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An Age Structured Malaria Transmission Dynamics Model Incorporating Severe and Uncomplicated Infections Compartments

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ABSTRACT

In this study, a mathematical model of malaria was formulated. System of ordinary differential equations were used to described the transmission dynamics of the malaria disease in an age-structured population. The model is proved to be mathematically well-posed and epidemiologically feasible. The Malaria free equilibrium was obtained, and the method of next generation matrix approach were employed to determine the basic reproduction number R_0. The analysis of the malaria-free equilibrium shows that; the system is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1.$ The approach by Castillo-Chavez was employed to determine the global stability analysis of the Malaria-free equilibrium. The result of the global stability analysis show that, the system is locally asymptotically stable when $R_0 < 1$ and unstable if $R_0 > 1.$ This indicates that, the Malaria transmission elimination is only possible if the threshold parameter R_0 is kept below unity. Therefore, all efforts must be geared *towards* ensuring that the minimum number of secondary infections is less than one in both children and adult populations respectively.

1. Introduction

Malaria is an infectious disease caused by the Plasmodium parasite, which is carried by the female Anopheles mosquito (Perko, Kebede & Mousa, 2022). There are about 400 different Plasmodium species, of these, four are the major cause of malaria in humans. These four species are *P. falciparum* and *P. vivax, P. ovale, and P. Malarie* which combined together contributed to about 95% of malaria infection (Dalu, Dennis, Chuwkuemeka, Chifurumnanya & Ugochi, 2022). As the main cause of malaria in Africa, *P. falciparum* has caused millions of deaths, accounting for more than 90% of the world's malaria mortality. The main symptoms of malaria are fever, fatigue, vomiting, and headaches (Li, Docile, Fisher, Pronyuk & Zhao, 2024). World Health Organization (WHO), estimates that the severe form of malaria has led to 194 000 deaths in 2021 alone in Nigeria, about 80% of these in children under the age of 5 years. This accounts for 31% of all malaria cases globally and 40% in the WHO African region. Nigeria has the highest burden of malaria globally, accounting for nearly 27% (68 million cases) of the global malaria burden and risk of transmission exists throughout the country, all year round (WHO, 2023). Preventive efforts such as vector control, insecticide-treated mosquito nets, seasonal malaria chemoprevention, and intermittent preventive treatment for infants

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and pregnant women are an important place to start reducing malaria transmission (Perko, Kebede & Mousa, 2022). Through the World Health Organization and many researchers' efforts, vaccines such as RTS, S/AS01 and PfSPZ exist among many others currently in clinical trials (Perko, Kebede & Mousa, 2022). Also, treatment guidelines have not changed for a long time, and many of the anti-malarial drugs currently have had cases of resistance (Perko, Kebede & Mousa, 2022). To achieve malaria elimination in Nigeria, there is a need to extend surveillance strategies to other secondary malaria vectors that have not received significant attention (Adeogun *et al.,* 2025). World Health Organization (2024) emphasized in its latest report that equitable access to preventive tools such as insecticide-treated nets, indoor residual spraying, and malaria vaccines are crucial to reducing transmission, especially among vulnerable populations.

1.1 Mathematical models of malaria

A new non-autonomous model incorporating diurnal temperature fluctuation is designed by Nwankwo and Okuonghae (2019). The model assessed the impact of different microclimate conditions on malaria transmission dynamics. A temperature-dependent mathematical model of malaria transmission with stage-structured mosquito population dynamics was developed by Traoré, Barro, Sangaré, and Traoré (2021). A mathematical model for the dynamics and control of malaria in Nigeria is studied by Collins and Duffy (2022). Their model incorporates drug resistance, treatment, and the use of mosquito nets as preventive strategies. Application of mathematical modelling to inform national malaria intervention planning in Nigeria was presented by Ozodiegwu, Ambrose, Galatas, B, Runge, and Nandi,. (2023). Comparison between different approaches of modelling the effects of temperature and rainfall on malaria transmission in high and low transmission settings was studied by Madito and Silal (2024). Effect of Environmental Immunity on Mathematical Modeling of Malaria Transmission between Vector and Host Population was presented by Olutimo, Mbah, Abass, and Adeyanju (2024). Adeogun, Babalola, Oyale, Oyeniyi, Omotayo and Izekor (2025) investigated the spatial distribution and geospatial modeling of potential spread of secondary malaria vectors species in Nigeria using recently collected empirical data. Malaria, climate variability, and interventions: modelling transmission dynamics was studied by Beloconi, Nyawanda, Bigogo, Khagayi, Obor and Danquah (2023). Grosso, Hens and Abrams (2025) presented an integrative review of the combined use of mathematical and statistical models for estimating malaria transmission parameters.

However, this research is an improvement on the work of Olutimo, Mbah, Abass and Adeyanju (2024). An age-structured mathematical model of malaria is developed. The uncomplicated, severe and treatment classes were incorporated into the model with the view to investigate its transmission dynamics, which was not presented in the reviewed literature presented in this study.

