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# A Mathematical Measure to Fight Against Malaria and Exterminate Anopheles Mosquitoes

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**Abstract:** Modelling the effects of three natural predators on the aquatic and adult anopheles' mosquitoes in the control of malaria transmission was aimed at eradicating anopheles' larva, pupa and adult anopheles' mosquito by introduction of natural predators "copepods, tadpoles and purple martins" (organism that eat up mosquito at larva, pupa, and adult stages), so that there should not be anopheles' adult mosquito for malaria transmission in our society. This new proposed model is a control flow diagram of predator-prey interaction model in mosquito life-cycle that considers an open population of mosquito and predators. The population is sub-divided based on mosquito life-cycle and natural predators. Under a mosquito life-cycle, the population is divided into four compartments, Egg compartment E(t), Larva compartment L(t), Pupa compartment P(t), and Adult compartment A(t), and natural predators, it is divided into three compartments, namely; Copepods $C_P(t)$, Tadpole $T_P(t)$ and Purple martins $P_M(t)$. These models provide understanding for control of malaria in our environments, especially when the models are based on the ecology of the vector population and sound understanding of variables and parameters relevant for transmission. The model equations were derived using the model variables and parameters. The stability analysis of the free equilibrium states were analyzed using equilibrium point, elimination, substitution methods, idea of Beltrami's and Diekmann's conditions. From the stability analysis of steady state, we observed that the model free equilibrium state is stable, this implies that the equilibrium point or steady state is stable and the stability of the model(3.13.1) – (3.13.8) means, there will not be anopheles adult mosquito in our society for malaria transmission and from the idea of Beltrami's and Diekmann's conditions we observed that the Determinant of the Jacobian matrix is greater than zero$(\mathrm{Det}\{j\} > 0)$,Trace of the Jacobian matrix is less than zero$(\mathrm{Tr}\{j\} < 0)$ and the basic reproduction number is less than one ( $R_0 < 1$) which implies that the model disease free equilibrium state is stable. Hence the number of larva that transform to pupa is almost zero and the number of pupa that develop to adult is minimal and number of adult that escape to vector stage are inconsequential, that means the life-cycle could be broken at the larva, pupa, and adult stages with the introduction of natural predators, with the natural implication there will not be anopheles adult mosquito for malaria transmission and we also use maple for symbolical and numerical solution and presented the results graphically. The contribution of this research work to knowledge is to bring out the control flow diagram of prey-predator interaction, mathematical models, Identify the ability to control and eradicate malaria through stability analysis and numerical experiments showing the effect of the introduction of three natural predators on the larva, pupa and adult stages of the adult Anopheles mosquito( biological inoffensive method) which will contribute to the eradication of adult anopheles' mosquito, which will also lead to the elimination of malaria in our society.

## I. Introduction

### 1. 1 Background to the Study

The *Anopheles* vector system in Nigeria and in sub-Saharan Africa is probably the strongest that exists for human *Plasmodium*. Contact with human vectors, particularly An. *gambiae* s.l., shows remarkable stability and flexibility, resulting in extremely high vaccination rates under different seasonal and geographic ecological conditions (Mokuolu *et al.,* 2018). Malaria remains a leading cause of death and disease in most tropical regions of the world, where it is endemic in 106 countries. In 2010, out of a total of 216 million cases of malaria, around 81% occurred in Africa and 13% in Southeast Asia1. The majority (91%) of the estimated 665,000 malaria deaths occur in Africa and primarily affect children under the age of five (86%). In America in 2010 there were more than 670,000 confirmed cases of malaria with 133 deaths from malaria. The transmission is active in 21 countries and puts approximately 20% of the US population at risk. Malaria severely limits economic development and is a cause of poverty in most countries where the disease is endemic. Malaria remains an ongoing problem in sub-Saharan Africa, and while great strides have been made over the past 15 years, millions of people are still at risk of contracting the parasite (Patouillard *et al.,* 2017).

Africa offers a stable and ecologically diverse ecosystem and hosts the world's highest vectors of malaria (Bernard *et al.,* 2020) and is expected to remain so in the future. Climate change (Adigun *et al.,* 2015). The main vectors of *Anopheles* malaria in sub-Saharan Africa are *Anopheles funestus* s.s. and three members of the *Anopheles gambiae* complex: *An. Gambiae* s.s., *Anopheles coluzzii* and

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*Anopheles arabiensis* (Molinaro *et al.*, 2015), which play a role in the transmission of malaria in their distribution area, e.g. the groups *Anopheles moucheti* and *Anopheles* nili (Rajeswari, 2017) and another of secondary or random vectors (Antonio-nkondjio *et al.*, 2006). Considering that the genus *Anopheles* includes more than 500 species worldwide, of which only a few are considered important species for the transmission of malaria (Garcia Guerra *et al.*, 2014).

The morphological identification of species is crucial for allocating scarce resources solely to the fight against malaria vectors. Species groups and species complexes are common within the genus *Anopheles* (Harbach & Besansky, 2014) and this complicates vector control because not all species in a complex share similar behaviors or similar roles in transmission malaria disease (Velickovic & Leicht, 2002).

Mosquitoes of the family *Culicidae* are considered a nuisance and a major public health problem because their females feed on human blood and therefore transmit extremely harmful diseases such as malaria, yellow fever and *filariasis* (Tsoka-Gwegweni & Okafor, 2014). They are estimated to transmit diseases to more than 700 million people each year and are responsible for the death of around 1 in 17 people ("Malaria Policy Advisory Committee to the WHO: Conclusions and Recommendations of Eighth Biannual Meeting (September 2015)," 2016). Effective transmission of mosquito-borne diseases requires successful contact between female mosquitoes and their hosts (Vanelle *et al.*, 2012a). Among *Anopheles*, members of the genus *Anopheles* are best known for their role in the global transmission of malaria and *filariasis* ("Malaria Policy Advisory Committee to the WHO: Conclusions and Recommendations of Fifth Biannual Meeting (March 2014)," 2014). Among these diseases, malaria, caused by the *Plasmodium* parasite, is one of the deadliest diseases in the world ("Malaria Policy Advisory Committee to the WHO: Conclusions and Recommendations of Sixth Biannual Meeting (September 2014)," 2015). ("Malaria Vaccine: WHO Position Paper, January 2016 – Recommendations," 2018) reported approximately 207 million cases of malaria in 2012, of which 200 million (80.0%) were on the affected continent. Patterns of disease spread, transmission, and intensity depend on the degree of urbanization and distance from vector breeding sites (MCNAMARA, 2005). The endemicity of malaria in each region is determined, among other things, by native *Anopheles* mosquitoes, their abundance, diet, resting behavior and *Plasmodium* infectivity (Atta & Reeder, 2014). The Federal Ministry of Health in Abuja reported that at least 50.0% of Nigerians suffer from some form of malaria, making it the most significant health problem in Nigeria (UM & AN, 2016). The high transmission rate and prevalence of malaria is the result of the various mosquito breeding sites, including convenient water reservoirs such as cans, old tires, tree holes, cisterns, open pools, drains, streams and ponds (McKenzie, 2014). Part of the fight is the official observance of April 25 each year, beginning in 2008, as World Malaria Day (CDC Weekly, 2020). Arms-only people face a variety of barriers when assessing malaria prevention, particularly with respect to knowledge of vector biology and ecology (Ingstad *et al.*, 2012).

Malaria vector mapping is important for malaria control. Indeed, the species composition and distribution, as well as other biological parameters, of mosquitoes in the ecological zones of Nigeria and in most malaria-endemic areas are in decline due to difficulties in the morphological identification of some complex species, knowledge of which is known for the vestal. Torah sea creatures, and around which they die to combat the prevalence of the disease in endemics (O.C *et al.*, 2017). Mosquitoes are responsible for the spread and transmission of various dangerous diseases such as malaria and *lymphatic filariasis*. It is known to infect over 700 million people and cause 1 million deaths each year, especially in developing regions of the world, including sub-Saharan Africa (Kumari, 2022). Despite years of control efforts, malaria remains a major public health threat in parts of sub-Saharan Africa, including Nigeria. About 97% of Nigeria's population is at risk of malaria, with malaria accounting for 60% of hospital outpatient visits and 30% of hospital admissions for children under five and pregnant women (M Dokunmu, 2019). Entomological studies that focus on the diversity, density, behavioral patterns, and temporal variation of *Anopheles* species have long been useful for identifying and monitoring malaria vectors (Moreno *et al.*, 2010). A combination of factors that determine a vector's ability to transmit malaria include: abundance, *anthropophilia*, *zoophilia*, susceptibility to infection by the malaria parasite, infection rate, and low male longevity (Speybroeck, 2011).

The vectoring capacity of a mosquito population largely determines the intensity of transmission of vector-borne diseases. Vector competence is also a crucial parameter for the pathogen to be transmitted. In human malaria, the vector systems are limited in number. Only female *Anopheles* can transmit *Plasmodium* to humans, and out of more than 450 known Anopheles species, 60 are considered true vectors in nature (Tainchum *et al.*, 2015). Vector capacity and competence also exhibit quantitative characteristics in the sense that some species play a major role in malaria transmission and others a minor role. Also at the species level, certain populations or mosquitoes may have different effects on transmission (Dev & Manguin, 2021). Research to understand the genetic determinants of skills and competencies has benefited greatly from the availability of the full genome sequence of *Anopheles gambiae* (Kuntworbe *et al.*, 2012), with identification of candidate genes ongoing. However, the various aspects of vectoring ability and competence have not been studied consistently, and some have been largely overlooked. There are 465 officially recognized species and over 50 unnamed members of species complexes. Approximately 70 of these species have the ability to transmit human malaria parasites (Townson, 2009) and 41 species are considered to be the dominant vector species complex capable of transmitting malaria at a level of public health concern. Knowledge of the main vectors of malaria and their bionomics in Africa remains a

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problem (Weiss *et al.,* 2014). Overall, medical reports have shown that mosquito-borne diseases are responsible for a significant impact on human morbidity and mortality worldwide (Mugoyela *et al.,* 2002).

The global burden is 207 million malaria cases per year with 627 000 deaths ("Malaria Policy Advisory Committee to the WHO: Conclusions and Recommendations of September 2012 Meeting," 2012), with sub-Saharan Africa being the most affected region. According to the latest WHO Malaria Report (2014), there were about 197 million malaria cases and about 584 000 deaths worldwide in 2014, mainly among African children. Au Nigeria, malaria remains a major public health concern with approximately two-thirds of the population living in malaria-prone areas (Scott, 2015). Among the malaria vectors from Nigeria, *Anopheles gambiae-*, *An. funestus-* and *An. arabiensis* complex to be the transmission of malaria in the country, although there are other non- and incidental vectors that are now blamed for the transmission of malaria (Awolola *et al.,* 2002). Two million deaths are attributed to the malaria pathogen in sub-Saharan Africa in general and in Nigeria in particular, one third of whom are children. Malaria is transmitted by the female *Anopheles* mosquito, which feeds on human blood. Much work has been done to genetically modify mosquitoes in the laboratory to prevent or block parasite transmission, which makes mosquitoes stubborn. It does this by inserting genes in the appropriate place to create a stable germ line. Advances in this area are relatively recent. (Wiese, 2012) presents an overview of transgenic mosquitoes to suppress malaria transmission. In 1967, however, the World Health Organization (WHO) recognized that global eradication of malaria was impossible for various reasons, and control shifted to combating killer diseases. Since the idea of eradicating mosquitoes was unrealistic, efforts focused on reducing and controlling their population below the threshold that would cause disease.

Additionally, for the first time in Africa, an entomological study went beyond the conventional practice of determining the parity and survival rates of adult *Anopheles* mosquitoes collected in the field, but also linked these variables to the duration of *Plasmodium sporogony* and estimated infectious life expectancy. Thus, from January 2005 to December 2006, blood-sucking female mosquitoes were collected in Ilorin, Nigeria. The *Anopheles gambiae* population in Ilorin is dominated by older mosquitoes with a high survival rate, indicating high vector potential for the species in this area. This information on the survival rate of *Anopheles gambiae* in relation to malaria transmission would encourage the development of a more targeted and informed vector control intervention. In 1963, the WHO team also conducted a large field trial of dichlovs in Kankiya district, Katsina province, northern Nigeria. (Zhang *et al.,* 2012) conducted the study on the conditions of malaria transmission. Malaria transmission has been shown to occur mainly or exponentially from August to December, but continues at very low levels in the remaining months, even when *Anopheles* densities are as low as 0.02 per hut. Mosquito is a common flying insect found all over the world. There are about 3,500 species of mosquitoes classified into 41 general ones. For the purposes of this work, the research is limited to the female genius of the *Anopheles* mosquito: of the approximately 430 species of *Anopheles*, only 30 to 40 transmit malaria in nature. Like all mosquitoes, *Anopheles* goes through four stages in their life cycle. Egg, larva, pupa and adult. The first three stages are aquatic and last from 5 to 14 days, depending on species and ambient temperature. In the terminal adult stage, the female *Anopheles* mosquito acts as a vector of malaria. The adult female can live up to a month but will probably not live more than 1-2 weeks in the wild. Work by (Mandal *et al.,* 2011), (Emmanuel & Omini, 2020) and reviews by Hassel and (Rosanda, 2012), Murdoch and (Tobin-West & Briggs, 2015) have expanded the topic of host-predator-parasitoid patterns and interactions. Therefore, this work focuses on modeling the effects of three natural predators on the aquatic and adult stages of *Anopheles* mosquitoes in the control of malaria transmission.

### 1.2 Motivation of the Study

Malaria remains a leading cause of death, with more than a million deaths per year in sub-Saharan Africa in general and Nigeria in particular. In Nigeria, malaria is responsible for approximately 30,000 deaths each year and accounts for 40% of public health expenditure. It is estimated that the cost of treating and preventing malaria in Nigeria is over a billion a year. However, with project work to model the effects of three natural predators on the aquatic and adult *anopheline* mosquitoes in controlling malaria transmission, the rate of malaria transmission will reduce or eliminate the parasite risk from malaria in our societies. Therefore, funds being spent on the malaria burden by the Nigerian government and the programmers of the WHO's Roll Back Malaria program are being concentrated elsewhere. These are a source of motivation for this research work.

### 1.3 Statement of the Research Problem

Mosquito control remains an important component of human and animal diseases. Vector-borne diseases are among the leading causes of morbidity and death, particularly in tropical and subtropical countries; Vector control through the use of insecticides plays a key role in the prevention and control of infectious diseases. *Anopheles* mosquitoes are the main vectors responsible for transmission of malaria in tropical and subtropical regions of the world including Nigeria and Nasarawa. There is currently little or no empirical data on the population dynamics of the *Anopheles* mosquito vector in the ecological environments of Nasarawa State. Malaria is characterized by its biological diversity, and this diversity is mainly due to anopheline mosquitoes, which are involved in transmission through their spread, behavior and vectoring ability. In Nigeria, because of their behavior, mosquitoes are considered public health

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enemies due to nuisance biting and noise pollution, insomnia, allergic reactions and disease transmission. They transmit human diseases such as malaria, yellow fever, dengue fever, hemorrhagic fever, *filariasis* and *encephalitis*. However, this has been limited by the development and spread of resistance and limited knowledge of mosquito biology. In addition, significant changes in resistance patterns have been observed in West Africa over the past 10 years. Currently, there is little or no empirical data on the population, dynamics, species composition, vector capacity, prevalence, and spread of malaria in Nasarawa State as a vector of *Anopheles* mosquitoes that would allow an assessment of the mosquito and malaria situation and malaria and malaria strategy. With this in mind, the work attempts to model the impact of three natural enemies on the aquatic stages and adults of Anopheles mosquitoes in controlling malaria transmission.

### 1.4 Aim and Objectives of the Study

The research aims to model the effects of three natural predators on the aquatic and adult stages of the *Anopheles* mosquito in the control of malaria transmission. In addition, the following specific objectives of the study are listed.

i. Introducing natural predators (copepods, tadpoles and purple martins) swallows into the life cycle of the *Anopheles* mosquito in the larva, pupa and adult stages
ii. Derivation of model equations for perturbing the life cycle of the *Anopheles* mosquito in the larva, pupa and adult stages.
iii. Determination of the mosquito-free steady-state stability of the model using the equilibrium point, Beltrami's and Diekman's conditions.
iv. Analyze, solve and run numerical simulations with graphical representation of results.

### 1.5 Significant of the Study

The study is important because it contributes to the latest research on the mathematical model of *Anopheles* mosquito eradication and malaria elimination. The study will help interested governmental and non-governmental organizations understand the impact of prevention strategies on specific malaria transmission routes. It will help scientists identify and implement three different species of *Anopheles* mosquitoes using sampling techniques to reduce malaria transmission.

### 1.6 Scope and Limitations of the Study

The model formulation models the effects of three natural enemies on the aquatic and adult stages of *Anopheles* mosquitoes in controlling malaria transmission. We use ordinary differential equations to model growth at each stage, from egg to adult. The work investigates the appropriate environmental conditions for different behaviors of the *Anopheles* mosquito, such as resting, swarming, egg-laying, biting and feeding. The data analysis methods used include the linear stability method and numerical experiments (ODE solver).

### 1.7 Definition of Operational Terms

**1.7.1 Natural predators:** The term "natural predators" is used for organisms that kill or injure other animals. For example, copepods, tadpoles and swallows are natural enemies of mosquitoes, and predators or parasites are natural enemies of insect pests. Spiders are natural enemies of stem borers. Moreover, pathogens are natural enemies.

**1.7.2 Copepods:** Copepods are tiny crustaceans (shrimp, crabs, lobsters and related species) that are widely distributed in both freshwater and saltwater habitats. They are voracious or zealous predators used to control mosquito production in water holding areas. Knowing where mosquitoes breed is very important for effective control of copepod mosquitoes.

**1.7.3 Tadpoles:** The aquatic larvae of frogs, toads, etc., which develop from a form of limbless tail

With external gills in a form with internal gills, limbs and a reduced tail. They are voracious or zealous predators used to control mosquito production in water holding areas. Knowing where mosquitoes breed is very important for effective tadpole control.

**1.7.4 Purple Swallow:** The Purple martins of the order Passerine is the largest swallow in North America. Despite their name, purple martins aren't actually purple. Their dark blue-black feathers have an iridescent sheen caused by the refraction of incident light, giving them a light blue to dark blue or purple appearance. In low light, they may even appear green.

**1.7.5 Adult mosquitoes:** Adult mosquitoes (family *Culicidae*) are slender, agile insects with long legs. An adult mosquito has the following three characteristics: a long proboscis (biting organ) protruding from the head and this proboscis is several times longer than the head itself.

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**1.7.6. Egg:** Mosquitoes of the genus *Culex* lay their eggs in the form of egg rafts that float on standing or stagnant bodies of water. An egg raft can hold 100 to 400 eggs. The eggs pass through the larval and pupal stages and feed on microorganisms before developing into flying midges.

**1.7.7 Larva:** Mosquito larvae have a well-developed head with mouth brushes for feeding, a large thorax and a segmented abdomen, they have no legs. Unlike other mosquitoes, Anopheles larvae have no respiratory mucosa and must therefore position themselves so that their bodies are parallel to the surface of the water. The larvae feed on microorganisms and organic matter in the water. The larvae reproduce through spiracles located on the eighth abdominal segment. *Anopheles* mosquito larvae have been found in freshwater or saltwater swamps, mangroves, etc. The larvae develop in four stages. At the end of each instars, the larva molts, i.e. it molts to allow further growth, after which it pupates.

**1.7.8 Pupa:** An insect in the intermediate stage between a larva and an adult in complete metamorphosis during which the insect is in a cocoon or a box, stops feeding and undergoes internal modifications.

**1.7.9 Malaria:** A group of chronic relapsing febrile illnesses in humans caused by the *homospermidine* blood parasite of the genus *Plasmodium*, transmitted by the bite of the *Anopheles* mosquito.

**1.7.10 Anopheles:** Genus of mosquitoes in the family *culicidae*; members are vectors of malaria, *dengue* and *filariasis*.

**1.7.11 Anopheline**: Pertaining to a mosquito of the genus *Anopheles* or a closely related genus.

**1.7.12 Plasmodium**: A genus of *protozoans* in the family *plasmodiidae* in which all the true malarial parasites are placed.

**1.7.13 Parasite**: An organism that lives in or on another organism of different species from which it derives nutrients and shelter.

**1.7.14 Oviposition**: Means of egg deposition especially by insects, fish and other organisms.

**1.7.15 Epidemiology**: The study of the mass aspect of disease.

**1.7.16 Vector**: An organism such as a mosquito or tick that transmits disease-causing microorganisms from an infected person or animal to another.

**1.7.17 Predator**: A carnivorous animal or any other organism that hunts kills and eats other animals in order to survive.

**1.7.18 Prey**: An animal or animals caught, killed, and eaten by another animal as food.

## II. Literature Review

### 2.1 Conceptual Framework

### 2.1.1 Basic Malaria Models

The genesis of modeling in malaria dates back to the introduction of the first model by Ross. Ross introduced the first deterministic differential equation model of malaria by dividing the human population into two compartments, namely: susceptible and infected compartments. The mosquito population also has only two compartments mentioned above, but they do not recover from infection due to their short lifespan. The temporal evolution of the proportion of individuals in infected classes is studied using two differential equations, one for humans and one for mosquitoes. An increase in mosquito mortality and a reduction in mosquito biting rate can reduce $R_0$. Ross' model describes the basic characteristics of malaria transmission and places the weight of transmission on mosquito-specific traits, paving the way for mosquito-based malaria control programs. The malaria parasite spends about 10 days inside a mosquito during its life cycle. Ross's simple model did not take into account this period of parasite latency in mosquitoes and their survival during this time. This led the model to predict a rapid progression of the human epidemic and a higher equilibrium prevalence of infectious mosquitoes.

Macdonald took into account the latency period and introduced the mosquito exposure class (Macdonald, 1965). Therefore, the $R_0$ of the Ross model decreases as the latency increases. In a natural extension of the models of Ross and Macdonald, Anderson and May considered the twenty-one (21) day latency period of the parasite in humans and included the class exposed in the human population in their model (Anderson *et al.,* 1991). This divided the host population as well as the mosquito population into three compartments: susceptible, exposed and infected classes. A comparative study of the Ross (RR), Macdonald (MC) and Anderson-May (AM) models for the prevalence of infected humans and mosquitoes. This shows that the inclusion of mosquito and human parasite latencies not only reduces the long-term prevalence of infected humans and infected mosquitoes (RR is highest and AM is lowest); Rates of progression to these terminally infected populations are also reduced. Even with this minimal complexity, these basic models can provide insight into the effect of different types of interventions on the dynamics of disease transmission. Predicting the Effects of Interventions in Reference Models Parameters of mosquito density, biting rates, and mosquito mortality

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rates are important in regulating the proportion of the human population that falls into the exposed and infected classes. The most important fact for any epidemiologist or public health worker is to have an idea of the relative impact of manipulating these parameters on the intensity of transmission, the measure of which is $R_O$.

### 2.1.2 History of Malaria, *Anopheles* Mosquitoes and Malaria Transmission

Malaria is a widespread and potentially deadly infectious disease in many tropical and subtropical regions. It is caused by the *Plasmodium* parasite, which is transmitted by female *Anopheles* mosquitoes when they bite humans to feed on blood for the development of their eggs. During the blood meal, the mosquito injects *sporozoites* into the bloodstream. Within minutes, the *sporozoites* invade liver cells, where each *sporozoite* develops into a tissue *schizont* containing 10,000 to 30,000 *merozoites*. After 1–2 weeks, the *schizont* ruptures and releases the *merozoites* into the bloodstream, which then invade the red blood cells. The clinical symptoms of malaria are due to the breakdown of red blood cells and the release of parasitic and cellular debris into the bloodstream. Note that human malaria is caused by five different species of *Plasmodium*: *P. falciparum*, *P. malariae*, *P. ovale*, *P. vivax* and *P. knowlesi*. However, P. *falciparum* is more prevalent in Africa and causes the highest mortality rate from the disease (Katiku *et al.*, 1998). The biology of the five *Plasmodium* species is generally similar and consists of two distinct stages: a sexual stage in the mosquito host and an asexual stage in the human host.

The World Health Organization (WHO) estimated that there were 214 million cases of malaria in 2015, resulting in approximately 438,000 deaths ("Malaria Policy Advisory Committee to the WHO: Conclusions and Recommendations of Seventh Biannual Meeting (March 2015)," 2015). In addition, in endemic regions, children under 5, pregnant women and non-immune adults are at increased risk of malaria mortality (Forouzannia & Gumel, 2014). In fact, there are currently over 100 countries at risk of malaria transmission and they are visited by over 125 million international travelers each year. International travelers to countries with persistent local transmission of malaria, coming from countries without transmission, are at high risk of malaria infection and its consequences due to lack of immunity. Migrants from countries with malaria transmission live in malaria-free countries and return to their home country to visit friends and family is at similar risk due to reduced or absent immunity. Despite major eradication efforts, malaria caused by P. *falciparum* remains a significant problem. A feature of *falciparum* malaria that complicates control efforts is clinical immunity: an immune response that develops on contact with parasites and provides protection against clinical symptoms of malaria despite the presence of the parasite (Schofield & Mueller, 2006). This immunity is not complete and one can lose it and become vulnerable after the exposure stops. Those who have acquired immunity can ingest and tolerate malaria parasites without developing clinical symptoms. They can become asymptomatic carriers of parasites and easily transmit parasites to mosquitoes (Keegan & Dushoff, 2013). In order to reduce the spread of infectious diseases, mathematical models have been proposed to study their dynamics (Nakata & Kuniya, 2010). Models can provide estimates of underlying parameters of a real-world problem that are difficult or expensive to obtain through experimentation or otherwise (Da *et al.*, 2014). They can predict whether the associated disease will spread or disappear in the population (Dabitao *et al.*, 2011). It can also assess the impact of a control measure and provide useful public health guidance for further disease eradication efforts. In terms of mathematical modeling of malaria, significant progress has been made in recent years since the first model introduced by Ronald Ross (Ross, 1911). According to Ross, malaria can be eradicated if the mosquito population can be reduced below a certain threshold. A few years later, Macdonald ("Malaria Control," 1957) improved Ross's model. The work has shown that reducing the number of mosquitoes in areas of intense transmission has little impact on the epidemiology of malaria.

## 2.2 Empirical Review of Literature

### 2.2.1 Detecting Malaria: Infection Versus Transmission

Since the 2011 malERA process, research has ranged from illuminating the basic biology of the development of sexual-stage parasites in humans and mosquitoes to evaluating operational approaches targeting infectious individuals in endemic communities. Additionally, a harmonized set of definitions relevant to malaria transmission and elimination has been developed (Gulland, 2016).

Malaria infection and transmission can be detected and measured with a variety of metrics. Their suitability and discriminatory power, however, can vary widely across settings and populations. To reliably confirm clinical malaria, a minimum diagnostic sensitivity of 200 parasites/μL bloods is required (Martelli *et al.*, 2015). Microscopy and some rapid diagnostic tests (RDTs) meet this threshold ("Who Knows Best about Malaria?," 2007). In the absence of fever, some individuals will have *parasitaemia* levels detectable by microscopy and RDTs. These asymptomatic infections are particularly common in areas of high transmission (i.e., above 25 clinical cases per week per 1,000 persons) ("PLOS Biology 2016 Reviewer and Editorial Board Thank You," 2017), where high levels of human immunity allow older individuals to carry relatively large parasite burdens chronically (Chen *et al.*, 2016). Such individuals would be detected within mass screen and treat (MSAT) programmes using currently available diagnostics. However, through the use of molecular amplification methods, it is now clear that many individuals harbour low-density malaria infections beneath the limit of detection of both microscopy and RDTs ("Lee W-C, Malleret B, Lau Y-L, *et al*. Glycophorin c

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(CD236R) Mediates Vivax Malaria Parasite Rosetting to Normocytes. Blood. 2014;123(18):E100-E109.," 2015). Meta-analyses indicate that molecular methods detect up to twice as many *P. falciparum* infections as RDT or microscopy (Et. al., 2021).

Lack of sensitivity of diagnostic detection is more acute for *P. vivax* infections, which circulate at lower parasite densities hampering accurate estimates of true prevalence. There are also other unique challenges presented by *P. vivax* that make characterizing its transmission reservoir problematic (WHO, 2015). *P. vivax* and *P. ovale* have a dormant liver stage, the *hypnozoite*, which is undetectable by currently available diagnostic methods. Periodic reactivation of *hypnozoites* results in repeated blood-stage infection (relapses) occurring weeks, or even years, following the initial infection. As control efforts reduce the incidence of *P. falciparum* cases, *P. vivax* cases can remain relatively stable and become a greater proportion of malaria cases overall (Anvikar *et al.,* 2010). However, several barriers to mass drug administration (MDA) for *P. vivax* exist. The 8-*aminoquinolines primaquine* and *tafenoquine* are the only known *anti-hypnozoite* drugs. Both drugs are contraindicated in pregnancy and individuals with glucose-6-phosphate *dehydrogenase* deficiency ("MalERA: An Updated Research Agenda for Diagnostics, Drugs, Vaccines, and Vector Control in Malaria Elimination and Eradication," 2017).

Diagnosis and treatment of clinical malaria is vital for disease control, particularly if this can be rapidly implemented to reduce the likelihood of *gametocyte* production. There is also a good public health rationale for identifying and treating 'asymptomatic' malaria detectable with microscopy or RDTs, as it is increasingly recognized that this is associated with ongoing morbidity [e.g., *anaemia*, increased susceptibility to bacterial infections, and cognitive function; reviewed in (Vanelle *et al.,* 2012a)]. If the aim is malaria elimination, the contribution of low-density infections to transmission needs to be considered given that, where data are available, low-density infections represent a significant proportion of malaria infections and can be the majority in low-endemic areas. It follows that the cost-effectiveness of existing or novel surveillance methods and interventions in reducing malaria transmission cannot be predicted or evaluated unless the relative contribution to transmission of three clinical/symptomatic malaria, two asymptomatic parasitaemia (detectable by microscopy or RDT), and three low-density parasitaemia (not detectable by microscopy or RDT) are estimated for a particular setting. With an increasingly diverse array of potential approaches for malaria elimination (Sinden, 2017), but with limited human and financial resources (Childs & Prosper, 2017), characterizing the contribution of low-density *parasitaemia* to transmission will help to focus elimination efforts.

### 2.2.2 Low-Density *Parasitaemia* and Transmission

Currently, there are insufficient field diagnostics to identify low-density submicroscopic *parasitaemia*, although different approaches are being evaluated for their performance and scalability (Gupta *et al.,* 2014). But even if all infected people are identified, it is necessary to understand who is infected by their mosquitoes and followers. I will understand the contribution of parasite density to the reservoir of infection for a typology of malaria essential to determine diagnostic sensitivity needs. It also affects how much effort a program puts into detecting and dealing with these infections, and when that effort is best spent. As mentioned above, when transmission decreases, the proportion of low-density parasites increases.

Recent evidence from Senegal also suggests that transmission efficiency increases with decreasing transmission intensity in both males and females. Currently, the only way to measure human infectivity is to feed humans colony-grown mosquitoes either directly (direct feeding test [DFA] ("Lee W-C, Malleret B, Lau Y-L, *et al.* Glycophorin c (CD236R) Mediates Vivax Malaria Parasite Rosetting to Normocytes. Blood. 2014;123(18):E100-E109.," 2015b) or infected human blood through a membrane ( fluid diet). (Traore *et al.,* 2021). For example, studies in Burkina Faso using DMFA found that 28.7% (25 of 87) of infected people were microscopically negative, causing 17.0% of mosquito-borne infections (Ouedraogo *et al.,* 2018). Weise, DFA studies in Thailand found that 21% (13 of 62) of individuals submicroscopic for *P. falciparum* or *P. vivax* were able to infect mosquitoes (Churcher *et al.,* 2015). Preliminary studies suggest that surveillance systems could be modified in the future to detect submicroscopic mosquito infections and target efforts to reduce transmission and infection in the area of transmission. Remains a major challenge for research. Furthermore, few empirical studies have quantified the proportion of the general population that is submicroscopic and infectious, especially in low transmission settings (i.e. i.e. less than 8 clinical cases per week per 1,000 people).

The cell is needed to determine when and where parasite trait density is deficient, to interrupt transmission, and to ensure diagnostic sensitivity for detection. Mathematical models suggest that conventional diagnostics can detect 55% of their reservoir of infection and with a 100-fold increase in detection sensitivity, i.e. h of 200 at 2 parasites/µl of blood, up to 95 % of infectious individuals.

(Churcher *et al.,* 2012).The level of diagnostic sensitivity could alter our understanding of the malaria transmission reservoir and enable the development and implementation of the best transmission interruption strategies to eliminate malaria.

### 2.2.3 Detecting *Gametocytes*

All malaria infections have the ability to produce *gametocytes*. Therefore, treating people who test positive for asexual parasites in community *chemotherapy* programs is a realistic program goal. However, research tools measuring *gametocytemia* are essential to advance our understanding of transmission biology and to define the populations and individuals driving transmission. Some studies

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suggest that transmission efficiency may increase when malaria prevalence decreases due to higher *gametocyte* densities. As the development of new transmission-blocking drugs and vaccines advances, it is necessary to understand the factors driving this transmission efficiency in order to determine in which settings interventions can be successfully pursued and/or implemented (Churcher *et al.,* 2015).

The programmatic applications of serology remain to be fully tested, although various approaches are being evaluated, including serological markers for adventitious infections (Ashley & Yeka, 2020). Il s'agit de are developing dynamic models that capture the impact of human population movements and could include multi-metric ensembles to allow self-consistent mapping across the range of transmission settings (Krishna *et al.,* 2017)

### 2.3 Theoretical Framework

Here are some of the theories that support this research work.

#### 2.3.1 Mosquito Malaria Theory

The mosquito theory of malaria (or sometimes the mosquito theory) is a scientific theory developed in the second half of the 19th century by Charles in 1851 which settled the question of the transmission of malaria. The theory essentially proposed that malaria was transmitted by mosquitoes, contrary to age-old medical dogma that malaria was caused by bad air or miasma. The first scientific idea was postulated by Charles E. Johnson in 1851, who argued that miasma was not directly related to malaria (Pearson, 2003). Although Johnson's hypothesis had been forgotten, the emergence and confirmation of Laveran's disease theory in the late 19th century began to shed new light. (Eldridge, 1925) revealed the implications of the discovery of filarial transmission in malaria, which supported Laveran's germ theory of malaria.

#### 2.3.2 Modern Theory

The work of (Croskerry *et al.,* 2014) presented the modern theory introduced in 1970 as the Mosquito-Borne Pathogen Theory (MBPT). This theory addresses specific biological or control issues involving different models of vector control, disease transmission or control with drugs or vaccines, pathogen evolution, and the management of virulence or drug resistance.

#### 2.3.3 Testing Theory

(Kelly-Hope & McKenzie, 2009) proposed the test theory. This theory posits the distinctive component of vector control that allows the potential intensity of pathogen transmission by each mosquito population to be assessed. This theory reveals a complementary approach to vector control through the indirect estimation of $R_0$ using other exposure field metrics (Okorie *et al.,* 2011).

#### 2.3.4 Critical Theory

(ROSS, 1905) postulated the theory of criticism. The critique of the theory exposes the challenges of ineffective translation of the applicability of the basic theory. It also uncovers any transmission involving the movement of pathogens, either through the movement of infected mosquitoes or the movement of infected hosts. However, based on the theory, one criticizes which factors determine the size of a focus or which scales characterize the transmission.

#### 2.3.5 Recasting Theory

Theory reformulation represents the development of a theory and it's testing of how actual transmission differs from mass action and how heterogeneity and poor mixing affect quantitative conclusions about control (Smith & Whittaker, 2014). This theory deals with the transmission and adaptation of large variations in time and space.

### 2.4 Related Models

(Lorimer, 2010), measures the length of each of the three stages (egg, larva and pupa) of mosquitoes in different breeding habitat using the equation

$$M = {}^{1}/_{(Le\ +Li\ +Lp)} \quad \ldots(2.1)$$

where;

M is the maturation rate of mosquitoes

Le is the period of egg stage.

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Li is the period of larva (L1-L4) stages.

Lp is the period of pupa stage.

Diekmann *et al.*, (2006) determined the basic reproduction number ($R_0$) using the equation.

$$F = \frac{\partial F_1(X_0)}{\partial xj},\ V = \frac{\partial V_i(X_0)}{\partial xj} \quad \ldots(2.2)$$

Where i, j = 1……m and where $X_0$ is the disease-free equilibrium. $FV^{-1}$ gives the rate at which infected individuals in xj produces new infection in $X_i$.

(Smith & Ellis McKenzie, 2004) examine the Statics and Dynamics of Malaria infection in *Anopheles*

mosquito and observed the following;

**i.** The survivorship and life span of anopheles mosquito is the proportion of a cohort mosquito that survive at age A, given by $\lambda A = e^{-ga}$, where $e^{-ga}$is the probability that individual mosquito survives one day. The average life span of mosquitoes is given by

$$\int_0^\infty \mathrm{gA}\lambda(A)\partial A = \frac{1}{g}, \quad \ldots \quad (2.3)$$

where gλ(A) is the proportion of mosquito that dies at age A, g = force of mortality, A = age, $\frac{1}{\mathrm{g}}$ = the average mosquito life span.

**ii.** The Human Feeding Stability Index (HFSI) and Human Blood Index (HBI) since a mosquito lives, $\frac{1}{\mathrm{g}}$ days and bites human once everyday.Where S = The stability index which is the number of bites given by a mosquito after it has become infectious, a = expected number of bites and a = Q where Q = proportion of bites taken on human and f = mosquito feeding rate. Human Blood Index is given by

$$\frac{\int_0^\infty \eta(A)dA}{\int_0^\infty \lambda(A)dA} = \frac{a}{a+g} \quad \ldots \quad (2.4)$$

Where η(A)λ = the fraction of mosquitoes in a population that survived to age A and bit a human. Note that the fraction of mosquitoes that feed on $\frac{f}{f+g}$ can be derived in the same way, assuming a = f, so that the human blood index (HBI) is a simple function of the rate of feeding mosquitoes and human lives. It can be understood as the ratio of two waiting times; time to first human bite or death$\frac{1}{a+g}$ and time to first human bite in surviving mosquitoes $\frac{1}{a}$.

**iii.** Proportion of infected mosquitoes is given by

$$\int_0^\infty v(A)\lambda(A)\frac{dA}{\lambda(A)} = \frac{acX}{g+acX} \quad \ldots (2.5)$$

Therefore, the proportion of infected mosquitoes is a ratio of two latencies: the latency to death or infection, $\frac{1}{g} + acx$, and the latency to infection in surviving mosquitoes$\frac{1}{acx}$, $v(A) = 1 - e^{-acxA}$ is the fraction of the surviving mosquito of age A that has already been infected, and V(A)λ(A) is the fraction of the original mosquito that is alive and infected. Let X be the proportion of contagious people, and since this is a static analysis, let X remain constant. c = the probability that an uninfected mosquito will become infected after biting an infectious human. Thus, mosquitoes are infected at the acX rate.

IV. Percentage of infectious mosquitoes $p_e = e^{-gnp_e}$ = the probability of surviving n days. n = duration of the incubation period $\mu(A) = \left(1 - e^{-acx(A-n)}A > n\right)$ is the fraction of infective mosquitoes of age A. A is $V(A)\lambda(A)$. Thus, the proportion of infective mosquitoes Z (also called *sporozoites*), or equivalently, the probability that an individual mosquito will ever become infectious, is given by

$$\int_0^\infty \beta(A)\lambda(\mathrm{A})\mathrm{dA}/\int_0^\infty \lambda(\mathrm{A})\mathrm{dA} = \frac{acX}{\mathrm{g+acX}}\, \mathrm{e}^{-\mathrm{gn}} \quad \ldots \quad (2.6)$$

Note that it is simply the product of the probabilities of ever becoming infected and, then, surviving the incubation period, $Z = YP_e$ Life time transmission potential, according to Smith and Mekenzie (2004) is given by

$$\beta = \int_0^\infty ba\mu(A)\lambda(A)dA = \frac{a^2bcXe^{-gn}}{\mathrm{g(g+acX)}} \quad \ldots \quad (2.7)$$

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b = the probability that an uninfected human becomes infected after being bitten by an infectious mosquito. $ba\mu(A)\lambda(A) =$ The expected reproductive output of a cohort of mosquito at age A. Life time transmission potential denoted $\beta$, is integrated over a mosquito life time by

$$V\frac{a^2bce^{-gn}}{g^2} = \frac{\partial\beta(X,\ldots)}{\partial X} \quad \ldots \quad (2.8)$$

Life time transmission potential for a mosquito is a function of the proportion of a human population that is infectious, X as well as other parameters. The curve $\beta$(X) is concave down with slope. Thus, VX $\beta$(X) the two differ by the factor thus

$$\left(1+\frac{acX}{g}\right)e^{-1}\left(\frac{h}{h+acX}\right) \quad \ldots \quad (2.9)$$

Please note that this is a three size product; Squared stability index $(S^2)$ , net transfer efficiency $(bc)$ and probability of surviving the incubation period $(p_e)$. Formula V describes the total Victorian capacity contribution of a single mosquito over its lifetime. The mosquito population can be combined into a single variable C(t) known as the Victorian capacity (GARRETT-JONES, 1964). The average number of bites per person per day is Ma: Macdonald (1995), where M: density of mosquitoes per human, a: average number of bites per day in humans by a single mosquito.

