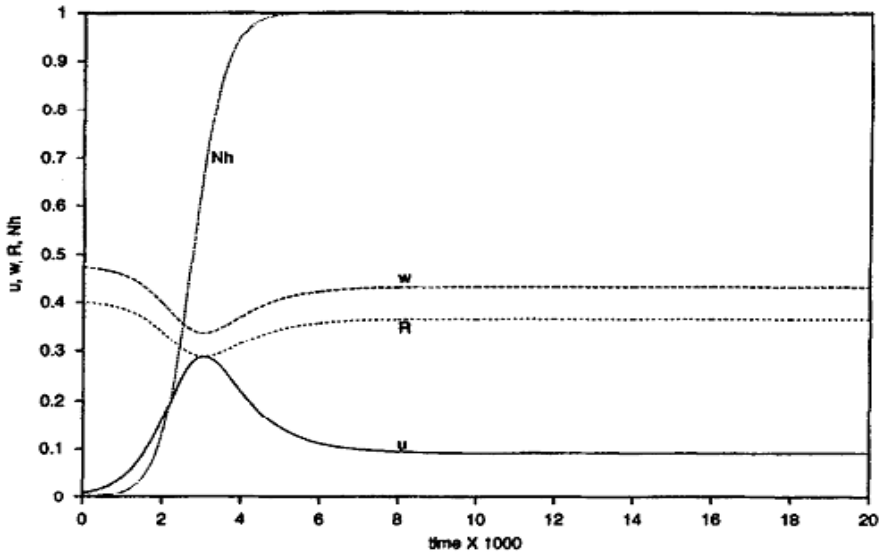

Figure 2: Long-Term Behavior of Proportions

Figure 2. Long-term behavior of the solutions shown in Figure 1. As more and more individuals lose their immunity and join the susceptible, the proportion of susceptible rises, after the initial drop, and then equilibrates at a final finite amplitude Steady state. There is a depression in the reservoir of infections as the proportions of susceptible peaks to its maximum.