The paper is categorized into different sections as follows: Introduction is presented in section one, model description, equations, variables and parameter description and the schematic diagram were presented in section two. The basic properties and model analysis were presented in section three. Discussion of Result is presented in section four while conclusion and references were presented in section five respectively.

2. Model diagram of Malaria

The schematic diagram of the Malaria is presented in Figure 1.

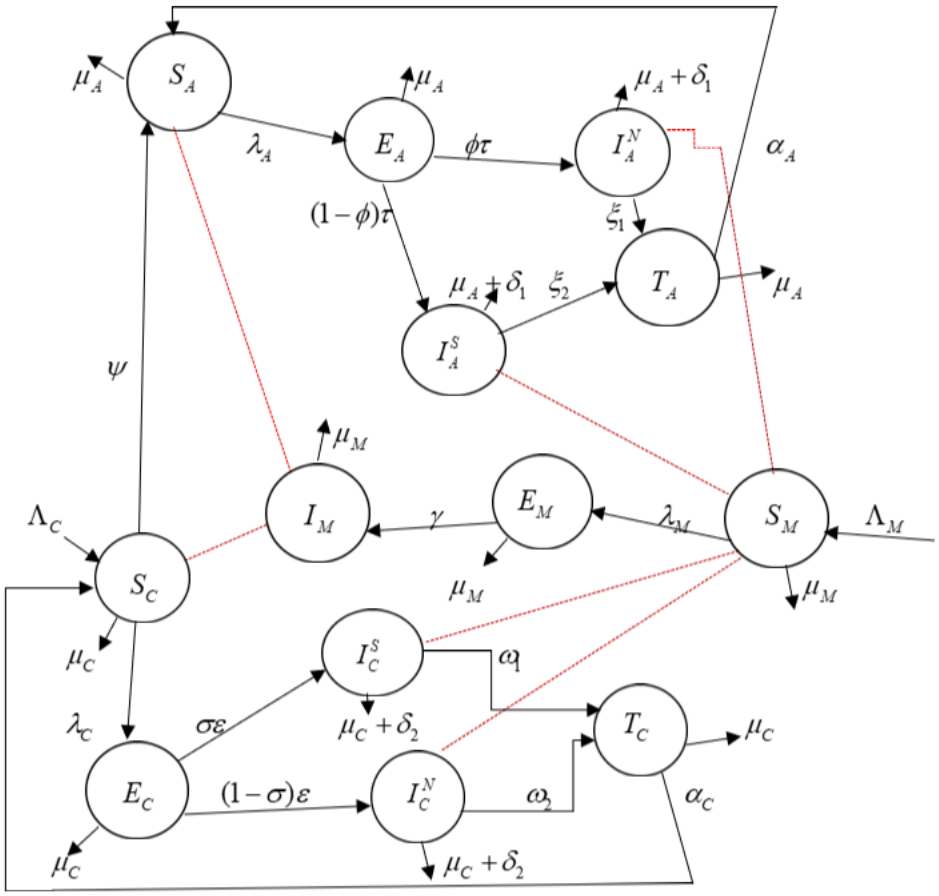

Figure 1: The schematic diagram of the malaria model

2.1 The Model Equations

$$\frac{dS_A}{dt} = \psi S_C + \alpha_A T_A - \lambda_A S_A - \mu_A S_A$$

$$\frac{dE_A}{dt} = \lambda_A S_A - (\phi\tau + \mu_A) E_A$$

$$\frac{dI_A^N}{dt} = \phi\tau E_A - (\xi_1 + \mu_A) I_A^N$$

$$\frac{dI_A^S}{dt} = (1-\phi)\tau E_A - (\xi_2 + \mu_A) I_A^S$$

$$\frac{dT_A}{dt} = \xi_1 I_A^N + \xi_2 I_A^S - (\mu_A + \alpha_A) T_A$$

$$\frac{dS_C}{dt} = \Lambda_C + \alpha_C T_C - \psi S_C - \lambda_C S_C - \mu_C S_C \quad (1)$$

$$\frac{dE_C}{dt} = \lambda_C S_C - (\sigma\varepsilon + \mu_C) E_C$$

$$\frac{dI_C^N}{dt} = \sigma\varepsilon E_C - (\omega_1 + \mu_C) I_C^N$$

$$\frac{dI_C^S}{dt} = (1-\sigma)\varepsilon E_C - (\omega_2 + \mu_C) I_C^S$$

$$\frac{dT_C}{dt} = \omega_1 I_C^N + \omega_2 I_C^S - (\mu_C + \alpha_C) T_C$$

$$\frac{dS_M}{dt} = \Lambda_M - \lambda_M S_M - \mu_M S_M$$

$$\frac{dE_M}{dt} = \lambda_M S_M - (\gamma + \mu_M) E_M$$

$$\frac{dI_M}{dt} = \gamma E_M - \mu_M I_M$$

with the initial conditions:

$$S_A > 0, E_A \geq 0, I_A^N \geq 0, I_A^S \geq 0, T_A^N \geq 0, T_A^S \geq 0, S_C > 0, E_C \geq 0, I_C^N \geq 0, I_C^S \geq 0, T_C^N \geq 0, T_C^S \geq 0,$$
$$S_M > 0, E_M \geq 0, I_M \geq 0.$$

where

$$\lambda_A = \frac{\vartheta_M \beta_A I_M}{N_H} \quad \lambda_C = \frac{\vartheta_M \beta_C I_M}{N_H} \text{ and } \quad \lambda_M = \frac{\vartheta\beta_M (I_A^N + I_A^S + I_C^N + I_C^S)}{N_H}$$

2.2 Variable/Parameter Description

The description of variables/parameters are presented in Table 1.