(Koram & Molyneux, 2007) examined the effect of socio-economic conditions associated with global warming on the dynamics of malaria transmission.

$$\frac{dx_1}{dt} = \mu + (\theta+\alpha)x_2 + \theta x_6 - (hy_3+\mu), \quad \ldots \quad (2.10)$$

$$\frac{dx_2}{dt} = \text{hy(t)}x_1 - (\theta+\gamma+\mu+\alpha)x_2, \quad \ldots \quad (2.11)$$

$$\frac{dx_3}{dt} = \gamma\, x_2 - (\gamma+\mu)x_3, \frac{dx_4}{dt} = \gamma\, x_3 + hy_{3(t)}x_3 + \theta_2 - (\theta_1+\mu)x_4, \quad \ldots \quad (2.12)$$

$$\frac{dx_5}{dt} = \theta_{1x4} - (hy_{3(t)} + \theta 2 + \mu). \quad \ldots \quad (2.13)$$

where;

$x_1$ = susceptible

$x_2$ = infectious human

$x_3$ = non-infectious human

$x_4$ = immune human

$x_5$ = partial immune

$\mu$ = natural rate

$\alpha$ = mortality rate

$\theta$ = natural mortality rate against malaria

$y_3$(t) = fraction of infectious mosquito

$\gamma$ = average period of infectious.

Dentinova (1989) examined the degree per day, dependent on the time for the preparation of a brood in mosquitoes (the pornographic cycle Gc) and time for biting according to Dentinova (1989) is given by equation

$$GC = 1 + \frac{Dd}{Ti-Tc} \quad \ldots \quad (2.14)$$

Where;

Dd=The number of degree days

Ti = The daily average temperature

Tc = Dependent on humidity.

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("May 19, 1999," 1999) examined the model, in which the vital rates are not necessarily equal, so that the disease-free equilibrium of population dynamics may grow exponentially, given by

$$\frac{dN}{dt} = (a - b)N \quad \ldots \quad (2.15)$$

Where;

N is the total population

**a** is the death rate

**b** is the natural mortality rate

(Ross & Fortini, 1981) examined the transmission of malaria in population dynamics of human and vector given by $\frac{dy}{dt} = \frac{b'}{n}$ f'y' (n - y) – (r + v) . . . (2.16)

Where;

N : total population sizes of a given time

y: total number of infected individuals

f: infected individuals who are not infectious

r: recovery — rate

μ: birth — rate

v: death — rate

where prime means the same values with respect to vector.

(Dietz, 1971) developed a transmission dynamics model of malaria defined as,

$(1 — \delta)^N h\ (t — N)x_1(t — N)$ i. e. $Q = (1 — \delta)^N h(t - N)$ . . . (2.17)

Where;

t — N: individual who are infected at time

$x_1(t — N)$: proportion of non- immune negative

h (t — N): inoculation rate

N: proportion of newly infected individual who survived the incubation period of days is approximately $(1 — \delta)^N$

Q: total number of infected people.

### 2.4.1 Entomological Measurement

According to the World Health Organization, malaria remains the leading cause of death among children in sub-Saharan Africa. Diseases in this region kill a child every 2 minutes (WHO, 2015). Recent studies have also shown that the abundance of *Anopheles* mosquito species is the most commonly used entomological measure to determine the relationship between vectors and malaria incidence at any site ((Urbinati & Iorio, 2016). Changes in the environment, especially climatic, have a major impact on the breeding habitats of various mosquito species, which affects the population density of adult mosquitoes (Bashar *et al.,* 2013). Climatic factors such as rainfall affect the abundance of adult mosquitoes by drastically altering the quality and quantity of breeding habitats. In order to determine the level of parasite activity and associated disease risk, the relationship between rainfall and mosquito abundance must be determined (Bashar *et al.,* 2013). A good understanding of the relationship between malaria remains a major public health threat in parts of sub-Saharan Africa, including Nigeria, despite years of control efforts. About 97% of the Nigerian population is at risk of malaria, with malaria accounting for 60% of hospital outpatient visits and 30% of hospital admissions for children under five and pregnant women (Ogbuehi & Ebong, 2015). Entomological studies focusing on the diversity, density, behavioral patterns and temporal variation of Anopheles species have long proven useful in identifying and monitoring malaria vectors (Sweileh *et al.,* 2017). Combinations of factors that determine a vector's ability to transmit malaria include: frequency, *anthropophilia*, bestiality, susceptibility to malaria parasite infection, infection rates, and female longevity (Aniedu, 1992). Recent studies have shown that

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the abundance of Anopheles mosquito species is the most commonly used entomological measure to determine the relationship between vectors and malaria incidence at a site (Urbinati & Iorio, 2016). Environmental changes, including climate change, have a major impact on the breeding habitats of various mosquito species and affect the population densities of adult mosquitoes (Bashar & Tuno, 2014). Climatic factors such as rainfall affect the abundance of adult mosquitoes by drastically altering the quality and quantity of breeding habitats. In order to determine the level of parasite activity and the associated disease risk, the relationship between rainfall and mosquito abundance must be determined (White, 2017). A good understanding of the relationship between rainfall and vector mosquito abundance will help to develop an effective and feasible vector control program in the communities studied, hence the need to determine the seasonal abundance of mosquito populations.

### 2.5 Etiology of Malaria

The first evidence of malaria parasites was found in early Paleocene mosquitoes preserved in amber around 30 million years ago ("Lee W-C, Malleret B, Lau Y-L, *et al.* Glycophorin c (CD236R) Mediates Vivax Malaria Parasite Rosetting to Normocytes. Blood. 2014;123(18):E100-E109.," 2015). The name malaria derives from "mal-aria" (Old Italian "bad air") and was probably first used by Leonardo Bruni in a publication in 1476 (JAMES & TATE, 1937). The discovery of the malaria parasite is attributed to Alphonse Laveran, who began his research in 1879 in a military hospital in Algeria. He found black pigments in the blood, as well as completely unknown bodies with certain properties that led him to believe they were parasites. However, he was able to perform studies on fresh blood without chemical reactions or staining processes (Al-Riyami & Al-Khabori, 2013). Laveran published his first major work on these parasites, Treatises of Palustral Fever, in 1884. The work showed that the parasites destroy red blood cells during development and alter the red pigment of malarial particles. Laveran had already established in 1894 that malaria had to go through a phase of its development in mosquitoes. However, it was an Army surgeon, Ronald Ross, who, while experimenting with less common species of mosquito larvae hatched in the laboratory and released to bite malaria patients, found bodies that were in the stage development of human malaria parasites in the stomach wall of this rare species of mosquito.

Laveran named the microscopic organism responsible for malaria *Oscillaria malariae* (Dashevskiy & Ramirez, 2015). Golgi observed in 1885 that all parasites present in the blood divided at regular intervals almost simultaneously, and that the division coincided with bouts of fever. He recognized that three types of malaria are caused by different *protozoa.* (Smith *et al.*, 2005) noted that Marchiafara and Celli were however the first to name this new organism *Plasmodium.*

### 2.6 Epidemiology of Malaria

The logical success of *An. gambiae* is largely dependent on its highly dynamic ecological behavior (White *et al.,* 2017), which has evolved over time to take advantage of specific tropical temperate climatic conditions that favor mosquito breeding and human-animal contact vectors.

*An. gambiae* is widespread in sub-Saharan Africa, its behavior and ecological adaptability vary considerably from site to site, dictated in part by spatio-temporal differences in seasonal climatic patterns (Monach, 2010). Such temporal fluctuations in the behavior of Anopheles vectors in response to seasonal changes in climatic conditions in an area are responsible for the enormous heterogeneity in the intensity of malaria transmission and the effectiveness of control measures (Webb, 2011). Studies have shown that during the rainy season, Anopheles mosquitoes tend to be more endophagic, more endophilic, and more cannibalistic to avoid hashish-related outdoor environmental conditions (Kamimura *et al.,* 2004). Also, during the rainy season, these mosquitoes breed more in natural larval habitats, such as (Paaijmans *et al.,* 2010). Local interactions of combinations of these important entomological factors of malaria transmission, caused by the behavioral responses of Anopheles mosquitoes to prevailing climatic conditions, will go a long way in determining vectorial efficiency and thus malaria transmission patterns, as well as the efficiency. Vector control measures implemented. *Anopheles* mosquitoes pose less of a threat to human health when they are forced to be zoophiles or breed in less productive and dangerous places. In addition, indoor residual spraying of insecticides such as *pyrethroids* and the use of insecticide-treated mosquito nets are more effective in controlling malaria vectors when these prefer to feed and rest indoors (Degen *et al.,* 2000). Nigeria, with its distinct annual tropical wet and dry seasons, coupled with local heterogeneity in the intensity and distribution of climatic factors such as rainfall, relative humidity and temperature in its different ecogeographical zones, is susceptible to induce behavioral changes in *Anopheles* mosquitoes at different times of the year, affecting the epidemiology of malaria and the effectiveness of vector control measures. However, patterns of Anopheles mosquito population dynamics have been elucidated in almost all geo-ecological zones of Nigeria (Venturini *et al.,* 2005). Similar variations in mosquito behavioral traits across seasons remain poorly understood based on the available evidence. Therefore, this study was undertaken to fill this information gap by examining some aspects of *An. gambiae* identified and compared, including peak season and local biting preferences, timing of development and survival to immature stage, susceptibility to insecticides, etc. during different seasons in north-central Nigeria.

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### 2.7 Global Indices of Malaria Cases

Malaria remains one of the leading causes of morbidity and mortality in sub-Saharan Africa and poses one of the greatest public health challenges in Africa. at risk of contracting malaria. In addition, one million deaths are recorded each year, 91% of which occur in sub-Saharan Africa (WHO, 2011).

Malaria is the third leading cause of death among children under five worldwide, after pneumonia and diarrheal diseases (WHO, 2013, 2014). According to WHO estimates, there were 655,000 deaths from malaria in 2010, with 91% in the African region and 86% in children under 5 years old. According to WHO estimates, in 2010 there were 216 million cases of malaria, 81% of them in the African region and affecting 3.3 billion people, or half of the world's population, in 106 countries and territories (WHO, 2014).

#### 2.7.1 Malaria in Nigeria

Malaria is *holoendemic* in Nigeria and accounts for 25% of infant mortality and 30% of infant mortality (Woo, 2003). Ninety-five percent of malaria infections in Nigeria are caused by *Plasmodium falciparum* and five percent by *Plasmodium malariae*. However, according to (Gallup & Sachs, 2001), malaria transmission is geographically specific. (Speybroeck, 2011), also reported that malaria vectors exhibit behavioral differences in different locations. Malaria is estimated to contribute to 11% of maternal deaths (WHO, 2012, 2013; 2014). There are approximately 100 million cases of malaria with over 300,000 deaths each year in Nigeria. In comparison, 215,000 people die each year from HIV/AIDS in Nigeria (WHO, 2015). Malaria is at risk for 97% of the Nigerian population. The remaining 3% of the population live in the malaria-free highlands. Malaria is a major public health problem in Nigeria, where it accounts for more cases and deaths than any other country in the world.

#### 2.7.2 Malaria Parasite in Human

Malaria is a vector-borne disease caused by *protozoan* parasites of the *genus Plasmodium*. There are four types of human malaria parasites, namely *p. falciparum, p. vivax, p. malariae* and *p. ovale*. Parasites are transmitted from person to person by female mosquitoes of the genus *Anopheles*. Different species are found in different regions (WHO, 2015). Transmission can occur seasonally, depending on vector population dynamics. The life cycle of the parasite begins with the inoculation of the parasite into human blood through the bite of a female *Anopheles* mosquito. Within half an hour, the *sporozoites* reach the liver and invade liver cells. Inside liver cells, *trophozoites* initiate their *intracellular asexual* division. At the end of this phase, thousands of *erythrocyte merozoites* are released from each liver cell. The time required to complete the tissue phase varies according to the infecting species (5 to 6 days for *p. falciparum*). *Merozoites* invade red blood cells (RBCs) and then grow through the ring, *trophozoite*, early *schizont*, and mature stages; Each mature *schizont* consists of thousands of *erythrocyte merozoites* (Wardrop *et al*., 2013). These *merozoites* are released after lysis of red blood cells and immediately invade uninfected *erythrocytes*. This whole invasion-multiplication-release-invasion cycle lasts about 48 hours in *p. falciparum* infections. The contents of the infected cell, released upon lyses of red blood cells, stimulate tumor necrosis factor and other cytokines, leading to the characteristic clinical manifestations of the disease. A small proportion of *merozoites* develop into *gametocytes*. Mature *gametocytes* appear in peripheral blood after a period of 8 to 11 days after the primary attack by *P. falciparum*. They increase in number for up to three weeks, then decrease, but circulate for several weeks. *Gametocytes* enter the mosquito when it bites an infected person. The malaria pathogen in the human vector of malaria is transmitted by mosquitoes of the genus *Anopheles*. Of the 360 species, there are about 45 with the ability to transmit malaria to humans. *Anopheles*es live worldwide, but malaria transmission occurs primarily in tropical and subtropical regions of the world. *Anopheles*-free always means malaria-free, but not the other way around. If the malaria pathogen enters the mosquito after a blood meal, the *gametocytes* continue their development (*sporogony*). Male and female gametes fuse and form a zygote. This develops into an *ookinete*, which penetrates the intestinal wall and develops into an *oocyst*. The *oocyst* divides asexually into numerous *sporozoites*, which reach the mosquito's salivary gland, where they can be transmitted during the mosquito's next blood meal. *Sporogony* in the mosquito lasts about 10-20 days depending on the air temperature, then the mosquito remains infectious for 1-2 months if it survives.

At a temperature below 15°C, there is no *sporogony*. Only the female mosquito takes a blood meal (male *Anopheles* feed on nectar) necessary for the development of eggs. Two to three days after the blood meal taken at night or at dawn, the female *Anopheles* mosquito lays about a hundred eggs (Agusto & Parshad, 2011).

Therefore, it can produce over 1,000 eggs in its multi-week lifespan. Eggs are always laid on the water surface, preferably in swamps or shallow water. They can also breed in water reservoirs or tree cavities. The oval eggs are a millimeter long and take about two weeks to develop into adult mosquitoes. They only travel short distances of a few kilometers. Their preferred location is near human habitation. There are behavioral differences between mosquito species that are important for studying the geographic distribution of the vector (Wardrop *et al.*, 2013).

The most important *Anopheles* species in Africa are members of the An. *gambiae* and An. funkier complex Five types of *An. gambiae* complex are vectors of malaria, and two of them (*An. gambiae* s.s. and *An. arabiensis*) are more common in Africa. -

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Saharan Africa. *In*. arabiensis *predominates* in dry areas and An. *gambiae* s.s in wetter areas. Their favorite breeding sites are sunny temporary ponds or rice paddies. An. *arabiensis* feeds on humans and animals, while An. *gambiae s.s* feeds efficiently on humans, prefers indoor places for biting and resting, and has a higher vector capacity than other species. Two saltwater species of the An. *gambiae* complex (*An. melas* and *An. merus*) are found in West and East Africa, respectively, where *An. merus* feeds mainly on animals and *An. merus. melas* bites people or animals. Another important vector of malaria in many parts of tropical and subtropical Africa is *An. funestus* of the *An. funestus* group. It feeds mainly on humans, resting and biting inside. It breeds in semi-permanent and permanent vegetated waters and swamps and is associated with the perennial transmission of malaria (Eckhoff, 2011).

### 2.8 Malaria Vector Ecology

Mosquitoes have been a problem for humans and animals throughout human history. About sixty different species of mosquitoes are present in the world (Defoliart, 1954). Among these genera, members of the *Anopheles*, *Culex*, *Aedes*, *Hemagogus* and *Mansonia* complexes are important pests in Nigeria (Oyewole *et al.*, 2007). Mosquitoes not only inflict stinging pain on humans, but also suck human blood and transmit pathogens, dying soon after mating.

The female mosquito bites humans and animals because they need blood to develop eggs. Males are short-lived, suck nectar and plant sap instead of blood, and die soon after mating. The *haematophagous* behavior of female mosquitoes is a public health problem. Several parasitic and viral diseases are transmitted by mosquitoes. Wuchereria bancrofti and Brugia malayi, which cause *lymphatic filariasis* in humans (WHO, 2010), are transmitted by members of the *Aedes*, *Culex* and *Mansonia* complexes. Yellow fever and dengue viruses are also transmitted by these mosquitoes (Eckhoff, 2012).

The flies' short range and their preferred lodging and breeding sites are responsible for large local variations in the geographic distribution of *Anopheles* mosquitoes. The effect of the environment on the malaria vector is further determined by rainfall and temperature, which affect the survival of the mosquito and the life cycle duration of the parasite in the vector.

#### 2.8.1 Temperature

Temperature affects the survival of the parasite throughout its life cycle in the *Anopheles* vector. All species have the shortest life cycle, around 27–31_C, with a range of 8 to 15–21 days depending on the species (Wardrop *et al.*, 2013). The lower the temperature, the longer the cycle. Below 19oC for *p. falciparum*, the parasites are unlikely to complete their cycle and thus further spread the disease. Temperature also modifies the vectorial capacity of *Anopheles* mosquitoes. Optimal temperature values of 22°C to 30°C prolong the lifespan of mosquitoes and increase the frequency of blood feeding by females to one meal every 48 hours. Higher temperatures also shorten the aquatic life cycle of mosquitoes from 20 to 7 days and shorten the time between hatching and ovipositor and the time between successive ovipositors (Gething *et al.*, 2011).

Temperature also affects the vector. In tropical climates, Anopheles eggs hatch within 2–3 days of being laid, while in cooler temperatures it may take 2–3 weeks. At low temperatures close to freezing, African vector populations are effectively wiped out and at very high temperatures, above 40°C, Anopheles are killed (Verhulst *et al.*, 2011). Due to all the temperature demands, malaria transmission becomes rarer at higher altitudes. There are no Anopheles above 2,500 meters near the equator and none above 1,500 meters in other regions.

#### 2.8.2 Precipitation and humidity

Rainfall and humidity greatly affect the living conditions of Anopheles (Helinski *et al.*, 2006). Temporary pools created by increased rainfall provide ideal conditions for vector breeding. However, rainfall can also destroy existing breeding sites: heavy rains can turn breeding ponds into streams, prevent mosquito eggs or larvae from developing, or simply wash eggs or larvae out of ponds (Lindsley *et al.*, 2005). Conversely, exceptional drought conditions can transform watercourses into ponds. The appearance of such opportunistic mosquito breeding grounds sometimes precedes epidemics. The interaction between precipitation, evaporation, runoff and temperature modulates ambient air humidity, which in turn affects the survival and activity of Anopheles mosquitoes. Mosquitoes can survive when the relative humidity is at least 50-60%. Higher levels extend the lifespan of mosquitoes, allowing them to infect more people. As an indirect indicator of humidity and precipitation, the vegetation index has proven to be a good indicator (Helinski *et al.*, 2006).

### 2.9 Distribution of *Anopheles* Mosquitoes

The distribution, frequency and underlying causative factors of mosquitoes vary from continent to continent. A review of the available literature shows that in South America, Dunn surveyed and conducted inspections in twenty-six towns and villages in various parts of the northern half of Venezuela to determine the extent to which mosquitoes were cultivated. Observations were also made at each site on the water supply system and other conditions that could affect the breeding and distribution of this species. The water supply system was such that it required the use of many reservoirs to store water in the houses, which created favorable conditions for the breeding of domestic mosquitoes in the apartments. The water reservoirs examined in the 23 cities bore the

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number 9616 and consisted of the following containers: 2,725 jars, 2,053 pots, 1,822 barrels, 1,083 basins, 824 filter stones, 288 reservoirs, 23 ornamental fountains and 798 containers various. Of these containers, baby mosquitoes were found in 2,752 or 28.61%. The positive containers included 1020 jars, 990 barrels, 278 pots, 232 pilas, 95 filter stones, 70 reservoirs, 5 ornamental fountains and 62 miscellaneous containers. However, in Europe (Poncon *et al*., 2008), it has been established that the probability of recurrence of malaria in an area depends on three factors: susceptibility, infectivity and susceptibility of vector and host. (Chaudhary *et al.*, 2003) determined the prevalence and distribution of *Anopheles* mosquitoes in 12 villages in the 4 *tehsils* of the arid district of Bikaner, India. Six guys, viz. *Anopheles subpictus* (34.7%), *An. stephensi* (33.3%), *An. culicifacies* (18.0%), *anularis* (12.1%), *an. pulcherrimus* (1.1%) and *An. barbirostris* (0.8%). *In. stephensi* was present all year round and the other species were present during the monsoon and post-monsoon periods. During the main winter period (December to January) it was only lit. *stephensi* present and in low density. *In. culicifacias* appeared only in spring and lasted until mid-November. *An. subpictus*, *An. pulcherrimus*, *An. barbirostris* and *An. annularis* were only found during the monsoon and post-monsoon periods. *An. subpictus* was the most abundant species during the monsoon, as was *An. stepihensi* during the spring season indoors.

Furthermore, (Tyagi, 2004) noted that there has been a recent resurgence of malaria in various parts of India and that the Tharp Desert in northwestern India is currently suffering from the effects of epidemics repeated annual outbreaks of malaria which occur with the introduction of canal irrigation works, in particular the Indira Gandhi Nahar Pariyojana (IGNP) massif. Before the advent of pipe irrigation, the interior of the Tharp Desert was only *An. stephensi*, which bred primarily in household and community groundwater reservoirs and transmitted low levels of malaria. Since the 1980s, extensive irrigation of three different canal systems has altered desert *physiography*, vector abundance, distribution, and capacity, leading to the emergence of *p. falciparum* in pristine desert levees of the Tar. Alterations of plant configuration, maintenance of high surface humidity and excessive channeling added to inadequate management of irrigation water have attracted several previously absent *Anophelines*, eg *An. culicificios*. According to (Cuamba *et al.*, 2006), malaria is responsible for 50% of all outpatient treatment and approximately 22% of all hospital deaths. PCR showed a preponderance of *An. gambiae* with indoor roosting densities ranging from 0.9 to 23.5 per house. From 403 to *gambiae* identified molecularly were 93.5% M-form and 6.5% S-form. M and S were sympatric at 4 sites, but no M/S hybrids were detected. *In. funestus* was found in a locality near Luanda. They concluded that *An. gambiae* M was the most important and widespread vector of malaria in the study areas.

However, in East Africa, (Minakawa et al., 2002) found that there are three species of malaria vectors in the Lake Victoria basin region, *An. gambiae*, *An. arabiensis* and *An. funestus*, but *An. arabiensis* is not present in the western highlands of Kenya. The range and relative frequency of *An. gambiae* and An. *arabiensis* were defined by climatic factors such as annual precipitation and annual and wet season temperatures. *In. gambiae* was more frequent in humid environments and *An. arabiensis* is more common in arid areas (Kirby *et al*., 2008). Thus, other biotic or abiotic factors are responsible for species composition variation at micro-geographic scale. (Verhulst *et al.*, 2011) collected day resting indoor mosquitoes and of those collected, 83 were *An. gambiae* s.l.

### 2.9.1 Monthly Distribution of *Anopheles* Mosquitoes

During the long rains of April to May and the short rains of November and December, mosquito populations were very high. Blood meal analysis for *An. gambiae* s.l. females showed a human blood index of 0.97. *In. gambiae* s.l. it breeds in the polluted waters of Nairobi and 95% of the larvae were An. *arabiensis. Anopheles arabiensis* was *anthropophilic* and therefore showed ecological flexibility within the species. (Charlwood *et al*., 2003), studied the survival of An. *funesto*, *An. gambiae* and *An. arabiensis* in the dry season in the Kilombero Valley, a dry savannah area in East Africa. *Anopheles gambiae* has only been found associated with humans in forest areas with high annual rainfall, while An. *funestus* was present in high density at the edge of the valley where large areas of standing water remained. A large population of *An. arabiensis* was present along the river system in the middle of the valley, and mosquitoes probably from this population were occasionally caught in villages bordering the valley. *Anopheles funestus* was the main vector of dry season malaria in the valley and remains in foci closely associated with clusters of dwellings. All three species were very common but otherwise hidden refugee populations.

(Cano *et al.*, 2006) reported from a small town in mainland Equatorial Guinea that malaria transmission varies from country to country and that there are also local temporal and spatial differences. A total of 1,173 *Anopheles* were captured: 279 *An. gambiae* s.l. (217 *An. gambiae* s.s. and one *Anopheles* melas), 777 *An. moucheti* and 117 *Anopheles carnevalei*. *In. Moucheti* turned out to be the main vector species. A significant correlation was found between the distance from the houses to the nearest water source (Ntem river or tributaries). (Himeidan *et al.*, 2004) reported that of the 4854 female Anopheles they collected, 4847 (99.9%) *An. arabiensis* and 7 (0.1%) *An. pharoensis* were. *An. arabiensis* female reproduces all year round, with 2 maximum densities, during the rainy season (158.4 females/space/day and 84.7 larvae/10 dives) and the irrigation season (136.8 females /space/day and 44.8 larvae/10 immersions). (Shililu et al., 1998) identified 13 species of Anopheles, including An. *gambiae* complex predominated the first year (75.6%, n=861) and the second year (91.9%, n=1262) of sampling. PCR showed that 99% (n=1309) of *An. gambiae* s.l. Specimens of *An. arabiensis*, indicating that it was the only extant member of the *Gambiae* complex.

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### 2.9.2 *Anopheles* Seasonal Distribution Patterns

The global proliferation of seasonal areas of potential malaria transmission, caused by the encroachment of seasonal areas into perennial areas and the spread of seasonal malaria into previously malaria-free areas, is of concern. The potential seasonal transmission of malaria is very likely to favor epidemics, resulting in widespread wasting, increased mortality and high morbidity in unprepared or unimmunized populations (Fraunholz, 2005). Malaria epidemics occur mainly in hypo- or *meso-endemic* areas. One of the characteristics of these epidemics is that they occur in cycles of 5 to 8 years; however, it is difficult to predict a cyclical epidemic because the cycles are far from regular. The most obvious indicators of a possible epidemic are meteorological and environmental factors, but reasonably good collection of relevant statistical data can detect them at an early stage and facilitate the initiation of appropriate action (Bruce-Chwatt & Bruce-Chwatt, 1950). West Africa also experiences a remarkable abundance and prevalence of mosquitoes and malaria, as evidenced by the literature of the following authors: (Takken *et al.,* 2002) reported that mosquito larval populations were found in rice fields, but Anopheles *gambiae* s.s. it was significantly more common in the early stages of rice development than later in the growing season. Post-harvest fallow land has also been found to serve as very suitable breeding sites for mosquitoes, leading to high populations of Anopheles mosquitoes. Although Anopheles mosquitoes were also found in the irrigation ditches, it is concluded that the rice fields were the main source of malaria transmission. Elsewhere in West Africa, (Arrighi *et al.,* 2009) conducted a longitudinal entomological study in two villages located in different ecological zones of Senegal (a Sahelian region and a Sudanese savannah). In both villages, there was *An. gambiae* s.l. the main vector, where *An. gambiae* in the savannas of *Wassadou* and *An. arabiensis* predominate in the Sahel region of Thiaye. Malaria transmission is mainly seasonal; with a higher male bite rate (ma) and entomological vaccination rate (h) in Wassadou than in Thiaye. A strong variation in the density of *An. gambiae* s.l. observed how females disappear in the dry season. A specific composition of *An. gambiae* s.l. has been observed with *An. gambiae* predominant in the rainy season and *An. arabiensis* generally more common in the dry season.

Additionally, (Nacher, 2012) report that entomological studies conducted over the past 30 years have shown that there was low malaria transmission in the suburbs of Dakar, Senegal, but very few cases in Dakar itself. Cases of transmission of malaria between permanent residents have been reported. From May 2005 to October 2006, 4,117 and 797 *An. gambiae* s.l. trapped in Bel-air or Ouakam. Three members of the complex were present: *An. arabiensis* (more than 98%), An. melas (less than 1%) and *An. gambiae* s.s. Molecular form M (less than 1%). The proportion of host-seekers trapped inside *An. gambiae* s.l. it was 17% in Bel Air and 51% in Ouakam. These data were consistent with clinical data from a Senegalese military hospital in Dakar (main hospital), where most malaria cases occurred between October and December. It was An's first record. Melas in (Awono-Ambéné & Robert, 1999) reports that 13 villages in the savanna zone and 21 villages in the forest zone of Côte d'Ivoire had an acrid density of *An. gambiae*, which is directly linked to the cultivation of rice in the interior valleys within a radius of 2 km from each village. Snapper population densities drop during the rainy season. In other words, the onset of the rainy season was accompanied by an increase in the density of mosquito bites. In the forest zone, in villages with a rice-growing cycle or without rice-growing, an annual population peak of *An. Gambia* has been observed. In villages with two rice cycles, a second peak was observed during the dry season (off-season) growth period. During the peak season cultivation period, rice cultivation and uncultivated lowland areas show a positive correlation with the bite density of *An. gambiae* in the villages of the savannah of Côte d'Ivoire. However, in the forest zone, the population density of An's prickles was correlated. *gambiae* was strongly correlated with surface water availability in inner rice-growing valleys, especially during transplanting, while the correlation with surface water availability in other lowlands (uncultivated) was weak or not significant. High density of Anopheles mosquitoes needed to maintain transmission (WHO, 2013). According to WHO (2013), areas of unstable (epidemic) malaria can be categorized into two distinct types of transmission patterns, highly seasonal but intense transmission with a more or less predictable pattern each year associated with explosive epidemics at intervals of five to ten years respectively, Highly seasonal with very little or no transmission for several years. These areas are also sometimes affected by dramatic and devastating epidemics that often result from environmental or weather changes most of the time, as this will increase the transmission of malaria (WHO, 2013).

### 2.10 Spatiotemporal Malaria Transmission

Malaria is transmitted through the bite of an infected female *Anopheles* mosquito, while the main vectors of malaria in Nigeria are: An. It is therefore highly dense, *anthropophilic* and a very important vector of malaria. *On. arabiensis* is dominant in the savannah ecotype. C'est prefers dry environment. It is also *zoophilic* and *exophilic*, while An. melas is the saltwater species. Il est is generally more *exophagous* and *zoophilic* and therefore a poorer vector than *An. gambiae* (Awolola *et al.,* 2005).

Within the zones and villages there was a high population of *Anophelines*, with more than 80% of the total *anophelines* collected from less than 20% of the villages and from only 10% of the sampled homes. Similarly, (Ngáng'a *et al.,* 2008) from Eritrea, where data showed the presence of only one peak transmission season for malaria between July and October for the highlands and the western lowlands. The highest vaccination rates were recorded in August and September (range = 0.29-43.6 infectious bits/person/month) at all sites during the biennium.

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In addition, a group of authors represented by (Vanelle *et al.,* 2012a) reported that a total of 12,937 individuals from 176 villages were screened for both *P. falciparum* and *P. vivax* parasite species using the optimal rapid diagnostic test. The prevalence of malaria was generally low, but highly focal and variable, with the proportion of *parasitemia* being 2.2% (range: 0.4% to 6.5%). Although there was no significant difference in age- or gender-specific prevalence rates, positive cases accounted for 7% of households and 90% of these were *P. falciparum*. Multivariate regression analyzes showed that mud-walled houses were positively associated with malaria infection. (Geall *et al.,* 2004). And of the 29,572 malaria vectors collected, 14,661 (49.5%) were *An. funestus*, 14,153 (47.9%) *An. gambiae* s.l. and 758 (2.6%) An. *mascarensis*. *Anopheles arabiensis*, *An. merus* et *An. funestus* were present in all villages, while *An. gambiae s.s* et *An. mascarensis* (a mosquito native to Madagascar) have only been found in the two villages surrounded by rice fields and in the Humid Région, respectively. *Anopheles funestus, An. gambiae* s.s. and on. *mascarensis* were infected more frequently. *On. funestus* was responsible for 90% of infectious bites.

The spatial distribution of the molecular forms of *An. gambiae* (M and S) and associated environmental factors related to disease prevalence, data collected showed that the M and S forms of *An. gambiae* in most places were sympathetic to similar weather and vegetation characteristics with Nigeria, hence the mosquito and malaria situations of the two countries are similar. A report by (Lonneux & Hamoir, 2010) showed that An. *gambiae s.s* mosquitoes are important vectors of *linfatic filariasis* (LF) and malaria in Ghana. However, the S-shape was more prevalent in the central region, while the M-shape was more prevalent in the northern and near-coastal savannah regions. The Republic of Niger experiences similar weather, climate and vegetation as the current study area, Nasarawa State. And one of the studies conducted in the arid Republic of Niger was that of (Labbo *et al.,* 2004) on members of the *An. gambiae* complex in three zones of the Republic of Niger. *On. funestus*, thought to have disappeared from the Republic of Niger, has reappeared in both Sudan and the Sahel. This has been attributed to the clearing of naturally forested savannahs and the enlargement of cultivated fields, leading to the alteration of surface characteristics and the construction of temporary ponds such as dams that improve water drainage. The situation of abundance, distribution and characterization of mosquito and malaria species in Nigeria differs when moving through the different ecological zones, for example (Onyabe & Conn, 2001) examined the distribution *ön. gambiae* and *An. superecological* zones of Nigeria (dry savannah in the north gradually gives way to humid forest in the south). They compared the study to the distribution found using samples of indoor women reported in a previous study over 20 years ago. In both years there was variation in species types within the 10 localities, but this observed variation was very high in only four of the 10 localities (Onyabe and Conn, 2001). The identity of the more common species changed between 1997 and 1999 in only three of 10 localities. *An. arabiensis* was most widespread in many savannah areas of southern Guinea, although it was absent there about 20 years ago. The data suggest that *An. arabiensis* has expanded its range. Similarly, (Adak *et al.,* 2005) state that the ecology and distribution of different mosquito species is necessary to determine the frequency of mosquito vectors and the prevalence of associated diseases. The distribution of different genera of mosquitoes in natural and man-made habitats and their relative abundances were studied between August 2002 and July 2003 at three focal points, namely Uromi, Ekpoma and Auchi, covering the regions of Esan and Etsako in west-central Nigeria. The study identified 17 vector species belonging to three genera (*Anopheles*, *Culex* and *Aedes*) as vectors of four human diseases prevalent in the study areas. A total of 736 mosquito larvae were found in artificial sources and 568 larvae were collected from natural sources. Swimming pools, plastics and metal boxes were the main artificial sources of mosquito larvae. (Oyewole *et al.,* 2007) collected a total of 790 an. gambiae (52.7%), 555 BC. arabiensis (37%) and group 155 An. funestus (10.3%). The indoor catch of 807 (53.8%) exceeded the outdoor catch of 693 (46.2%), which was mainly An. rivulorum and An. arabiensis. Biting activity observed indoors was significantly higher than outdoors ($p<0.05$) with a ratio of 10.1:9.60 indoors to outdoors. The malaria vectors involved were *An. arabiensis, An. gambiae s.s.* and a *funestus s.s* with overall infection rates of 2.3%, 2.5% and 2.9%, respectively. Elsewhere in Nigeria, (Fakoorziba *et al.,* 2009). Mosquitoes in the permanent dormitories of Nnamdi Azikiwe University, Awka. A total of 1265 mosquitoes composed of 5 species of mosquitoes were collected in the form of larvae. 72 adult mosquitoes, including 3 species of mosquitoes, were collected from the university accommodation. *Anopheles gambiae* had the highest percentage of indoor biting and dormant mosquitoes at 50 (69.45%). (Okwa *et al.,* 2009) noted, however, that two of the problems with Anopheles control in Nigeria are the diversity of Anopheles vectors and the size of Nigeria. Accordingly, Anopheles distribution and malaria transmission dynamics were compared among four ecotypes in Nigeria during the rainy season. Five species were identified among 16,410 Anopheles collected. NAIL. *gambiae s.s* constituted between 29.2% and 36.6% of the population of each zone. All five species identified transmitted *P. falciparum.* NAIL. *gambiae s.s* had the highest rate of *sporozoites*. The most infected mosquitoes have been found in the rainforest.

(Alaba & Alaba, 2009) believe that the occurrence of malaria varies with climate and this affects the survival of *Anopheles* or otherwise. Also, tropical areas, including Nigeria, have the best combination of adequate rainfall, temperature, and humidity, which facilitates the breeding and survival of Anopheles mosquitoes. The prevalence of malaria varies between regions of the world and even within the same country. This is facilitated by variation in parasite-vector-human transmission dynamics that favor or limit the transmission of Plasmodium infection and the associated risk of disease and death. Of the four Plasmodium species that infect humans: *P. falciparum, P. vivax, P. malariae,* and *P. ovale*. *Plasmodium falciparum* causes most of the severity and death associated with malaria, which is most prevalent in sub-Saharan Africa, where Nigeria has the largest population. And that malaria accounts

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for about 50% of outpatient consultations, 15% of all hospitalizations and the leading cause of death in the country (National Action Plan to Fight Malaria 1996-2001). More importantly, it has become a social and economic problem, consuming an estimated US$3.5 million of government resources and US$2.3 million from other stakeholders in control efforts in 2003 (WHO, 2005) . About 50% of Nigerians experience at least one episode a year. However, government figures show an average of four seizures per person per year (WHO, 1995 and 2002). The situation is aggravated by the resistance of *Plasmodium* to first-line antimalarial drugs (WHO, 2000). The Nigeria Malaria Report 2005 indicates that malaria has increased in recent years, with around 1.12 million cases reported in the early 1990s, 2.25 million cases in 2000 and around 2.61 million cases reported in 2003.

### 2.11 The Epidemiology of Malaria in Nasarawa North-Central Nigeria

Malaria in the Guinea savanna varies in a clear seasonal pattern (Samdi *et al.*, 2005). The relatively dry savannah of northern Nigeria exhibits strong seasonality in malaria transmission, peaking during the rainy season. Consequently, the North experiences an unstable *hypoendemic* or *mesoendemic* malaria (Molta *et al.*, 2007). Malaria is considered "seasonal" when potential transmission occurs between 1 and 7 months in a year (Fraunholz, 2005). The prevalence of malaria in Nasarawa can be classified as *mesoendemic*. Since previous work showed that the prevalence of *Plasmodium* infection in the communities studied was 40.2% (80/200) in Nasarawa (Samdi *et al.*, 2005) was enough. This classification is based on the cumulative prevalence determined for the study population according to the World Health Organization classification of endemic malaria, which characterizes the dry season while high levels occur during the rainy season.

Additionally, (Molta *et al.*, 2007b) identified this strong seasonality in a study of the pattern of childhood cerebral malaria in northeastern Nigeria, with ninety-five (95) percent of infected patients occurring between June and November, with a peak in October. The seasonal pattern observed in the Guinea savanna contrasts with the humid forest areas of Nigeria, where malaria transmission is high throughout the year and is therefore considered 'persistent'. While malaria is potentially transmitted 8 to 12 months a year ("Malaria Deaths Following Inappropriate Malaria Chemoprophylaxis—United States, 2001," 2001), these areas, especially rural settlements, are often located in areas where malaria transmission is stable and little affected by climate change, and Malaria vectors in this part of Nigeria are highly infectious, highly *anthropophilic* and long-lived, for example, in a September 1997 survey of the coastal region of Ibeshe, Lagos State, 1,068 out of 1,118 ( 96%) female *Anopheles* mosquitoes from the survey were dissected. positive (Mokuolu *et al.*, 2018). A similar study was conducted in the Sahel around the same time of year (July-September), which coincides with the peak of malaria transmission. Only 2.4% of *Anopheles* females were positive in ELISA (Samdi et al., 2012). However, vectors in the Guinean and Sahelian savannahs coincidentally show a high number of infective bites per person, about 60–100 infective bites per person per year in the savannah ecotype and about 30–60 infective bites per person per year in the forest. The cumulative entomological inoculation rate in the Sudanese savanna reached a maximum of 145 *sporozoite*-positive bites in one year (including 132 in the rainy season) (Mokuolu *et al.*, 2018). Studies in Garki District, Kano State had estimated that malaria transmission is sustained when the human population receives approximately 0.33 infective bites per person, i.e. transmission will be maintained as long as every person in the population is infected once every 3 years (Emmanuel *et al.*, 2018). There are large seasonal, annual and local variations in the extent of malaria transmission in the Sudanese savannah. (Chuma *et al.*, 2010) also found during their six-year malaria project in Garki, Kano State, that the cumulative prevalence of malaria was very high, reaching 100% in the age group of 1 to 8 years old. This could be attributed to the high transmission rates caused by mosquito bites. The ability of mosquitoes to transmit malaria from person to person was between 200 and 2000 times greater than the critical level needed to maintain malaria as an endemic disease (MOLINEAUX *et al.*, 2001).