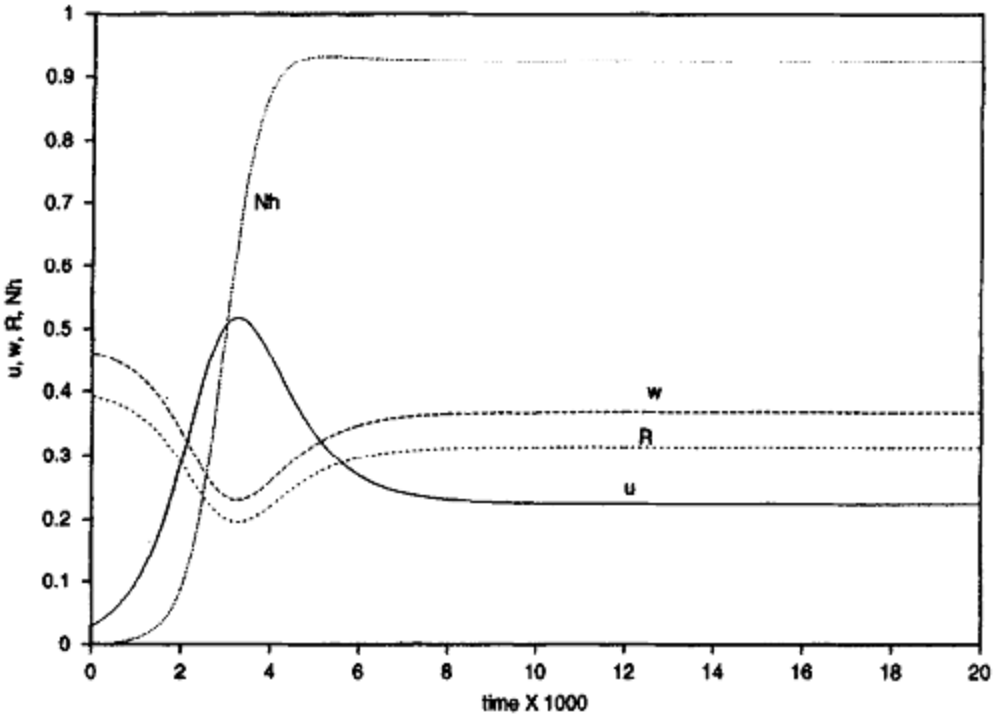

Figure 3: Behavior for Smaller Values of R0R_0R0

Figure 3. Diagram showing the long-term behavior of the proportions in the human Populations. ξ = 1.3554 and all other parameters are as in Figure 1. R_O, = 20.57. Observe that the proportions in the reservoir of infections are smaller in this case than in those of Figures 1 and 2. A large R_O as is the case in Figures 1 and 2, implies that more individuals end up being infected. Because of disease related deaths, the total human population does not reach its carrying capacity, here normalized to one of ξ.

Figure 4 show that the long-term proportion in the vector population. The parameters as in figure 1 .The initial conditions are explained in the text. With this parameter regime more than 50% of the mosquitoes are infectious indicating the endemicity and prevalence of infection.

Figure 2. The solutions' long-term behavior as displayed in Figure 1. Following the initial decline, the fraction of susceptible people increases as more and more people lose their immunity and become susceptible, eventually equilibrating at a final finite amplitude. Stable condition. A decline

has been observed in the reservoir of infections, with the percentage of vulnerable reaches its highest point.

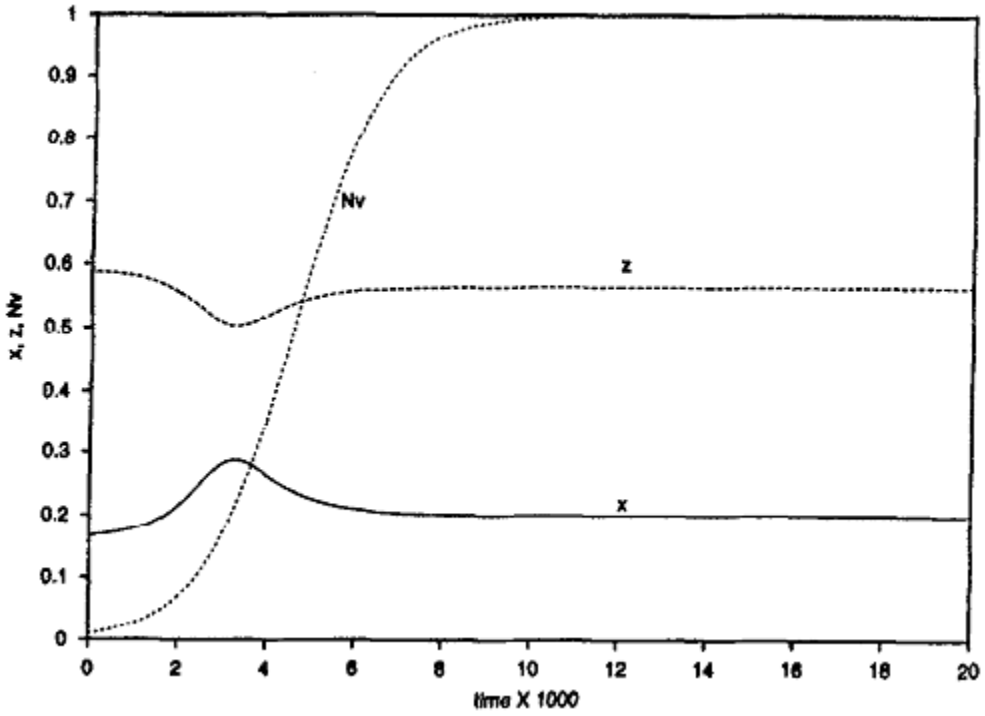

Figure 4: Long-Term Behavior in Mosquito Population

Figure 4: Diagram showing the long-firm proportions of the vector population. Figure 1's parameters are used. Under these parameters, about 50% of the mosquito population is infected, indicating the prevalence and endemicity of infection.