Variables/Parameters	Description
S_A	Susceptible adult population
S_C	Susceptible children below the age of puberty
E_A	Pre-infectious adult population

E_C	Pre-infectious children population
I_A^N	Infected non-severe adult population
I_C^N	Infected non-severe children population
I_A^S	Severely-infected adult population
I_C^S	Severely-infected children population
T_A	Treated adult population
T_C	Treated children population
S_M	Susceptible Mosquito population
E_M	Pre-infectious mosquito population
I_M	Infected mosquito population
ψ	Maturity rate from children to adult population
α_A	Rate at which treated adult population loss their immunity
β_A	Transmission probability of the susceptible adult population
ϑ_M	Per capita biting rate of the infected mosquito into the susceptible adult population
μ_A	Natural death rate in adult human population
μ_C	Natural death rate in children
μ_M	Natural death rate in mosquito population
τ	Progression rate into non-severe infected adult population
ϕ	Progression rate into severely-infected adult population
$\xi_i (i=1,2)$	Treatment rate of adult population
$\delta_i (i=1,2)$	Disease induced death rate
Λ_C	Recruitment rate of susceptible children population
α_C	Rate at which treated children loss immunity
ϑ	Per capita biting rate of the susceptible mosquito into the infected adult and children population
β_C	Transmission probability of the susceptible children population
ε	Progression rate into non-severe infected children population
σ	A proportion of individuals leaving the pre-infectious children population into the non-severe infected class
ω_1	Treatment rate in non-severe infected children population
ω_2	Treatment rate in severely infected children population

γ	Progression rate from pre-infectious into infectious mosquito population
Λ_M	Recruitment rate into the susceptible mosquito population

2.3 The Model Description

The total human population is sub-divided into the pool of susceptible adult population S_A, the susceptible children below the age of puberty S_C, the pre-infectious adult population E_A, the pre-infectious children population E_C, the infected non-severe adult I_A^N, the infectious non-severe children I_C^N, the severely-infected adult population I_A^S, the severely-infected children I_C^S, the non-severe and severely treated adult populations T_A, the non-severe and severely treated children populations T_C. Thus, the human population is given by

$$N_H = S_A + S_C + E_A + E_C + I_A^N + I_C^N + I_A^S + I_C^S + T_A + T_C \qquad (2)$$

The total mosquito population is sub-divided into Susceptible mosquito population S_M, pre-infectious mosquito population E_M, and the infected and infectious mosquito population I_M. Thus, the mosquito population is given by

$$N_M = S_M + E_M + I_M \qquad (3)$$

The susceptible adult population is increased through attaining the age of maturity from the population of susceptible children S_C, at the rate ψ and through treatment of infected and infectious populations at the rate α_A since treatment does not confer permanent immunity. This population is decreased by coming into contact with infected and infectious mosquito I_M, and transfer of the newly infected persons into the pre-infectious population E_A, by the force of infection λ_A, where,

$$\lambda_A = \frac{\vartheta_M \beta_A S_A I_M}{N_H} \qquad (4)$$

where ϑ_M is the per capita mosquito biting rate on susceptible adult population, β_A is the transmission probability of malaria into the susceptible adult population.

This population is further reduced by natural death at a rate μ_A. The equation becomes,

$$\frac{dS_A}{dt} = \psi S_C + \alpha_A T_A - \lambda_A S_A - \mu_A S_A \qquad (5)$$

The population of the pre-infectious adult population E_A, is increased by the transfer of the newly infected persons λ_A. The population is decreased by the migration of persons in this class into the non-severe infected and infectious adult population I_A^N, at the rate τ. A fraction of this population moves to the severely infected and infectious adult population at a proportion ϕ where $(0 \le \phi \le 1)$. The population is further decreased by natural death at the rate μ_A. So that, the equation becomes,

$$\frac{dE_A}{dt} = \lambda_A S_A - (\phi\tau + \mu_A)E_A \tag{6}$$

The population of non-severe infected and infectious adult I_A^N, is increased by a fraction of individuals from pre-infectious adult population E_A at a proportion $\phi\tau$. The same population is decreased by the treatment of individuals in this class and by their migration into the treatment populations at a rate ξ_1 and is further decreased by the natural death at a rate μ_A and due to malaria induced death at a rate δ_1. Thus,

$$\frac{dI_A^N}{dt} = \phi\tau E_A - (\xi_1 + \mu_A + \delta_1)I_A^N \tag{7}$$

The population of severely-infected and infectious adult I_A^S, is increased by a fraction of individuals from pre-infectious adult population E_A, at a proportion $(1-\phi)\tau$. The same population is decreased by the treatment of individuals in this class and by their migration into the treatment populations at the rate ξ_2 and is further decreased by the natural death at a rate μ_A and due to malaria induced death at a rate δ_1. Then,

$$\frac{dI_A^S}{dt} = (1-\phi)\tau E_A - (\xi_2 + \mu_A + \delta_1)I_A^S \tag{8}$$