### 2.12 Distribution of *Anopheles* Vectors in Different Ecological Settings in Nigeria

Malaria biodiversity and vector distribution of northern Nigeria, characterized by harsh environmental conditions during the dry season with sparse grassland vegetation in arid and savanna regions, as reported by (Lamidi *et al.*, 2018), who identified and determined *Anopheles* mosquito species in Nguru, Yobe State. Distribution and relative abundance over the months of the year. *Anopheles gambiae* (1145); *An. funestus* (1220) and *An. arabiensis* (827) were the main predominant species in the city. *An. gambiae* was particularly abundant during the wet months, followed by *An. funestus* at the end of the rainy season and *later. arabiensis* during the driest months. Based on observation of the monthly *Anopheles* distribution and supporting data on malaria prevalence, the three species appear to complement each other, maintaining malaria endemicity in the city. The study showed malaria vectors throughout the year due to the favorable environmental conditions in the arid zone of Nigeria.

### 2.13 Malaria-Related Mortality, Morbidity and Immunity

The incubation period of *P. falciparum* malaria (the time between parasite inoculation and the first medical symptoms) is approximately 8 to 15 days. The main symptoms of all forms of malaria are (periodic) bouts of fever. The most severe form of malaria morbidity is cerebral malaria, which is characterized by coma with detectable *parasitaemia* and is accompanied by obstruction of the capillaries of the central nervous system. Cerebral malaria is a serious complication of clinical malaria in areas where malaria transmission is 10 to 20 infectious bites per year. Other serious complications include severe anemia, acute kidney

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failure or failure, liver or lung problems, jaundice, and gastrointestinal symptoms such as abdominal pain, nausea, vomiting, diarrhea, or constipation (Warrell, 2003).

Acquired immunity develops after repeated infections. Adults can tolerate the parasites without developing symptoms. Babies are protected by maternal antibodies for the first 3 to 6 months of life. Until they develop their own immunity, they are vulnerable to clinical episodes of malaria. Infant mortality in areas with high endemic rates of malaria is high (Kalipeni & Drakakis-Smith, 1993). Pregnancy causes suppression of immunity. A high parasitaemia is observed during the first pregnancy and decreases during subsequent pregnancies (Brabin *et al.,* 2002). Maternal malarial infection is an important cause of miscarriage and stillbirth, which reduces a newborn's chances of survival.

### 2.14 Measures of Malaria Endemicity and Transmission

Malaria prevalence is the most widely used measure of endemicity. Prevalence data are obtained through community surveys of people being tested for the presence of parasites in their blood. The acquisition of partial immunity in older children and adults in malaria-endemic areas means that this measure is age dependent. Prevalence is only an indirect measure of the extent of malaria transmission, as malaria infections can vary in duration. A direct measure of transmission is the incidence of the disease, ie the number of new cases of malaria diagnosed per unit of time and per person. Incidence data collected from health facilities may be biased because it may reflect patient access to these facilities. They also depend on accurate estimates of the population at risk.

The most common entomological measure of malaria transmission is the entomological inoculation rate (EIR), which is defined as the number of *sporozoite*-positive mosquito bites per person per unit of time (usually one year) and is the product of *Anopheles* density, human bite, and *sporozoite* index (the number of infective mosquitoes) (Smith *et al.,* 2007). The human bite rate can be measured by catches from human baits or mosquito traps. One of the best documented studies on malaria transmission was conducted between 1971 and 1973 in the Garki region of northern Nigeria (MOLINEAUX *et al.,* 2001). Using Garki's data, a mathematical model was formulated (Stehr, 1998) that makes 6 predictions on the age-specific prevalence of *P. falciparum* in humans based on vector fitness. It can be used to link multiple measures of transmission (including vectorial capacity and entomological vaccination coverage) and malaria prevalence.

### 2.15 Malaria Mortality Measures

There are four main ways to measure malaria mortality: from clinical records, when the cause of death is identified; observe the increase in mortality during malaria epidemics; observe the reduction in mortality as malaria is brought under control; or calculate the mortality necessary to maintain the observed level of the sickle cell gene in a balanced polymorphism (Molineaux, 2012).

Clinical registers in Africa almost never contain post-mortem series and, worse, are biased because they only come from tertiary care facilities and very rarely include young children and infants. The fact that most people die outside of hospital and the limited availability of beds for children in Africa make it clear that death certificates are a poor measure of malaria mortality (Shulman *et al.,* 2002). Interactions between malaria and other diseases in malaria-endemic areas make it difficult to quantify malaria-related mortality. Malaria can be a relevant risk factor for many deaths, even if it is not the direct cause ("NETTLETON v. MOLINEAUX," 1876). In addition, low birth weight is a major risk factor for infant mortality and is known to result from both prematurity and intrauterine growth retardation due to maternal malaria infection during pregnancy. (SMITH et *al.,* 2006) emphasized that it is just as important to consider the relationship between malaria endemicity and all-cause mortality as its relationship to malaria-specific deaths.

### 2.16 Spatial Epidemiology of Malaria

Spatial epidemiology is the study of the spatial/geographic distribution of disease occurrence and its relationship to potential risk factors. The origins of space epidemiology date back to 1855 with the pioneering work of Snow on the transmission of cholera. He mapped cholera cases as well as the location of water sources in London and showed that contaminated water was the main cause of the disease. Spatial analysis was used primarily in the 19th and 20th centuries in plotting observed cases or disease rates (Townson, 2009). Newer methods use computer-assisted mapping methods, satellite data and modern statistical methods and allow an integrated approach to tackle both tasks; Infer the geographic spread of a disease and predict it in new locations.

Spatial epidemiological tools applied to malaria research can identify areas of high malaria transmission and assess potential environmental and other risk factors that may explain spatial differences. Clarifying the relationship between the environment and malaria helps predict the impact of environmental changes on malaria risk, including the impact of global warming and human interventions (dams, changes in agriculture, urbanization, etc.). Understanding the environmental aspects of malaria is important for effective antimalarial interventions that not only directly target the parasite, but also the vector mosquito and its living conditions. Malaria spread maps provide estimates of disease burden and help evaluate intervention programs.

### 2.17 Vector *Anopheles* Mosquitoes

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The biology of the main African vectors of malaria has been part of the literature for more than 50 years. Vectors have been variously described and identified as subspecies, forms, varieties, races, etc. These were made in relation to morphological differences, distribution, biology, ecology and behavior, among others. In West and Central Africa, five different species are considered the main vectors of malaria: *An. gambiae, An. arabiensis, An. funesto, An. nili* and An. *moucheti* At least 4 to 5 other species are considered as important or locally important secondary vectors (Sameer Dixit *et al.,* 2018), e.g. *An. paludis, An. Hancocki, An. mela* and others.

### 2.17.1 *Anopheles-Gambiae* Complex

*An. gambiae sensu stricto* (ss), *An. arabiensis* and An. melas are Anopheles complexes found in West and Central Africa. *In. gambiae* predominates in humid environments, while *An. arabiensis* is more common in drier areas, but they are sympatric over a wide area. The saltwater species *An. melas* breeds in mangroves along the west coast of Africa south to Namibia (Coetzee *et al.,* 2000). Species are identified based on fixed paracentric inversions or PCR-based diagnostic tools, detecting species-specific sequence differences in the ribosomal DNA intergenic spacer region (rDNA-IGS). Also, *karyotype* distributions naturally indicate. *Gambiae* for large and persistent deviations from Hardy-Weinberg equilibrium, as some *heterokaryotypes* are deficient or even completely absent. For this reason, five chromosomal forms exist in West Africa, designated in *non-Linnean nomenclature* as Bamako, Bissau, Forest, Mopti and Savanna (Favia *et al.,* 2001). Recently, rDNA-IGS analysis identified robust sequence differences between sympatric and synchronous *chromosomal* forms of Savannah, Bamako and Mopti populations in Mali and Burkina Faso, leading to the designation of two *non-panmictic* molecular forms called S. and M. forms. Both molecular forms are found in West and Central Africa (Favia *et al.,* 2001). All specimens from *Mopti* identified so far belong to molecular form M; However, outside of Mali and Burkina Faso, the M form may have *chromosomal* arrangements typical of the Bissau, forest, or savannah forms. Molecular form S may also carry standard chromosomes indicative of Forest form or typical Savanna and Bamako karyotypes. Although some very rare M/S hybrids have been found in Sierra Leone, Mali and Cameroon, evidence of reproductive isolation between molecular forms is widespread enough to suggest early speciation (Favia *et al.,* 2001) or , for example, in southern Cameroon. , A population genetic study based on microsatellite DNA markers reported a large genetic difference between sympatric M and S populations, both within the standard forest *chromosomal* form of *An. gambiae* (Menze *et al.,* 2018). Insecticide resistance has been reported in almost all West African countries (Favia *et al.,* 2001).

A copy of *An. arabiensis* from Burkina Faso was also found to carry the resistance allele. Other resistance mechanisms (resistant AChE, esterases, oxidases, Rdl, GST) have also been identified in populations of *An. Gambiae* have been described in West and Central Africa (Favia *et al.*, 2001).

### 2.17.2 *Anopheles Funestus* Group

The *funestus* group includes at least eleven species: *An. funesto Giles, An. vaneedeni Gillies* and *Coetzee, An. rivulorum Leeson, An.* similar to *rivulorum, An. Leesoni Evans, An. Confusion Evans* and *Leeson, An. parensis Gillies, An Service Brucei, An. Aruni Sobti, An. fuscivenosus Leeson* and an Asian member *An. fluviatilis* James (Favia *et al.,* 2001). These species are not all sympatric. Originally, members of the group could only be distinguished by their *karyotype* (Moody *et al.,* 2000). More recently, however, simpler than differing PCR-based tests have been developed among group members. For example, the PCR test based on species-specific single nucleotide polymorphisms (SNPs) in the internal transcribed spacer region 2 (ITS2).

*Anopheles funestus* is widespread in sub-Saharan West Africa. It has been known since the 1930s that this group is made up of several very closely related species, distinguished only by very minor morphological characteristics at the larval or adult stage (Rossi *et al.,* 2008) or by a recently developed PCR test (Coetzee & Fontenille, 2004) NAIL. *Funesto, An. Leesoni, An. rivulorum* and *An. brucei* are found in West and Central Africa. Their biology and vectorial capacity are very different. With the exception of *An. funestus*, these species feed on animals, not humans, so they are generally not vectors of malaria. In 2003, (COHUET et al., 2003) described a new taxon closely related to *An. rivulorum* on the basis of biological, morphological and genetic characteristics. This taxon, provisionally similar to *An. rivulorum*, recorded in Burkina Faso and Cameroon; it differs from *An. rivulorum* from South Africa and does not appear to play a role in the transmission of malaria.

*Anopheles funestus* itself is highly *polymorphic*, both biologically and genetically, with at least 11 *paracentric chromosomal* inversions on *chromosomes* 2 and 3. In Burkina Faso, *An. funestus* exhibits enormous *Hardy-Weinberg disequilibrium* and *vessel disequilibrium* between investments, which *(*Ayala *et al., 2009)* to describe two chromosomal forms called Kiribina and Folonzo based on the presence and association of paracentric inversions. In Senegal, you recognized 3 chromosomal populations with different anthropophilic activities and sometimes found in sympathy. In Cameroon, a line of inversion frequencies has been reported ranging from humid forest in the south (with inverted Folonzo-like populations) to dry savannahs in the north (with standard Kiribina populations), with both forms exhibiting *heterozygous* deficiency severe when they are nice. All these data suggest a restricted gene flow between the chromosomal forms of *An. Funkier* However, several observations from Cameroon (and East Africa) report no signs of sympathy between *Folonzo* and *Kiribina*, and the *heterokaryotypes* reported were, in fact, the expected

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frequencies within the populations. The use of *microsatellite* markers in Senegal and Cameroon showed that gene flow between chromosomal forms is allowed and suggested isolation due to geographic distance between populations. These results suggest that *heterozygous* deficits at *chromosomal* loci are primarily locus-specific and arise due to environmental selection on the inversions themselves (or the genes they contain) (Ayala *et al.,* 2009). *Pyrethroid* resistance has not been reported in West African populations of *An. funestus*, unlike the findings in *Mozambique* and South Africa, which seriously hampers vector control.

### 2.18 Habitats of Mosquito Larvae

Habitat and climate determine which species of mosquitoes are found in an area. The needs of the larvae can be very specific and vary considerably. Mosquito larvae are found in many habitats. Each habitat produces and displays a seasonal trajectory of mosquito species. There are about four different types of mosquito habitats, e.g. running water, transient water, and permanent water containing habitats.

#### 2.18.1 Running Water

Mosquito larvae consume a lot of energy to avoid being pushed out of waterways when the water level rises sharply. The tropical *genus Chagasia* and some species of Anopheles breed in waterways. Although at. *quadrimaculatus*, *Culex territans* and *Uranotaenia sapphirina* breed in streams, preferring other habitats. The larvae attach themselves to the vegetation along the banks to avoid being carried away by water currents.

#### 2.18.2 Transitional Water

*Aedes* and *Psorophora* use temporary water sources, such as flooded areas, snow puddles, and ditches, as breeding sites because their eggs cannot withstand drying out. Their life cycles require alternating wet and dry periods. Opportunistic species such as *Culex* can reproduce even during a prolonged period of flooding. Transient water masses are subject to changes in water quality, causing different species of mosquitoes to use the same reservoir over a period of time.

#### 2.18.3 Continuous Water

These bodies of water (also called semi-permanent) are present for long periods of time and support characteristic aquatic vegetation such as cattails, bulrushes and bulrushes. *Anopheles*, *Culex*, *Culiseta*, *Coquillettidia* and *Uranotaenia* breed in continuous water to prevent their eggs from drying out. *Aedes* adults lay their eggs near the edge of swamps or in clumps of vegetation and must be flooded to drown the eggs and hatch. The species present, the vegetation and the water quality change with the seasons.

#### 2.18.4 Containers

Containerized aquatic habitats are found in natural environments, such as B. water stored by plants (bromeliads), and in man-made environments such. B. Water in tires. Container habitats are based on the containers themselves. Tree hollows usually have tannin-enriched water that is typically clear, with rotting wood at the bottom. Many species of hollow trees also use artificial containers such as tires because they provide weather protection and are more common. Man-made containers are a convenient way to transport a species of mosquito to a location outside of its natural habitat.

### 2.19 Susceptibility to Becoming a Disease Vector

Some species of Anopheles are poor carriers of malaria because the parasites they contain do not develop (or do not develop at all). Laboratory experiments have made it possible to select strains of *An. gambiae* resistant to infection by *Plasmodium* parasites. The immune system of refractory strains is able to kill malaria parasites after they have penetrated the stomach wall of the mosquito. The genetic mechanism of this response is currently being investigated. It is hoped that one day, genetically engineered malaria-resistant mosquitoes could control or even eliminate malaria, replacing wild mosquitoes that are not resistant to the *Plasmodium* parasite.

### 2.20 Preferred Blood Meal Source

An important behavioral factor is that a species of *Anopheles* prefers to feed on humans (*anthropophily*) or animals other than animals (*zoophily*). *Anopheline* mosquitoes are more likely to transmit malaria parasites from person to person. Most Anopheles mosquitoes are neither exclusively *anthropophilic nor zoophilic* (Liverani et al., 2017). The main vectors of malaria in Africa, *An. gambia* and *An. funestus*, are highly *anthropophilic* and therefore two of the most effective malaria vectors in the world. Once ingested by a mustache, *Plasmodium* parasites grow inside the mustache before becoming infectious to humans. Depending on the type of parasite and the temperature, the extrinsic incubation period is between 10 and 21 days. If a female is to survive beyond the extrinsic incubation period, she cannot transmit Plasmodium parasites. It is difficult to directly determine the lifespan of mosquitoes in the wild. The daily survival of *An. gambiae* in Tanzania was between 0.77 and 0.84 per day, meaning that at the end of a day between 77% and 84% survived (Liverani *et al.,* 2017). Assuming this survival rate is constant throughout a mosquito's adult life,

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only about 10% of female *An. gambiae* survived plus an extrinsic incubation period of 14 days. If the survival rate is 0.9, plus 20% of whiskers surviving after an extrinsic incubation period of 14 days. Spraying litter indoors may impair malaria transmission due to adult longevity which may be an effect due to the adult whisker population.

### 2.21 Patterns of Eating and Resting Behavior

Some *Anopheles* mosquitoes are active at dusk or dawn, while others are nocturnal (active only at night). Some *Anopheles* mosquitoes feed internally (*endophages*), while others are *exophages.* After all, some mosquitoes prefer to nest indoors (*endophiles*) while others prefer to nest outdoors (*exophiles*), but this varies by region, local vector ecotype, Vector chromosomal composition, and host type and local microclimatic conditions vary. Insecticide-treated mosquitoes (ITNs) and improved housing designs that prevent mosquito entry (e.g, window mosquitoes) can reduce biting by nocturnal *endophagous Anopheles* mosquitoes. *Endophilic* whiskers are easily controlled by spraying interior areas with remaining effective insecticides. On the contrary, *exophagic* and *exophilic* vectors are better controlled in the destruction of mosquito breeding sites (Liverani *et al.,* 2017).

### 2.22 Vector Capacity and Competence in Malaria Transmission

The vector capacity of a mosquito population largely determines the intensity of vector-borne disease transmission. Vector competence is also a crucial parameter for the transmission of pathogens. In human malaria, vector systems are limited in number.

Only female *Anopheles* mosquitoes are capable of transmitting *Plasmodium* to humans, and out of more than 450 known species of *Anopheles* mosquitoes, 60 are considered true vectors in nature (Dev & Manguin, 2021). Vector capacity and competence also exhibit quantitative characteristics as some species play an important role in malaria transmission and others play a minor role. Even at the species level, certain populations or mosquitoes may have differential effects on transmission (Dev & Manguin, 2021). Research aimed at understanding the genetic determinants of ability and competence has greatly benefited from the availability of the complete *Anopheles gambiae genome* sequence (Benito & Rubio, 2002), with the identification of candidate genes in progress. However, the various aspects of vector capability and competence have not been consistently studied and some have been largely overlooked. For example, rapid advances have recently been made in mosquito immunity and odor genetics (Christophides, 2005), however, the genetic determinants of parasite virulence and mosquito adaptation to the human environment remain narrower areas of research. In addition, evolutionary pressures on vectors, including forces exerted by the parasites they transmit, can have important implications for malaria transmission and are rarely considered for their impact on malaria control measures. Here we discuss the main aspects of vector capacity and competence and the evolutionary forces that influence them in *Anopheles*: vector longevity; the duration of *sporological* development; contact between the mosquito and the appropriate vertebrate host for the parasite; and vector susceptibility/resistance to the parasite.

### 2.23 Vector Lifetime

The development of malaria parasites in vector mosquitoes requires the passage of two *epithelia* and results in thousands of parasites (Vanelle *et al.,* 2012b). Therefore, the development of *sporoges* could induce some degree of virulence and compromise the host fitness of the vector. The cost of adaptation to infection can be expressed as reduced survival or reduced fertility, but an impact on survival would have a much greater impact on malaria transmission, as the vector must live long enough to become infectious. . Several mechanisms of *Plasmodium* virulence against mosquito vectors are expected. Some have been tested, but mainly in experimental *Plasmodium Anopheles* systems. The results on reduced life expectancy are not consistent with many studies showing that vector survival is not affected by infection, but some show the opposite (Ferguson & Read, 2002).

### 2.24 Resistance to Insecticides

Indoor spraying with insecticides and ITNs are the methods to eliminate mosquito bites indoors. However, prolonged exposure to an insecticide has led to resistance. Mosquito resistance to some insecticides was not discovered until a few years after insecticides were introduced. There are over 125 species of mosquitoes that have shown resistance to one or more insecticides, frustrating global malaria eradication campaigns. Appropriate use of insecticides in mosquito control can significantly stabilize the development and spread of resistance. However, this is constantly thwarted by the inappropriate use of insecticides in agriculture, which has long contributed to the resistance of mosquito populations. Therefore, all control measures should include an initial search for insecticide and drug resistance by mosquitoes and *Plasmodium* (Corbel *et al.,* 2002).

#### 2.24.1 Vector longevity, Insecticide Resistance and Control of Malaria Transmission

Insecticides reduce the lifespan of mosquitoes, the most important parameter of vectorial capacity. However, insecticide resistance limits the effectiveness of vector control measures and can interact with the parasite (Ayala & Coluzzi, 2005).

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### 2.24.2 New Strategies Could Limit the Emergence of Resistance.

The use of insecticides for agriculture or public health generates strong selection pressure (Antonio-nkondjio *et al.,* 2006). Multiple resistance mechanisms have arisen independently in malaria vectors and/or have been able to spread despite strong barriers to gene flow. For example, in An, several mutational events were selected independently. Gambiae were transmitted to other members of the *Gambiae* complex by *introgression* (Diabate & Tripet, 2015).

### 2.24.3 Impact of Insecticide Resistance on Infection Costs

Genetic resistance to insecticides has been shown to affect the level of infection in the invertebrate host. For example, several studies have shown that insecticide-resistant *Culexes* were more heavily infected with *Wolbachia* and suffered higher infection costs (Chapman *et al.,* 2006). In contrast, in the case of filarial infection, insecticide-resistant mosquitoes were less infected than susceptible ones. Insecticide resistance may affect parasite transmission in mosquitoes by altering potential redo responses in several tissues, and it has therefore been suggested that it may provide direct protection against infection (McCarroll & Hemingway, 2002). To our knowledge, no studies have been published on the relationship between insecticide resistance in *Anopheles* mosquitoes and the level of infection/cost of *Plasmodium*, although they would be very relevant for the control of malaria.

### 2.25 Rate of Human-Mosquito Contact and Human Bite

The density of vectors in contact with humans and the preference of host animals for mosquito blood meals are closely linked. The *anthropophilic* behavior of *An. gambiae* is an important factor due to its high vectorial capacity. The hypothesis developed by *Coluzzi* explains the increase in contact between humans and *An. gambiae* a few thousand years ago and the drastic changes in vectorial capacity that followed (Ayala & Coluzzi, 2005). 3,000 years ago, widespread penetration of forests began with Bantu peoples, who established agriculture through logging. The ancestors of *gambiae*, previously unable to survive in forests, could find suitable sunny breeding sites and invade this new ecological niche. At the same time, strong selective pressure against livestock due to *trypanosomiasis* meant that humans were the most common large vertebrate hosts available in these areas (Ayala and Coluzzi, 2005). By providing the breeding grounds and blood meal of newly arrived Anopheles, humans served as "food and shelter" and selected for the highly specialized species *An. gambiae*, whose biology became heavily dependent on humans. This specialization in people was selected differently among members of the *An. Gambiae* Complex. The adaptation of these different species to different environments and their associated feeding behavior has been accompanied by the fixation of different chromosomal arrangements known to protect the co-adapted alleles from recombination.

The association between chromosomal inversions and host preference provides evidence for a genetic basis for feeding behavior (TIRADOS *et al.*, 2006) and makes it susceptible to selective forces. The rapid adaptation of *An. Gambia s.s.* to humans and the specialization of members of the complex to different environments is a clear example of its genetic diversity and plasticity.

The adaptation of the ancestors of *An. gambiae* to humans was accompanied by a dramatic increase in *P. falciparum* transmission. The traditional view of the story emphasizes the benefit of the vector's adaptation to its vertebrate host, but this could also be the result of the parasite's selective pressure to increase its transmission, which may have enhanced the specialization. Several experiments have demonstrated the ability of Plasmodium to alter the feeding behavior of the mosquito host. Vectors show a preference for biting gametocyte-infected human hosts and pregnant females (which are generally more infected) and infected vectors are more aggressive (OBISIKE, 2020). One might think that the proportion of infected mosquitoes in the wild would not allow a strong selective pressure of the parasite on the behavior of mosquitoes. However, given the daily mortality rate of An. gambiae (estimated at 10-18% (Dr. Ramesh M *et al.*, 2020)) and the long development of sporozoites (10-14 days), the commonly observed infection rate of *sporozoites* of 5% means that a large proportion of *An. gambiae* are in contact with the parasite throughout their lifetime, suggesting that feeding behavior may be under selective pressure to increase parasite transmission.

The genetic determinants of adaptation to the human environment and the feeding behavior of malaria vectors remain largely unknown. Current research is based on the hypothesis that smell plays a crucial role in behavior, at least in the choice of the host mosquito for a particular blood meal.

Recent descriptions of cellular and molecular odorous components (Ali H. Hallem *et al.,* 2019) offer promising avenues for understanding how mosquitoes select their vertebrate host for a blood meal, and therefore the potential to modify their feeding behavior to adapt. . to limit the transmission of malaria.

### 2.26 Mosquito Vector Control

More than 120 years after the discovery of *Plasmodium* by Laveran, malaria remains a major public health problem in sub-Saharan Africa. Between 1955 and 1968, the attempt to control malaria consisted of achieving the global eradication of malaria by indoor residual spraying (IRS) of each dwelling with residual insecticides (DDT, DLN, HCH, various *organophosphates* ). This program did not include sub-Saharan Africa, which was in the pre-eradication phase due to, among other things, a lack of funds and technical

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and operational problems. The program mentioned above was transformed in 1969 into the fight against malaria with 4 technical variants dealing exclusively with diagnosis and treatment. The 1992 WHO global strategy recommended not only case management, but also selective and sustained vector control for the prevention of malaria. Insecticide-treated nets (ITNs) and other materials, as well as IRS, which are still effective and widely used in several countries, mainly in southern Africa (Craig *et al.,* 2005) and Burundi, were then used to fight malaria. This approach stopped the 1987 malaria epidemic in Madagascar and KwaZulu Natal, South Africa. At the 2000 Africa Malaria Summit in Abuja, it was agreed to use appropriate and sustainable measures to strengthen health systems. The Summit agreed that 60% of the risk of malaria should be eliminated by 2005, particularly among children under five and pregnant women, through personal and community protective measures, such as the use of ITNs and other measures available at low cost. cost to prevent infection and disease.

In West African countries, Anopheles mosquitoes are controlled through the large-scale use of ITNs and other impregnated materials, as they are effective in reducing the incidence of malaria and the overall infant mortality in countries like Ghana (Pates *et al.,* 2005). Additionally, trials have shown a profound effect of permethrin-treated nets in Ghana, Kenya (Mansell *et al.,* 2006) and with impregnated curtains in Burkina Faso and there was no subsequent rebound in mortality even after several years of ITN use. With 60-80% of the population covered, even people not covered by ITNs can be protected against malaria if they live in treated facilities or within 300m.

### 2.27 Mosquito and Malaria Control

Understanding the biology and behavior of Anopheles mosquitoes can help understand how malaria is transmitted and can aid in the development of appropriate control strategies. Factors that affect a mosquito's ability to transmit malaria include its innate susceptibility to *Plasmodium*, host choice, and lifespan. The susceptibility of *Anophelines* to insecticides and their preferred feeding and resting habitats should be carefully considered when designing control programs.

Although malaria has existed since time immemorial, it was eradicated in Europe, North America, the Caribbean and parts of Asia and South Central America during the first regional eradication campaigns in the late 1940s. Achieved similar results in sub-Saharan Africa.

## III. Materials and Methods

### 3.1 Materials

The responsibility for the interpretation and use of the materials for larva collections lies with the target area so that larval habitants can be accessed and treated.

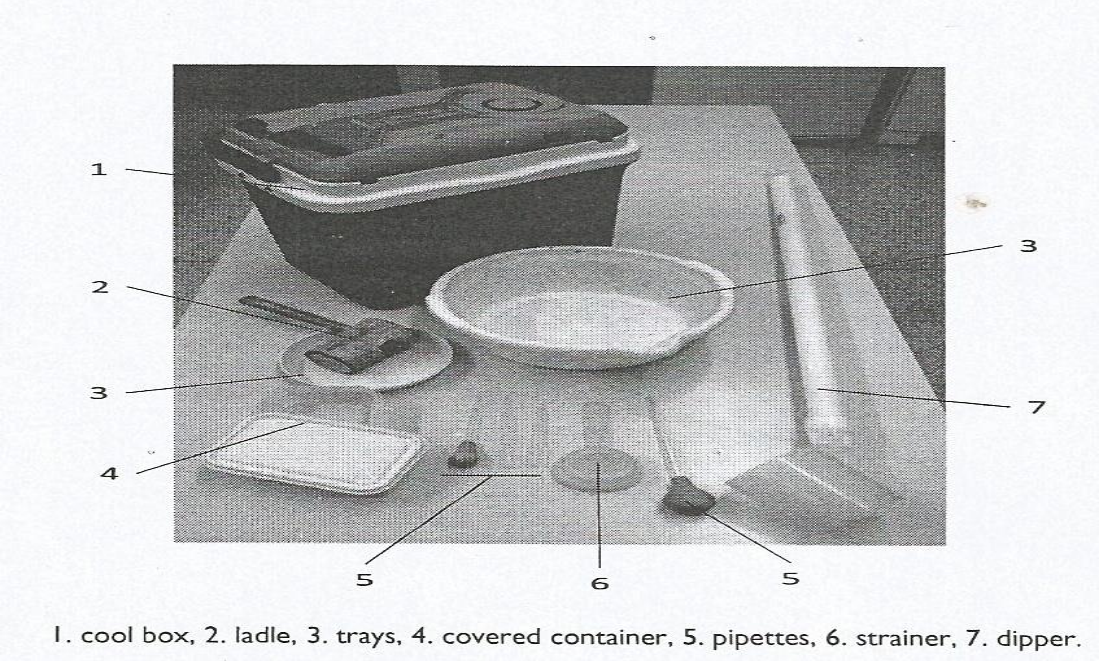


Figure3.1: Main Materials for Larva Collections

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There are seven (7) materials for larva collection from the above figure such as cool box, ladle, trays, covered container, pipettes, strainer and dipper.

**Cool Box:** Mosquitoes breed as larvae in cooler water. There are good larvicide like *diflubenzuron* of Bayer, available for this purpose.

**Ladle:** Ladle material are used for larvae collect sample of larvae, larva densities before treatment are taken by ladle.

**Trays:** The mosquito mass-rearing trays was designed to provide a large surface area to evaluate stress on larvae and pupae during the collection.

**Covered Container:** The container-inhabiting *Aedes* mosquitoes are the major vectors to detect larvae and pupae and associated socioeconomic surveys to collect.

**Pipette:** The net is an effective means of collecting *anopheline* larvae and was to use a pipette to remove all the mosquito larvae that were at.

**Strainer:** The purpose of this material is to provide specific and laboratory-reared mosquito larvae of known age or instars.

**Dipper:** Dipper for the collection of mosquito larvae and pupae is a patented telescoping water sampling dipper primarily intended for use by vector field.

### 3.2 Developmental Stages of *Anopheles* Mosquito

There are four stages in the life cycle of a mosquito which includes egg, larva, pupa and adult. During its life-cycle the mosquito undergoes two changes from larva to pupa and from pupa to adult (metamorphoses). The developmental stage of a mosquito is part of materials.

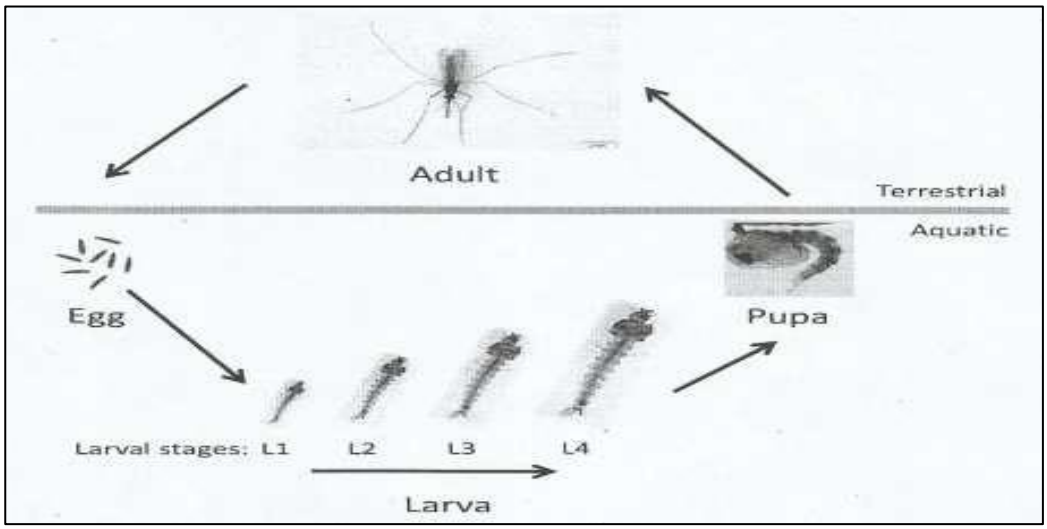


Figure 3.2: Stages of the life cycle of the Anopheles Mosquito

**Egg Stage**

a) The adult female *Anopheles* mosquito mates once and lays eggs throughout its life.

b) Females should feed on blood every 2-3 days. Blood is needed to develop eggs. Females lay a series of eggs before the next blood meal.

c) Eggs are laid in batches of 50 to 200 eggs in water (rain ponds, ponds, banks, lakes, etc.).

d) The hatching time of the eggs depends largely on the temperature.

e) At about 30°C, the eggs hatch into larvae in about 2-3 days.

f) In temperate zones (16oC), about 7-14 days

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**Larva Stage**

a) The larva has a well-developed head with "mouth brushes" for feeding (filtering). The larva feeds on micro-agents (eg algae, bacteria) and organic matter in the water in which it reproduces.

b) The Anopheles larva does not have a respiratory siphon. It is parallel to the surface of the water to breathe.

c) There are four stages of larval development called instars (designated L1 to L4, Fig. 3.2).

d) Development from larva to pupa takes 5-10 days at normal tropical temperatures, depending on the species. Water temperature affects the time required for development, which is shorter in warmer waters.

**Pupa Stage**

a) The pupa is comma-shaped and stays on the surface of the water.

b) It has a pair of breathing trumpets through which it breathes when on the surface.

c) During this phase, there is no feeding, but the pupa is mobile and responds to stimuli.

d) It is the dormant or inactive phase during which there is a great transformation from aquatic life to surface life and extra-aquatic life.

e) The pupa stage lasts about 2 to 5 days.

**Adult Stage**

a) The adult animal usually emerges from the pupa at dusk.

b) After emerging from the pupa, the adult mosquito rests briefly to harden its body.

c) Both male and female mosquitoes feed on nectar for energy.

d) After mating, the female mosquito seeks blood for the development of her eggs. In some species, one diet is enough to develop the eggs. In other species, two feedings are necessary, at least for the development of the first eggs.

e) The time between the egg and the adult Anopheles can vary from 7 days at 31°C to 20 days at 20°C

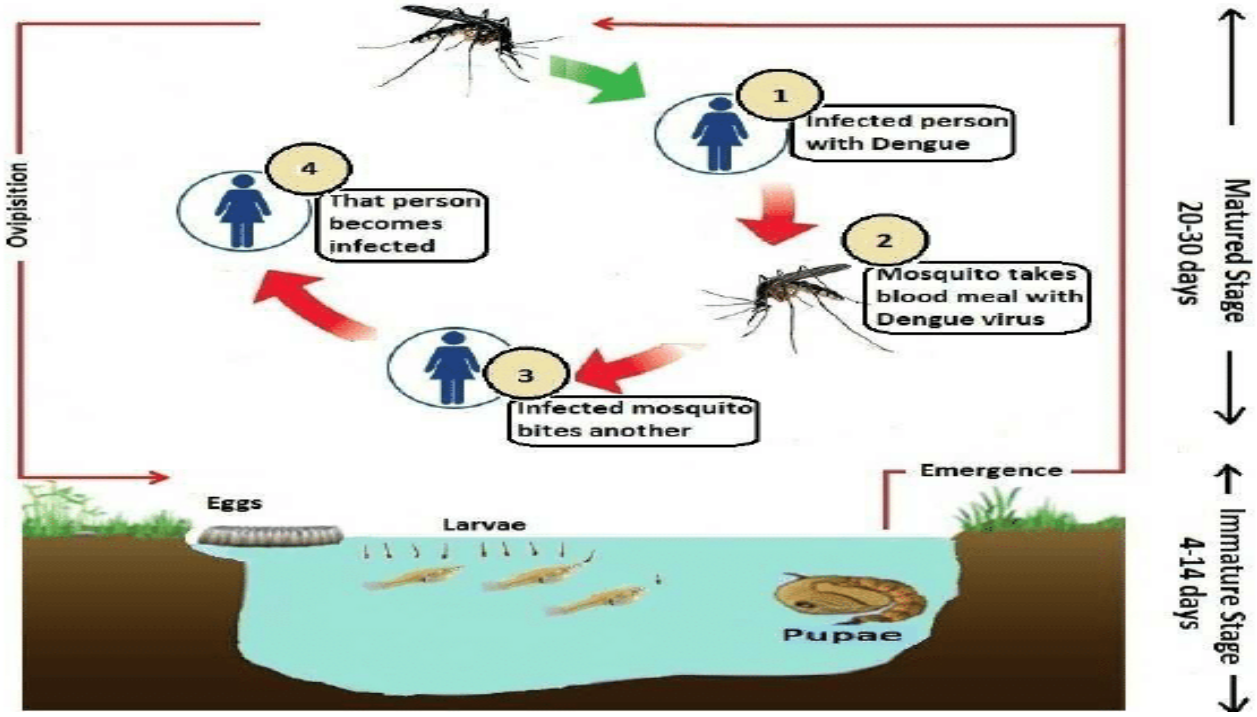


Fig: 3.3 Mosquito Life-cycle with Predator and Disease Transmission among Human

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### 3.3 Malaria Parasite

The malaria parasite is a vector-borne disease caused by *protozoan* parasites of the *genus Plasmodium*. There are four types of human malaria parasites: *P. falciparum, P. vivax, P.* malariae and *P. ovale*. Parasites are transmitted from person to person by female mosquitoes of the *genus Anopheles*.

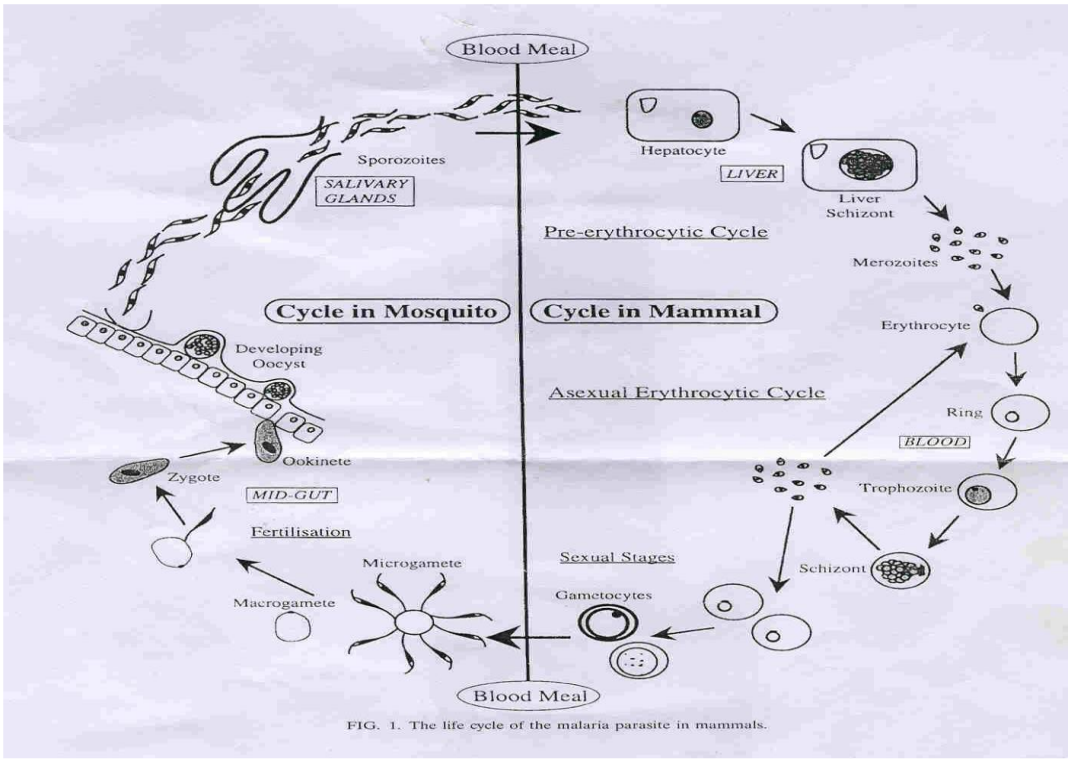

FIG. 1. The life cycle of the malaria parasite in mammals.

Figure 3.4: The life cycle of the Malaria Parasite. (Source: Phillips 2001).