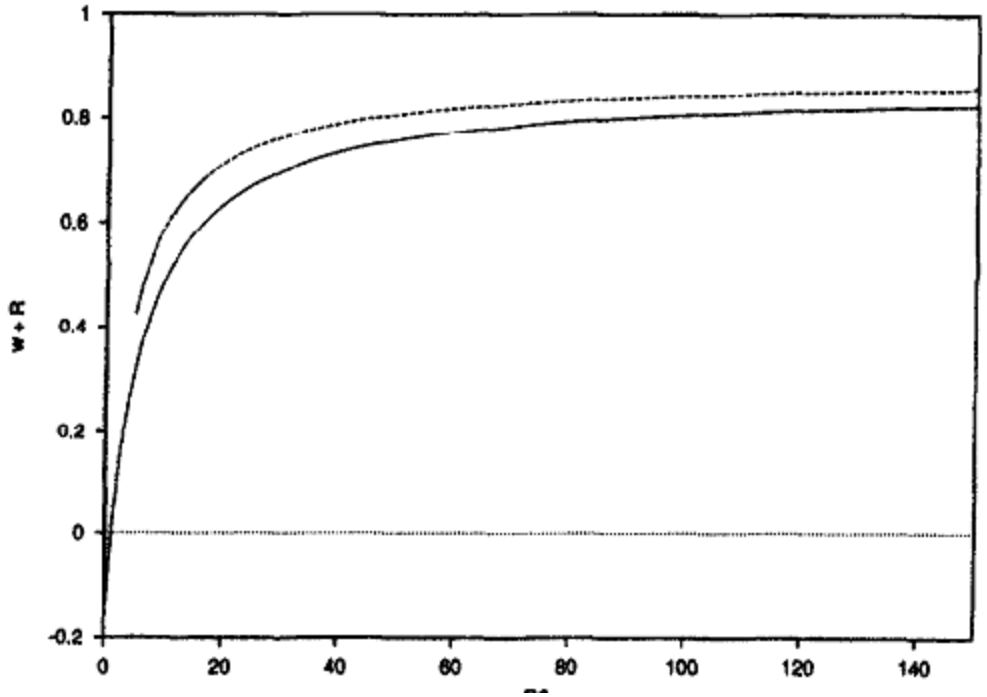

Figure 5: Reservoir of Infection as a Function of R0R_0R0

According to our model, even significant changes in $\tilde{R}_o$ which are frequently brought about by widespread insecticide spraying to lower mosquito populations—will only have a negligible impact on prevalence rates if $\tilde{R}_o$ is more than 50. This behavior has broad implications for the fight against endemic malaria since it indicates that significant decreases in mosquito populations won't have much of an impact if the initial contact rate remains significantly higher than its crucial value of one. This is particularly true if these mosquitoes are allowed time to heal.

CHAPTER SIX

6. CONCLUSION AND REMARKS

The model developed in this paper was SIERS model, which gives the dynamics and transmission of the endemic malaria to study other vector transmitted diseases. We began off briefly by revising available literature on the last work on this area.

The mathematical model was established for malaria in particular and vector borne disease in general. The unrealistic approximation of a constant population size is often made. Even though, our first target had been to study malaria transmission, and endemic malaria dynamics our model has implications to other infectious disease of humans like dengue fever, yellow fever and sleeping sleekness.

We reformulated the differential equation model in terms of proportion of susceptible, incubating, infectious and individuals in both the vector and human populations, and this model showed unique solutions. After that we explained the existence of realistic equilibria. The outcome of our model is fit to the models previously analyzed models. The threshold parameter $\tilde{R}_0$ and the disease can prevail if and only if $\tilde{R}_0 > 1$.

If $\tilde{R}_0 < 1$, then the disease- free equilibrium always exists and is locally stable. Again

If the $\tilde{R}_0 > 1$, then the disease-free equilibrium always unstable. And the disease – free equilibrium is globally stable when $\tilde{R}_0 < 1$.Hence, $\tilde{R}_0 > 1$

The existence of an endemic equilibrium showed, which implies that locally and asymptotically stable. The confirmation of numerical simulations shows this outcome.

Our model's outputs conformed to the trend of earlier models that were examined. The sickness can only continue if and only if the threshold parameter, &, is greater than one. There is always a disease-free equilibrium, which is locally stable while $\tilde{R}_0$ is less than 1 .