The population of treated adult T_A grow due the treatment of non-severe and severely infected adult populations at a rate ξ_1 and ξ_2 respectively. The population is decreased by the transfer of individuals into susceptible adult population S_A and due to natural death at rate μ_A. Thus,

$$\frac{dT_A}{dt} = \xi_1 I_A^N + \xi_2 I_A^S - (\mu_A + \alpha_A)T_A \tag{9}$$

The population of susceptible children S_C is increased through birth at the rate Λ_C and through migration of treated children's who were previously infected with malaria at the rate α_C. The population is reduced due to new infection

acquired at the rate λ_C. The population is decreased by the transfer of children who are above the puberty age at the rate ψ into the susceptible adult population S_A and due to natural death at the rate μ_C.

$$\text{where } \lambda_C = \frac{\vartheta_M \beta_C I_M}{N_H} S_C \tag{10}$$

With ϑ_M being the per capita biting ate of the mosquito on the susceptible children population S_C and β_C is the transmission probability. Thus,

$$\frac{dS_C}{dt} = \Lambda_C + \alpha_C T_C - \psi S_C - \lambda_C S_C - \mu_C S_C \tag{11}$$

The population of pre-infectious children E_C is increased due to new infection acquired by the susceptible children at a rate λ_C. The same population is decreased as a result of transfer of the pre-infectious into the infected and infectious non-severe children population I_C^N at a rate ε with σ being the fraction moving out from the pre-infectious class. The population is further decreased because of natural death at a rate μ_C. Therefore,

$$\frac{dE_C}{dt} = \lambda_C S_C - (\sigma\varepsilon + \mu_C) E_C \tag{12}$$

The population of non-severe infected children I_C^N is increased due to progression of a fraction of pre-infectious children at a proportion $\sigma\varepsilon$ where $0 \le \sigma \le 1$. This population is decreased by treatment and the transfer of the treated children at a rate ω_1. The population is further decreased because of natural death at a rate μ_C and due to malaria-induced death at a rate δ_2. Hence,

$$\frac{dI_C^N}{dt} = \sigma\varepsilon E_C - (\omega_1 + \mu_C) I_C^N \tag{13}$$

The population of severely- infected children I_C^S is increased due to progression of a fraction of pre-infectious children at a proportion $(1-\sigma)\varepsilon$ where $0 \le \sigma \le 1$. This population is decreased by treatment and the transfer of the treated children at a rate ω_2. The population is further decreased because of natural death at a rate μ_C and due to malaria-induced death at a rate δ_2. So that,

$$\frac{dI_C^S}{dt} = (1-\sigma)\varepsilon E_C - (\omega_2 + \mu_C)I_C^S \tag{14}$$

The population of treated children T_C grow due to the treatment of non-severe and severely infected children's populations at a rate ω_1 and ω_2 respectively. The population is decreased by the transfer of individuals into susceptible children population S_C at a rate α_C and due to natural death at a rate μ_C. Thus,

$$\frac{dT_C}{dt} = \omega_1 I_C^N + \omega_2 I_C^S - (\mu_C + \alpha_C)T_C \tag{15}$$

The susceptible mosquito population S_M is increased through maturation of the egg-larva-pupa into adult mosquito at a rate Λ_M. This population decreased due to new infection acquired through contacts between susceptible mosquito, infected adult and infected children both severe and non-severe populations by the force of infection λ_M, where

$$\lambda_M = \frac{\vartheta\beta_M(I_A^N + I_A^S + I_C^N + I_C^S)}{N_H} S_M \tag{16}$$

Where ϑ is the per capita biting rate of the susceptible mosquito on infected adult and infected children populations, β_M is the transmission probability through blood meals of infected individuals into the susceptible mosquito. This population is further decreased through natural death at a rate μ_M. Thus,

$$\frac{dS_M}{dt} = \Lambda_M - \lambda_M S_M - \mu_M S_M \tag{17}$$

The population of pre-infectious mosquito is increased through migration of newly infected mosquitoes λ_M from the susceptible population. This population decreased as a result of progression into infected mosquito I_M at a rate γ and is further reduced due to natural death at a rate μ_M. This gives,

$$\frac{dE_M}{dt} = \lambda_M S_M - (\gamma - \mu_M)E_M \tag{18}$$

Finally, the population of infected and infectious mosquito I_M grow due to progression of pre-infectious mosquito population E_M at a rate γ and is decreased as a result of natural death at a rate μ_M. Hence this is governed by the equation