The life cycle of the parasite begins with the inoculation of the parasite into human blood through the bite of a female *Anopheles* mosquito. Within half an hour, the *sporozoites* reach the liver and invade liver cells. Inside liver cells, *trophozoites* initiate their intracellular asexual division. At the end of this phase, thousands of *erythrocyte merozoites* are released from each liver cell. The time required to complete the tissue phase varies according to the infecting species (5 to 6 days for P. *falciparum*). *Merozoites* invade red blood cells (RBCs) and then grow through the ring, *trophozoite*, early *schizont*, and mature stages; Each mature *schizont* consists of thousands of *erythrocyte merozoites* (Wardrop *et al*., 2013). These *merozoites* are released after *lysis* of red blood cells and immediately invade uninfected *erythrocytes*. This whole invasion-multiplication-release-invasion cycle lasts about 48 hours in *P. falciparum* infections. The contents of the infected cell, released upon *lysis* of red blood cells, stimulate tumor necrosis factor and other cytokines, leading to the characteristic clinical manifestations of the disease. A small proportion of *merozoites* develop into gametocytes. Mature gametocytes appear in peripheral blood after a period of 8 to 11 days after the primary attack by *P. falciparum*. They increase in number for up to three weeks, then decrease, but circulate for several weeks. Gametocytes enter the mosquito when it bites an infected person. The malaria pathogen in the human vector of malaria is transmitted by mosquitoes of the genus *Anopheles*. Of the 360 species, there are about 45 with the ability to transmit malaria to humans. *Anopheles* lives worldwide, but malaria transmission occurs primarily in tropical and subtropical regions of the world. *Anopheles*-free always means malaria-free, but not the other way around. If the malaria pathogen enters the mosquito after a blood meal, the gametocytes continue their development (*sporogony*). Male and female *gametes* fuse and form a *zygote*. This develops into an *ookinete*, which penetrates the intestinal wall and develops into an *oocyst*. The *oocyst* divides asexually into numerous *sporozoites*, which reach the mosquito's salivary gland, where they can be transmitted during the mosquito's next blood meal. *Sporogony* in the mosquito lasts about 10-20 days depending on the air temperature, then the mosquito remains infectious for 1-2 months if it survives.

At a temperature below 15°C, there is no *sporogony*. Only the female mosquito takes a blood meal (male *Anopheles* feed on nectar) necessary for the development of eggs. Two to three days after the blood meal taken at night or at dawn, the female *Anopheles* mosquito lays about a hundred eggs. Therefore, it can produce over 1,000 eggs in its multi-week lifespan. Eggs are always laid on the water surface, preferably in swamps or shallow water. They can also breed in water reservoirs or tree cavities. The oval eggs

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are a millimeter long and take about two weeks to develop into adult mosquitoes. They only travel short distances of a few kilometers. Their preferred location is near human habitation. There are behavioral differences between mosquito species that are important for studying the geographic distribution of the vector (Wardrop *et al*., 2013).

### 3.4 Natural Predator

The term "natural predator" is used for organisms that kill or injure other animals. For example, copepods, tadpoles and barn swallows are natural enemies of mosquitoes, predators or parasites are natural enemies of insect pests. Spiders are natural enemies of stem borers. Furthermore, pathogens are natural enemies. In this work, we interrupt the life cycle of the *Anopheles* mosquito with some natural enemies, which is a biological mosquito control method, thus reducing or eliminating the mosquito threat. We will briefly explain the three forms of natural predators that will be used for the success of this work.

#### 3.4.1 Copepods

Copepods are small crustaceans (shrimps, crabs, lobsters, and relatives) found in both freshwater and saltwater habitats. They are voracious or timid predators that control the production of mosquitoes in water retention areas. Knowing where mosquitoes breed is very important for effective control of copepod mosquitoes.

Figure 3.5: Copepods

#### 3.4.2 Tadpoles

Aquatic larva of frogs, toads, etc., which develops from a limbless tail form with external gills to one with internal gills, limbs and a reduced tail. They are voracious or shy predators that controlled mosquito production in water holding areas. Knowing where mosquitoes breed is very important for effective mosquito control with tadpoles.

Figure 3.6: Tadpoles

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### 3.4.3 Purple Martins

The purple swallow is a passerine bird in the swallow family *Hirundae*. It is the largest swallow in North America. Despite their name, Purple Martins are not actually purple. Their dark bluish-black feathers have an iridescent sheen caused by the refraction of incoming light, giving them a light blue to navy blue or dark purple appearance. In certain lighting conditions, they may even appear green. They are known for their speed, agility, and distinctive combination of rapid kicking and gliding patterns. Approaching their nesting site, they tumble from the sky at high speed, wings folded**.**

Figure 3.7: Purple Martins

### 3.5 Microscope

A microscope is a laboratory instrument used to examine objects too small to be seen with the naked eye. Microscopy is the science of examining small objects and structures under a microscope. Microscopic means invisible to the eye unless assisted by a microscope. In this work, we use it to observe the different stages of the life cycle of mosquitoes.

Figure 3.8: Viewing the Larva Stages of Mosquito Using Binocular Microscope

### 3.6 Methods

The modified model is used to study the uniqueness, existence, stability analysis of disease-free steady states, analyze, solve and perform numerical simulations showing a graphical representation of the results, three methods would be used. We would use

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the point of equilibrium or steady state, Beltrami conditions, Dikeman conditions and finally we would use Maple software to display the results when three natural predators are introduced simultaneoulsy.

### 3.7 Sampling Methods for Collecting Larvae

There are several methods for sampling larvae. The application of individual sampling methods depends on the type and type of hatchery and is described in the following sections. The larval collector should approach the hatchery with caution, as any disturbance will cause the larvae and pupae to descend and become inaccessible. It is important that the collector does not cast a shadow on the water. If the larvae and pupae are moving, it may be necessary to remain still until they swim again.

#### 3.7.1 Diving (Dipping) procedures

1. This method is generally used to sample relatively large bodies of water, such as swamps, ditches, streams and rice fields.
2. The bucket should be gently lowered at an angle of approximately 45° to minimize disturbance and skim the surface of the water or gently lowered to allow water and nearby larvae to drain into the bucket. Be careful not to spill any water when you take the bucket out of the water.
3. The larvae should be removed from the spoon with a pipette and transferred to a properly labeled bottle or vial.
4. If vegetation appears in the hatchery, the collector should agitate the water and allow the larvae to swim to the bottom, then remove some vegetation to create a clear area for sampling and wait a few minutes before to continue sampling as previously described. . To calculate the larval density, note the number of baths in each hatchery. Also consider the time required for collection.

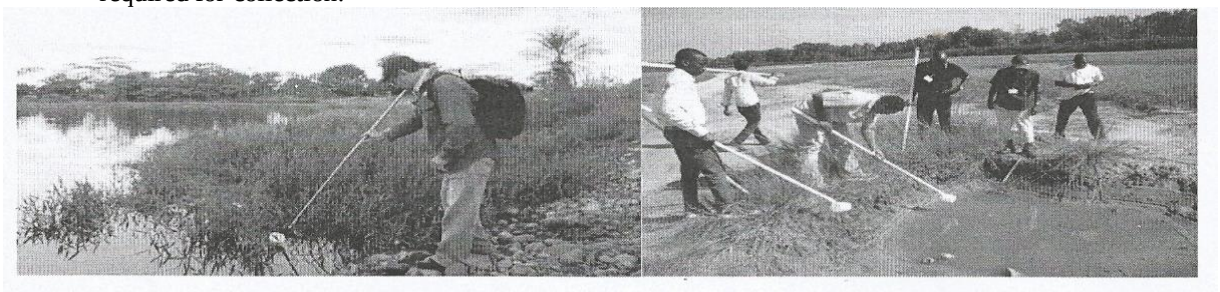

Figure3.9: Sample Collection (Larva) by Dipping

#### 3.7.2 Mode of Remuneration (Netting Method)

This method consists of using a fine-mesh net attached to a handle, with a plastic bottle or hose attached to the end. It is usually used to collect larvae and pupae in larger bodies of water such as ponds and small lakes. The net should be held at approximately a 45º angle to the water surface and pulled across the surface. The larvae and pupae are collected in the plastic bottle at the end.

Figure 3.10: Sample Collection (Larva) by Netting

#### 3.7.3 Pipetting Method

This method is used to collect larvae from small breeding sites, such as small puddles, hoof prints, containers, plant axils, and tree cavities.

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Figure 3.11: Larva Collection by Pipetting (left) and Introduction of Natural Predator to the Larvae Tray (right)

Figure 3.12: Showing Larva Rearing Container (left) and Larva Food (right).

### 3.8 Description of the Model

This new model is a control flowchart of the predator-prey interaction model in the mosquito life cycle that considers an open population of mosquitoes and predators. The population is subdivided according to the life cycle of mosquitoes and natural predators. In the life cycle of a mosquito, the population is divided into four compartments: Egg compartment E(t), Larval compartment L(t), Pupal compartment P(t), Adult compartment A(t) and natural Predator divided into three divisions. Copepods $C_P(t)$, Tadpoles $T_P(t)$, and Purple Martins $P_M(t)$.

Mathematical models provide a solid understanding of planning and risk controls in heterogeneous settings, especially when the models are based on vector population ecology and a solid understanding of entomological parameters relevant to transmission. Research conducted by (Killeen & Chitnis, 2014) that mathematical models have also played an important role in understanding the epidemiology of malaria and other infectious diseases; that mathematical models also provide an accurate quantitative description of complex nonlinear processes and a method to relate the individual infection process to the incidence of disease or infection in a population over time, yielding insights important on the introduction of natural predator to increase the interruption of the life cycle of the Anopheles mosquito at the larval, pupal and Adult stages, thereby reducing or eradicating the mosquitoes. This introduction of natural enemies reduces malaria by the biting vector. They work by reducing the intensity of malaria transmission or eradicating malaria. The classification of a natural enemy as predator or parasite largely depends on the number of prey or hosts attacked or consumed the reproductive strategy and other details of the system, in which there are many similarities in characteristics of the natural predator and in the model, to study them. Mathematical modeling of malaria is a challenging area

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of applied mathematics due to its peculiarities in Africa and particularly in Nigeria. Millions of people die of malaria every year. Mosquitoes are resistant to most vaccines we have today. It is important to develop preventives/methods to fight against malaria and mosquitoes in general.

Therefore, each of the two population compartments above is divided into classes below;

A(t) = Number of adult mosquitoes at time(t)

E(t) = Number of eggs at time(t)

L(t) = Number of larvae at time(t)

P(t) = Number of pupae at time(t)

$C_P(t)$ = Number of natural predator for larva (Copepods)

$T_P(t)$ = Number of natural predator for pupa (Tadpoles)

$P_M(t)$ = Number of natural predator for adult (Purple martins)

$N_1 =$ Total population for mosquitoes at time t, $N_1 = A(t) + L(t) + P(t) + E(t)$

$N_2 =$ Total population for predator at time t, $N_2 = P_m(t) + C_P(t) + T_p(t)$

$N =$ Total population at time t, $N(t) = N_1(t) + N_2(t)$

An adult female mosquito interact sexually with males or vice versa at a rate called the incidence rate, given by η. $b_1$is the natural birth rate of the adult class, $\beta_1$ is the induced mortality rate of copepods due to chemical and environmental conditions of the adult class and $\mu_1$ is the natural mortality rate of the adult class. σ is the fraction in which the egg is harsh to larva, $\beta_2$ is the induced mortality rate of the egg due to the chemical and environmental conditions of the egg class, and $\mu_2$ is the natural mortality rate of the compartment to eggs. λ is the fraction at which the larvae transform to pupate, $\beta_3$ is the induced mortality rate of the larvae due to the chemical and environmental conditions of the larval class, and $\mu_3$ is the natural mortality rate of the larvae. $\pi$ is the fraction at which the pupa transforms into an adult, $\beta_3$ is the induced death rate of the pupa due to chemical and environmental conditions of the pupal class, and $\mu_3$ is the natural death rate of the pupa compartment. $b_2$ is the natural birth rate of the copepod class, $\beta_6$ is the induced mortality rate of copepods due to chemical and environmental conditions of the copepod class, $\mu_6$ is the natural death rate of the copepod compartment and α is the probability at which mosquito larvae eaten by copepods. $b_3$ is the natural birth rate of the tadpole class, $\beta_7$ is the induced mortality rate of the tadpoles due to the chemical and environmental conditions of the tadpole class, $\mu_7$ is the natural death rate of tadpoles compartment and ω is the probability at which adult mosquito are eaten up by purple martins. $b_4$ is the natural birth rate of purple martins class, $\beta_5$ is the induce death rate of purple martins due to chemical and environment conditions of purple martins class and $\mu_5$ is the natural death rate of purple martins compartment and γ is the probability at which mosquito adult are eaten up by purple martins.

### 3.9 Table 3.1: Model Variables and Parameters Defined

In table below, variables and parameters used in the new model are defined

| **Variables** | **Description** |
|---|---|
| A(t) | Number of adult mosquitoes at time(t) |
| E (t) | Number of eggs at time(t) |
| L(t) | Number of larvae at time(t) |
| P(t) | Number of pupae at time(t) |
| N(t) | Total population |
| $\boldsymbol{C_P}$(t) | Number of natural Predator for larva at time(t) (Copepods) |
| $T_p$(t) | Number of natural Predator for pupa at time(t)(Tadpoles) |
| $P_m$(t) | Number of natural Predator for Adult at time(t) (Purple Martins) |
| **Parameters** | **Description** |

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| | |
|---|---|
| $b_1$ | Natural birth rate of adult class |
| $b_2$ | Natural birth rate of copepods class |
| $b_3$ | Natural birth rate of tadpoles' class |
| $b_4$ | Natural birth rate of purple martins' class |
| $\mu_1$ | Natural death rate of adult class |
| $\mu_2$ | Natural death rate of egg class |
| $\mu_3$ | Natural death rate of larva class |
| $\mu_4$ | Natural death rate of pupa class |
| $\mu_5$ | Natural death rate of purple martins' class |
| $\mu_6$ | Natural death rate of copepods class |
| $\mu_7$ | Natural death rate of tadpoles' class |
| $\beta_1$ | Induce death rate of adult due to chemical and environmental conditions |
| $\beta_2$ | Induce death rate of egg due to chemical and environmental conditions |
| $\beta_3$ | Induce death rate of larva due to chemical and environmental conditions |
| $\beta_4$ | Induce death rate of pupa due to chemical and environmental conditions |
| $\beta_5$ | Induce death rate of purple martins' due to chemical and environmental conditions |
| $\beta_6$ | Induce death rate of copepods due to chemical and environmental conditions |
| $\beta_7$ | Induce death rate of tadpoles' due to chemical and environmental conditions |
| $\eta$ | The incidence rate (the rate at which adult mosquitoes oviposit) |
| $\sigma$ | The proportion at which egg harsh to larva |
| $\boldsymbol{\lambda}$ | The proportion of larva that transform to pupa |
| $\boldsymbol{\pi}$ | The proportion of pupa that transform to adult |
| $\alpha$ | The probability at which mosquito larva are eaten up by copepods |
| $\omega$ | The probability at which mosquito pupa are eaten up by tadpoles |
| $\gamma$ | The probability at which mosquito adult are eaten up by purple matins |
| **C** | The average temperature of the water culture |
| $N_L$ | Number of larva been eaten up by copepods at time(t) |
| $N_P$ | Number of pupa been eaten up by tadpoles at time(t) |
| $N_A$ | Number of adult been eaten up by purple martins at time(t) |

### 3.10 Model Assumptions

When formulating the model, the following assumptions were made

1) The total population of Anopheles mosquitoes consists of four populations such as egg, larva, pupa and adult.
2) The total population of natural predators consists of three populations such as copepods, tadpoles and purple martins.
3) The parasite of a mosquito, transmitted from one mosquito to another, is transmitted only through the host, this is called horizontal transmission.

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4) Predators can consume infinite amounts of prey.
5) Emigration and immigration of the Anopheles mosquito population does not occur in this population; however, the population increases only by the natural birth rate and decreases only by the natural death rate and also due to environmental factors.
6) The prey population grows exponentially when the predator is absent.
7) The Anopheles mosquito is thought to transmit malaria only through direct contact.
8) The predator population will starve in the absence of the prey population

The following diagram describes the flux control of the predator-prey interaction; It will be useful in formulating models.

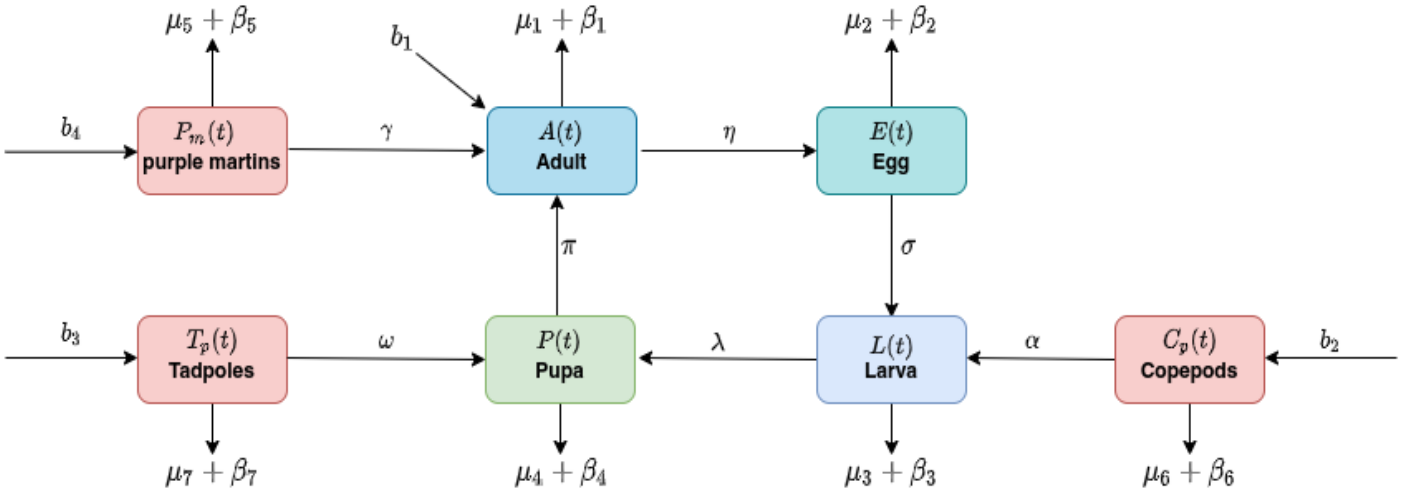


Figure 3.13: Flow Control Diagram of Predator-prey Interaction Model in Mosquito Life-Cycle

### 3.11 Predator-Prey Model

It can be argued that predators and prey are the building blocks of bioecosystems and ecosystems because biomasses have developed from their resource masses. Species compete, evolve and disperse simply to find resources to sustain their struggle for existence. Depending on your specific application setup, plant-herbivore, parasite-host, tumor cell (virus) and immune system interactions, susceptible infections, etc. They deal with general gain-loss interactions and therefore may have applications outside of ecosystems. When seemingly competitive interactions are carefully studied, they are often in fact a form of predator-prey interaction in disguise. The predation rate (p) is the size of the predator population multiplied by the capital mortality rate. $\frac{dP}{dt} = -qp$ Where p is the predator population size and q is the per capita mortality rate.

**Theory 3.1**

The Lotka-Volterra model assumes that a predator's rate of prey consumption is directly proportional to prey frequency. This means that predator feeding is limited only by the amount of prey in the area. Some examples of predators and prey are lions and zebra, bear and fish, and fox and rabbit.

The words "predators" and "prey" almost always refer to animals that eat animals, but the same concept also applies to plants: bears and berries, rabbit and lettuce, grasshopper and leaves.

### 3.12 A General Predator-Prey Model

Consider two populations whose sizes at reference time t are denoted by x(t) and y(t) respectively. The x and y functions can indicate population counts or concentrations (numbers per area) or some other scaled measure of population size, but they are considered continuous functions. Changes in population size over time are described by the time derivatives $\dot{x} = \frac{dx}{dt}$ and $\dot{y} = \frac{dy}{dt}$, respectively, and a general model of the interacting populations is written in terms of two differential equations autonomous

$\dot{x} = xf(x, y)$ and $\dot{y} = yg(x, y)$ {ie the time t does not appear explicitly in the functions xf(x,y) and yg(x,y)}. The functions f and g denote the respective per capita growth rates of the two species. We assume that $\frac{df(x,y)}{dy} < 0$ and $\frac{dg(x,y)}{dx} > 0$. This general environment is often referred to as the Kolmogorov predator-prey model (Henson *et al.*, 2003).

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### 3.13 The Model Equations

From the above assumptions and flowchart, the following equations were derived

#### 3.13.1 Model Equations for Mosquito Life-Cycle

$$\frac{dA}{dt} = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) \quad \ldots \quad (3.13.1)$$

$$\frac{dE}{dt} = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) \quad \ldots \quad (3.13.2)$$

$$\frac{dL}{dt} = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) \quad \ldots \quad (3.13.3)$$

$$\frac{dP}{dt} = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) \quad \ldots \quad (3.13.4)$$

#### 3.13.2 Model Equations for Natural Predators

$$\frac{dC_P}{dt} = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) \quad \ldots \quad (3.13.5)$$

$$\frac{dT_p}{dt} = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) \quad \ldots \quad (3.13.6)$$

$$\frac{dP_m}{dt} = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \quad (3.13.7)$$

#### 3.13.3 Model Equation for Total Population

$$N_1 = A(t) + L(t) + P(t) + E(t)$$

$$N_2 = P_m(t) + C_P(t) + T_p(t)$$

$$N(t) = N_1(t) + N_2(t))$$

$$N(t) = A(t) + L(t) + P(t) + E(t) + P_m(t) + C_P(t) + T_p(t)$$

$$\frac{dN}{dt} = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \quad (3.13.8)$$

## IV. Results and Discussion

### 4.1 Results

#### 4.1.1 Existence and Uniqueness of the Disease-Free Steady State of the model

Here, we would determine the model-free steady-state stability (MFE) by considering the model variables and parameters and using the model equations. Since we have nonlinear eight systems of equations or deterministic ordinary differential equations, we know that it is impossible to obtain an analytical solution of these systems. Therefore, we used the idea of equilibrium point, Beltrami and Diekmann conditions and we also used Maple software to graph the results.

The Mosquito Free Equilibrium (MFE) state of the model by zeroing the left-hand sides of equations (3.13.1-3.13.8), the following model equations associated with (3.13) are given below;

$$b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) = 0 \quad \ldots \quad (4.1.1)$$

$$\eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) = 0 \quad \ldots \quad (4.1.2)$$

$$\sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) = 0 \quad \ldots \quad (4.1.3)$$

$$\lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) = 0 \quad \ldots \quad (4.1.4)$$

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$$b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) = 0 \quad \ldots \quad (4.1.5)$$

$$b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) = 0 \quad \ldots \quad (4.1.6)$$

$$b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) = 0 \quad \ldots \quad (4.1.7)$$

$$b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) = 0 \quad \ldots \quad (4.1.8)$$

Looking at the system of equations, it is now clear that if we have eight unknowns Variables whose values are to be determined; it must be possible to express them in two equations where the two unknowns can be related. To solve a pair of simultaneous equations, two main methods are used as follows:

**Method 4.1.1: Substitution Method**

In the substitution method, one of the two unknowns becomes the subject of the formula in one of the equations. This will then be substituted into the second equation to have a simple equation with one unknown. The equation is then solved linearly to obtain a value, and so the obtained value is substituted to obtain the other unknown.

**Method 4.1.2: Elimination Method**

In the elimination method, one of the two unknowns (which is not present) is eliminated by adding or subtracting the two equations. Note that each unknown to be eliminated must have the same (same) coefficient to facilitate (allow) addition or subtraction.

At this point, we make $C_P(t), T_p(t)\ and\ P_m(t)$ the subject of the formula from equation (4.1.5)-(4.1.7)

From equation (4.1.5), we have

$$\frac{dC_P}{dt} = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t)$$

$$\Rightarrow b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) = 0$$

$$\Rightarrow (\mu_6 + \beta_6 + \alpha)C_P(t) = b_2$$

$$\Rightarrow C_p(t) = \frac{b_2}{(\mu_6 + \beta_6 + \alpha)} \quad \ldots \quad (4.1.9)$$

From equation (4.1.6), we have

$$\frac{dT_p}{dt} = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t)$$

$$\Rightarrow b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) = 0$$

$$\Rightarrow (\mu_7 + \beta_7 + \omega)T_p(t) = b_3$$

$$\Rightarrow T_p(t) = \frac{b_3}{(\mu_7 + \beta_7 + \omega)} \quad \ldots \quad (4.1.10)$$

From equation (4.1.7), we have

$$\frac{dP_m}{dt} = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t)$$

$$\Rightarrow b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) = 0$$

$$\Rightarrow (\mu_5 + \beta_5 + \gamma)P_m(t) = b_4$$

$$\Rightarrow P_m(t) = \frac{b_4}{(\mu_5 + \beta_5 + \gamma)} \quad \ldots \quad (4.1.11)$$

At this point, the substitution method is used to solve the system of equations (4.1.1, 4.1.2, 4.1.3, 4.1.4 and 4.1.8).

From equation (4.1.1), we have

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$$\frac{dA}{dt} = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t)$$

$$\Rightarrow \quad b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) \ = 0$$

$$\Rightarrow (\mu_1 + \beta_1 + \eta)A(t) = b_1 + \gamma P_m(t) + \pi P(t)$$

$$\Rightarrow (\mu_1 + \beta_1 + \eta)A(t) - \pi P(t) = b_1 + \gamma P_m(t)$$

$$where\ P_m(t) = \frac{b_4}{(\mu_5 + \beta_5 + \gamma)}$$

$$let\ M_1 = \ (\mu_1 + \beta_1 + \eta)\ and\ M_5 = (\mu_5 + \beta_5 + \gamma), we\ have$$

$$\Rightarrow M_1 A(t) - \pi P(t) = b_1 + \gamma M_1 \left\{\frac{b_4}{M_5}\right\}$$

$$\Rightarrow M_1 M_5 A(t) - M_5 \pi P(t) = M_5 b_1 + \gamma M_1 b_4 \qquad \ldots \ (4.1.12)$$

From equation (4.1.2), we have

$$\frac{dE}{dt} = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t)$$

$$\Rightarrow \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) = 0$$

$$Let\ M_2 \ = (\mu_2 + \beta_2 + \sigma), we\ have$$

$$\Rightarrow \eta A(t) - M_2\, E(t) = 0 \qquad \ldots \ (4.1.13)$$

From equation (4.1.3), we have

$$\frac{dL}{dt} = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t)$$

$$\Rightarrow \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) = 0$$

$$where\ C_p(t) = \frac{b_2}{(\mu_6 + \beta_6 + \alpha)}\ , M_3 = \ (\mu_3 + \beta_3 + \lambda)\ and\ M_6 = (\mu_6 + \beta_6 + \alpha), we\ have$$

$$\Rightarrow \sigma E(t) - M_3 L(t) = -\alpha \frac{b_2}{M_6}$$

$$\Rightarrow M_6 \sigma E(t) - M_3 M_6 L(t) = -\alpha b_2 \qquad \ldots \ (4.1.14)$$

From equation (4.1.4), we have

$$\frac{dP}{dt} = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t)$$

$$\Rightarrow \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) \ = 0$$

$$where\ T_p(t) = \frac{b_3}{(\mu_7 + \beta_7 + \omega),} M_4 = \ (\mu_4 + \beta_4 + \pi)\ and\ M_7 = (\mu_7 + \beta_7 + \omega), we\ have$$

$$\Rightarrow \lambda L(t) + \omega \frac{b_3}{(\mu_7 + \beta_7 + \omega)} - (\mu_4 + \beta_4 + \pi)P(t) \ = 0$$

$$\Rightarrow \lambda L(t) + \omega \frac{b_3}{M_7} - M_4 P(t) \ = 0$$

$$\Rightarrow M_7 \lambda L(t) + \omega b_3 - M_4 M_7 P(t) \ = 0$$

$$\Rightarrow M_7 \lambda L(t) - M_4 M_7 P(t) \ = -\omega b_3 \qquad \ldots \ (4.1.15)$$

From equation (4.1.8), we have

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$$\frac{dN}{dt} = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t)$$

$$\Rightarrow b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) = 0$$

$$where\ P_m(t) = \frac{b_4}{(\mu_5 + \beta_5 + \gamma)}, C_p(t) = \frac{b_2}{(\mu_6 + \beta_6 + \alpha)}, T_p(t) = \frac{b_3}{(\mu_7 + \beta_7 + \omega),}\ M_1 = (\mu_1 + \beta_1 + \eta), M_2 = (\mu_2 + \beta_2 + \sigma), M_3 = (\mu_3 + \beta_3 + \lambda), M_4 = (\mu_4 + \beta_4 + \pi), M_5 = (\mu_5 + \beta_5 + \gamma), M_6 = (\mu_6 + \beta_6 + \alpha)\ and\ M_7 = (\mu_7 + \beta_7 + \omega)\ we\ have$$

$$\Rightarrow b_1 - (\mu_1 + \beta_1)A(t) - (\mu_2 + \beta_2)E(t) - (\mu_3 + \beta_3)L(t) - (\mu_4 + \beta_4)P(t) + b_2 - (\mu_6 + \beta_6)\frac{b_2}{(\mu_6 + \beta_6 + \alpha)} + b_3 - (\mu_7 + \beta_7)\frac{b_3}{(\mu_7 + \beta_7 + \omega)} + b_4 - (\mu_5 + \beta_5)\frac{b_4}{(\mu_5 + \beta_5 + \gamma)} = 0$$

$$\Rightarrow b_1 + b_2 + b_3 + b_4 - (\mu_1 + \beta_1)A(t) - (\mu_2 + \beta_2)E(t) - (\mu_3 + \beta_3)L(t) - (\mu_4 + \beta_4)P(t) - (\mu_6 + \beta_6)\frac{b_2}{(\mu_6 + \beta_6 + \alpha)} - (\mu_7 + \beta_7)\frac{b_3}{(\mu_7 + \beta_7 + \omega),} - (\mu_5 + \beta_5)\frac{b_4}{(\mu_5 + \beta_5 + \gamma)} = 0$$

$$let\ M_1{}^* = (\mu_1 + \beta_1), M_2{}^* = (\mu_2 + \beta_2),\ M_3{}^* = (\mu_3 + \beta_3),\ M_4{}^* = (\mu_4 + \beta_4), we\ have$$

$$\Rightarrow b_1 + b_2 + b_3 + b_4 - M_1{}^* A(t) - M_2{}^* E(t) - M_3{}^* L(t) - M_4{}^* P(t) - M_6{}^* \frac{b_2}{M_6} - M_7{}^* \frac{b_3}{M_7} - M_5{}^* \frac{b_4}{M_5} = 0$$

$$let\ b_1 + b_2 + b_3 + b_4 = b_0{}^*, we\ have$$

$$\Rightarrow M_1{}^* A(t) + M_2{}^* E(t) + M_3{}^* L(t) + M_4{}^* P(t) = b_0{}^* + M_5{}^* \frac{b_4}{M_5} + M_6{}^* \frac{b_2}{M_6} + M_7{}^* \frac{b_3}{M_7}$$

$$\Rightarrow M_1{}^* M_5 M_6 M_7 A(t) + M_2{}^* M_5 M_6 M_7 E(t) + M_3{}^* M_5 M_6 M_7 L(t) + M_4{}^* M_5 M_6 M_7 P(t) = b_0{}^* + M_5{}^* M_6 M_7 b_4 + M_6{}^* M_5 M_7 b_2 + M_7{}^* M_5 M_6 b_3 \quad \ldots (4.1.16)$$

In synopsis we have the following equations

$$C_p(t) = \frac{b_2}{(\mu_6 + \beta_6 + \alpha)} = \frac{b_2}{M_6} \quad \ldots (4.1.9)$$

$$T_p(t) = \frac{b_3}{(\mu_7 + \beta_7 + \omega)} = \frac{b_3}{M_7} \quad \ldots (4.1.10)$$

$$P_m(t) = \frac{b_4}{(\mu_5 + \beta_5 + \gamma)} = \frac{b_4}{M_7} \quad \ldots (4.1.11)$$

$$\Rightarrow M_1 M_5 A(t) - M_5 \pi P(t) = M_5 b_1 + \gamma M_1 b_4 \quad \ldots (4.1.12)$$

$$\Rightarrow \eta A(t) - M_2 E(t) = 0 \quad \ldots (4.1.13)$$

$$\Rightarrow M_6 \sigma E(t) - M_3 M_6 L(t) = -\alpha b_2 \quad \ldots (4.1.14)$$

$$\Rightarrow M_7 \lambda L(t) - M_4 M_7 P(t) = -\omega b_3 \quad \ldots (4.1.15)$$

$$\Rightarrow M_1{}^* M_5 M_6 M_7 A(t) + M_2{}^* M_5 M_6 M_7 E(t) + M_3{}^* M_5 M_6 M_7 L(t) + M_4{}^* M_5 M_6 M_7 P(t) = b_0{}^* + M_5{}^* M_6 M_7 b_4 + M_6{}^* M_5 M_7 b_2 + M_7{}^* M_5 M_6 b_3 \quad \ldots (4.1.16)$$

From equation (4.1.13), we make A(t) the subject of the formula and substitute into equation (4.1.12), we have

$$\eta A(t) - M_2 E(t) = 0$$

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$$A(t) = \frac{M_2\, E(t)}{\eta}$$

$$M_1 M_5 A(t) - M_5 \pi P(t) = M_5 b_1 + \gamma M_1 b_4$$

$$\Rightarrow M_1 M_5 \frac{M_2\, E(t)}{\eta} - M_5 \pi P(t) = M_5 b_1 + \gamma M_1 b_4$$

$$\Rightarrow M_1 M_2\, M_5 E(t) - M_5 \pi \eta P(t) = M_5 b_1 \eta + \gamma M_1 b_4\, \eta \qquad \ldots (4.1.17)$$

From equation (4.1.15), we make L(t) the subject of the formula and substitute to equation (4.1.14), we have

$$M_7 \lambda L(t) - M_4 M_7 P(t) = -\omega b_3$$

$$M_7 \lambda L(t) = M_4 M_7 P(t) - \omega b_3$$

$$L(t) = \frac{M_4 M_7 P(t) - \omega b_3}{M_7 \lambda}$$

$$\Rightarrow M_6 \sigma E(t) - M_3 M_6 L(t) = -\alpha b_2$$

$$\Rightarrow M_6 \sigma E(t) - M_3 M_6 \left(\frac{M_4 M_7 P(t) - \omega b_3}{M_7 \lambda}\right) = -\alpha b_2$$

$$\Rightarrow M_6 M_7 \lambda \sigma E(t) - M_3 M_4 M_6 M_7 P(t) - M_3 M_6 \omega b_3 = -M_7 \lambda \alpha b_2$$

$$\Rightarrow M_6 M_7 \lambda \sigma E(t) - M_3 M_4 M_6 M_7 P(t) = M_3 M_6 \omega b_3 - M_7 \lambda \alpha b_2 \qquad \ldots (4.1.18)$$

Solve equation (4.1.17) and (4.1.18) simultaneously.