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Appendix

Figure 1: Initial Behavior of Proportions and Total Human Population over Time

```
matlab
Copy code
% Parameters
X = 0.00184;
p = 0.35;
r = 0.2;
a = 0.3;
g = 2.0;
c = 4.066;
s = 1.062;
e = 2.4;
k = 10.0;

% Initial conditions
u = 0.85; % Susceptible
w = 0.05; % Incubating
R = 0.04; % Immune
z = 0.999999; % Infectious mosquitoes
z_mos = 0.000001; % Infectious humans

% Time span
tspan = [0 0.5]; % Adjust as per requirement
[t, y] = ode45(@(t,y) model_equations(t, y, X, p, r, a, g, c, s,
e, k), tspan, [u w R z z_mos]);

% Plotting
figure;
```

```
plot(t, y(:, 1), 'b', t, y(:, 2), 'g', t, y(:, 3), 'r', t, y(:,
4), 'k', t, y(:, 5), 'm');
legend('Susceptible', 'Incubating', 'Immune', 'Infectious
mosquitoes', 'Infectious humans');
xlabel('Time');
ylabel('Proportion');
title('Initial Behavior of Proportions Over Time');
```

Figure 2: **Long-Term Behavior of Proportions**

```
matlab
Copy code
% Time span (longer duration for long-term behavior)
tspan = [0 16]; % Adjust as per requirement
[t, y] = ode45(@(t,y) model_equations(t, y, X, p, r, a, g, c, s,
e, k), tspan, [u w R z z_mos]);

% Plotting
figure;
plot(t, y(:, 1), 'b', t, y(:, 2), 'g', t, y(:, 3), 'r', t, y(:,
4), 'k', t, y(:, 5), 'm');
legend('Susceptible', 'Incubating', 'Immune', 'Infectious
mosquitoes', 'Infectious humans');
xlabel('Time');
ylabel('Proportion');
title('Long-Term Behavior of Proportions Over Time');
```

Figure 3: **Long term of Behavior of the proportion in the human populations**

```
matlab
Copy code
% Adjust parameters to reflect smaller R0
```

```
R0 = 20.57; % Example value
% You may need to adjust `s`, `e`, etc., based on your
calculations.

% Time span
tspan = [0 16];
[t, y] = ode45(@(t,y) model_equations(t, y, X, p, r, a, g, c, s,
e, k), tspan, [u w R z z_mos]);

% Plotting
figure;
plot(t, y(:, 1), 'b', t, y(:, 2), 'g', t, y(:, 3), 'r', t, y(:,
4), 'k', t, y(:, 5), 'm');
legend('Susceptible', 'Incubating', 'Immune', 'Infectious
mosquitoes', 'Infectious humans');
xlabel('Time');
ylabel('Proportion');
title('Behavior for Smaller R0');
```

Figure 4: **Long-Term Behavior in Mosquito Population**

```
matlab
Copy code
% Time span
tspan = [0 20];
[t, y] = ode45(@(t,y) model_equations(t, y, X, p, r, a, g, c, s,
e, k), tspan, [u w R z z_mos]);

% Plotting
figure;
plot(t, y(:, 4), 'k', t, y(:, 5), 'm');
legend('Infectious mosquitoes', 'Infectious humans');
```

```
xlabel('Time');
ylabel('Proportion');
title('Long-Term Behavior in Mosquito Population');
```

Figure 5: Reservoir of Infection as a Function in the human population

```
matlab
Copy code
% Range of R0 values
R0_values = linspace(1, 100, 100); % Adjust range as necessary
reservoir = zeros(size(R0_values));

for i = 1:length(R0_values)
    R0 = R0_values(i);
    % Recalculate the system's steady state based on the new R0
    [t, y] = ode45(@(t,y) model_equations(t, y, X, p, r, a, g,
c, s, e, k), tspan, [u w R z z_mos]);
    reservoir(i) = y(end, 2) + y(end, 3); % w + R
end

% Plotting
figure;
plot(R0_values, reservoir, 'b');
xlabel('R0');
ylabel('w + R');
title('Reservoir of Infection as a Function of R0');
```