$$\frac{dI_M}{dt} = \gamma E_M - \mu_M I_M \tag{19}$$

3. The Basic Properties of the Malaria model

The state variables in model (1) are all time dependent and all the parameters are positive (non-negative). Notably, the equations which described the rate of change of the total adult (N_A), children (N_C), and the Mosquito (N_M) populations are:

$$\frac{dN_A}{dt} = \psi - \mu_A N_A$$

$$\frac{dN_C}{dt} = \Lambda_C - \mu_C N_C \tag{20}$$

$$\frac{dN_M}{dt} = \Lambda_M - \mu_M N_M$$

Theorem 1: Let $S_A > 0, E_A \geq 0, ...I_M \geq 0.$ The solutions $S_A, E_A, ...I_M$ of the model system (1) are positive for $t \geq 0$ the system (1) in the region D is positively invariant and all solutions starting in D approach, enter or stay in $D.$

Proof: Using the approach by Bhunu et al. (2009), theorem 1 will be proved as follows:

Under the given initial conditions, it is easy to prove that the components of system (1) are positive, otherwise, we assume a contradiction: that there exists a first time

$t_1 : S_A(t_1) = 0, \frac{dS_A}{dt_1} < 0,\ E_A(t) > 0,\ I_A^N(t) > 0,\ I_A^S(t) > 0,\ ...I_M(t) > 0,$ for $0 < t < t_2,$ or assume there exist

$t_2 : E_A(t_2) = 0,\ \frac{dE_A}{dt_2} < 0,\ S_A(t) > 0,\ I_A^N(t) > 0,\ I_A^S(t) > 0,\ ...I_M(t) > 0,$ for $0 < t < t_3,$ or assume there exist

$t_3 : I_A^N(t_3) = 0,\ \frac{dI_A^N}{dt_3} < 0,\ S_A(t) > 0,\ E_A(t) > 0,\ I_A^S(t) > 0,\ ...I_M(t) > 0,$ for $0 < t < t_1,$ or assume there exist

$t_4 : I_A^S(t_4) = 0,\ \frac{dI_A^S}{dt_4} < 0,\ S_A(t) > 0,\ E_A(t) > 0,\ I_A^N(t) > 0,\ ...I_M(t) > 0,$ for $0 < t < t_4,$ or assume there exist

$t_5 : T_A(t_5) = 0,\ \frac{dT_A}{dt_5} < 0,\ S_A(t) > 0,\ E_A(t) > 0,\ I_A^N(t) > 0,\ ...I_M(t) > 0,$ for $0 < t < t_5,$ or assume there exist

$t_6 : S_C(t_6) = 0, \frac{dS_C}{dt_6} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_6$, or assume there exist

$t_7 : E_C(t_7) = 0, \frac{dE_C}{dt_7} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_7$, or assume there exist

$t_8 : I_C^N(t_8) = 0, \frac{dI_C^N}{dt_8} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_8$, or assume there exist

$t_9 : I_C^S(t_9) = 0, \frac{dI_C^S}{dt_8} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_9$, or assume there exist

$t_{10} : T_C(t_{10}) = 0, \frac{dT_C}{dt_{10}} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_{10}$, or assume there exist

$t_{11} : S_M(t_{11}) = 0, \frac{dS_M}{dt_{11}} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_{11}$, or assume there exist

$t_{12} : E_M(t_{12}) = 0, \frac{dE_M}{dt_{12}} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_{12}$, or assume there exist

$t_{13} : I_M(t_{13}) = 0, \frac{dI_M}{dt_{13}} < 0, S_A(t) > 0, E_A(t) > 0, I_A^N(t) > 0, ...I_M(t) > 0,$ for $0 < t < t_{13}$, or assume there exist

Thus, from the first equation of model (1),

$$\frac{dS_A}{dt} = \psi S_C + \alpha_A T_A > 0 \tag{21}$$

This is a contradiction to the earlier assumption made and it implies that S_A is positive.

From the second equation of the model (1) we have

$$\frac{dE_A}{dt} = \lambda_A S_A > 0 \tag{22}$$

This is a contradiction to the earlier assumption made and it implies that E_A is positive.

From the third equation of the model (1) we get,

$$\frac{dI_A^N}{dt} = \phi\tau E_A > 0 \tag{23}$$

This is a contradiction to the earlier assumption made and it implies that I_A^N is positive.

From the fourth equation of model (1) we have

$$\frac{dI_A^S}{dt} = (1-\phi)\tau E_A > 0 \tag{24}$$

This is also a contradiction from the assumption made and it implies that I_A^S is positive.

From the fifth equation of model (1) we have

$$\frac{dT_A}{dt} = \xi_1 I_A^N + \xi_2 I_A^S > 0 \tag{25}$$

This is a contradiction from the assumption made and it implies that T_A is positive.

From the sixth equation of the model (1), we have

$$\frac{dS_C}{dt} = \Lambda_C + \alpha_C T_C > 0 \tag{26}$$

This is a contradiction from the assumption made and it implies that S_C is positive.