Multiply equation (4.1.17) by $M_6 M_7 \lambda \sigma$, we have

$$M_1 M_2\, M_5 E(t) - M_5 \pi \eta P(t) = M_5 b_1 \eta + \gamma M_1 b_4\, \eta$$

$$\Rightarrow M_6 M_7 \lambda \sigma \{M_1 M_2\, M_5 E(t) - M_5 \pi \eta P(t) = M_5 b_1 \eta + \gamma M_1 b_4\, \eta\}$$

$$\Rightarrow M_1 M_2\, M_5 M_6 M_7 \lambda \sigma E(t) - M_5 M_6 M_7 \lambda \sigma \pi \eta P(t) = M_5 M_6 M_7 \lambda \sigma b_1 \eta + M_1 M_6 M_7 \lambda \gamma \sigma b_4\, \eta \quad \ldots (a)$$

Multiply equation (4.1.18) by $M_1 M_2\, M_5$, we have

$$\Rightarrow M_6 M_7 \lambda \sigma E(t) - M_3 M_4 M_6 M_7 P(t) = M_3 M_6 \omega b_3 - M_7 \lambda \alpha b_2$$

$$\Rightarrow M_1 M_2\, M_5 \{M_6 M_7 \lambda \sigma E(t) - M_3 M_4 M_6 M_7 P(t) = M_3 M_6 \omega b_3 - M_7 \lambda \alpha b_2\}$$

$$\Rightarrow M_1 M_2\, M_5 M_6 M_7 \lambda \sigma E(t) - M_1 M_2\, M_5 M_3 M_4 M_6 M_7 P(t) = M_1 M_2\, M_5 M_3 M_6 \omega b_3 - M_1 M_2\, M_5 M_7 \lambda \alpha b_2 \ldots (b)$$

Subtract equation (a) from equation (b), we have

$$\Rightarrow M_1 M_2\, M_5 M_6 M_7 \lambda \sigma E(t) - M_5 M_6 M_7 \lambda \sigma \pi \eta P(t) = M_5 M_6 M_7 \lambda \sigma b_1 \eta + M_1 M_6 M_7 \lambda \gamma \sigma b_4\, \eta \qquad \ldots a$$

$$\Rightarrow M_1 M_2\, M_5 M_6 M_7 \lambda \sigma E(t) - M_1 M_2\, M_5 M_3 M_4 M_6 M_7 P(t) = M_1 M_2\, M_5 M_3 M_6 \omega b_3 - M_1 M_2\, M_5 M_7 \lambda \alpha b_2 \ldots b$$

$$\Rightarrow M_1 M_2\, M_5 M_3 M_4 M_6 M_7 P(t) - M_5 M_6 M_7 \lambda \sigma \pi \eta P(t) = M_5 M_6 M_7 \lambda \sigma b_1 \eta + M_1 M_6 M_7 \lambda \gamma \sigma b_4\, \eta + M_1 M_2\, M_5 M_7 \lambda \alpha b_2 - M_1 M_2\, M_5 M_3 M_6 \omega b_3$$

$$\Rightarrow \{M_1 M_2\, M_5 M_3 M_4 M_6 M_7 - M_5 M_6 M_7 \lambda \sigma \pi \eta\} P(t) = M_5 M_6 M_7 \lambda \sigma b_1 \eta + M_1 M_6 M_7 \lambda \gamma \sigma b_4\, \eta + M_1 M_2\, M_5 M_7 \lambda \alpha b_2 + M_1 M_2\, M_5 M_3 M_6 \omega b_3$$

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$$P(t) = \frac{M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta + M_1M_2\,M_5M_7\lambda\alpha b_2 + M_1M_2\,M_5M_3M_6\omega b_3}{M_5M_6M_7(M_1M_2\,M_3M_4 - \lambda\sigma\pi\eta)}$$

Put P(t) into equation (a), we have

$$M_1M_2\,M_5M_6M_7\lambda\sigma E(t) - M_5M_6M_7\lambda\sigma\pi\eta P(t) = M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta$$

$$\Rightarrow M_1M_2\,M_5M_6M_7\lambda\sigma E(t) - M_5M_6M_7\lambda\sigma\pi\eta\left(\frac{M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta + M_1M_2\,M_5M_7\lambda\alpha b_2 + M_1M_2\,M_5M_3M_6\omega b_3}{M_1M_2\,M_5M_3M_4M_6M_7 - M_5M_6M_7\lambda\sigma\pi\eta}\right)$$
$$= M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta$$

$$\Rightarrow M_1M_2\,M_5M_6M_7\lambda\sigma E(t) - M_5M_6M_7\lambda\sigma\pi\eta\left(\frac{M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta + M_1M_2\,M_5M_7\lambda\alpha b_2 + M_1M_2\,M_5M_3M_6\omega b_3}{M_1M_2\,M_5M_3M_4M_6M_7 - M_5M_6M_7\lambda\sigma\pi\eta}\right)$$
$$= M_5M_6M_7\lambda\sigma b_1\eta + M_1M_6M_7\lambda\gamma\sigma b_4\,\eta$$

$$E(t) = \frac{M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1\eta + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2}{M_1M_2\,M_5M_3M_4M_6M_7 - M_5M_6M_7\lambda\sigma\pi\eta}$$

$$E(t) = \frac{\eta(M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1 + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2\,M_3 - \lambda\sigma\pi\eta)}$$

Substitute E(t) in equation (4.1.13), we have

$$\eta A(t) - M_2\,E(t) = 0$$

$$\Rightarrow \eta A(t) - M_2\left(\frac{\eta(M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1 + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2\,M_3 - \lambda\sigma\pi\eta)}\right) = 0$$

$$\Rightarrow \eta A(t) = M_2\left(\frac{\eta(M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1 + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2\,M_3 - \lambda\sigma\pi\eta)}\right)$$

$$\Rightarrow A(t) = M_2\left(\frac{M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1 + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2}{M_5M_6M_7(M_1M_2\,M_3 - \lambda\sigma\pi\eta)}\right)$$

Settle E(t) into equation (4.1.14), we have

$$M_6\sigma E(t) - M_3M_6L(t) = -\alpha b_2$$

$$\Rightarrow M_6\sigma\left(\frac{\eta(M_3M_4M_6M_7\gamma b_4 + M_3M_4M_5M_6M_7b_1 + M_5M_6\omega\pi b_3 + M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2\,M_3 - \lambda\sigma\pi\eta)}\right) - M_3M_6L(t) = -\alpha b_2$$

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$$\Rightarrow M_6\sigma\left(\frac{\eta(M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right)+\alpha b_2=M_3M_6L(t)$$

$$\Rightarrow M_3M_6L(t)=M_6\sigma\left(\frac{\eta(M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right)+\alpha b_2$$

$$\Rightarrow L(t)=\frac{M_4M_6M_7\gamma\eta\sigma b_4+M_4M_5M_6M_7\eta\sigma b_1+M_5M_6\omega\pi\sigma b_3+M_1M_2M_4M_5M_7\alpha b_2}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}$$

Therefore, the total population becomes

$$M_1{}^*M_5M_6M_7A(t)+M_2{}^*M_5M_6M_7E(t)+M_3{}^*M_5M_6M_7L(t)+M_4{}^*M_5M_6M_7P(t)=b_0{}^*+M_5{}^*M_6M_7b_4+M_6{}^*M_5M_7b_2+M_7{}^*M_5M_6b_3$$

Insert A(t), E(t), L(t), P(t), $C_P(t)$, $T_P(t)$ and $P_M(t)$, into equation (4.1.16),we have

$$M_1{}^*M_5M_6M_7A(t)+M_2{}^*M_5M_6M_7E(t)+M_3{}^*M_5M_6M_7L(t)+M_4{}^*M_5M_6M_7P(t)=b_0{}^*+M_5{}^*M_6M_7b_4+M_6{}^*M_5M_7b_2+M_7{}^*M_5M_6b_3$$

The following results were obtained manually.

$$where\ A(t)=M_2\left(\frac{M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right)$$

$$E(t)=\frac{\eta(M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}$$

$$L(t)=\frac{M_4M_6M_7\gamma\eta\sigma b_4+M_4M_5M_6M_7\eta\sigma b_1+M_5M_6\omega\pi\sigma b_3+M_1M_2M_4M_5M_7\alpha b_2}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}$$

$$P(t)=\frac{M_5M_6M_7\lambda\sigma b_1\eta+M_1M_6M_7\lambda\gamma\sigma b_4\,\eta+M_1M_2M_5M_7\lambda\alpha b_2+M_1M_2M_5M_3M_6\omega b_3}{M_5M_6M_7(M_1M_2M_3M_4-\lambda\sigma\pi\eta)}$$

$$C_p(t)=\frac{b_2}{(\mu_6+\beta_6+\alpha)}=\frac{b_2}{M_6}$$

$$T_p(t)=\frac{b_3}{(\mu_7+\beta_7+\omega)}=\frac{b_3}{M_7}$$

$$P_m(t)=\frac{b_4}{(\mu_5+\beta_5+\gamma)}=\frac{b_4}{M_7}$$

Total population is giving as;

$$\begin{aligned}M_1{}^*M_5M_6M_7&\left\{M_2\left(\frac{M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right)\right\}\\&+M_2{}^*M_5M_6M_7\left\{\frac{\eta(M_3M_4M_6M_7\gamma b_4+M_3M_4M_5M_6M_7b_1+M_5M_6\omega\pi b_3+M_5M_7\lambda\alpha\pi b_2)}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right\}\\&+M_3{}^*M_5M_6M_7\left\{\frac{M_4M_6M_7\gamma\eta\sigma b_4+M_4M_5M_6M_7\eta\sigma b_1+M_5M_6\omega\pi\sigma b_3+M_1M_2M_4M_5M_7\alpha b_2}{M_5M_6M_7(M_1M_2M_3-\lambda\sigma\pi\eta)}\right\}\\&+M_4{}^*M_5M_6M_7\left\{\frac{M_5M_6M_7\lambda\sigma b_1\eta+M_1M_6M_7\lambda\gamma\sigma b_4\,\eta+M_1M_2M_5M_7\lambda\alpha b_2+M_1M_2M_5M_3M_6\omega b_3}{M_5M_6M_7(M_1M_2M_3M_4-\lambda\sigma\pi\eta)}\right\}\\&=b_0{}^*+M_5{}^*M_6M_7b_4+M_6{}^*M_5M_7b_2+M_7{}^*M_5M_6b_3\end{aligned}$$

Maple result for A(t), E(t), L(t), and P(t) are shown below;

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$$solve\left(\left\{M_1\cdot \mathrm{A}(t)-\pi\cdot \mathrm{P}(t)=\mathrm{b}_1+\frac{\gamma\cdot b_4}{M_5},\ \eta\cdot A(t)-M_2\cdot E(t)=0,\ \sigma\cdot E(t)-M_3\cdot L(t)=-\frac{\alpha\cdot b_2}{M_6},\right.\right.$$
$$\left.\left.\lambda\cdot L(t)-M_4\cdot P(t)=-\frac{\varpi\cdot b_3}{M_7}\right\},[A(t),E(t),L(t),P(t)]\right)$$

$$\left[\left[A(t)=\right.\right.$$
$$-\frac{M_2\left(M_7\gamma b_4 M_6 M_4 M_3+M_7 b_1 M_5 M_6 M_4 M_3+\varpi b_3 M_6 M_5 M_3\pi+\lambda M_7 M_5\alpha b_2\pi\right)}{M_5\left(\pi\eta\lambda\sigma-M_1 M_2 M_3 M_4\right)M_7 M_6},$$

$$E(t)=$$

$$-\frac{\eta\left(M_7\gamma b_4 M_6 M_4 M_3+M_7 b_1 M_5 M_6 M_4 M_3+\varpi b_3 M_6 M_5 M_3\pi+\lambda M_7 M_5\alpha b_2\pi\right)}{M_5\left(\pi\eta\lambda\sigma-M_1 M_2 M_3 M_4\right)M_7 M_6},$$

$$L(t)=$$

$$-\frac{1}{M_5\left(\pi\eta\lambda\sigma-M_1 M_2 M_3 M_4\right)M_7 M_6}\left(M_7 M_1 M_2\alpha b_2 M_5 M_4+M_7\gamma b_4\eta\sigma M_6 M_4\right.$$

$$\left.+M_7 b_1 M_5\eta\sigma M_6 M_4+\eta\varpi\sigma b_3 M_6 M_5\pi\right),P(t)=$$

$$\left.-\frac{M_1 M_2 M_3 M_5\varpi b_3 M_6+\lambda M_7 M_1 M_2\alpha b_2 M_5+\lambda M_7\gamma b_4\eta\sigma M_6+\lambda M_7 b_1 M_5\eta\sigma M_6}{M_5\left(\pi\eta\lambda\sigma-M_1 M_2 M_3 M_4\right)M_7 M_6}\right]$$

$$\left.\right]$$

Maple result for A(t), E(t), L(t), P(t) and N(t) are shown below

$$solve\left(\left\{M_1\cdot \mathrm{A}(t)-\pi\cdot \mathrm{P}(t)=\mathrm{b}_1+\frac{\gamma\cdot b_4}{M_5},\ \eta\cdot A(t)-M_2\cdot E(t)=0,\ \sigma\cdot E(t)-M_3\cdot L(t)=-\frac{\alpha\cdot b_2}{M_6},\right.\right.$$
$$\left.\left.\lambda\cdot L(t)-M_4\cdot P(t)=-\frac{\varpi\cdot b_3}{M_7},A(t)+E(t)+L(t)+P(t)+\frac{b_2}{M_6}+\frac{b_3}{M_7}+\frac{b_4}{M_5}=N(t)\right\}\right.$$
$$\left.,[A(t),E(t),L(t),P(t),N(t)]\right)$$

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$$\left[\left[A(t) = -\frac{M_2\left(M_7\gamma b_4 M_6 M_4 M_3 + M_7 b_1 M_5 M_6 M_4 M_3 + \varpi b_3 M_6 M_5 M_3 \pi + \lambda M_7 M_5 \alpha b_2 \pi\right)}{M_5\left(\pi\eta\lambda\sigma - M_1 M_2 M_3 M_4\right) M_7 M_6},\right.\right.$$

$$E(t) = -\frac{\eta\left(M_7\gamma b_4 M_6 M_4 M_3 + M_7 b_1 M_5 M_6 M_4 M_3 + \varpi b_3 M_6 M_5 M_3 \pi + \lambda M_7 M_5 \alpha b_2 \pi\right)}{M_5\left(\pi\eta\lambda\sigma - M_1 M_2 M_3 M_4\right) M_7 M_6},$$

$$L(t) = -\frac{1}{M_5\left(\pi\eta\lambda\sigma - M_1 M_2 M_3 M_4\right) M_7 M_6}\left(M_7 M_1 M_2 \alpha b_2 M_5 M_4 + M_7\gamma b_4\eta\sigma M_6 M_4\right.$$

$$\left. + M_7 b_1 M_5\eta\sigma M_6 M_4 + \eta\varpi\sigma b_3 M_6 M_5\pi\right), P(t) = -\frac{M_1 M_2 M_3 M_5\varpi b_3 M_6 + \lambda M_7 M_1 M_2\alpha b_2 M_5 + \lambda M_7\gamma b_4\eta\sigma M_6 + \lambda M_7 b_1 M_5\eta\sigma M_6}{M_5\left(\pi\eta\lambda\sigma - M_1 M_2 M_3 M_4\right) M_7 M_6},$$

$$N(t) = -\frac{1}{M_6 M_7 M_5\left(\pi\eta\lambda\sigma - M_1 M_2 M_3 M_4\right)}\left(\lambda M_7 M_1 M_2\alpha b_2 M_5\right.$$
$$+ M_7\gamma b_4\eta M_6 M_4 M_3 + M_7 b_1 M_5\eta M_6 M_4 M_3 + \eta\varpi b_3 M_6 M_5 M_3\pi$$
$$+ \lambda M_7 M_5\alpha b_2 M_2\pi + M_7 b_1 M_5\eta\sigma M_6 M_4 + \eta\varpi\sigma b_3 M_6 M_5\pi + M_7\gamma b_4\eta\sigma M_6 M_4$$
$$+ \lambda M_7 M_5\alpha b_2\eta\pi + M_1 M_2 M_3 M_5\varpi b_3 M_6 + M_7 M_1 M_2\alpha b_2 M_5 M_4$$
$$+ M_7\gamma b_4 M_6 M_4 M_3 M_2 + M_7 b_1 M_5 M_6 M_4 M_3 M_2 + \varpi b_3 M_6 M_5 M_3 M_2\pi$$
$$+ b_3 M_6 M_5 M_4 M_3 M_2 M_1 + b_4 M_6 M_7 M_4 M_3 M_2 M_1 + b_2 M_7 M_5 M_4 M_3 M_2 M_1$$
$$+ \lambda M_7\gamma b_4\eta\sigma M_6 + \lambda M_7 b_1 M_5\eta\sigma M_6 - \lambda b_3 M_6 M_5\eta\sigma\pi - \lambda b_4 M_6 M_7\eta\sigma\pi$$
$$\left.\left. - \lambda b_2 M_7 M_5\eta\sigma\pi\right)\right]\right]$$

#### 4.1.2 Mosquito-Free Steady-State Stability Analysis Using Beltrami Conditions

The Beltrami conditions state that if the determinants of the Jacobian are greater than zero and the trace elements of the Jacobian are less than zero, then the mosquito-free equilibrium stability analysis model is stable; otherwise it is unstable.

Before using result to construct the mosquito-free equilibrium stability analysis $E_P$ using Beltrami's conditions, the following theorems are given without proof.

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**Theorem 4.1**

Let $\mathcal{R}$ be a commutative sub ring of ${}^{n}F^{n}$, where F is a field (or a commutative ring) and $M \in {}^{n}F^{n}$.

$LET\ M = \begin{pmatrix} A & B \\ C & D \end{pmatrix}$, where A, B, C, D are $n \times n$ block matrices over F, so that $M \in {}^{2n}F^{2n}$. Suppose that C = 0, the $n \times n$ zero matrices, then

(a). $det_F M = det_F \begin{pmatrix} A & B \\ O & D \end{pmatrix} = det_F AD = det_F A.\, det_F D$

(b). $Trace_F M = Trace_F \begin{pmatrix} A & B \\ 0 & D \end{pmatrix} = Trace_F(A + D) = Trace_F(A) + Trace_F(D)$

**Proof:** (See Silvester, 2000).

**Theorem 4.**2

The eigenvalues $\lambda_{1,2}$ of a 2 by 2 matrix satisfy J satisfy $Re\lambda_{1,2} < 0$ if and only if $Det(J) > 0 and\ Trace(J) < 0$. they are pure imaginary if and only if $Trace(J) = 0$. moreover the eigenvalues fulfills the following conditions $\lambda_1 < 0 < \lambda_2\ or\ \lambda_2 < 0 < \lambda_1\ if\ and\ only\ if\ Det(J) < 0$

**Proof:** (See page 255 of Geland, 2012).

**Theorem 4.3**

Let A be an $n \times n$ matrix with n distinct eigenvalues $\lambda_1, \lambda_2, \ldots, \lambda_n$, and J(A) the Jacobian matrix of A evaluated at equilibrium state $E_P$

1) If the eigenvalues of the Jacobian Matrix J(A) all have non-negative real parts, then the equilibrium state is stable or predictable otherwise it is unstable.
2) If at least one of the eigenvalues of the Jacobian Matrix J(A) has real parts less than zero, then the equilibrium state is unstable or uncertain

**Proof:** (See Thomas, 2008).

### 4.1.3 Equations of the Model Associated with Equations (3.13.1) – (3.13.8)

We study the behavior of the system (3.13.1)-(3.13.8) around the mosquitoes free equilibrium state $Let\ E_P = (A_0, E_0, L_O, P_O, C_{PO}, T_{PO}, P_{MO}\ and\ N_O)$ be the equilibrium points of the model, we linearized stability approach.
Let

$$f_1 = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) \quad \ldots \ (4.3.1)$$

$$f_2 = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) \quad \ldots \ (4.3.2)$$

$$f_3 = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) \quad \ldots \ (4.3.3)$$

$$f_4 = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) \quad \ldots \ (4.3.4)$$

$$f_5 = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) \quad \ldots \ (4.3.5)$$

$$f_6 = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) \quad \ldots \ (4.3.6)$$

$$f_7 = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \ (4.3.7)$$

$$f_8 = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \ (4.3.8)$$

Then

$$f_1 = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t)$$

$$\frac{\partial f_1}{\partial A} = -(\mu_1 + \beta_1 + \eta), \frac{\partial f_1}{\partial E} = 0, \frac{\partial f_1}{\partial L} = 0, \frac{\partial f_1}{\partial P} = \pi, \frac{\partial f_1}{\partial C_P} = 0, \frac{\partial f_1}{\partial T_P} = 0, \frac{\partial f_1}{\partial P_M} = \gamma, \frac{\partial f_1}{\partial N} = 0 \quad \ldots \ (4.3.9)$$

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$$f_2 = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t)$$

$$\frac{\partial f_2}{\partial A} = \eta, \frac{\partial f_2}{\partial E} = -(\mu_2 + \beta_2 + \sigma), \frac{\partial f_2}{\partial L} = 0, \frac{\partial f_2}{\partial P} = 0, \frac{\partial f_2}{\partial C_P} = 0, \frac{\partial f_2}{\partial T_P} = 0, \frac{\partial f_2}{\partial P_M} = 0, \frac{\partial f_2}{\partial N} = 0 \quad \ldots (4.3.10)$$

$$f_3 = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t)$$

$$\frac{\partial f_3}{\partial A} = 0, \frac{\partial f_3}{\partial E} = \sigma, \frac{\partial f_3}{\partial L} = -(\mu_3 + \beta_3 + \lambda), \frac{\partial f_3}{\partial P} = 0, \frac{\partial f_3}{\partial C_P} = \alpha, \frac{\partial f_3}{\partial T_P} = 0, \frac{\partial f_3}{\partial P_M} = 0, \frac{\partial f_3}{\partial N} = 0 \quad \ldots (4.3.11)$$

$$f_4 = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t)$$

$$\frac{\partial f_4}{\partial A} = 0, \quad \frac{\partial f_4}{\partial E} = 0, \frac{\partial f_4}{\partial L} = \lambda, \quad \frac{\partial f_4}{\partial P} = -(\mu_4 + \beta_4 + \pi), \frac{\partial f_4}{\partial C_P} = 0, \frac{\partial f_4}{\partial T_P} = \omega, \frac{\partial f_4}{\partial P_M} = 0, \frac{\partial f_4}{\partial N} = 0 \quad \ldots (4.3.12)$$

$$f_5 = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t)$$

$$\frac{\partial f_5}{\partial A} = 0, \frac{\partial f_5}{\partial E} = 0, \frac{\partial f_5}{\partial L} = 0, \frac{\partial f_5}{\partial P} = 0, \frac{\partial f_5}{\partial C_P} = -(\mu_6 + \beta_6 + \alpha), \frac{\partial f_5}{\partial T_P} = 0, \frac{\partial f_5}{\partial P_M} = 0, \frac{\partial f_5}{\partial N} = 0 \quad \ldots (4.3.13)$$

$$f_6 = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t)$$

$$\frac{\partial f_6}{\partial A} = 0, \frac{\partial f_6}{\partial E} = 0, \frac{\partial f_6}{\partial L} = 0, \frac{\partial f_6}{\partial P} = 0, \frac{\partial f_6}{\partial C_P} = 0, \frac{\partial f_6}{\partial T_P} = -(\mu_7 + \beta_7 + \omega), \frac{\partial f_6}{\partial P_M} = 0, \frac{\partial f_6}{\partial N} = 0 \quad \ldots (4.3.14)$$

$$f_7 = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t)$$

$$\frac{\partial f_7}{\partial A} = 0, \frac{\partial f_7}{\partial E} = 0, \frac{\partial f_7}{\partial L} = 0, \frac{\partial f_7}{\partial P} = 0, \frac{\partial f_7}{\partial C_P} = 0, \frac{\partial f_7}{\partial T_P} = 0, \frac{\partial f_7}{\partial P_M} = -(\mu_5 + \beta_5 + \gamma), \frac{\partial f_7}{\partial N} = 0 \quad \ldots (4.3.15)$$

$$\begin{aligned} f_8 = {} & b_1 - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \pi P(t) \\ & - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + \alpha C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + \omega T_p(t) + b_4 \\ & - (\mu_5 + \beta_5 + \gamma)P_m(t) + \gamma P_m(t) \end{aligned}$$

$$\frac{\partial f_8}{\partial A} = -(\mu_1 + \beta_1), \frac{\partial f_8}{\partial E} = -(\mu_2 + \beta_2), \frac{\partial f_8}{\partial L} = -(\mu_3 + \beta_3), \frac{\partial f_8}{\partial P} = -(\mu_4 + \beta_4), \frac{\partial f_8}{\partial C_P} = -(\mu_6 + \beta_6), \frac{\partial f_8}{\partial T_P} = -(\mu_7 + \beta_7), \frac{\partial f_7}{\partial P_M}$$
$$= -(\mu_5 + \beta_5), \frac{\partial f_8}{\partial N} = 0 \quad \ldots (4.3.16)$$

### 4.1.4 Jacobian Matrix (J) Associated with Model Equations (4.3.9) – (4.3.16)

**Theorem 4.4**

The mosquito's free equilibrium state of the model (4.3.9) – (4.3.16) is locally asymptotically stable if $R_o < 1$ and the following threshold conditions hold $(i).R_1 < 1\ (ii).R_2 < 1\ (i).R_3 < 1$, otherwise $E_P$ is unstable.

**Proof**: The Jacobian matrix of the system is given below

$$\boldsymbol{J} = \begin{pmatrix} -(\boldsymbol{\mu_1 + \beta_1} + \eta) & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\pi} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\gamma} & \boldsymbol{0} \\ \boldsymbol{0} & -(\boldsymbol{\mu_2 + \beta_2 + \sigma}) & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\sigma} & -(\boldsymbol{\mu_3 + \beta_3 + \lambda}) & \boldsymbol{\alpha} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\lambda} & -(\boldsymbol{\mu_4 + \beta_4 + \pi}) & \boldsymbol{0} & \boldsymbol{\omega} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & -(\boldsymbol{\mu_6 + \beta_6 + \alpha}) & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & -(\boldsymbol{\mu_7 + \beta_7 + \omega}) & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & -(\boldsymbol{\mu_5 + \beta_5 + \gamma}) & \boldsymbol{0} \\ -(\boldsymbol{\mu_1 + \beta_1}) & -(\boldsymbol{\mu_2 + \beta_2}) & -(\boldsymbol{\mu_3 + \beta_3}) & -(\boldsymbol{\mu_4 + \beta_4}) & -(\boldsymbol{\mu_6 + \beta_6}) & -(\boldsymbol{\mu_7 + \beta_7}) & -(\boldsymbol{\mu_5 + \beta_5}) & \boldsymbol{0} \end{pmatrix}$$

If the Jacobian is evaluated in the free equilibrium state of the mosquito, then the criterion required for a stable equilibrium (by Theorem 4.2 and Theorem 4.3) is that the determinant of the Jacobian be positive and the trace of the Jacobian be negative .

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$$Det(J)$$
$$= \begin{pmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 & \pi & 0 & 0 & \gamma & 0 \\ 0 & -(\mu_2+\beta_2+\sigma) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_3+\beta_3+\lambda) & \alpha & 0 & 0 & 0 \\ 0 & 0 & \lambda & -(\mu_4+\beta_4+\pi) & 0 & \omega & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\mu_6+\beta_6+\alpha) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_1+\beta_1) & -(\mu_2+\beta_2) & -(\mu_3+\beta_3) & -(\mu_4+\beta_4) & -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{pmatrix}.$$

Using the result of Theorem 4.1 above, we partition the matrix $(Z)$ represented in equation (4.4.2) above as follows:

$$Z = \begin{pmatrix} A & \cdots & B \\ \vdots & \ddots & \vdots \\ C & \cdots & D \end{pmatrix} \qquad \ldots \ (4.4.3)$$

where A, B, C and D are block matrices defined as follows

$$Det(J)$$
$$= \begin{pmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 & \pi & 0 & 0 & \gamma & 0 \\ 0 & -(\mu_2+\beta_2+\sigma) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_3+\beta_3+\lambda) & \alpha & 0 & 0 & 0 \\ 0 & 0 & \lambda & -(\mu_4+\beta_4+\pi) & 0 & \omega & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\mu_6+\beta_6+\alpha) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_1+\beta_1) & -(\mu_2+\beta_2) & -(\mu_3+\beta_3) & -(\mu_4+\beta_4) & -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{pmatrix}$$

$$A = \begin{bmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 & \pi \\ 0 & -(\mu_2+\beta_2+\sigma) & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_3+\beta_3+\lambda) \\ 0 & 0 & \lambda & -(\mu_4+\beta_4+\pi) \end{bmatrix}, \quad B = \begin{bmatrix} 0 & 0 & \gamma & 0 \\ 0 & 0 & 0 & 0 \\ \sigma & 0 & 0 & 0 \\ 0 & \omega & 0 & 0 \end{bmatrix},$$

$$C = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -(\mu_1+\beta_1) & -(\mu_2+\beta_2) & -(\mu_3+\beta_3) & -(\mu_4+\beta_4) \end{bmatrix} \text{ and } D$$
$$= \begin{bmatrix} -(\mu_6+\beta_6+\alpha) & 0 & 0 & 0 \\ 0 & -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{bmatrix} \qquad \ldots (4.4.5)$$

The determinant of a matrix is a scalar or numeric value associated with any square matrix, which can be a real or complex number, positive, negative or zero. The determinant is usually denoted by det(A) or |A|.

We have that the determinant of the Jacobian matrix (J) is given by

$$A = \begin{bmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 & \pi \\ 0 & -(\mu_2+\beta_2+\sigma) & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_3+\beta_3+\lambda) \\ 0 & 0 & \lambda & -(\mu_4+\beta_4+\pi) \end{bmatrix} \qquad \ldots (4.4.6)$$

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$$Det(A) = -(\mu_1 + \beta_1 + \eta)\begin{bmatrix} -(\mu_2 + \beta_2 + \sigma) & 0 & 0 \\ 0 & \sigma & -(\mu_3 + \beta_3 + \lambda) \\ 0 & \lambda & -(\mu_4 + \beta_4 + \pi) \end{bmatrix} - 0\begin{bmatrix} 0 & 0 & 0 \\ 0 & \sigma & -(\mu_3 + \beta_3 + \lambda) \\ 0 & 0 & -(\mu_4 + \beta_4 + \pi) \end{bmatrix}$$
$$+ 0\begin{bmatrix} 0 & -(\mu_2 + \beta_2 + \sigma) & 0 \\ 0 & 0 & -(\mu_3 + \beta_3 + \lambda) \\ 0 & 0 & -(\mu_4 + \beta_4 + \pi) \end{bmatrix} - 0\begin{bmatrix} 0 & -(\mu_2 + \beta_2 + \sigma) & 0 \\ 0 & 0 & \sigma \\ 0 & 0 & \lambda \end{bmatrix}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)\begin{bmatrix} -(\mu_2 + \beta_2 + \sigma) & 0 & 0 \\ 0 & \sigma & -(\mu_3 + \beta_3 + \lambda) \\ 0 & \lambda & -(\mu_4 + \beta_4 + \pi) \end{bmatrix}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)\left\{-(\mu_2 + \beta_2 + \sigma)\begin{bmatrix} \sigma & -(\mu_3 + \beta_3 + \lambda) \\ \lambda & -(\mu_4 + \beta_4 + \pi) \end{bmatrix} - 0\begin{bmatrix} 0 & -(\mu_3 + \beta_3 + \lambda) \\ 0 & -(\mu_4 + \beta_4 + \pi) \end{bmatrix} + 0\begin{bmatrix} 0 & \sigma \\ 0 & \lambda \end{bmatrix}\right\}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)\left\{-(\mu_2 + \beta_2 + \sigma)\begin{bmatrix} \sigma & -(\mu_3 + \beta_3 + \lambda) \\ \lambda & -(\mu_4 + \beta_4 + \pi) \end{bmatrix}\right\}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)\{-(\mu_2 + \beta_2 + \sigma)[-(\mu_4 + \beta_4 + \pi)\sigma + (\mu_3 + \beta_3 + \lambda)\lambda]\}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)\{(\mu_2 + \beta_2 + \sigma)(\mu_4 + \beta_4 + \pi)\sigma - (\mu_2 + \beta_2 + \sigma)(\mu_3 + \beta_3 + \lambda)\lambda\}$$

$$Det(A) = \{-(\mu_1 + \beta_1 + \eta)(\mu_2 + \beta_2 + \sigma)(\mu_4 + \beta_4 + \pi)\sigma - (\mu_1 + \beta_1 + \eta)(\mu_2 + \beta_2 + \sigma)(\mu_3 + \beta_3 + \lambda)\lambda\}$$

$$Det(A) = -\{(\mu_1 + \beta_1 + \eta)(\mu_2 + \beta_2 + \sigma)(\mu_3 + \beta_3 + \lambda)\lambda + (\mu_1 + \beta_1 + \eta)(\mu_2 + \beta_2 + \sigma)(\mu_4 + \beta_4 + \pi)\sigma\}$$

$$Det(A) = -(\mu_1 + \beta_1 + \eta)(\mu_2 + \beta_2 + \sigma)\{(\mu_3 + \beta_3 + \lambda)\lambda + (\mu_4 + \beta_4 + \pi)\sigma\}$$

$$B = \begin{bmatrix} 0 & 0 & \gamma & 0 \\ 0 & 0 & 0 & 0 \\ \sigma & 0 & 0 & 0 \\ 0 & \omega & 0 & 0 \end{bmatrix} \quad \ldots (4.4.7)$$

$$Det(B) = \begin{bmatrix} 0 & 0 & \gamma & 0 \\ 0 & 0 & 0 & 0 \\ \sigma & 0 & 0 & 0 \\ 0 & \omega & 0 & 0 \end{bmatrix} = 0$$

$$C = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -(\mu_1 + \beta_1) & -(\mu_2 + \beta_2) & -(\mu_3 + \beta_3) & -(\mu_4 + \beta_4) \end{bmatrix} \quad \ldots (4.4.8)$$

$$Det(C) = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -(\mu_1 + \beta_1) & -(\mu_2 + \beta_2) & -(\mu_3 + \beta_3) & -(\mu_4 + \beta_4) \end{bmatrix} = 0$$

$$D = \begin{bmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 & 0 & 0 \\ 0 & -(\mu_7 + \beta_7 + \omega) & 0 & 0 \\ 0 & 0 & -(\mu_5 + \beta_5 + \gamma) & 0 \\ -(\mu_6 + \beta_6) & -(\mu_7 + \beta_7) & -(\mu_5 + \beta_5) & 0 \end{bmatrix} \quad \ldots (4.4.9)$$

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$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)\begin{bmatrix} -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{bmatrix} - 0\begin{bmatrix} 0 & 0 & 0 \\ 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_6+\beta_6) & -(\mu_5+\beta_5) & 0 \end{bmatrix}}$$
$$\boldsymbol{+ 0\begin{bmatrix} 0 & -(\mu_7+\beta_7+\omega) & 0 \\ 0 & 0 & 0 \\ -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & 0 \end{bmatrix} - 0\begin{bmatrix} 0 & -(\mu_7+\beta_7+\omega) & 0 \\ 0 & 0 & -(\mu_5+\beta_5+\gamma) \\ -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & -(\mu_5+\beta_5) \end{bmatrix}}$$

$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)\begin{bmatrix} -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{bmatrix}}$$

$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)\Big\{-(\mu_7+\beta_7+\omega)\begin{bmatrix} -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_5+\beta_5) & \lambda \end{bmatrix} - 0\begin{bmatrix} 0 & 0 \\ -(\mu_7+\beta_7) & 0 \end{bmatrix}}$$
$$\boldsymbol{- 0\begin{bmatrix} 0 & -(\mu_5+\beta_5+\gamma) \\ -(\mu_7+\beta_7) & -(\mu_5+\beta_5) \end{bmatrix}\Big\}}$$

$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)\Big\{-(\mu_7+\beta_7+\omega)\begin{bmatrix} -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_5+\beta_5) & \lambda \end{bmatrix}\Big\}}$$

$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)\{(\mu_7+\beta_7+\omega)(\mu_5+\beta_5+\gamma)\lambda\}}$$

$$\boldsymbol{Det(D) = -(\mu_6+\beta_6+\alpha)(\mu_7+\beta_7+\omega)(\mu_5+\beta_5+\gamma)\lambda}$$

$$\boldsymbol{Det(J) = detA.detD}$$
$$\boldsymbol{= -\{(\mu_1+\beta_1+\eta)(\mu_2+\beta_2+\sigma)(\mu_3+\beta_3+\lambda)\lambda}$$
$$\boldsymbol{+ (\mu_1+\beta_1+\eta)(\mu_2+\beta_2+\sigma)(\mu_4+\beta_4+\pi)\sigma\}\{-(\mu_6+\beta_6+\alpha)(\mu_7+\beta_7+\omega)(\mu_5+\beta_5+\gamma)\lambda\}}$$

$$\boldsymbol{Det(J) = detA.detD}$$
$$\boldsymbol{= \{(\mu_1+\beta_1+\eta)(\mu_2+\beta_2+\sigma)(\mu_3+\beta_3+\lambda)\lambda}$$
$$\boldsymbol{+ (\mu_1+\beta_1+\eta)(\mu_2+\beta_2+\sigma)(\mu_4+\beta_4+\pi)\sigma\}\{(\mu_6+\beta_6+\alpha)(\mu_7+\beta_7+\omega)(\mu_5+\beta_5+\gamma)\lambda\} > 0}$$

Similarly, the Trace of the Jacobian Matrix (J) is given by

$$Trace(J) = Trace\begin{pmatrix} A & 0 \\ 0 & D \end{pmatrix} = Trace(A+D) = Trace(A) + Trace(D)$$

$$\boldsymbol{A = \begin{bmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 & \pi \\ 0 & -(\mu_2+\beta_2+\sigma) & 0 & 0 \\ 0 & 0 & \sigma & -(\mu_3+\beta_3+\lambda) \\ 0 & 0 & \lambda & -(\mu_4+\beta_4+\pi) \end{bmatrix}}$$

$$\boldsymbol{Trace(A) = -(\mu_1+\beta_1+\eta) - (\mu_2+\beta_2+\sigma) - (\mu_4+\beta_4+\pi) + \sigma}$$

$$\boldsymbol{Trace(A) = -\{(\mu_1+\beta_1+\eta) + (\mu_2+\beta_2) + (\mu_4+\beta_4+\pi)\} < 0}$$

$$\boldsymbol{D = \begin{bmatrix} -(\mu_6+\beta_6+\alpha) & 0 & 0 & 0 \\ 0 & -(\mu_7+\beta_7+\omega) & 0 & 0 \\ 0 & 0 & -(\mu_5+\beta_5+\gamma) & 0 \\ -(\mu_6+\beta_6) & -(\mu_7+\beta_7) & -(\mu_5+\beta_5) & 0 \end{bmatrix}}$$

$$Trace(D) = -(\mu_6+\beta_6+\alpha) - (\mu_7+\beta_7+\omega) - (\mu_5+\beta_5+\gamma)$$

$$Trace(D) = -\{(\mu_6+\beta_6+\alpha) + (\mu_7+\beta_7+\omega) + (\mu_5+\beta_5+\gamma)\}$$

$$Trace(J) = Trace(A) + Trace(D) = -\{(\mu_2+\beta_2) + (\mu_4+\beta_4+\pi)\} - \{(\mu_6+\beta_6+\alpha) + (\mu_7+\beta_7+\omega) + (\mu_5+\beta_5+\gamma)\}$$

$$Trace(J) = Trace(A) + Trace(D) = -\{(\mu_2+\beta_2) + (\mu_4+\beta_4+\pi) + (\mu_6+\beta_6+\alpha) + (\mu_7+\beta_7+\omega) + (\mu_5+\beta_5+\gamma)\} < 0$$

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### 4.1.5 Result for Beltrami Conditions

Since the determinants of the contact matrix are greater than zero and the trace of the contact matrix is less than zero, we conclude that the disease-free steady-state stability analysis of the model is stable, which shows that the rate of malaria parasites in our society will be reduced.

### 4.1.6 Mosquito-Free Steady-State Stability Analysis Using Diekmann Conditions

Diekmann's conditions state that if the fundamental reproduction number is less than one ($R_0 < 1$), the stability analysis of the free equilibrium state is stable; otherwise unstable. Calculation of the basic reproduction number $R_0$. In this section, we first rearrange the model equations (3.13.1) – (3.13.8) starting with the mosquito life cycle equations to the equations of the natural predators giving the following equations.

$$\frac{dA}{dt} = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) \quad \ldots \quad (4.5.1)$$

$$\frac{dE}{dt} = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) \quad \ldots \quad (4.5.2)$$

$$\frac{dL}{dt} = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) \quad \ldots \quad (4.5.3)$$

$$\frac{dP}{dt} = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) \quad \ldots \quad (4.5.4)$$

$$\frac{dC_P}{dt} = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) \quad \ldots \quad (4.5.5)$$

$$\frac{dT_p}{dt} = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) \quad \ldots \quad (4.5.6)$$

$$\frac{dP_m}{dt} = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \quad (4.5.7)$$

$$\begin{aligned}\frac{dN}{dt} = {} & b_1 - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \pi P(t) \\ & - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + \alpha C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + \omega T_p(t) + b_4 \\ & - (\mu_5 + \beta_5 + \gamma)P_m(t) + \gamma P_m(t) \quad \ldots \quad (4.5.8)\end{aligned}$$

To compute the basic reproduction number $(R_0)$ of the model (4.5.1) - (4.5.8), we employ the next generation method as applied in Diekman *et al.,* (2009) and Van den Driessche and Watmough, (2000). From equations (4.5.1) – (4.5.8), using their approached we have that

$$f_1 = b_1 + \gamma P_m(t) + \pi P(t) - (\mu_1 + \beta_1 + \eta)A(t) \quad \ldots \quad (4.5.9)$$

$$f_2 = \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) \quad \ldots \quad (4.5.10)$$

$$f_3 = \sigma E(t) + \alpha C_P(t) - (\mu_3 + \beta_3 + \lambda)L(t) \quad \ldots \quad (4.5.11)$$

$$f_4 = \lambda L(t) + \omega T_p(t) - (\mu_4 + \beta_4 + \pi)P(t) \quad \ldots \quad (4.5.12)$$

$$f_5 = b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) \quad \ldots \quad (4.5.13)$$

$$f_6 = b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) \quad \ldots \quad (4.5.14)$$

$$f_7 = b_4 - (\mu_5 + \beta_5 + \gamma)P_m(t) \quad \ldots \quad (4.5.15)$$

$$\begin{aligned}f_8 = {} & b_1 - (\mu_1 + \beta_1 + \eta)A(t) + \eta A(t) - (\mu_2 + \beta_2 + \sigma)E(t) + \sigma E(t) - (\mu_3 + \beta_3 + \lambda)L(t) + \lambda L(t) + \pi P(t) \\ & - (\mu_4 + \beta_4 + \pi)P(t) + b_2 - (\mu_6 + \beta_6 + \alpha)C_P(t) + \alpha C_P(t) + b_3 - (\mu_7 + \beta_7 + \omega)T_p(t) + \omega T_p(t) + b_4 \\ & - (\mu_5 + \beta_5 + \gamma)P_m(t) + \gamma P_m(t) \quad \ldots \quad (4.5.16)\end{aligned}$$

The basic reproduction number $R_0$ is defined as the number of mosquitoes an individual would bite during the period of malaria transmission, assuming all others are susceptible. $R_O = 1$ is a threshold below which the generation of secondary cases is insufficient to maintain malaria in humans. $R_O < 1$, the number of mosquitoes will decrease from generation to generation and the

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mosquitoes will be released, and if $R_O > 1$, the number of mosquitoes will increase from generation to generation and the transmission of malaria will continue.

$F_i$ is the attacked compartments' of the mosquitoes' life cycles (Adult, Larva and Pupa) equations (4.5.9, 4.5.11 and 4.5.12).

$$\frac{\partial f_1}{\partial A} = -(\mu_1 + \beta_1 + \eta), \frac{\partial f_1}{\partial L} = 0, \frac{\partial f_1}{\partial P} = 0 \quad \ldots (4.5.17)$$

$$\frac{\partial f_2}{\partial A} = 0, \frac{\partial f_2}{\partial L} = -(\mu_3 + \beta_3 + \lambda), \frac{\partial f_2}{\partial P} = 0 \quad \ldots (4.5.18)$$

$$\frac{\partial f_3}{\partial A} = 0, \quad \frac{\partial f_3}{\partial L} = \lambda, \quad \frac{\partial f_3}{\partial P} = -(\mu_4 + \beta_4 + \pi) \quad \ldots (4.5.19)$$

$V_i$ is the predators compartments' (Copepods, Tadpoles and Purple Martins) equations (4.5.13) – (4.5.15).

$$\frac{\partial f_4}{\partial C_P} = -(\mu_6 + \beta_6 + \alpha), \frac{\partial f_4}{\partial T_P} = 0, \frac{\partial f_4}{\partial P_M} = 0 \quad \ldots (4.5.20)$$

$$\frac{\partial f_5}{\partial C_P} = 0, \frac{\partial f_5}{\partial T_P} = -(\mu_7 + \beta_7 + \omega), \frac{\partial f_5}{\partial P_M} = 0 \quad \ldots (4.5.21)$$

$$\frac{\partial f_6}{\partial C_P} = 0, \frac{\partial f_6}{\partial T_P} = 0, \frac{\partial f_6}{\partial P_M} = -(\mu_5 + \beta_5 + \gamma) \quad \ldots (4.5.22)$$

Using linearization method, the associated matrices at mosquito's free equilibrium($E_O$), after taking partial derivatives.

$$F = \left[\frac{\partial f_i}{\partial x_j}(E_O)\right] \; and \; V = \left[\frac{\partial v_i}{\partial x_j}(E_O)\right] \quad \ldots (4.5.23)$$

With $1 \leq i, j \leq m$ and m is the number of attacked classes, in particular $m = 3$, we have

$$F = \begin{bmatrix} -(\mu_1 + \beta_1 + \eta) & 0 & 0 \\ 0 & -(\mu_3 + \beta_3 + \lambda) & 0 \\ 0 & \lambda & -(\mu_4 + \beta_4 + \pi) \end{bmatrix} \quad \ldots (4.5.24)$$

$$V = \begin{bmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 & 0 \\ 0 & -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & 0 & -(\mu_5 + \beta_5 + \gamma) \end{bmatrix} \quad \ldots (4.5.25)$$

The inverse of non-singular matrix denoted ($A^{-1}$), is the matrix B such that AB=BA=1. It is given as;

$$A^{-1} = \frac{1}{|A|}(AdjA) \quad \ldots (4.5.26)$$

Note that the inverse of a non-square matrix and singular matrix does not exist.

If the inverse of V is given as

$$V = \begin{bmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 & 0 \\ 0 & -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & 0 & -(\mu_5 + \beta_5 + \gamma) \end{bmatrix}$$

$$V^{-1} = \frac{1}{|V|}(AdjV) \quad \ldots (4.5.27)$$

$$|V| = \begin{vmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 & 0 \\ 0 & -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix}$$

$$|V| = -(\mu_6 + \beta_6 + \alpha)\begin{vmatrix} -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} - 0\begin{vmatrix} 0 & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} + 0\begin{vmatrix} -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & 0 \end{vmatrix}$$

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$|V| = -(\mu_6 + \beta_6 + \alpha)(\mu_7 + \beta_7 + \omega)(\mu_5 + \beta_5 + \gamma)$

The minor of an element $v_{ij}$ of a determinant of a matrix V is the determinant obtained from V by deleting the row and column where $v_{ij}$ occurs. It is denoted by $M_{ij}$.

Minor of matrix

$$V = \begin{vmatrix} V_{11} & V_{12} & V_{13} \\ V_{21} & V_{22} & V_{23} \\ V_{31} & V_{32} & V_{33} \end{vmatrix} \quad \ldots (4.5.28)$$

$$M_{11} = \begin{vmatrix} V_{22} & V_{23} \\ V_{32} & V_{33} \end{vmatrix}, M_{12} = \begin{vmatrix} V_{21} & V_{23} \\ V_{31} & V_{33} \end{vmatrix}, M_{13} = \begin{vmatrix} V_{21} & V_{22} \\ V_{31} & V_{32} \end{vmatrix}, \quad \ldots (4.5.29)$$

$$M_{21} = \begin{vmatrix} V_{12} & V_{13} \\ V_{32} & V_{33} \end{vmatrix}, M_{22} = \begin{vmatrix} V_{11} & V_{13} \\ V_{31} & V_{33} \end{vmatrix}, M_{23} = \begin{vmatrix} V_{11} & V_{12} \\ V_{31} & V_{32} \end{vmatrix}, \quad \ldots (4.5.30)$$

$$M_{31} = \begin{vmatrix} V_{12} & V_{13} \\ V_{22} & V_{23} \end{vmatrix}, \quad M_{32} = \begin{vmatrix} V_{11} & V_{13} \\ V_{21} & V_{23} \end{vmatrix}, M_{33} = \begin{vmatrix} V_{11} & V_{12} \\ V_{21} & V_{22} \end{vmatrix} \quad \ldots (4.5.31)$$

$$M_{11} = \begin{vmatrix} V_{22} & V_{23} \\ V_{32} & V_{33} \end{vmatrix} = \begin{vmatrix} -(\mu_7 + \beta_7 + \omega) & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} = (\mu_7 + \beta_7 + \omega)(\mu_5 + \beta_5 + \gamma)$$

$$M_{12} = \begin{vmatrix} V_{21} & V_{23} \\ V_{31} & V_{33} \end{vmatrix} = \begin{vmatrix} 0 & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} = 0$$

$$M_{13} = \begin{vmatrix} V_{21} & V_{22} \\ V_{31} & V_{32} \end{vmatrix} = \begin{vmatrix} 0 & -(\mu_7 + \beta_7 + \omega) \\ 0 & 0 \end{vmatrix} = 0$$

$$M_{21} = \begin{vmatrix} V_{12} & V_{13} \\ V_{32} & V_{33} \end{vmatrix} = \begin{vmatrix} 0 & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} = 0$$

$$M_{22} = \begin{vmatrix} V_{11} & V_{13} \\ V_{31} & V_{33} \end{vmatrix} = \begin{vmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 \\ 0 & -(\mu_5 + \beta_5 + \gamma) \end{vmatrix} = (\mu_6 + \beta_6 + \alpha)(\mu_5 + \beta_5 + \gamma)$$

$$M_{23} = \begin{vmatrix} V_{11} & V_{12} \\ V_{31} & V_{32} \end{vmatrix} = \begin{vmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 \\ 0 & 0 \end{vmatrix} = 0$$

$$M_{31} = \begin{vmatrix} V_{12} & V_{13} \\ V_{22} & V_{23} \end{vmatrix} = \begin{vmatrix} 0 & 0 \\ -(\mu_7 + \beta_7 + \omega) & 0 \end{vmatrix} = 0$$

$$M_{32} = \begin{vmatrix} V_{11} & V_{13} \\ V_{21} & V_{23} \end{vmatrix} = \begin{vmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 \\ 0 & 0 \end{vmatrix} = 0$$

$$M_{33} = \begin{vmatrix} V_{11} & V_{12} \\ V_{21} & V_{22} \end{vmatrix} = \begin{vmatrix} -(\mu_6 + \beta_6 + \alpha) & 0 \\ 0 & -(\mu_7 + \beta_7 + \omega) \end{vmatrix} = (\mu_6 + \beta_6 + \alpha)(\mu_7 + \beta_7 + \omega)$$

The cofactor of an element $V_{ij}$ is a signed minor of the determinant. The sign of the minor is +ve if i+j is even and -ve if i+j is odd and it is denoted as $C_{ij}$

$$C_{ij} = \begin{vmatrix} V_{11} & V_{12} & V_{13} \\ V_{21} & V_{22} & V_{23} \\ V_{31} & V_{32} & V_{33} \end{vmatrix} = \begin{vmatrix} +V_{11} & -V_{12} & +V_{13} \\ -V_{21} & +V_{22} & -V_{23} \\ +V_{31} & -V_{32} & +V_{33} \end{vmatrix}$$

$$AdjV = V_c^T = \begin{vmatrix} (\mu_7 + \beta_7 + \omega)(\mu_5 + \beta_5 + \gamma) & 0 & 0 \\ 0 & (\mu_6 + \beta_6 + \alpha)(\mu_5 + \beta_5 + \gamma) & 0 \\ 0 & 0 & (\mu_6 + \beta_6 + \alpha)(\mu_7 + \beta_7 + \omega) \end{vmatrix}$$

The Adjoint or Adjugate of a matrix is obtained by transposing the matrix of the cofactors and is denoted by $AdjV = V_c^T$ $\ldots (4.5.32)$

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$$\mathbf{V^{-1}} = \frac{\mathbf{1}}{|\mathbf{V}|}(\mathbf{AdjV}) = -\frac{\mathbf{1}}{(\boldsymbol{\mu_6}+\boldsymbol{\beta_6}+\boldsymbol{\alpha})(\boldsymbol{\mu_7}+\boldsymbol{\beta_7}+\boldsymbol{\omega})(\boldsymbol{\mu_5}+\boldsymbol{\beta_5}+\boldsymbol{\gamma})}$$

$$\begin{pmatrix} (\boldsymbol{\mu_7}+\boldsymbol{\beta_7}+\boldsymbol{\omega})(\boldsymbol{\mu_5}+\boldsymbol{\beta_5}+\boldsymbol{\gamma}) & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & (\boldsymbol{\mu_6}+\boldsymbol{\beta_6}+\boldsymbol{\alpha})(\boldsymbol{\mu_5}+\boldsymbol{\beta_5}+\boldsymbol{\gamma}) & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & (\boldsymbol{\mu_6}+\boldsymbol{\beta_6}+\boldsymbol{\alpha})(\boldsymbol{\mu_7}+\boldsymbol{\beta_7}+\boldsymbol{\omega}) \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} -\frac{1}{(\mu_6+\beta_6+\alpha)} & 0 & 0 \\ 0 & -\frac{1}{(\mu_7+\beta_7+\omega)} & 0 \\ 0 & 0 & -\frac{1}{(\mu_5+\beta_5+\gamma)} \end{pmatrix}$$

Then the next generation matrix denoted by $FV^{-1}$ is given as

$$FV^{-1} = \begin{pmatrix} -(\mu_1+\beta_1+\eta) & 0 & 0 \\ 0 & -(\mu_3+\beta_3+\lambda) & 0 \\ 0 & \lambda & -(\mu_4+\beta_4+\pi) \end{pmatrix} \begin{pmatrix} -\frac{1}{(\mu_6+\beta_6+\alpha)} & 0 & 0 \\ 0 & -\frac{1}{(\mu_7+\beta_7+\omega)} & 0 \\ 0 & 0 & -\frac{1}{(\mu_5+\beta_5+\gamma)} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)} & 0 & 0 \\ 0 & \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)} & 0 \\ 0 & -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)} \end{pmatrix} \quad \ldots \text{ (4.5.33)}$$

We fine the eigenvalues of $FV^{-1}$ by setting the determinant of

$$|FV^{-1} - \lambda I| = 0 \quad \ldots \text{ (4.5.34)}$$

Equation of the form $FV^{-1}X = \lambda X$ occur, where $FV^{-1} = [v_{ij}]$ is a square matrix and λ is a number (scalar). Clearly, $X = 0$ is a solution for any value of λ and is not normally useful. For non-trivial solutions, i.e $X \neq 0$, the values of λ are called the eigenvalues, characteristic values or latent roots of the matrix $FV^{-1}$and the corresponding solutions of the given equations $FV^{-1}X = \lambda X$ are called the eigenvectors or characteristic vectors of $FV^{-1}$.$|FV^{-1} - \lambda I|$is called the characteristic determinant of $FV^{-1}$ and $|FV^{-1} - \lambda I| = 0$ is the characteristic equation. On expanding the determinant, this gives a polynomial of degree n and the solution of the characteristic equation gives the values of λ i.e. the eigenvalues of $FV^{-1}$.

$$|FV^{-1} - \lambda I| = \left| \begin{pmatrix} \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)} & 0 & 0 \\ 0 & \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)} & 0 \\ 0 & -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)} \end{pmatrix} - \lambda \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix} \right|$$

$$|FV^{-1} - \lambda I| = \left| \begin{pmatrix} \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)} & 0 & 0 \\ 0 & \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)} & 0 \\ 0 & -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)} \end{pmatrix} - \begin{pmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{pmatrix} \right|$$

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$$|FV^{-1}-\lambda I| = \begin{vmatrix} \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}-\lambda & 0 & 0 \\ 0 & \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda & 0 \\ 0 & -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda \end{vmatrix}$$

$$\left(\frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}-\lambda\right)\begin{vmatrix} \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda & 0 \\ -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda \end{vmatrix} - 0\begin{vmatrix} 0 & 0 \\ 0 & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda \end{vmatrix} + 0\begin{vmatrix} 0 & \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda \\ 0 & -\frac{\lambda}{(\mu_7+\beta_7+\omega)} \end{vmatrix} = 0$$

$$\left(\frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}-\lambda\right)\begin{vmatrix} \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda & 0 \\ -\frac{\lambda}{(\mu_7+\beta_7+\omega)} & \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda \end{vmatrix} = 0$$

$$\left(\frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}-\lambda\right)\left(\frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda\right)\left(\frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda\right) = 0$$

$$\left(\frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}-\lambda\right) = 0 \; implies \; that \;\; \lambda_1 = \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)} \qquad \ldots \; (4.5.35)$$

$$\left(\frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}-\lambda\right) = 0 \; implies \; that \;\; \lambda_2 = \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)} \qquad \ldots \; (4.5.36)$$

$$\left(\frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)}-\lambda\right) = 0 \; implies \; that \;\; \lambda_3 = \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)} \qquad \ldots \; (4.5.37)$$

$$R_0 = \frac{(\mu_1+\beta_1+\eta)}{(\mu_6+\beta_6+\alpha)}, \; R_1 = \frac{(\mu_3+\beta_3+\lambda)}{(\mu_7+\beta_7+\omega)}, \; and \;\; R_2 = \frac{(\mu_4+\beta_4+\pi)}{(\mu_5+\beta_5+\gamma)} \qquad \ldots (4.5.38)$$

It should be noted that $(\mu_6+\beta_6+\alpha) > (\mu_1+\beta_1+\eta), (\mu_7+\beta_7+\omega) > (\mu_3+\beta_3+\lambda) \; and \; (\mu_5+\beta_5+\gamma) > (\mu_4+\beta_4+\pi)$ and the following conditions holds $R_0 < 1, \; R_1 < 1, and \;\; R_2 < 1$, where $R_0, R_1 \; and \; R_2$ are defined in equation (4.5.38).

### 4.1.7 Result for Diekmann Conditions

Since $R_O < 1$ under Diekmann's conditions, the stability analysis of the free equilibrium state is stable. Since $\alpha = \frac{N_L(t)}{L}, \omega = \frac{N_P(t)}{P} \; and \; \gamma = \frac{N_A(t)}{A}$, With natural implication, it means that the rate at which the proportion of mosquito larvae turn into pupae and pupae into adults is low, almost equal to zero, there will be no adult Anopheles mosquito for malaria transmission in our society, when more natural predators introduced to feed on larvae, pupae and adult mosquito.

### 4.1.8 List of Numerical Experiments of the Model

The following experiments are carried out

**Experiment 1**: Effect of introducing one natural predator, copepod on mosquitoes' larva ($C_p = 500 \; T_p = 0, and \;\; P_m = 0$).

**Experiment 2**: Effect of introducing two natural predators, copepod and tadpole on mosquitoes' larva and pupa respectively ($C_p = 500, T_p = 500 \; and P_m = 0$).

**Experiment 3**: Effect of introducing three natural predators, copepod, tadpole and purple martins on mosquitoes' larva, pupa and adult respectively ($C_p = 500, T_p = 500 \; and \; P_m = 130$).

**Experiment 4:** Comparison of the effect of introducing one, two and three natural predator on larva.

**Experiment 5:** Comparison of the effect of introducing two and three natural predator on pupa.

**Experiment 6**: Effect of introducing one natural predator, tadpole on mosquitoes' pupa($T_p = 500$).

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**Experiment 7**: Effect of introducing two natural predators, tadpole and purple martins on mosquitoes' pupa and adult respectively ($C_p = 0, T_p = 500\ and\ P_m = 130$).

**Experiment 8:** Comparison of the effect of introducing one, two and three natural predator on pupa.

**Experiment 9:** Comparison of the effect of introducing two and three natural predator on adult.

**Experiment 10**: Effect of introducing one natural predator, purple martins on mosquitoes' adult( $P_m = 130, C_p = 0, and\ T_p = 0$ ).

**Experiment 11**: Effect of introducing two natural predators, copepod and purple martins on mosquitoes' larva and adult respectively ($C_p = 500, T_p = 0\ and\ P_m = 130$).

**Experiment 12:** Comparison of the effect of introducing one, two and three natural predator on adult.

**Experiment 13:** Comparison of the effect of introducing two and three natural predator on adult.

**Experiment 14**: Effect of introducing low rate of natural predators, copepod, on mosquitoes' larva ($C_p = 500$).

**Experiment 15**: Effect of introducing high rate of natural predators, copepod, on mosquitoes' larva ($C_p = 2000$).

**Experiment 16**: comparison of the effect of introducing low and high rate of natural predators, copepod, on mosquitoes' larva.

**Experiment 17**: Effect of introducing low rate of natural predator, copepod, on mosquitoes' pupa ($T_p = 2000$).

**Experiment 18**: Effect of introducing high rate of natural predator, tadpole, on mosquitoes' pupa ($T_p = 2000$)

**Experiment 19**: Comparison of the effect of introducing low and high rate of natural predators, tadpole on mosquitoes' pupa.

**Table4.1: Numerical values of the variables and parameters**

| Variables/Parameters | Values | Source |
|---|---|---|
| A(t) | 500 | Assumed |
| E (t) | 100000 | Guerra, (2014) |
| L(t) | 90000 | Assumed |
| P(t) | 80000 | Assumed |
| N(t) | 270000 | Assumed |
| $\mathbf{C_P}$(t) | 500 | Practical |
| $T_p$(t) | 500 | Practical |
| $P_m$(t) | 130 | Assumed |
| $b_1$ | 0.02 | Olivier, (202) |
| $b_2$ | 0.21 | Gearty, (2021) |
| $b_3$ | 0.9 | Calef, (1973) |
| $b_4$ | 0.5 | Joshua,( 1971) |
| $\mu_1$ | 0.4 | Mathews, (2020) |
| $\mu_2$ | 0.3 | Clements, (1981) |
| $\mu_3$ | 0.2 | Couret, (2014) |
| $\mu_4$ | 0.1 | Mondragon, (2020) |
| $\mu_5$ | 0.5 | Jervis, (2019) |
| $\mu_6$ | 0.02 | Charyl, (2011) |
| $\mu_7$ | 0.01 | Szekely, (2022) |

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| | | |
|---|---|---|
| $\beta_1$ | $40^oC(0.3)$ | Beck-Johnson,, (2013) |
| $\beta_2$ | $37^oC(0.57)$ | Sukiato, (2019) |
| $\beta_3$ | $28^oC(0.0110)$ | Adam, (2014) |
| $\beta_4$ | $28^oC(0.0110)$ | Adam, (2014) |
| $\beta_5$ | $25^oC$ (0.13) | Fred, (2014) |
| $\beta_6$ | $40^oC(0.01)$ | Jiang, (2014) |
| $\beta_7$ | $35^oC(0.02)$ | Halsbank-Lenk,(2014) |
| $\eta$ | 0.002 | Practical |
| $\sigma$ | 0.00004 | Practical |
| $\lambda$ | 0.00005 | Practical |
| $\pi$ $\pi$ | 0.01 | Practical |
| $\alpha$ | 0.5 | Practical |
| $\omega$ | 0.5 | Practical |
| $\gamma$ | 0.9 | Practical |

**Experiment 1:** Effect of introducing one natural predator, copepod on mosquitoes' larva.

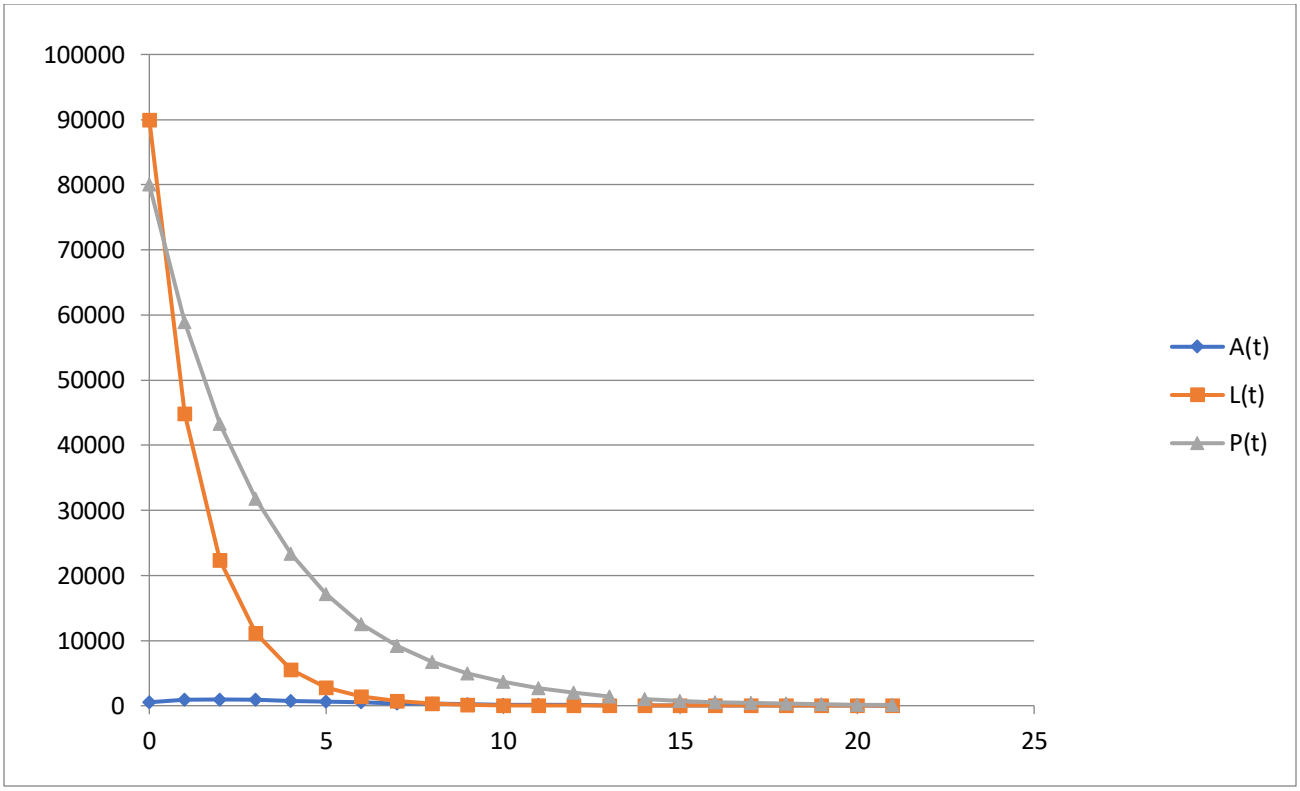


**Figure 4.1:** Number of mosquitoes' larva when one natural predator, copepod was introduced($C_p = 500, T_p = 0, P_m = 0, \alpha = 0.5, \mu_6 = 0.02,\ \beta_6 = 0.01\ and\ b_2 = 0.21$).

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**Table 4.2**: Function values at $t \in [0,21]$ when one natural predator, copepod was introduced to mosquitoes' larva.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 836.5 | 898.0 | 830.8 | 713.5 | 586.4 | 468.3 | 366.6 | 282.9 | 215.9 | 163.5 | 123.0 | 92.1 | 68.7 | … | 6.1 |
| L(t) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 0.0 |
| P(t) | 80000 | 58678.5 | 43038.9 | 31567.5 | 23153.4 | 16981.9 | 12455.4 | 9135.4 | 6700.4 | 4914.4 | 3604.4 | 2643.7 | 1939.0 | 1422.2 | … | 119.1 |

**Experiment 2:** Effect of introducing two natural predators, copepod and tadpole on mosquitoes' larva and pupa respectively.

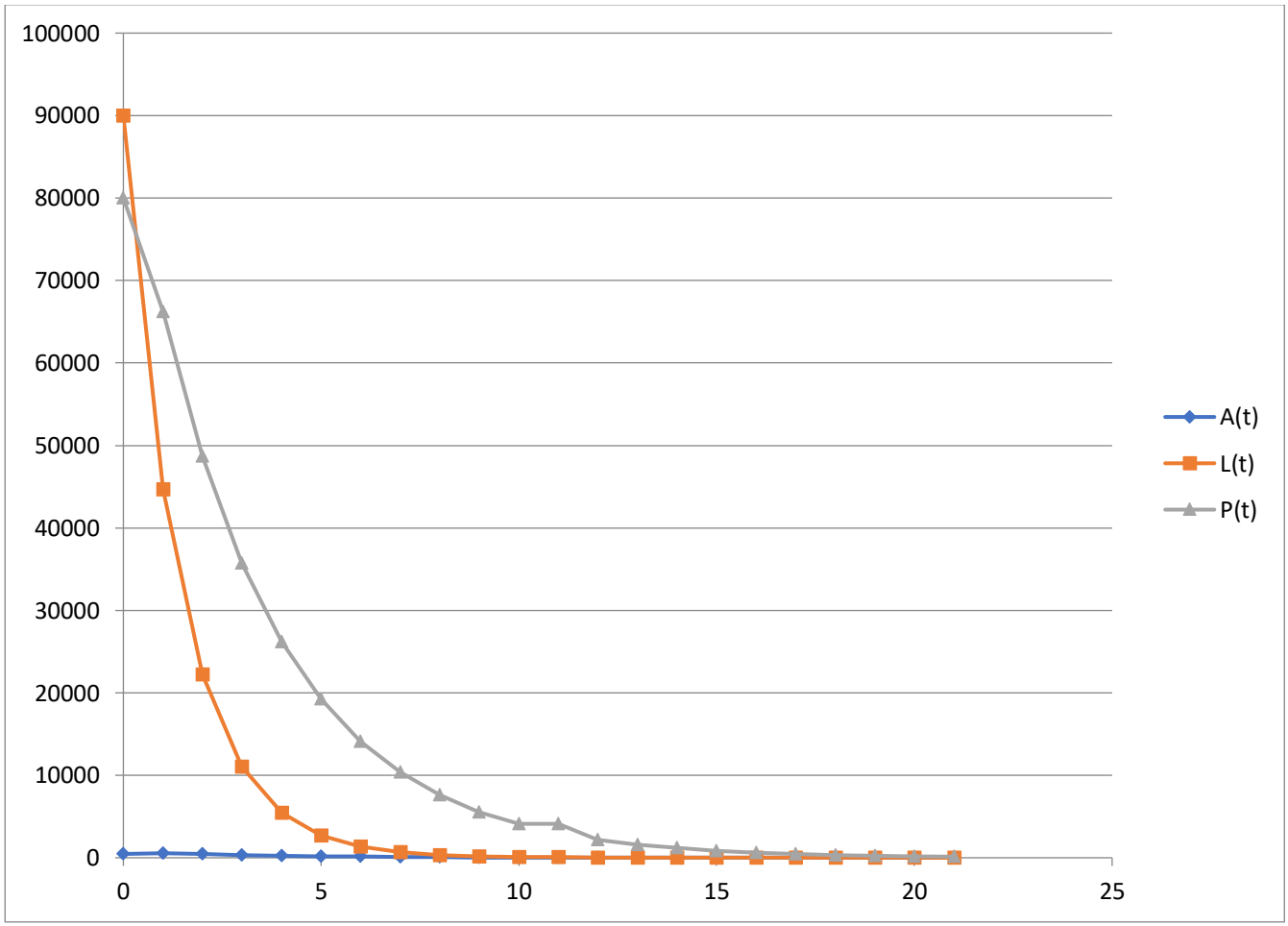


**Figure 4.2:** Number of mosquitoes' larva and pupa when two natural predators, copepod and tadpole are introduced respectively($C_p = 500, T_p = 500,\ P_m = 0,\ \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01,\ b_2 = 0.21, \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02\ and\ b_3 = 0.9$)

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**Table 4.3**: Function values at $t \in [0,21]$ when two natural predators, copepod and tadpole are introduced to mosquitoes' larva and pupa respectively.

| T | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | ... | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 481.5 | 382.2 | 287.7 | 212.9 | 156.6 | 115.0 | 84.4 | 61.9 | 45.4 | 33.3 | 24.4 | 17.9 | 13.1 | ... | 1.1 |
| L(t) | 90000 | 44864.9 | 22341.9 | 11117.6 | 5529.3 | 2749.0 | 1366.3 | 679.0 | 337.4 | 167.6 | 83.3 | 41.4 | 20.6 | 10.2 | ... | 0.0 |
| P(t) | 80000 | 58905.7 | 43280.4 | 31769.3 | 23309.5 | 17099.2 | 12542.3 | 9199.4 | 6747.4 | 4948.9 | 3629.8 | 2662.3 | 1952.6 | 1432.1 | ... | 119.9 |

**Experiment 3:** Effect of introducing three natural predators, copepod, tadpole and purple martins on mosquitoes' larva, pupa and adult.

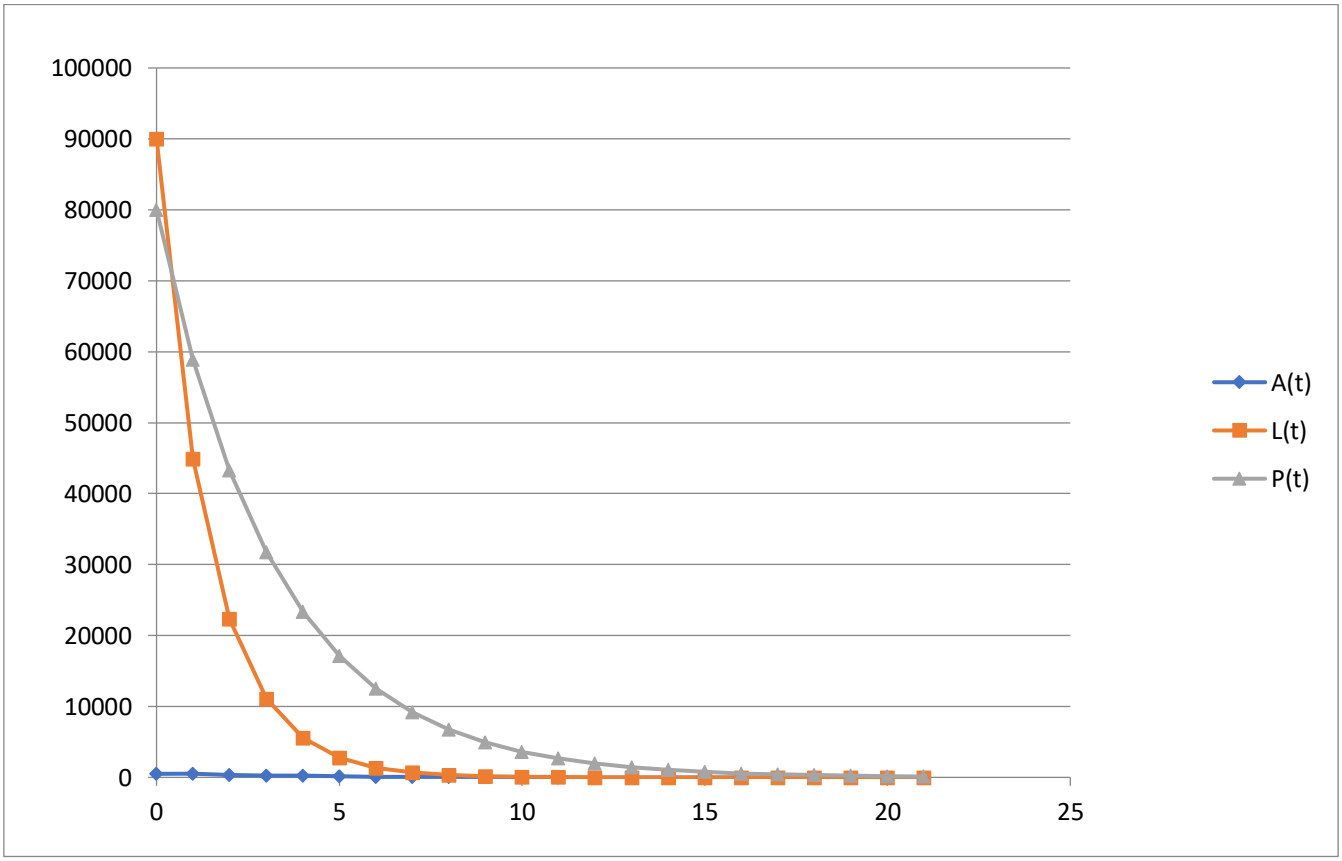


**Figure 4.3:** Number of mosquitoes' larva, pupa and adult, when three natural predators, copepod, tadpole and purple martins are introduced respectively $(C_p = 500, T_p = 500,\ P_m = 130, \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01,\ b_2 = 0.21,\ \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02, b_3 = 0.9,\ \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13\ and\ b_4 = 0.5)$.

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**Table 4.4**: Function values at $t \in [0,21]$ when three natural predators, copepod, tadpole and purple martins are introduced respectively.

| T | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 893.2 | 954.3 | 873.8 | 743.6 | 606.5 | 481.5 | 375.2 | 288.5 | 219.7 | 166.0 | 124.7 | 93.2 | 69.4 | … | 6.2 |
| L(t) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 0.0 |
| P(t) | 80000 | 58896.1 | 43263.4 | 31751.5 | 23294.1 | 17086.9 | 12532.9 | 9192.4 | 6742.2 | 4945.1 | 3627.0 | 2660.2 | 1951.1 | 1431.0 | … | 119.8 |

**Experiment 4:** Comparison of the effect of introducing one, two and three natural predator, copepod on mosquitoes' larva.

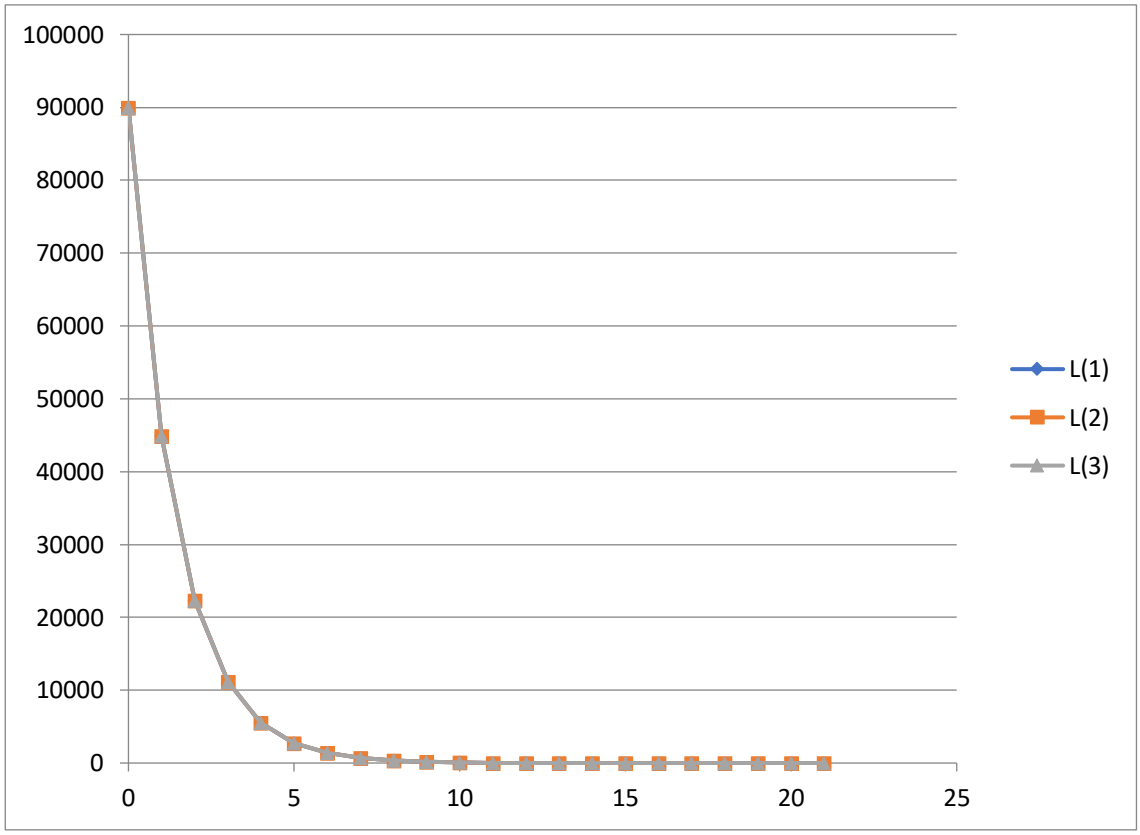


**Figure 4.4:** Number of mosquitoes' larva when one, two and three natural predators tadpoles are compared on larva respectively ($T_{1,2,\&3} = 500,\ \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02,\ and\ b_3 = 0.9$)

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**Table 4.5**: Function values at $t \in [0,21]$ when one, two and three natural predators, copepod, tadpole and purple martins are compared respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| L(1) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 6.2 |
| L(2) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 0.0 |
| L(3) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 119.8 |

**Experiment 5:** Comparison of the effect of introducing two and three natural predator, purple martins on mosquitoes' adult.

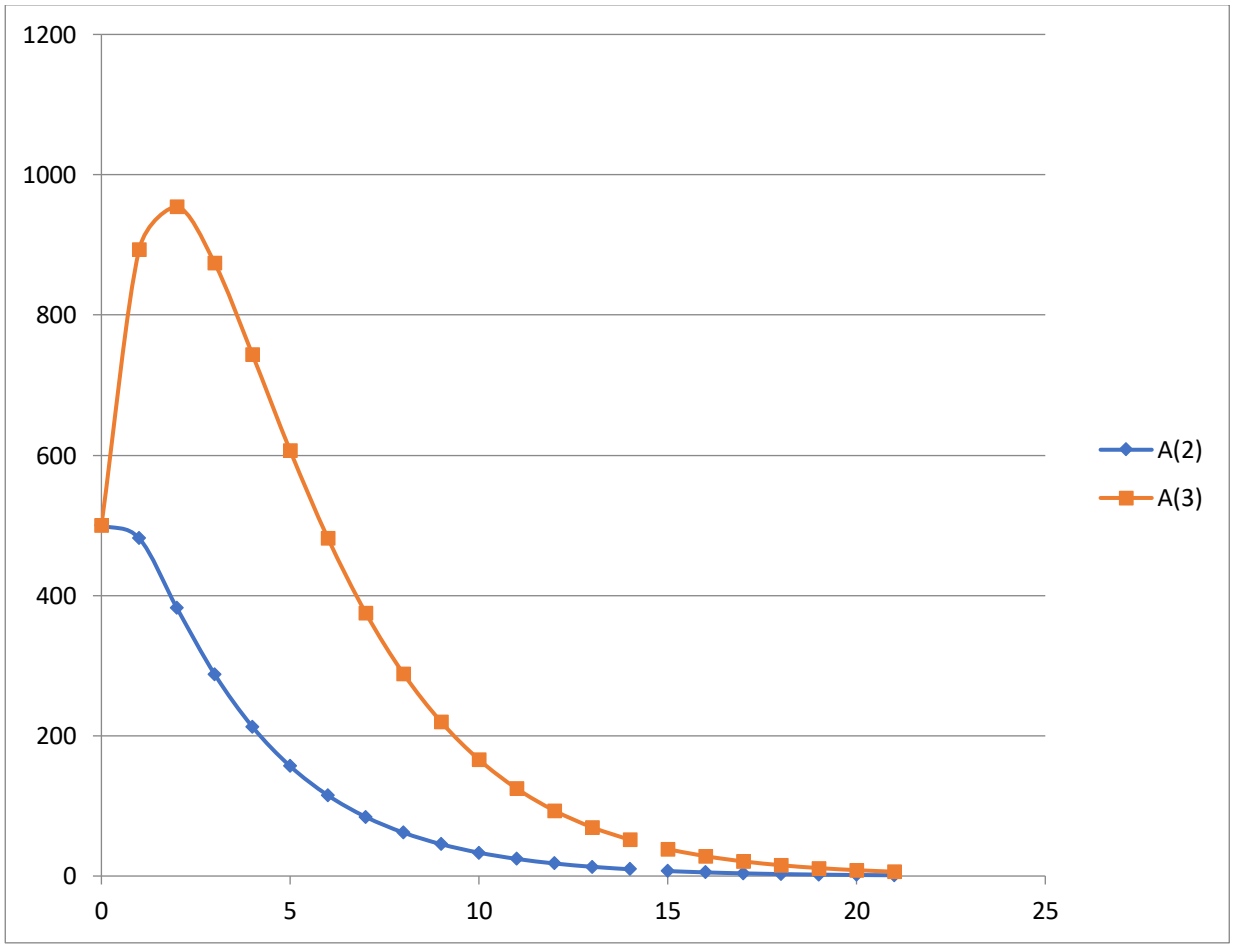


**Figure 4.5:** Number of mosquitoes' adult when two and three natural predator, purple martins are compared respectively ( $P_{2\&3} = 500, \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13, and\ \ b_4 = 0.5$).