From the seventh equation of model (1) we have

$$\frac{dE_C}{dt} = \lambda_C S_C > 0 \tag{27}$$

This is a contradiction from the assumption made and it implies that E_C is positive.

From the eight equations of model (1) we have

$$\frac{dI_C^N}{dt} = \sigma\varepsilon E_C > 0 \tag{28}$$

This is a contradiction from the assumption made and it implies that I_C^N is positive.

From the ninth equations of the model (1) we have

$$\frac{dI_C^S}{dt} = (1-\sigma)\varepsilon E_C > 0 \tag{29}$$

This is a contradiction from the assumption made and it implies that I_C^S is positive.

From the tenth equation of the model (1) we have

$$\frac{dT_C}{dt} = \omega_1 I_C^N + \omega_2 I_C^S > 0 \tag{30}$$

This is a contradiction from the assumption made and it implies that T_C is positive.

From the eleventh equation of the model (1) we have

$$\frac{dS_M}{dt} = \Lambda_M > 0 \tag{31}$$

This is a contradiction from the assumption made and it implies that S_M is positive.

From the twelve equations of the model (1) we have

$$\frac{dE_M}{dt} = \lambda_M S_M > 0 \tag{32}$$

This is a contradiction from the assumption made and it implies that E_M is positive.

From the thirteenth equations of the model (1) we have

$$\frac{dI_M}{dt} = \gamma E_M > 0 \tag{33}$$

This is a contradiction from the assumption made and it implies that I_M is positive.

Therefore, all the solutions to the system of model (1) remain positive for all given non-negative initial conditions.

Recall that, $\frac{dN_A}{dt} = \psi - \mu_A N_A$, $\frac{dN_C}{dt} = \Lambda_C - \mu_C N_C$, and $\frac{dN_M}{dt} = \Lambda_M - \mu_M N_M$, these indicates that,

$N_A \to \frac{\psi}{\mu_A}$, $N_C \to \frac{\Lambda_C}{\mu_C}$, and $N_M \to \frac{\Lambda_M}{\mu_M}$ are bounded. Epidemiological evidence pointed that the model (1) can be studied in the feasible region:

$$\Gamma = \{(S_A, E_A, I_A^N, I_A^S, T_A, S_C, E_C, I_C^N, I_C^S, T_C, S_M, E_M, I_M) \in \Re_+^{13} : N_A(t) \le \frac{\psi}{\mu_A}, N_C(t) \le \frac{\Lambda_C}{\mu_C}, N_M(t) \le \frac{\Lambda_M}{\mu_M}\} \quad (34)$$

Which is positively-invariant with respect to the system (1). This implies that the solution to system (1) with initial condition in Γ, will remain in Γ at all time $t \ge 0$. It sufficiently follows from Theorem (1) and this result to conveniently establish the following lemma:

Lemma (1): The region Γ is positively-invariant for the model (1) with initial conditions in $\Re_+^{13}$.

3.1 Disease-free equilibrium

At disease free equilibrium, the equation of the model (1) is set to zero as follow:

$$\frac{dS_A}{dt} = \frac{dE_A}{dt} = \frac{dI_A^N}{dt} = \frac{dI_A^S}{dt} = \ldots \frac{dI_M}{dt} = 0 \quad (35)$$

$$S_A^* = \frac{\psi \Lambda_C}{\mu_A(\psi + \mu_c)}, S_C^* = \frac{\Lambda_C}{(\psi + \mu_c)}, S_M^* = \frac{\Lambda_M}{\mu_M} \quad (36)$$

Therefore, the disease-free equilibrium of the Malaria model (1) is

$$E^* = \left(\frac{\psi \Lambda_C}{\mu_A(\psi + \mu_c)}, 0, 0, 0, 0, \frac{\Lambda_C}{(\psi + \mu_c)}, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0, \right) \quad (37)$$

3.2 Basic reproduction number

The basic reproduction number represents the secondary infection that an infectious individual can produce during his/her life time as an infectious person. Thus, $R_0 = \rho(FV^{-1})$, where ρ stands for the dominant eigenvalue. The method of next generation method is employed to determine the R_0. Where

$$F=\begin{pmatrix}\lambda_A S_A\\0\\0\\\lambda_C S_C\\0\\0\\\lambda_M S_M\\0\end{pmatrix} \quad \text{and} \quad V=\begin{pmatrix}(\phi\tau+\mu_A)E_A\\(\xi_1+\mu_A)I_A^N-\phi\tau E_A\\(\xi_2+\mu_A)I_A^S-(1-\phi\tau)E_A\\(\sigma\varepsilon+\mu_C)E_C\\(\omega_1+\mu_C)I_C^N-\sigma\varepsilon E_C\\(\omega_2+\mu_C)I_C^S-(1-\sigma)\varepsilon E_C\\(\gamma-\mu_M)E_M\\\mu_M I_M-\gamma E_M\end{pmatrix} \tag{38}$$