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**Table 4.6**: Function values at $t \in [0,21]$ when two and three natural predators, purple martins are compared respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(1) | 500 | 836.5 | 898.0 | 830.8 | 713.5 | 586.4 | 468.3 | 366.6 | 282.9 | 215.9 | 163.5 | 123.0 | 92.1 | 68.7 | … | 6.1 |
| A(2) | 500 | 481.5 | 382.2 | 287.7 | 212.9 | 156.6 | 115.0 | 84.4 | 61.9 | 45.4 | 33.3 | 24.4 | 17.9 | 13.1 | … | 1.1 |
| A(3) | 500 | 893.2 | 954.3 | 873.8 | 743.6 | 606.5 | 481.5 | 375.2 | 288.5 | 219.7 | 166.0 | 124.7 | 93.2 | 69.4 | … | 6.2 |

**Experiment 6:** Effect of introducing one natural predator, tadpole on mosquitoes' pupa.

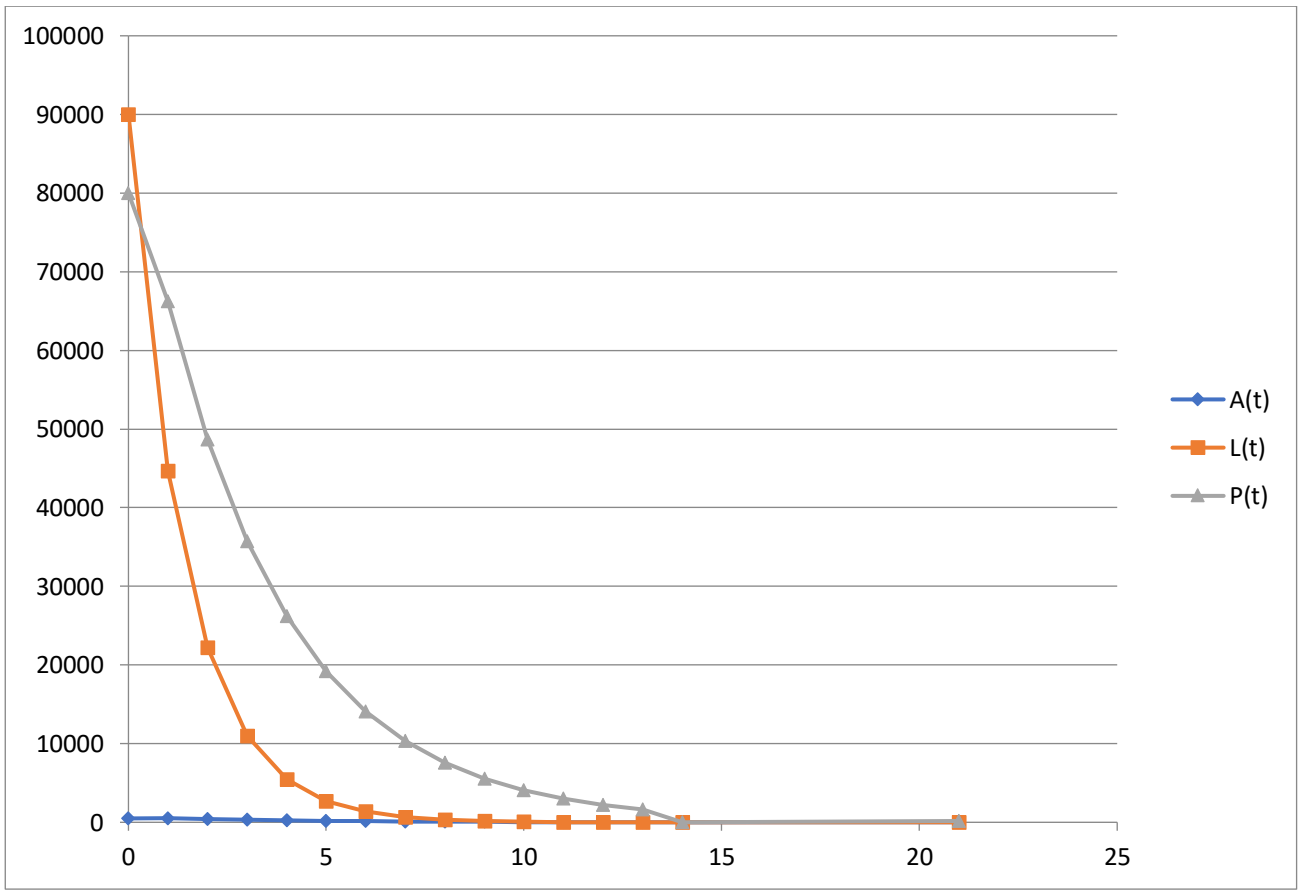


**Figure 4.6:** Number of mosquitoes' pupa when one natural predator, tadpole was introduced to mosquito pupa ( $T_m = 500$ $C_p = 0$, $P_m = 0$, $\omega = 0.5$, $\mu_7 = 0.01$, $\beta_7 = 0.02$, $and$ $b_3 = 0.9$)

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**Table 4.7**: Function values at $t \in [0,21]$ when one natural predator, tadpole was introduced to mosquitoes' pupa.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 526.2 | 425.9 | 322.5 | 239.1 | 176.0 | 129.3 | 94.9 | 69.6 | 51.0 | 37.4 | 27.5 | 20.1 | 14.8 | … | 1.2 |
| L(t) | 90000 | 44694.9 | 22196.2 | 11023.1 | 5474.4 | 2718.8 | 1350.3 | 670.7 | 333.1 | 165.5 | 82.2 | 40.8 | 20.3 | 10.1 | … | 0.0 |
| P(t) | 80000 | 66241.1 | 48661.7 | 35716.7 | 26205.1 | 19223.0 | 14100.1 | 10342.0 | 7585.4 | 5563.6 | 4080.6 | 2992.9 | 2195.1 | 1610.0 | … | 134.8 |

**Experiment 7:** Effect of introducing two natural predators, tadpole and purple martins on mosquitoes' pupa and adult respectively.

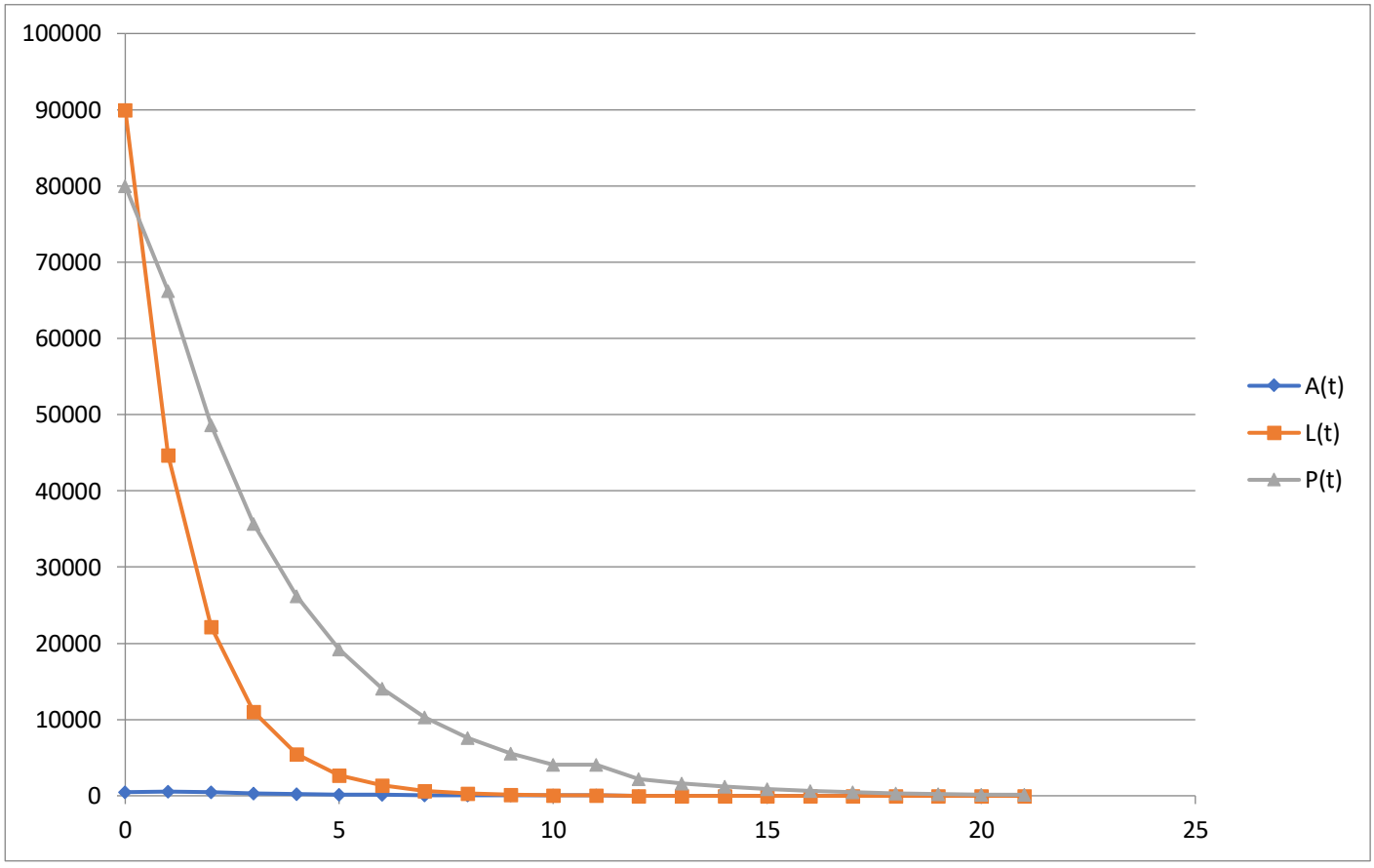


**Figure 4.7:** Number of mosquitoes' pupa and adult when two natural predators, tadpole and purple martins are introduced respectively ($T_p = 500, P_m = 130,\ C_p = 0, \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02,\ b_3 = 0.9,\ \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13, and\ b_4 = 0.5$).

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**Table 4.8**: Function values at $t \in [0,21]$ when two natural predators, tadpole and purple martins are introduced to mosquitoes' pupa and adult.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 550.6 | 445.2 | 334.4 | 245.9 | 179.8 | 131.4 | 96.0 | 70.2 | 51.4 | 37.6 | 37.6 | 20.2 | 14.8 | … | 1.2 |
| L(t) | 90000 | 44694.9 | 22196.2 | 11023.1 | 5474.4 | 2718.8 | 1350.3 | 670.7 | 333.1 | 165.5 | 82.2 | 82.2 | 20.3 | 10.1 | … | 0.0 |
| P(t) | 80000 | 66240.1 | 48659.9 | 35714.8 | 26203.4 | 19221.6 | 14099.0 | 10341.2 | 7584.8 | 5563.1 | 4080.3 | 4080.3 | 2195.0 | 1609.9 | … | 134.8 |

**Experiment 8:** Comparison of the effect of introducing one, two and three natural predator on pupa.

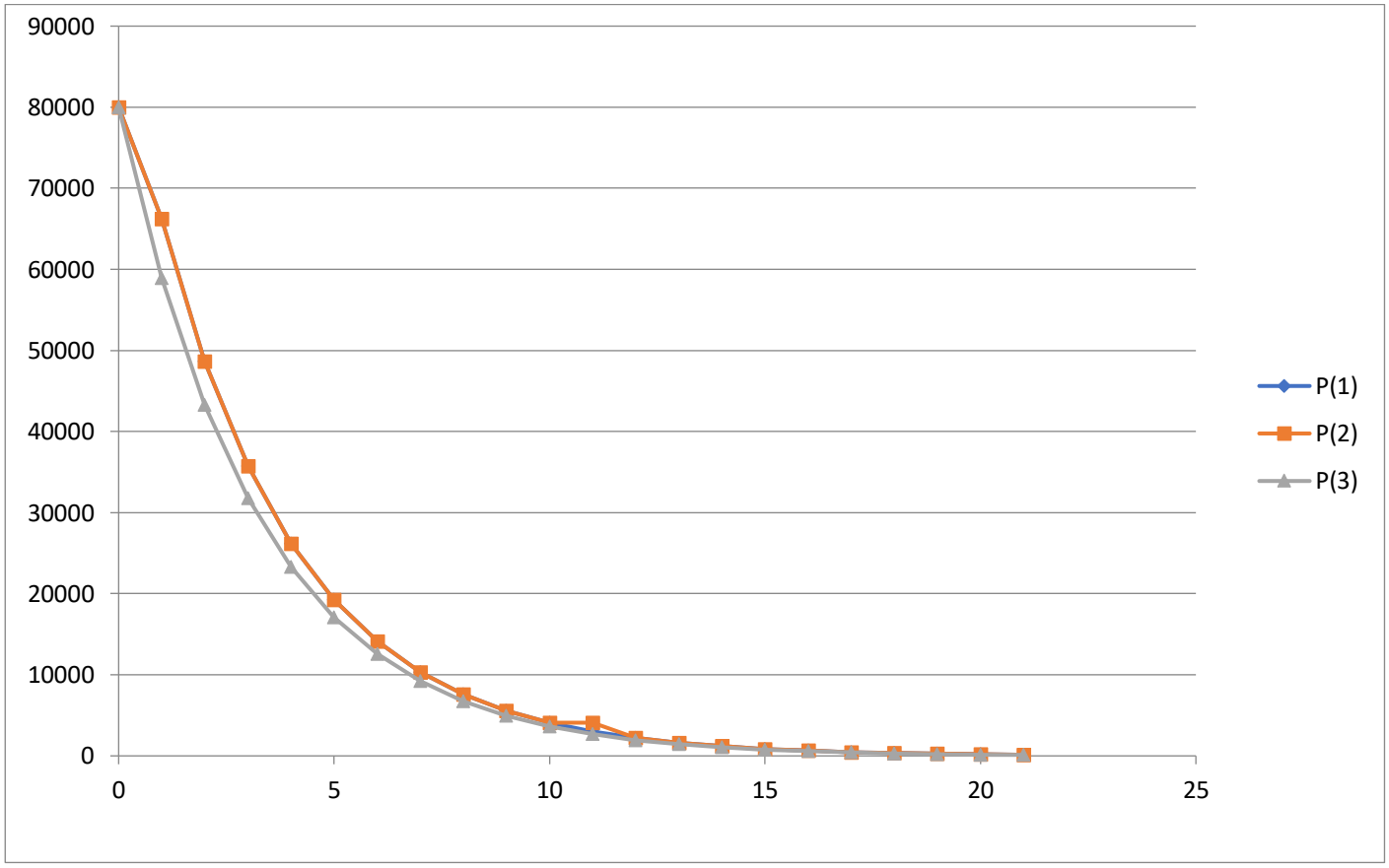


**Figure 4.8:** Number of mosquitoes' pupa when one, two and three natural predators are compared respectively ( $T_{1,2\,\&3} = 500$, $\omega = 0.5$, $\mu_7 = 0.01$, $\beta_7 = 0.02$, $and\ b_3 = 0.9$).

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**Table 4.9**: Function values at $t \in [0,21]$ when one, two and three natural predators, copepod, tadpole and purple martins are compared on mosquitoes' pupa respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P(1) | 80000 | 66241.1 | 48661.7 | 35716.7 | 26205.1 | 19223.0 | 14100.1 | 10342.0 | 7585.4 | 5563.6 | 4080.6 | 2992.9 | 2195.1 | 1610.0 | … | 134.8 |
| P(2) | 80000 | 66240.1 | 48659.9 | 35714.8 | 26203.4 | 19221.6 | 14099.0 | 10341.2 | 7584.8 | 5563.1 | 4080.3 | 4080.3 | 2195.0 | 1609.9 | … | 134.8 |
| P(3) | 80000 | 58896.1 | 43263.4 | 31751.5 | 23294.1 | 17086.9 | 12532.9 | 9192.4 | 6742.2 | 4945.1 | 3627.0 | 2660.2 | 1951.1 | 1431.0 | … | 119.8 |

**Experiment 9:** Comparison of the effect of introducing two and three natural predator purple martins on adult.

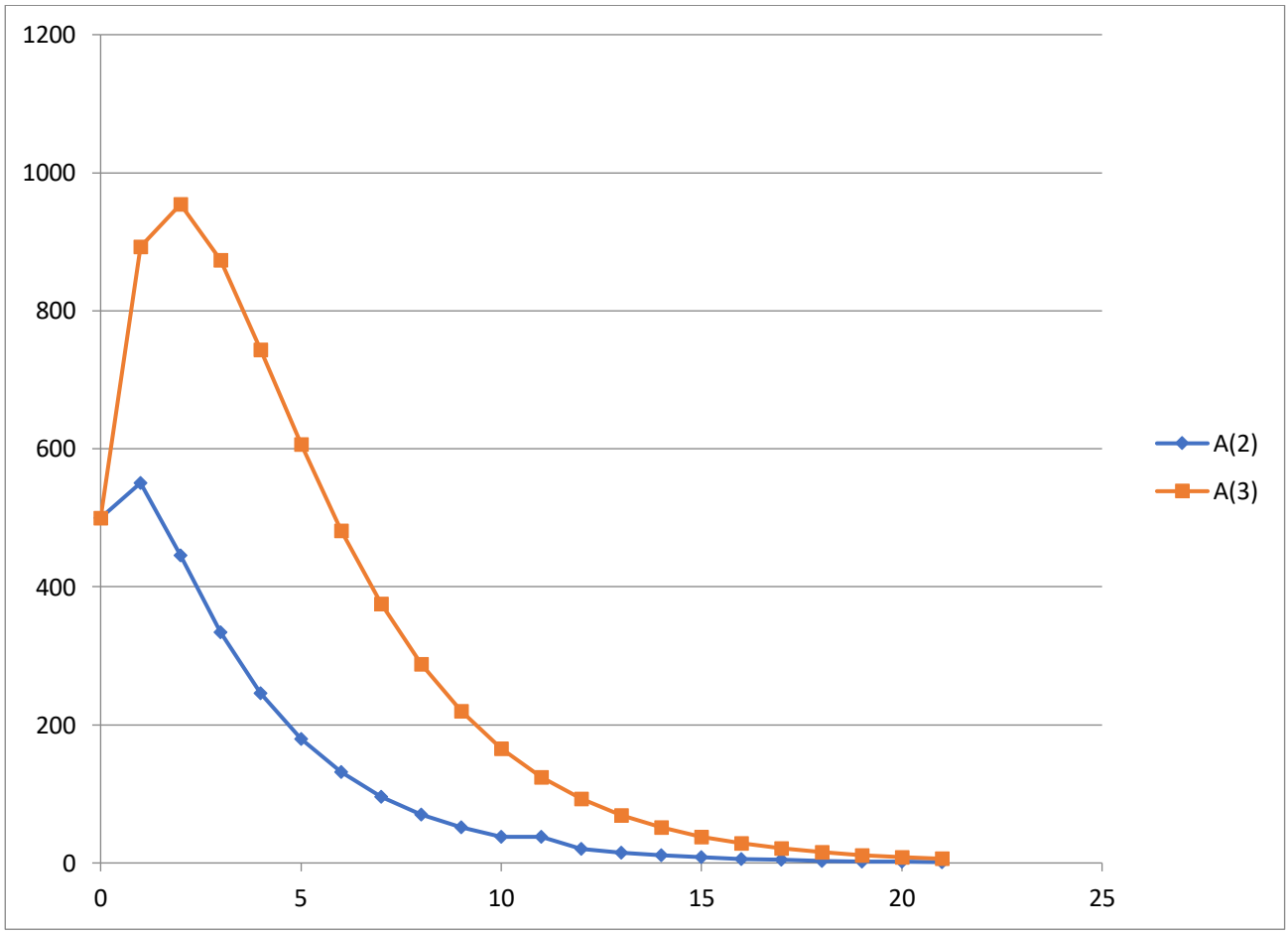


**Figure 4.9:** Number of mosquitoes' adult when two and three natural predators, purple martins are compared respectively ( $P_{2\&3} = 130, \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13\ and\ b_4 = 0.5$).

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**Table 4.10**: Function values at $t \in [0,21]$ when two and three natural predators, tadpole and purple martins are compared on mosquitoes' adult's respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(2) | 500 | 550.6 | 445.2 | 334.4 | 245.9 | 179.8 | 131.4 | 96.0 | 70.2 | 51.4 | 37.6 | 37.6 | 20.2 | 14.8 | … | 1.2 |
| A(3) | 500 | 893.2 | 954.3 | 873.8 | 743.6 | 606.5 | 481.5 | 375.2 | 288.5 | 219.7 | 166.0 | 124.7 | 93.2 | 69.4 | … | 6.2 |

**Experiment 10:** Effect of introducing one natural predator, purple martins on mosquitoes' adult.

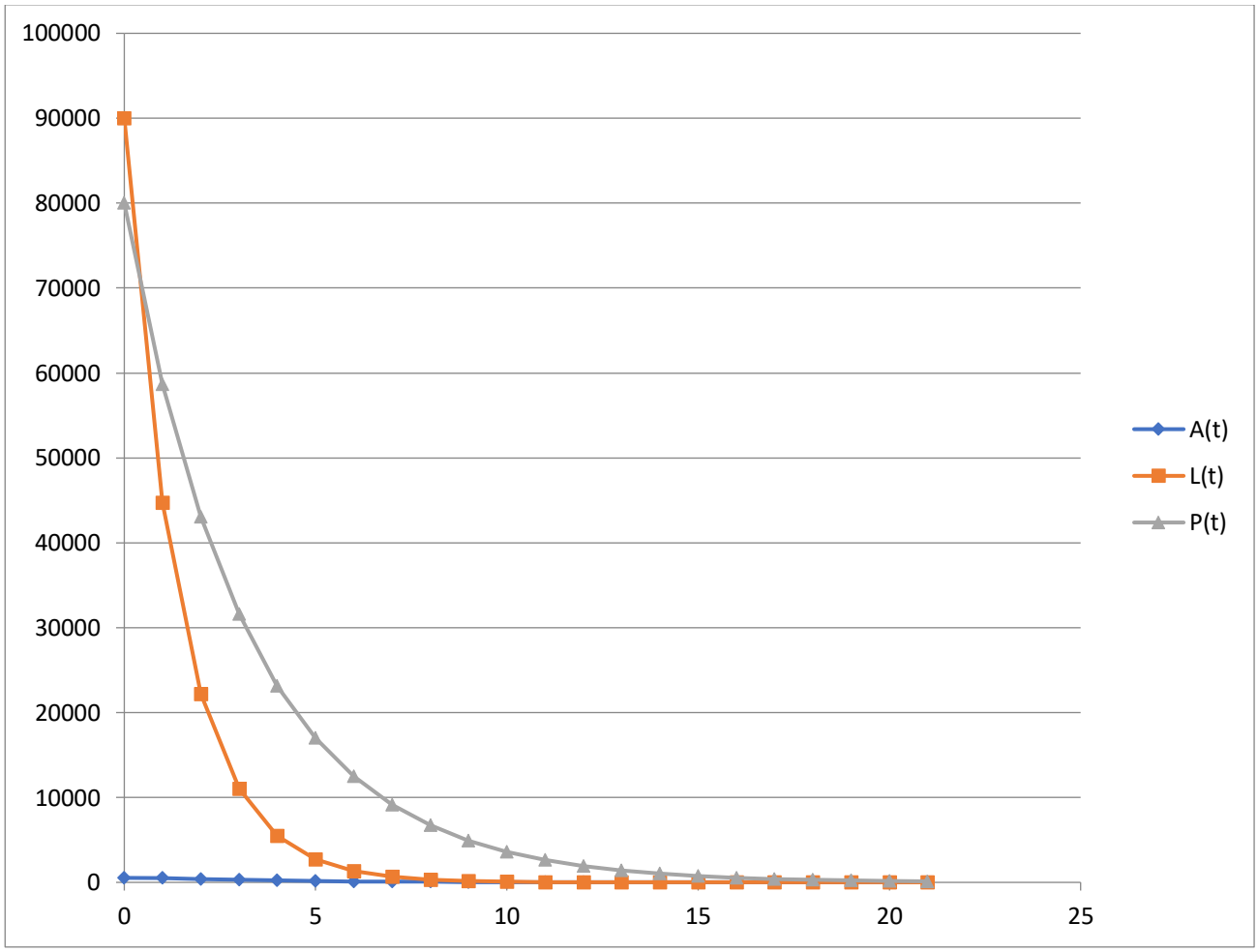


**Figure 4.10:** Number of mosquitoes' adult when one natural predator, purple martins was introduced to mosquito adult ( $P_m = 130, C_p = 0, T_p = 0, \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13\ and\ b_4 = 0.5$).

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**Table 4.11**: Function values at $t \in [0,21]$ when one natural predator, purple martins was introduced to mosquitoes' adult.

| T | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 505.0 | 399.9 | 298.1 | 218.4 | 159.4 | 116.3 | 84.9 | 62.1 | 45.4 | 33.3 | 24.4 | 17.8 | 13.1 | … | 1.1 |
| L(t) | 90000 | 44694.9 | 22196.2 | 11023.1 | 5474.4 | 2718.8 | 1350.3 | 670.7 | 333.1 | 165.5 | 82.2 | 40.8 | 20.3 | 10.1 | … | 0.0 |
| P(t) | 80000 | 58678.5 | 43038.9 | 31567.4 | 23153.4 | 16981.9 | 12455.4 | 9135.4 | 6700.4 | 4914.4 | 3604.4 | 2643.7 | 1939.0 | 1422.2 | … | 119.1 |

**Experiment 11:** Effect of introducing two natural predators, copepod and purple martins on mosquitoes' larva and adult respectively.

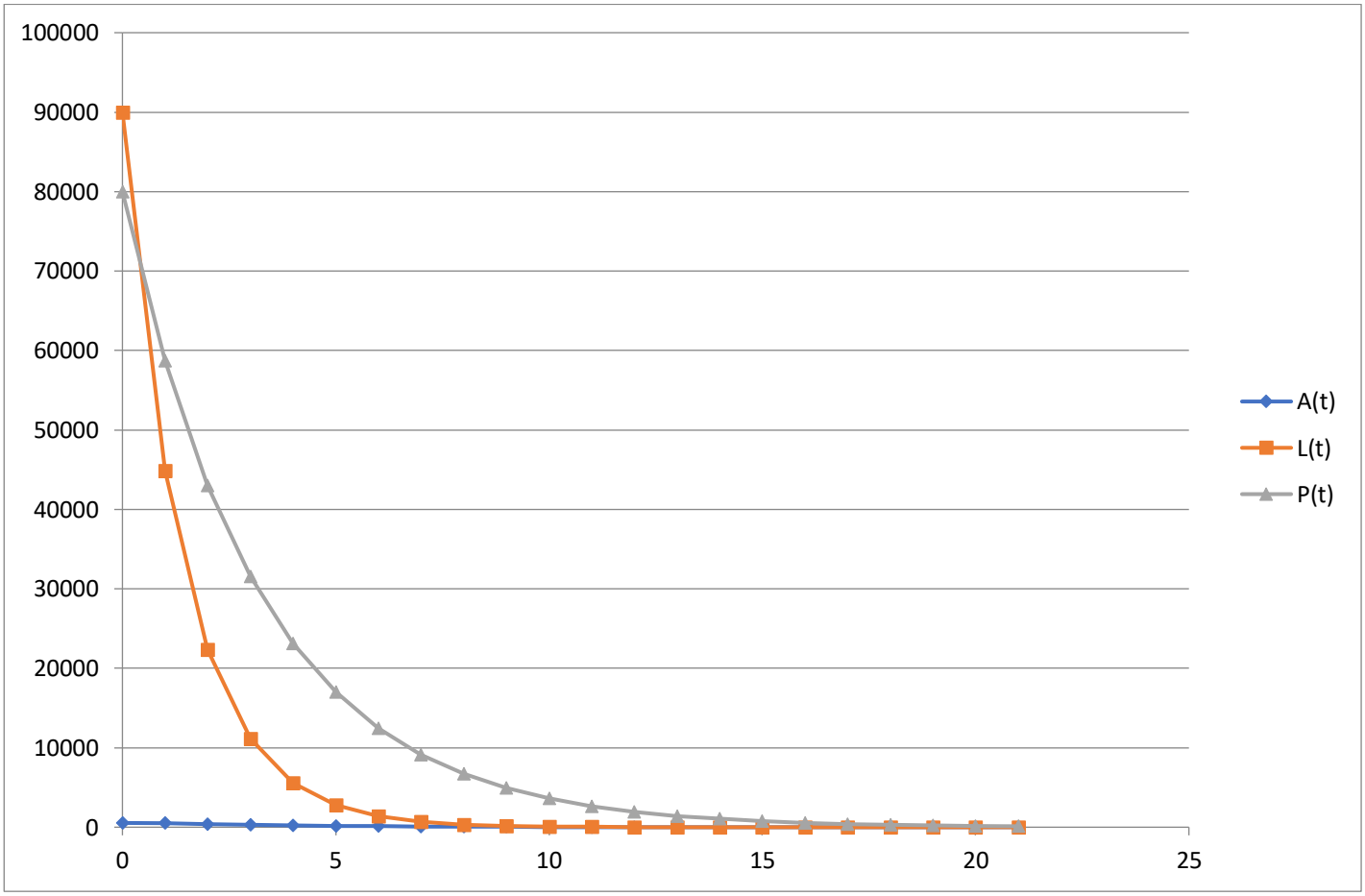


**Figure 4.11:** Number of mosquitoes' larva and adult when two natural predators, copepod and purple martins are introduced respectively ($C_p = 500, P_m = 130,\ T_p = 0,\ \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01,\ b_2 = 0.21,\ \gamma = 5,\ \mu_5 = 0.5,\ \beta_5 = 0.13\ and\ b_4 = 0.5$).

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**Table 4.12**: Function values at $t \in [0,21]$ when two natural predators, copepod and purple martins are introduced to mosquitoes' larva and adult respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 505.0 | 399.9 | 298.1 | 218.4 | 159.4 | 116.3 | 84.9 | 62.1 | 45.4 | 33.3 | 24.4 | 17.8 | 13.1 | … | 1.1 |
| L(t) | 90000 | 44864.9 | 22341.9 | 11117.6 | 5529.3 | 2749.0 | 1366.3 | 679.0 | 337.4 | 167.6 | 83.3 | 41.4 | 20.6 | 10.2 | … | 0.0 |
| P(t) | 80000 | 58678.5 | 43038.9 | 31567.4 | 23153.4 | 16981.9 | 12455.4 | 9135.4 | 6700.4 | 4914.4 | 3604.4 | 2643.7 | 1939.0 | 1422.2 | … | 119.1 |

**Experiment 12:** Comparison of the effect of introducing one, two and three natural predator on adult.

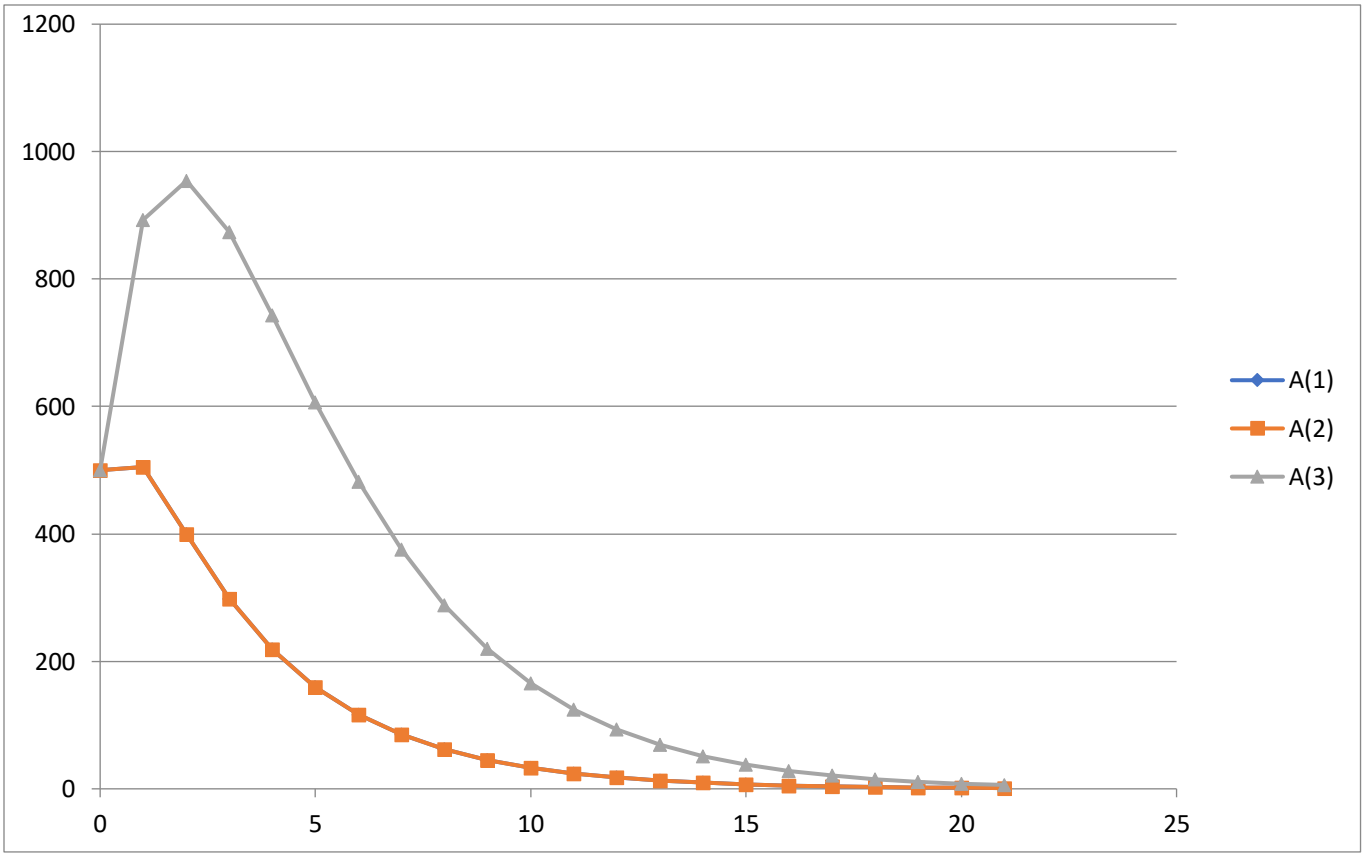


**Figure 4.12:** Number of mosquitoes' adult's when one, two and three natural predator, purple martins are compared respectively ( $P_{1,2\&3} = 130,\ \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02,\ and\ b_3 = 0.9$).

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**Table 4.13**: Function values at $t \in [0,21]$ when adult's in one, two and three natural predators, copepod, tadpole and purple martins are compared respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(1) | 500 | 505.0 | 399.9 | 298.1 | 218.4 | 159.4 | 116.3 | 84.9 | 62.1 | 45.4 | 33.3 | 24.4 | 17.8 | 13.1 | … | 1.1 |
| A(2) | 500 | 505.0 | 399.9 | 298.1 | 218.4 | 159.4 | 116.3 | 84.9 | 62.1 | 45.4 | 33.3 | 24.4 | 17.8 | 13.1 | … | 1.1 |
| A(3) | 500 | 893.2 | 954.3 | 873.8 | 743.6 | 606.5 | 481.5 | 375.2 | 288.5 | 219.7 | 166.0 | 124.7 | 93.2 | 69.4 | … | 6.2 |

**Experiment 13:** Comparison of the effect of introducing two and three natural predator, copepod on larva.

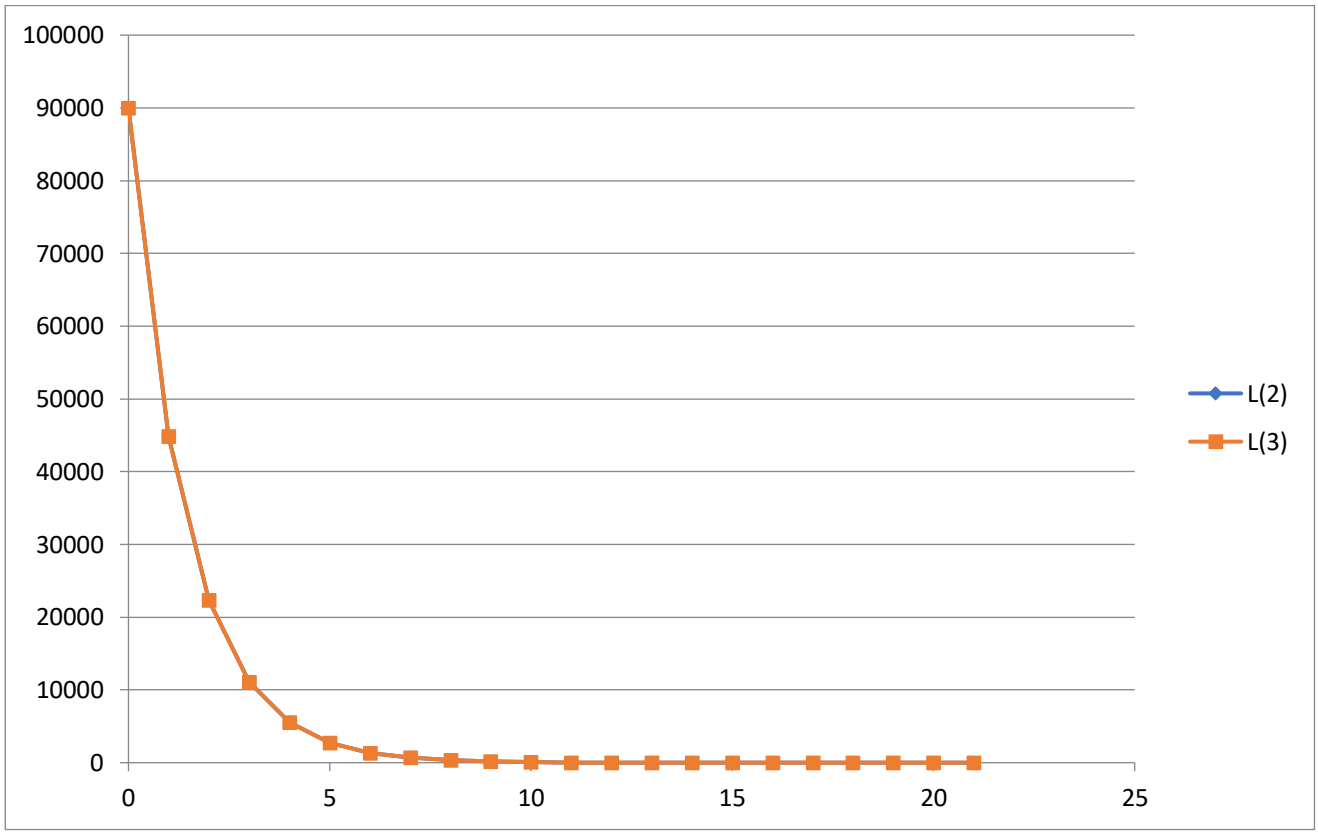


**Figure 4.13:** Number of mosquitoes' larva when, two and three natural predators are compared respectively ( $C_{2\&3} = 500,\ \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02\ and\ b_3 = 0.9$).

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**Table 4.14**: Function values at $t \in [0,21]$ when larva's in two and three natural predators, are compared respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| L(2) | 90000 | 44864.9 | 22341.9 | 11117.6 | 5529.3 | 2749.0 | 1366.3 | 679.0 | 337.4 | 167.6 | 83.3 | 41.4 | 20.6 | 10.2 | … | 0.0 |
| L(3) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | .. | 0.0 |

**Experiment 14**: Effect of introducing low rate of natural predators, copepod on mosquitoes' larva $(C_p = 200)$.

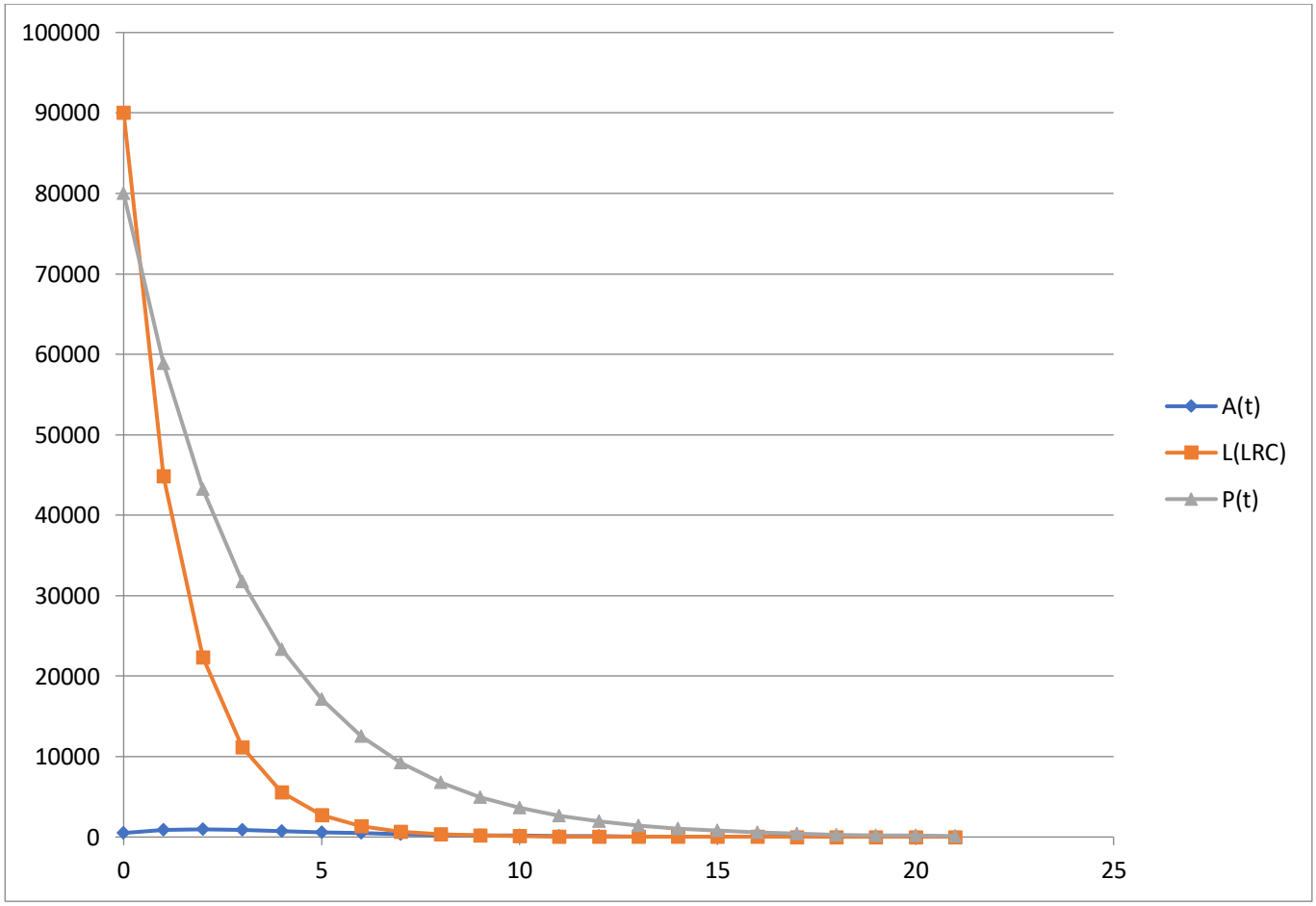


**Figure 4.14:** Number of mosquitoes' larva when low rate of natural predator, copepod was introduced to mosquitoes' larva ($C_p = 200, T_p = 0, P_m = 0, \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01\ and\ \ b_2 = 0.21$).