and the dominant eigenvalue is

$$R_0=\sqrt{\frac{\gamma\vartheta\beta_M\left[\vartheta_M\beta_C r_1 r_2 r_3\left(k_3 r_6+k_4 r_5\right)+\vartheta_M\beta_A r_4 r_5 r_6\left(k_1 r_3+k_2 r_2\right)\right]}{r_1 r_2 r_3 r_4 r_5 r_6 r_7}} \tag{39}$$

where $r_1=(\phi\tau+\mu_A), r_2=(\xi_1+\mu_A), r_3=(\xi_2+\mu_A), r_4=(\sigma\varepsilon+\mu_C), r_5=(\omega_1+\mu_C)$
$r_6=(\omega_2+\mu_C), r_7=(\gamma+\mu_M), r_8=\mu_M, k_1=\phi\tau, k_2=(1-\phi\tau), k_3=\sigma\varepsilon, k_4=(1-\sigma)\varepsilon.$

Lemma 1: Following the method by Driessche and Watmough (2002), the Malaria-free equilibrium is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

The basic reproduction number R_0, is a threshold parameter that represent the dynamics of the malaria transmission. The local asymptotic stability indicates that a small number of the outbreak if the $R_0<1$ will not generate higher disease spread, while a relatively threshold greater than one will bring about higher influx of the malaria disease within the population.

3.3 Global stability analysis of the malaria-free equilibrium

To determine the global asymptotic stability analysis of the malaria-free equilibrium, we follow the approach by Castillo-Chavez and Feng (2001). To achieve this, two conditions were set and the system (1) is re-written as follows:

$$\left.\begin{aligned}\frac{dX}{dt}&=F(X,Z)\\\frac{dZ}{dt}&=G(X,Z), G(X,0)=0\end{aligned}\right\} \tag{40}$$

where $X=(S_A,S_C,S_M)$ represent the uninfected adult, children and the mosquito populations respectively and $X\in R^3$, while $Z=(E_A,I_A^N,I_A^S,E_C,I_C^N,I_C^S,E_M,I_M)$ represent the infected adults, children and the mosquito populations alike and $Z\in R^8$.

The Malaria-free equilibrium is $E^*=\left(\frac{\psi\Lambda_C}{\mu_A(\psi+\mu_c)},\frac{\Lambda_C}{(\psi+\mu_c)},\frac{\Lambda_m}{\mu_m}\right)$ (41)

The following two conditions must be satisfied to guarantee the global asymptotic stability:

H_1: for $\frac{dX}{dt}=F(X,0)$, X^0 is globally asymptotically stable.

$H_2: G(X,Z)=CZ-\hat{G}(X,Z)$, where $\hat{G}(X,Z)\geq 0$, for $(X,Z)\in\Gamma$

where $C=D_ZG(x^0,0)$ is an $M-matrix$ (the off-diagonal element of C is non-negative) and Γ is a biologically feasible region.

Lemma 2: The point $K^*=\left(X^0,0\right)$ is called stable global equilibrium point whenever $R_0<1$ and the conditions H_1 and H_2 hold.

The following theorem is formed on the basis of Lemma 2,

Theorem 2: Suppose $R_0<1.$ The Malaria-free equilibrium is globally asymptotically stable.

Proof: based on condition of comparison theorem,

$\frac{dX}{dt}=F(X,Z)\Rightarrow X\in R^3$ can be written as follows:

$$F(X,Z)=\begin{pmatrix}\psi S_C+\alpha_A T_A-\lambda_A S_A-\mu_A S_A\\ \Lambda_C+\alpha_C T_C-\psi S_C-\lambda_C S_C-\mu_C S_C\\ \Lambda_M-\lambda_M S_M-\mu_M S_M\end{pmatrix} \quad (42)$$

At Malaria-free equilibrium, $\frac{dX}{dt}=F(X,0)$ Thus, the uninfected class becomes

$E^*=\left(\frac{\psi\Lambda_C}{\mu_A(\psi+\mu_c)},\frac{\Lambda_C}{(\psi+\mu_c)},\frac{\Lambda_m}{\mu_m}\right)$ which is verifiable. With

$S_A \to \frac{\psi\Lambda_C}{\mu_A(\psi+\mu_c)}, S_C \to \frac{\Lambda_C}{(\psi+\mu_c)}, S_M \to \frac{\Lambda_m}{\mu_m}$ as $t \to \infty$. This implies a global convergence of the system (1) on the region Γ. This satisfied H_1, which is the first of the two conditions. In the same fashion, the second condition H_2 of the infected classes, $Z \in R^8$ requires that $\hat{G}(X,Z) \geq 0 \ \forall (X,Z) \in \Gamma$. Thus,

$$G(X,Z) = \begin{bmatrix} \lambda_A S_A - (\phi\tau + \mu_A)E_A \\ \phi\tau E_A - (\xi_1 + \mu_A)I_A^N \\ (1-\phi)\tau E_A - (\xi_2 + \mu_A)I_A^S \\ \lambda_C S_C - (\sigma\varepsilon + \mu_C)E_C \\ \sigma\varepsilon E_C - (\omega_1 + \mu_C)I_C^N \\ (1-\sigma)\varepsilon E_C - (\omega_2 + \mu_C)I_C^S \\ \lambda_M S_M - (\gamma + \mu_M)E_M \\ \gamma E_M - \mu_M I_M \end{bmatrix} \tag{43}$$

From the second condition H_2, $G(X,Z) = CZ - \hat{G}(X,Z)$, where C is M-matrix and Z is a column vector; computing the Jacobian of (42) at the malaria-free equilibrium,

$$CZ = \begin{bmatrix} -z_1 & 0 & 0 & 0 & 0 & 0 & 0 & d_2 \\ \phi\tau & -z_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ (1-\phi)\tau & 0 & -z_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -z_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma\varepsilon & -z_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & (1-\sigma)\varepsilon & 0 & -z_6 & 0 & 0 \\ 0 & d_1 & d_1 & 0 & d_1 & d_1 & -z_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma & -\mu_M \end{bmatrix} \begin{pmatrix} E_A \\ I_A^N \\ I_A^S \\ E_C \\ I_C^N \\ I_C^S \\ E_M \\ I_M \end{pmatrix} \tag{44}$$

where $d_1 = \frac{\vartheta\beta_M}{N_H} S_M \quad d_2 = \frac{\vartheta_M \beta_A}{N_H} S_A \quad z_1 = (\phi\tau + \mu),\ z_2 = (\xi_1 + \mu_A),\ z_3 = (\xi_2 + \mu_A),\ z_4 = (\sigma\varepsilon + \mu_C),$
$z_5 = (\omega_1 + \mu_C),\ z_6 = (\omega_2 + \mu_C),\ z_7 = (\omega_2 + \mu_C).$

Since all the off-diagonal element of C are non-negative, it can be concluded that it is a Metzler matrix. Therefore,

$$G(X,Z) = \begin{pmatrix} G_1(X,Z) \\ G_2(X,Z) \\ G_3(X,Z) \\ G_4(X,Z) \\ G_5(X,Z) \\ G_6(X,Z) \\ G_7(X,Z) \\ G_8(X,Z) \end{pmatrix} = \begin{pmatrix} \vartheta_M \beta_A I_M \left(1 - \frac{S_A}{N_H}\right) \\ 0 \\ 0 \\ \vartheta_M \beta_C I_M \left(1 - \frac{S_C}{N_H}\right) \\ 0 \\ 0 \\ \vartheta\beta_M \left(I_A^N + I_A^S + I_C^N + I_C^S\right)\left(1 - \frac{S_M}{N_H}\right) \\ 0 \end{pmatrix} \tag{45}$$

It is obvious that, $G_i(X,Z) \geq 0,$ for $i = 1,2,...8.$ therefore, $S_{A0} \geq S_A, S_{C0} \geq S_C$ and $S_{M0} \geq S_M.$ It is also clear that C is an M-matrix, and this satisfied the conditions H_1 and $H_2.$ It is therefore sufficient to say that K^* is globally asymptotically stable when $R_0 < 1.$ This completes the proof.

4. Discussion of Result

An age-structured malaria model is presented. The model considered three populations namely, the adult human, the children before the age of puberty and the mosquito populations respectively. According to WHO (2023), 80% of overall death from malaria occurred in children of age 1-5 years. Therefore, this model is developed to explore the dynamics of malaria transmission taking into cognisance the population of children. The dynamics of the disease transmission is studied through the analytical solution of system (1). The preliminary investigation of the malaria model (1) reveals that, it's positively-invariant and epidemiologically feasible to study the behaviour of the system in the region Γ and it will remain in Γ at all-time $t \geq 0.$ The malaria free equilibrium point of the model (1) was obtained. The basic reproduction number of the system was also established using next generation matrix approach. The steady state analysis was carried out. It's shown that, the system (1) is locally asymptotically stable when the basic reproduction number $R_0 < 1$ and unstable otherwise. In the same fashion, the global stability analysis also reveals that, the system is globally asymptotically stable if $R_0 < 1$ and unstable otherwise. The result indicates that, once malaria transmission is confined within a small region, the disease may die out. Similarly, if the transmission is narrowed-down to a less than one person per all contacts between infected mosquito and susceptible adult and

children's populations the disease spread can be halted. To control malaria disease, there is an absolute need for understanding its transmission and the basic reproduction number as a major factor (Olitumo *et al.,* 2024). Therefore, keeping the secondary infection at the barest minimum is extremely important in combating the spread of malaria disease.

5. Conclusion

A deterministic model of malaria disease is formulated. The model is proved to be positively-invariant and biologically feasible in the region Γ. The Malaria-free equilibrium is obtained. The method of next generation matrix was employed to determine the basic reproduction number R_0, which is a threshold parameter for the spread of the disease. The stability analysis is proved to be locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. It is understood based on the result of the analysis that, the disease can be eradicated if there is at most less than one transmission by a single infectious individual within the given population.

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Conflict of Interest

The Authors declare that, there is no conflict of interest.

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