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**Table 4.15**: Function values at $t \in [0,21]$ when low rate of natural predator, copepod was introduced to mosquitoes' larva.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 893.2 | 954.3 | 873.8 | 743.6 | 606.5 | 481.5 | 375.2 | 288.5 | 219.7 | 166.0 | 124.7 | 93.2 | 69.4 | … | 6.2 |
| L(LRC) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 0.0 |
| P(t) | 80000 | 58896.1 | 43263.4 | 31751.5 | 23294.1 | 17086.9 | 12532.9 | 9192.4 | 6742.2 | 4945.1 | 3627.0 | 2660.2 | 1951.1 | 1431.0 | … | 119.8 |

**Experiment 15**: Effect of introducing high rate of natural predator, copepod on mosquitoes' larva ($C_p = 2000$).

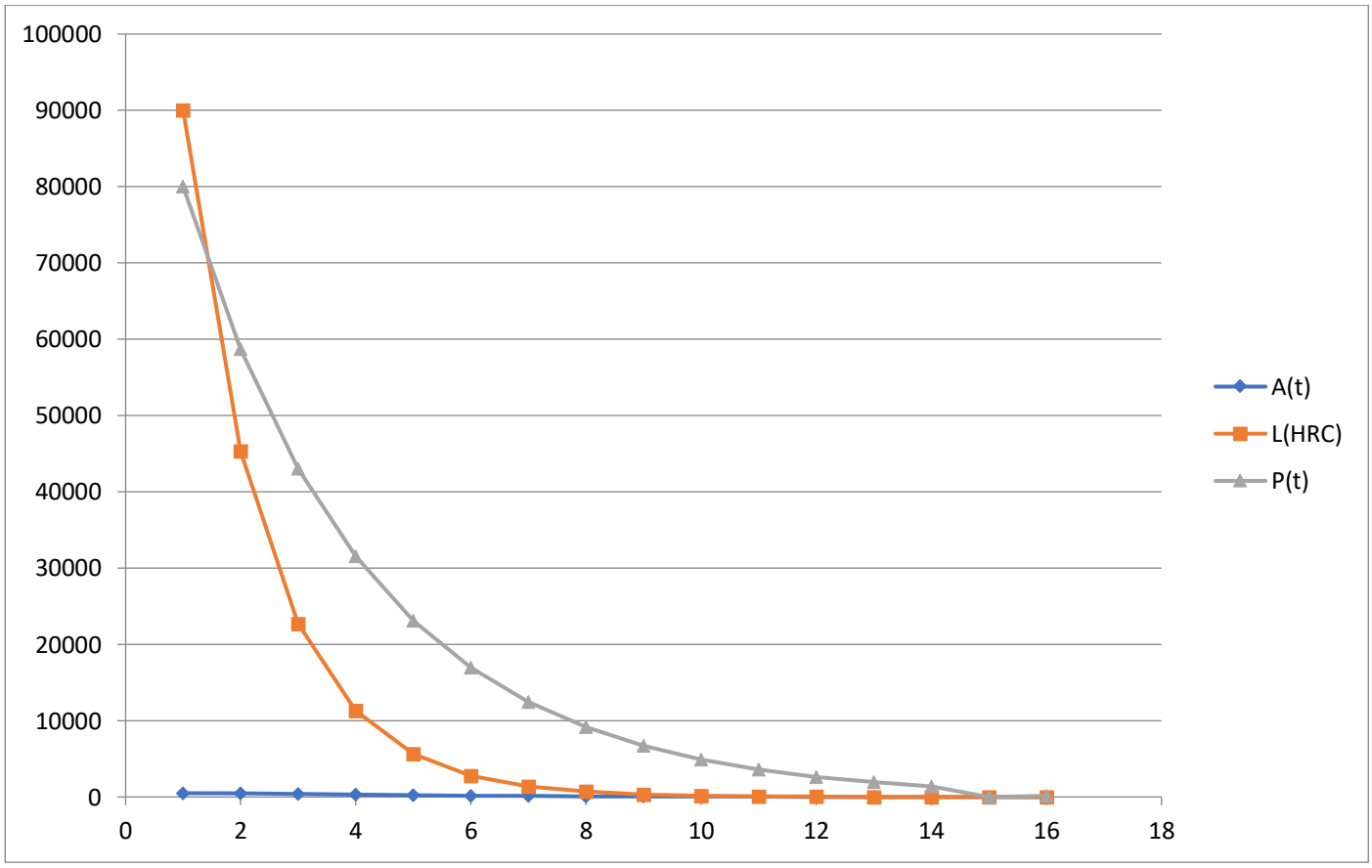


**Figure 4.15:** Number of mosquitoes' larva when high rate of natural predator, copepod was introduced to mosquitoes' larva( $C_p = 2000,\ \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01\ and\ b_2 = 0.21$).

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**Table 4.16**: Function values at $t \in [0,21]$ when high rate of natural predator, copepod was introduced to mosquitoes' larva.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 480.6 | 380.6 | 286.1 | 211.5 | 155.6 | 114.2 | 83.8 | 61.5 | 45.1 | 33.1 | 24.3 | 17.8 | 13.0 | … | 1.1 |
| L(HRC) | 90000 | 45320.1 | 22695.0 | 11327.5 | 5642.7 | 2807.5 | 1395.9 | 693.8 | 344.7 | 171.3 | 85.1 | 42.3 | 21.0 | 10.4 | … | 0.0 |
| P(t) | 80000 | 58678.5 | 43039.0 | 31567.5 | 23153.4 | 16982.0 | 12455.5 | 9135.5 | 6700.4 | 4914.4 | 3604.5 | 2643.7 | 1939.0 | 1422.2 | … | 119.1 |

**Experiment 16**: Effect of introducing low rate of natural predator, tadpole on mosquitoes' pupa ($T_p = 200$).

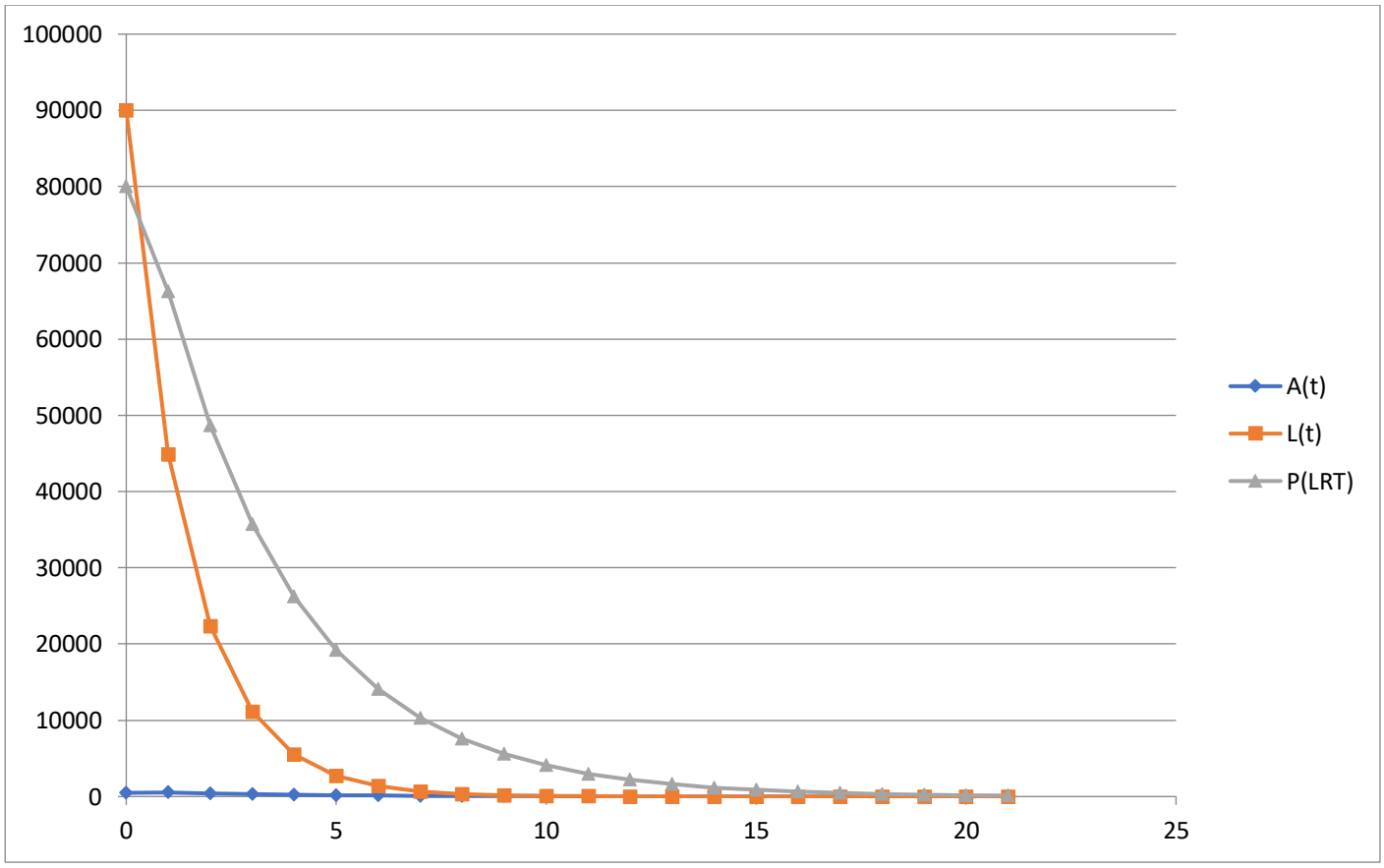


**Figure 4.16:** Number of mosquitoes' pupa when low rate of natural predator, tadpole was introduced ( $T_p = 200$, $\omega = 0.5$, $\mu_7 = 0.01$, $\beta_7 = 0.02$ *and* $b_3 = 0.9$).

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**Table 4.17**: Function values at $t \in [0,21]$ when low rate of natural predator, tadpole was introduced to mosquitoes' pupa.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 526.2 | 425.9 | 322.5 | 239.1 | 176.0 | 129.3 | 94.8 | 69.6 | 51.0 | 37.4 | 27.5 | 20.1 | 14.8 | … | 1.2 |
| L(t) | 90000 | 44864.9 | 22341.9 | 11117.6 | 5529.3 | 2749.0 | 1366.3 | 679.0 | 337.4 | 167.6 | 83.3 | 41.4 | 20.6 | 10.2 | … | 0.0 |
| P(LRT) | 80000 | 66240.2 | 48659.9 | 35714.8 | 26203.4 | 19221.6 | 14099.0 | 10341.2 | 7584.8 | 5563.1 | 4080.3 | 2992.7 | 2195.0 | 1609.9 | … | 134.8 |

**Experiment 17**: Comparison of the effect of introducing low and high rate of natural predator, copepod on mosquitoes' larva.

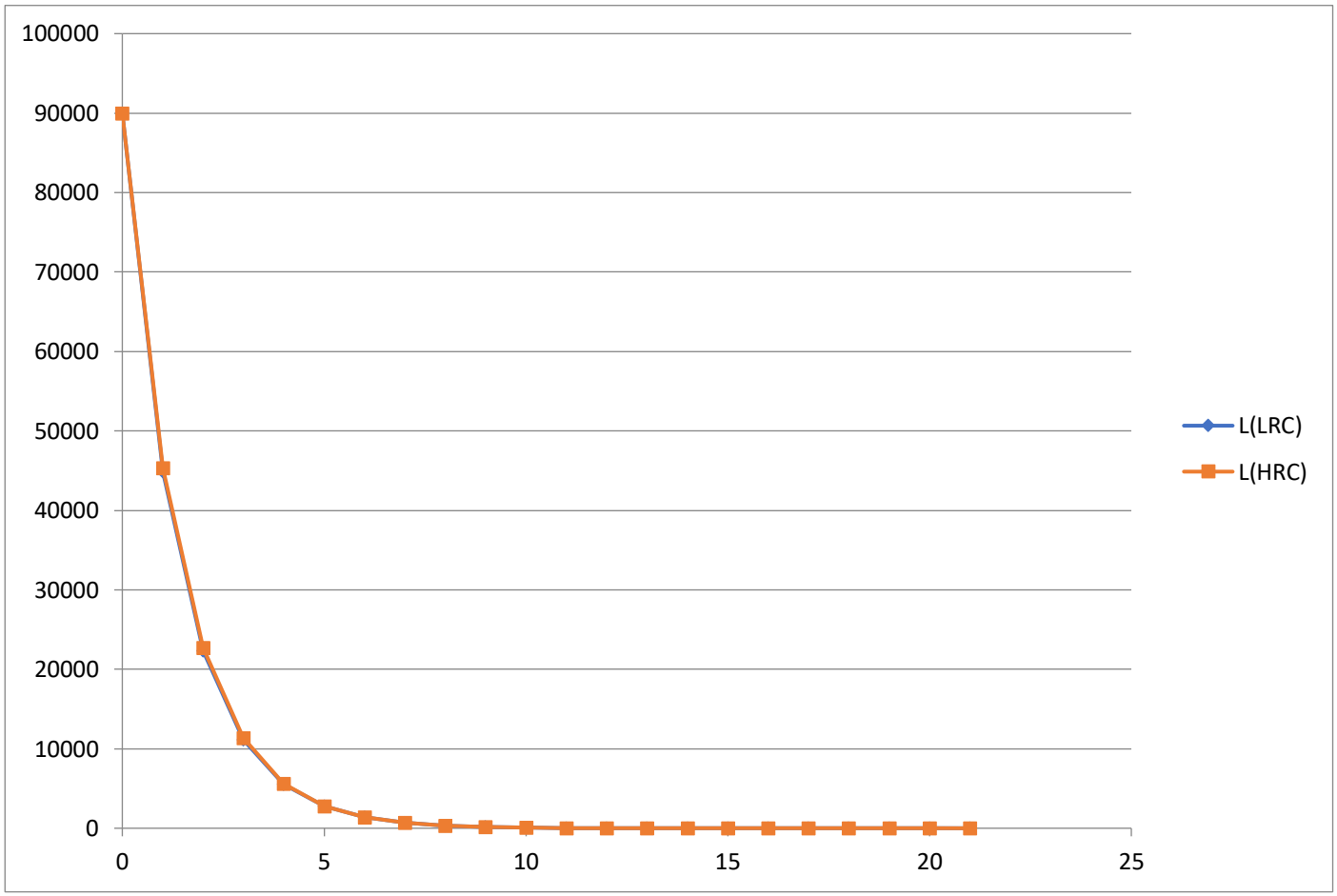


**Figure 4.17:** Number of mosquitoes' larva when low and high rate of natural predator, copepod are compared respectively ( $C_{L\&H} = 200\&2000,\ \alpha = 0.5,\ \mu_6 = 0.02,\ \beta_6 = 0.01\ and\ b_2 = 0.21$

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**Table 4.18**: Function values at $t \in [0,21]$ when low and high rate of natural predator, copepod are compared to mosquitoes' larva respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| L(LRC) | 90000 | 44877.3 | 22346.3 | 11117.1 | 5527.4 | 2747.2 | 1365.1 | 678.2 | 336.9 | 167.4 | 83.1 | 41.3 | 20.5 | 10.2 | … | 0.0 |
| L(HRC) | 90000 | 45320.1 | 22695.0 | 11327.5 | 5642.7 | 2807.5 | 1395.9 | 693.8 | 344.7 | 171.3 | 85.1 | 42.3 | 21.0 | 10.4 | … | 0.0 |

**Experiment 18**: Effect of introducing high rate of natural predator, tadpole on mosquitoes' pupa ($T_p = 2000$).

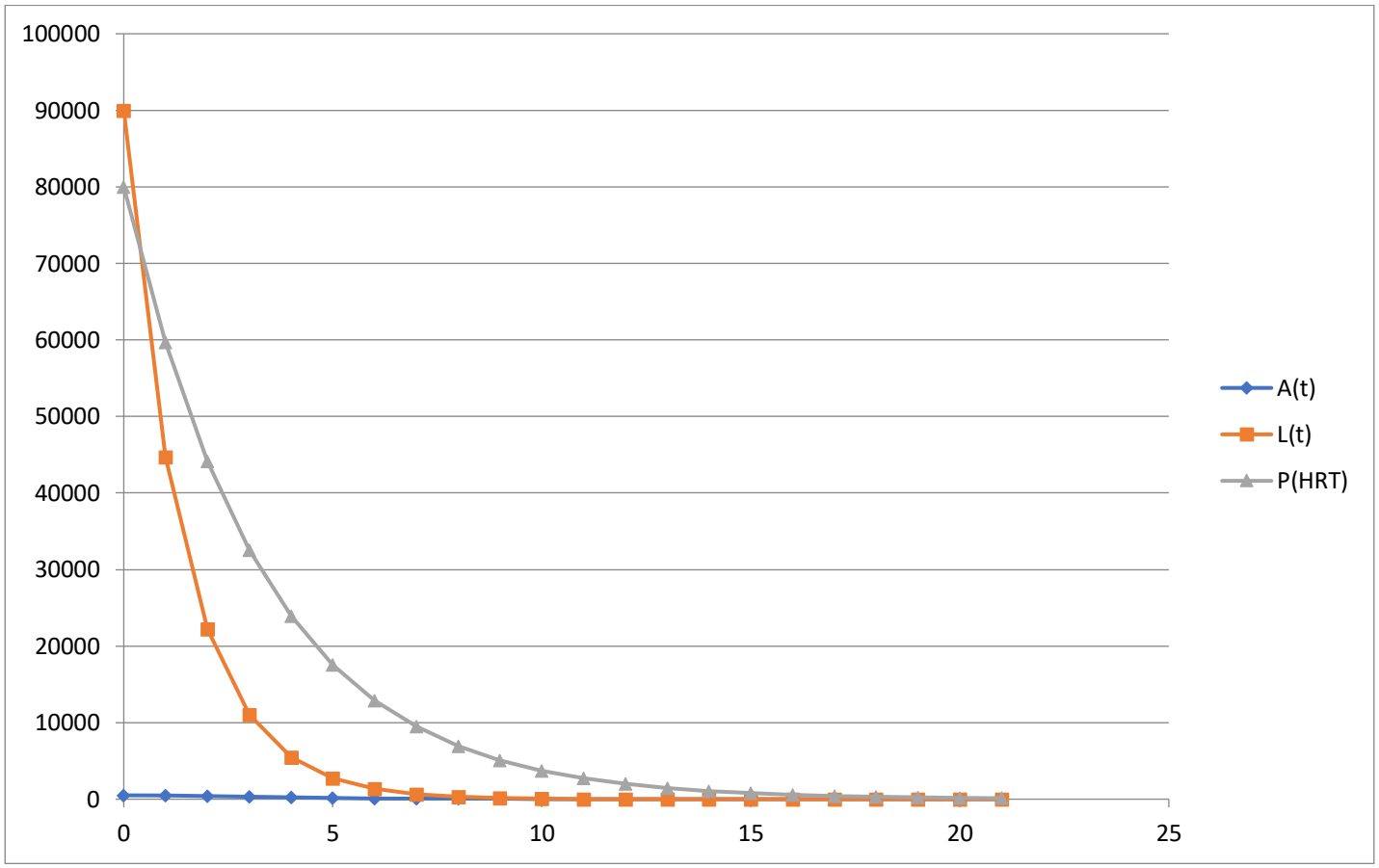


**Figure 4.18:** Number of mosquitoes' pupa when high rate of natural predator, tadpole was introduced to mosquitoes' pupa ( $T_p = 2000,\ \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02\ and\ b_3 = 0.9$).

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**Table 4.19**: Function values at $t \in [0,21]$ when high rate of natural predator, tadpole was introduced to mosquitoes' pupa.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A(t) | 500 | 484.5 | 387.6 | 293.5 | 218.0 | 160.8 | 118.2 | 86.8 | 63.7 | 46.8 | 34.3 | 25.2 | 18.5 | 13.5 | … | 1.1 |
| L(t) | 90000 | 44694.9 | 22196.2 | 11023.1 | 5474.4 | 2718.8 | 1350.3 | 670.7 | 333.1 | 165.5 | 82.2 | 40.8 | 20.3 | 10.1 | … | 0.0 |
| P(HRT) | 80000 | 59671.3 | 44166.7 | 32555.4 | 23942.8 | 17587.0 | 12909.7 | 9472.8 | 6949.5 | 5097.8 | 3739.3 | 2742.7 | 2011.6 | 1475.5 | … | 123.6 |

**Experiment 29**: Comparison of the effect of introducing low and high rate of natural predator, tadpole on mosquitoes' pupa.

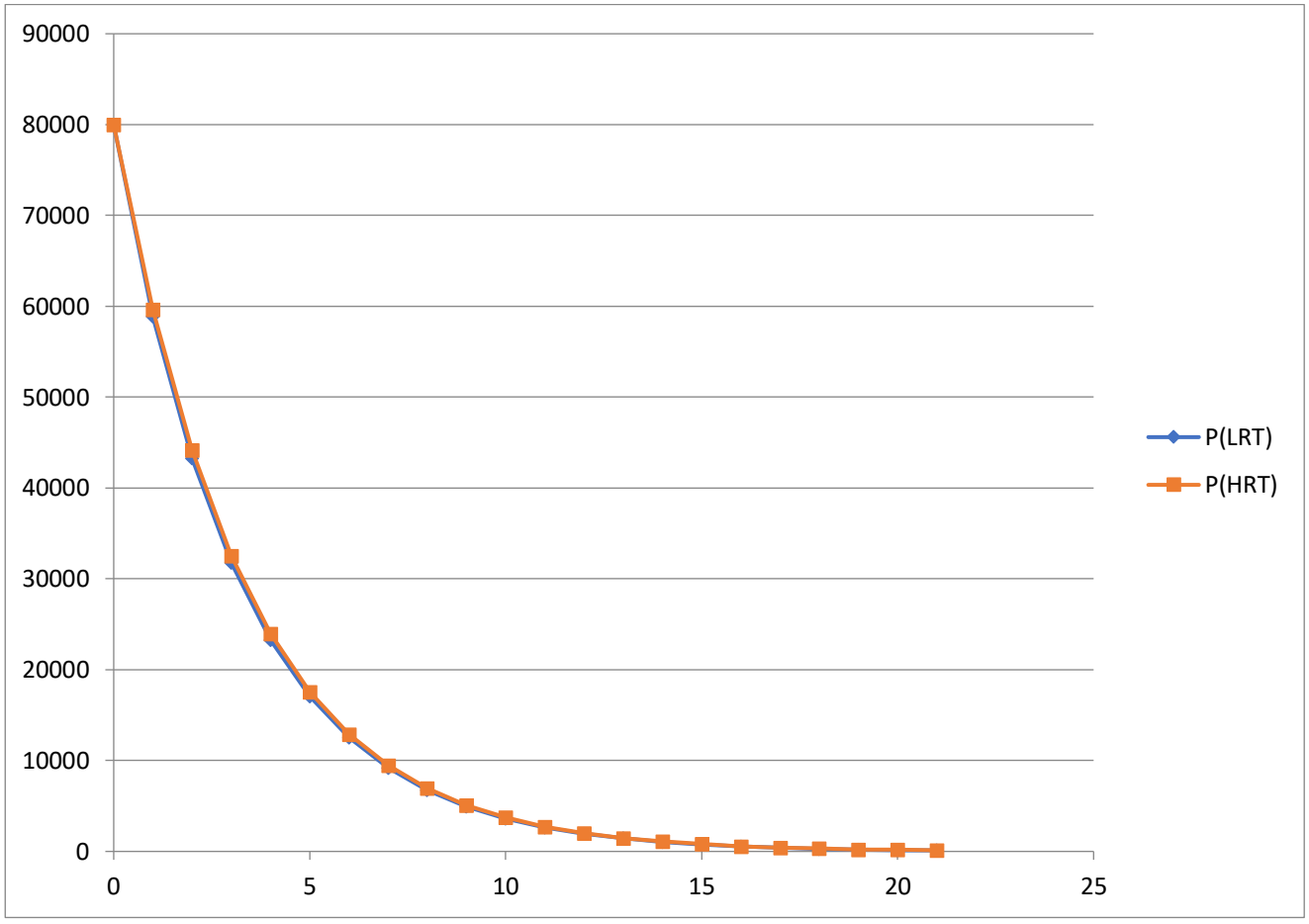


**Figure 4.19:** Number of mosquitoes' pupa when low and high rate of natural predator, tadpole is compared respectively ( $T_{L\&H} = 200\&2000, \omega = 0.5,\ \mu_7 = 0.01,\ \beta_7 = 0.02\ and\ b_3 = 0.9$)

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**Table 4.20**: Function values at $t \in [0,21]$ when low and high rate of natural predators, tadpole are compared to mosquitoes' pupa respectively.

| t | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | … | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P(LRT) | 80000 | 58896.1 | 43263.4 | 31751.5 | 23294.1 | 17086.9 | 12532.9 | 9192.4 | 6742.2 | 4945.1 | 3627.0 | 2660.2 | 1951.1 | 1431.0 | … | 119.8 |
| P(HRT) | 80000 | 59671.3 | 44166.7 | 32555.4 | 23942.8 | 17587.0 | 12909.7 | 9472.8 | 6949.5 | 5097.8 | 3739.3 | 2742.7 | 2011.6 | 1475.5 | … | 123.6 |

**4.2 Discussion of Results**

A prototype of a mathematical model for the control of malaria by interrupting the life cycle of the Anopheles mosquito through the use of biological enemies at the larva, pupa and adult stages is presented. We used elimination and substitution methods to verify the existence and uniqueness of the model equations, we performed the disease free steady state stability analysis of the model using equilibrium point idea, Beltrami conditions, Diekman conditions and also used Maple software for symbolic solution, numerically and plotted the results showing the effects of the introduction of three natural predators (copepods, tadpoles and house swallows) at the larva, pupa and adult stages. From the result, we see that the stability analysis of the free equilibrium state is stable, with the natural implication; there will be no adult female Anopheles mosquito for malaria transmission in our society.

The new model used the parameters shown in Table 3.1. These variables and parameters are chosen with the thresholds obtained in the steady-state disease-free stability analysis of the model. In the analytic output, model analysis showed the existence of a unique disease-free steady state $(E_O)$ that is locally and asymptotically stable when $R_O < 1$. We also identified the basic reproduction number $R_O$ in terms of model parameters. These threshold variables and parameters mentioned in Table 3.1 above should be considered when implementing the above model to provide control measures to reduce the prevalence of malaria parasites in our society and consequently eradicate the disease of mosquito in Nigeria. In the numerical results, numerical experiments performed using the variables and parameter values in Table 4.1 and applying disease-free steady state stability conditions $(E_O)$ yield the following result:

In Experiment 1, the effect of introducing a natural predator, copepod, on mosquito larvae was studied, and the numerical values of variables and parameters were analyzed as shown in Table 4.1 and Table 4.2 solved and numerical simulation with Graphical representation of the result was performed as in figure 4.1. Predator was introduced, this indicates that the number of larvae has decreased significantly and they have pupated.

In experiment 2, the effect of introducing two natural predators, copepods and tadpoles, on mosquito larvae and pupae was studied, and the numerical values of the variables and parameters were those given in the tables 4.1 and 4.4 which have been analyzed, solved and executed numerically with the result plotted as in Figure 4.2 When two natural predators were introduced, the number of pupa to larva was greatly reduced and the transformation of pupae to adults was minimal.

In Experiment 3, the effect of introducing three natural predators (copepods, tadpoles and purple swallows) on mosquito larvae, pupae and adults was studied, and the numerical values of variables and parameters were analyzed in Table 4.1 and Table 4.6 with the solution shown in Figure 3.3 and run a numerical simulation, plotting the result as shown in Figure 3.3 when three natural enemies are introduced simultaneously. Infection in the adult Anopheles mosquito population is significantly slowed down and thus eradicated, and the probability of transmission from the adult Anopheles mosquito to the human population is very low.

In Experiment 4, in which the effects of the introduction of one, two and three natural predators on the larvae were compared, the numerical values of the variables and parameters shown in Table 4.1 were analyzed and resolved, and a numerical simulation was performed with graphical representation of the results, shown in Figure 4.4 when one, two and three predators were examined. This result shows that the infection rate in Figure 4.4 decreases significantly to avoid reinjection of malaria, which is the prevention strategy in the fight against malaria.

In Experiment 5, the comparison of the effect of the introduction of two and three natural predators, swallows, on adult mosquitoes and the numerical values of the variables and parameters in Table 4.1 were studied, analyzed, solved and carried out a numerical simulation with the graph result, as shown in Figure 4.5 when two and three natural predators were introduced respectively. The

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result shows that the infection rate in Figure 4.5 decreases significantly. To prevent reinjection with malaria, the transmission rate must be close to zero.

In Experiment 6, the effect of introducing a natural predator, tadpoles, on mosquito pupae was studied, and the numerical values of variables and parameters were examined as shown in Table 4.1 and Figure 4.10, and a numerical simulation with graph has been analyzed, solved and run. The presentation of the result and the graphical result in Figure 3.6 shows that when a natural predator, the tadpole, has been introduced, the infection in the Anopheles mosquito adult population has slowed and the probability of transmission from nymph to adult Anopheles mosquito population is very low.

In Experiment 7, the effect of the introduction of two natural predators, tadpoles and swallows, on mosquito pupae and adults was studied, and the numerical values of the variables and parameters were those given in Table 4.1 and Table 4.12, they have been analyzed and resolved numerically using a graphical simulation. The result is shown in Figure 4.7, which confirms that the infection in the adult Anopheles mosquito population slows down significantly and the probability of becoming an adult Anopheles mosquito is very small.

In Experiment 8, comparing the effect of introducing one, two and three natural enemies into mosquito pupae, and the numerical values shown in Table 4.1 and the graphical result shown in Figure 4.8 when one, two and three natural enemies are present respectively. The result shows that the infection rate in Figure 4.8 decreases significantly. To avoid reinjection of malaria, the transmission rate must be close to zero.

In experiment 9, a numerical simulation was analyzed, solved and carried out with a graphical representation of the result in the comparison of the effect of introducing two and three natural enemies on adult mosquitoes and the numerical values of the variables and parameters shown in Table 4.1. Figure 4.9 show that the infection rate drops enough to prevent malaria infection.

In Experiment 10, the effect of introducing a natural predator, purple swallow, on adult mosquitoes was studied, and the numerical values of variables and parameters were analyzed as shown in Table 4.1 and Table 4.16, resolved and numerical simulation with plot. The result shown in Figure 4.10 after the introduction of a natural predator, the purple swallow, is quite stagnant in adult Anopheles mosquitoes and the transmission rate is very weak.

In Experiment 11, the effect of the introduction of two natural predators, copepods and purple swallows, on mosquito larvae and adults respectively, was examined, and the numerical values of variables and parameters were examined, as shown in Tables 4.1 and 4.18. Solve and run a numerical simulation with a graphical representation of the result shown in Figure 4.11 when two natural predators, copepods and purple swallows, are introduced. Infection in the adult Anopheles mosquito population is significantly slowed down and thus eradicated, and the probability of transmission from the pupa to the adult Anopheles mosquito population is very poor.

In Experiment 12, the comparison of the effect of introducing one, two and three natural predators on adult mosquitoes and the numerical values in Table 4.1 were analyzed and solved, and a numerical simulation was performed with a graphical representation of the result as indicated in Figure 4.12 at the introduction of one, two and three natural predators. The result shows that the infection rate decreases significantly to prevent new malaria disorder.

In Experiment 13, the effect of the introduction of two and three natural predators on mosquito larvae was compared with the numerical values of the variables and parameters presented in Table 4.1, and a numerical simulation with graphical representation was analyzed, resolved and carried out and results run, shown in Figure 4.13, when two and three natural predators were introduced, respectively. The result shows that the infection rate decreases to prevent malaria disease.

In Experiment 14, the effect of introducing a low rate of natural predators, copepods, on mosquito larvae was investigated, and the numerical values of variables and parameters were presented in Tables 4.1 and 4.22, analyzed, solved and a numerical simulation was played with a graphical representation of the result as shown in Figure 4.14. Low rate of natural predators, copepods have been introduced. Infection in adult Anopheles mosquitoes has decreased and the percentage of transmission is deep.

In Experiment 15, the effect of introducing a high level of natural predators, copepods, on mosquito larvae was studied, and the numerical values of variables and parameters were analyzed, solved and executed. as shown in Table 4.1 and Table 4.24, and a numerical simulation with a graphical representation of the result, as shown in Figure 4.15. High levels of natural predators, copepods, have been introduced. Infection in adult mosquitoes of the Anopheles family is fairly stagnant and is therefore well on the way to eliminating malaria illness.

In Experiment 16, the effect of introducing a low number of natural predators, tadpoles, into the mosquito pupa was investigated, and the numerical values of variables and parameters are shown in Tables 4.1 and 4.26, and the graphical result is shown in Figure 4.16. Low rate of natural predators, introduction of copepods decreased infection in adult Anopheles mosquitoes.

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In experiment 17, the effects of the introduction of low and high rate of natural predators, copepods, on mosquito larvae were studied; analyzed, solved and numerical simulations were carried out with numerical values of variables and parameters, as shown in Table 4.1. The representation of the result in Figure 4.18 has shown when low and high levels of natural predators, copepods, were introduced. The infection in adult mosquitoes of the Anopheles family is quite stagnant due to the low and high rate of natural predators introduced at the same time, therefore in the process of elimination, and the percentage of transmission is almost nil.

In Experiment 18, the effect of introducing a high rate of natural predators, tadpoles, into the mosquito pupa and the numerical values of the variables and parameters presented in Table 4.1 and Table 4.29 were analyzed and resolved, and performed a numerical simulation in the graphical representation of Figure 4.18 when a high level of the natural predator, the tadpole, was introduced. Infection in adult mosquitoes of the Anopheles family was almost zero.

In Experiment 19, the comparison of the effects of introducing low and high rate of natural predators, tadpoles, into the mosquito pupa and the numerical values of the variables and parameters as shown in Table 4.1 and the graphical result in Figure 4.19 shows that low and high rates of natural predators, copepods, were introduced. Infection in adult mosquitoes of the Anopheles family is fairly stagnant, and therefore on the way to elimination, and the percentage of transmission is downward.

Considering the total population, the effect of the introducing three natural predators, one, two and three, on the larva, larva and pupa and larva, pupa and adult (copepods, tadpoles and martens) respectively, (compare Figure 4.1, 4.2 and 4.3 with Figure 4.4 and 4.5). The infectious agent content is greatly reduced and the infection of the egg, larva and pupa is eradicated, but persists at a low level in the adult Anopheles mosquito.

When assessing the total population, the effect of introducing two natural predators one and two on pupae, pupae and adults (tadpoles and house swallows) was examined (compare Figure 4.6 and 4.7 with Figure 4.8 and 4.9). The infectious agent content is greatly reduced and the infection of the egg, larva and pupa is eradicated, but persists at a low level in the adult Anopheles mosquito.

When assessing the total population, the effect of introducing two natural predators, one and two, on the adult, larva and adult (swallow and copepod) respectively (compare Figure 4.10 and 4.11 with Figure 4.12 and 4.13). The infectious agent content is greatly reduced and the infection of the egg, larva and pupa is eradicated, but persists at a low level in the adult Anopheles mosquito.

When examining the total population, the effect of introducing low and high rate natural predators (copepods) on the larvae was introduced and studied (compare Figure 4.14 and 4.15 with Figure 4.17). The infectious agent content is greatly reduced and the infection of the egg, larva and pupa is eradicated, but persists at a low level in the adult Anopheles mosquito.

When analyzing the total population, the impact on the introduction of pupae of a high and low rate of natural predators, one (tadpole) was introduced and examined (compare Figure 4.16 and 4.18 with Figure 4.19). The infectivity of the adult Anopheles mosquito remains at a low level.

Finally, to understand the effects of introducing three natural enemies (copepods, tadpoles and house swallows) on larvae, pupae and adults when three natural enemies are introduced each, Figures 4.1, 4.2, 4.3, 4.4, 4.5…4.19 specify the representations to deliver. It could be clearly observed that the transmission speed was reduced to the indispensable minimum. This could be achieved since research should focus on formulating models that capture preventive strategies based on stability analysis to prevent the onset of the disease and thus eradicate it.

## V. Summary, Conclusion and Recommendation

### 5.1 Summary

Modelling the effects of three natural predators on the aquatic and adult stages of anopheles mosquitoes in the control of malaria transmission is presented. In chapter one, we discuss the prevalence of mosquitoes in our society, where two million deaths are due to malaria parasites in sub-Saharan Africa in general and Nigeria in particular, one third of which are children. We have discussed the developmental stages of Anopheles mosquito which are egg, larva, pupa and adult. We have mentioned the conditions in which mosquitoes breed, mosquitoes breed faster in areas with high humidity. In chapter two, we review the associated models, the conceptual framework, the empirical literature review and the theoretical framework of this research work. In chapter three, we discuss materials and methods used, sampling methods for larvae collection, model formulation and description, define model variables and parameters, make assumptions and represent the model showing the flow control diagram of the prey-predator interaction in the life of the mosquito. Three natural predators were introduced (copepods, tadpoles and purple martins) at larva, pupa and adult stages into the model and derived model equations for mosquito life cycle, predators and the total population.

In chapter four, we used elimination and substitution methods to verify the existence and uniqueness of the model equations, we performed the disease free steady state stability analysis of the model using equilibrium point idea, Beltrami conditions, Diekman conditions and also used Maple software for symbolic solution, numerically and plotted the results showing the effects of the

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introduction of three natural predators (copepods, tadpoles and house swallows) at the larva, pupa and adult stages. From the result, we see that the stability analysis of the free equilibrium state is stable, with the natural implication; there will be no adult female Anopheles mosquito for malaria transmission in our society.

### 5.2 Conclusion

We find out that based on the conditions of the Beltrami condition, when the determinants of the Jacobian matrix are greater than zero and the trace is less than zero, the disease-free steady-state stability analysis is stable and Diekmann's conditions which indicate when $R_0 < 1$, the steady state stability analysis without disease is stable. We conclude that if the natural predators introduced are large, the number of larvae leading to pupae will be zero and the number of pupae developing into adults will be zero, which will short the life cycle of the interrupted Anopheles mosquito. Therefore, in our community, there will be no adult Anopheles mosquitoes for the transmission of malaria parasites.

### 5.3 Recommendations

Recommendations for the results of this work are given below.

1) Emphasis should be placed on prevention models and strategies that capture the impact of adopting three natural predators (copepods, tadpoles and purple swallows) in the larva, pupa and adult stages of the adult female Anopheles mosquito.
2) Effective treatment strategies should focus on the larva, pupa and adult stages of anopheles adult mosquito.

### 5.4 Contributions to Knowledge

Here are the knowledge articles:

1. Modeling the effects of three natural predators on the aquatic and adult stages of anopheles mosquitoes in the control of malaria transmission.
2. Construction of the control flow diagram of prey-predator interaction.
3. Formulation of the mathematical formula.
4. Identify the ability to control and eradicate malaria through stability analysis.
5. Numerical experiments showing the effect of the introduction of three natural predators (copepods, tadpoles and purple swallow) on the larva, pupa and adult stages of the adult Anopheles mosquito.

### 5.5 Recommendation for Future Research

We observed that λ is inversely proportional to α and this just means that if we increase the value of α and ω it would decrease the value of λ thus truncating the value of the larva and pupa leading to the next stage. From the disease-free steady-state stability analysis of the governing equations (3.13.1) - (3.13.8) and based on the assumptions of the model (3.10), we saw that the Steady-state disease-free stability analysis of the disease is stable when Det(J) > 0, Tr(J) < 0, and $R_O < 1$.

The recommendation for future research is based on fuzzy structures of dynamic nature when$R_O \geq 1$. The basic reproduction number $R_O$ can describe the dynamics of malaria transmission of the disease, with an overall stable disease-free state when $R_O < 1$. Whereas for $R_O > 1$ the endemic equilibrium state becomes globally stable.

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