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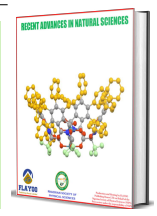

# Asymptotic perturbation and sensitivity analysis of malaria transmission in Nigeria: a mathematical model

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ABSTRACT

This article analyzes malaria dynamics among high-risk groups in Nigeria, with the human population divided into five compartments and the mosquito population divided into two compartments. The resulting system of nonlinear ordinary differential equations is examined by asymptotic perturbation and sensitivity-analysis methods. The perturbation analysis describes the behavior of the system at the malaria-free equilibrium (MFE) and the effect of a small perturbation around that state. The first-order perturbation solution indicates exponential growth in disease prevalence, showing that the MFE is unstable when infection is introduced. The sensitivity analysis shows that preventing mosquitoes from surviving long enough to become infectious is the most effective way to reduce the malaria transmission cycle. The infected adult male compartment, $I_M$, also acts as an important long-term reservoir and is the second most influential factor in the model. The estimated basic reproduction number satisfies $\mathcal{R}_0 > 1$, indicating a high potential for malaria spread and the need for effective control measures.



## 1. INTRODUCTION

According to Nundu *et al.* [1], malaria is a mosquito-borne disease caused by five protozoa: *Plasmodium falciparum*, *P. vivax*, *P. malariae*, *P. ovale*, and the more recently implicated *P. knowlesi*. According to Snow and Garcia [2, 3], *P. falciparum* accounts for more than 90% of global malaria mortality and remains a major threat to health systems worldwide. The World Health Organization's (WHO) World Malaria Report (2019) estimates 228 million cases of malaria worldwide, with 405,000 deaths recorded in 2018—many under the age of 5 [4]. Malaria is endemic in more than 90 countries and affects more than 40% of the world's population [3].

Children aged 3–6 months have relatively strong immunity against malaria because of the passive transfer of maternal antibodies through the placenta; this immunity wanes after about 6 months, leaving children vulnerable to malaria infection until their own immunity develops [5, 6]. Studies by Mbewe *et al.* and Takken *et al.* [7, 8] showed that children are more prone to malaria, although less attractive to female *Anopheles* mosquitoes than adults because of factors such as body size and metabolism. Pregnant women are more attractive to mosquitoes than children under five and non-pregnant adults. This is associated with factors that are elevated during pregnancy, including body chem-

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istry, odor, carbon dioxide production, and body heat [9].

A study carried out in Nigeria by Ayodele *et al.* [10] reported that malaria infection severity and prevalence appear to be higher in males than in females because of differences in exposure to malaria vectors and immunity to parasitic infection. Akanbi *et al.* [11] showed that adult males expose their bodies more than females, especially during hot weather, thereby increasing their likelihood of mosquito bites. Further investigations have suggested causes of sex differences, including male production of chemicals that attract mosquitoes, estrogen-mediated enhancement of anti-plasmodial immune responses in females [12], and testosterone-mediated suppression of anti-plasmodial immune responses in males [13]. These findings are consistent with Zuk and McKean [14], who noted that female immunity to parasitic diseases is higher than male immunity because of genetic and hormonal factors.

A model developed by Duve *et al.* [15] incorporates a class of vaccinated humans and an influx of infected immigrants. Sensitivity indices computed for $\mathcal{R}_0$ point to strategies for reducing mosquito–human contact. The results under different vaccine-coverage levels showed that, in the absence of infected immigrants, a malaria-free society can be achieved by increasing childhood vaccination and preventing the entry of infected humans. The optimal-control results showed that, if the influx of infected humans is completely stopped, the best way to reduce malaria incidence is the combined use of vaccination, personal protective equipment, and treatment.

In 2021, an age-structured model for the transmission dynamics of malaria with asymptomatic individuals and infected immigrants was analyzed. The model without infected immigrants showed that the disease-free equilibrium is stable whenever $\mathcal{R}_0 < 1$. The sensitivity results for the basic reproduction number indicated that children's parameters are more sensitive than adult parameters, whereas the model does not admit a disease-free equilibrium in the presence of infected immigrants. The sensitivity results revealed that the parameters for asymptomatic infected humans are more critical than those for infected immigrants. Therefore, strategies targeting asymptomatic carriers and infected immigrants are needed to reduce malaria transmission [16]. Extending this work, Kalula *et al.* [17] incorporated control measures to analyze optimal-control strategies and temperature variations without considering the roles of children under 5 years of age and pregnant women with malaria.

Azu-Tungmah *et al.* [18] modified the age-structured Rose model, incorporating four human compartments: susceptible individuals, infectious children under five, infectious individuals over five, and infectious pregnant women, while the mosquito population was divided into susceptible and infectious sections, providing a detailed view of malaria transmission dynamics. They performed a sensitivity analysis showing that malaria can be controlled or possibly eliminated if parameters such as mosquito biting rates, recruitment rate, density-dependent natural mortality rate for mosquitoes, and human recovery rates are controlled [18].

Extending the work of Azu-Tungmah *et al.* [18], Robert *et al.* [19] modified the mathematical model by including children below 5 years of age, pregnant women, and non-pregnant humans (males and females above 5 years), together with the influence of temperature, to study mosquito-biting behavior. Their simulations showed that an increase in temperature also increased infection rates in both human and mosquito populations. Thus, they concluded that higher temperatures increase malaria infection rates in both human and mosquito populations, with children below 5 years of age and pregnant women having distinct impacts.

The models formulated by Azu-Tungmah *et al.* [18] and Robert *et al.* [19] did not investigate malaria infection severity and prevalence in males and females separately because the class of non-pregnant adults was not partitioned. This work therefore modifies the mathematical model formulated by Azu-Tungmah *et al.* [18], following the findings in Refs. [10–13], by splitting non-pregnant adults into adult males and non-pregnant adult women, performing a sensitivity analysis, and obtaining the series solution of the model by the perturbation method.

## 2. THE EXTENDED MALARIA MODEL

The model follows the basic structure stated by Azu-Tungmah *et al.* [18], with the inclusion of one compartment to reflect the reports in Refs. [10–13]. For simplicity, some of the parameters of the model formulated by Azu-Tungmah *et al.* [18] are modified in the extended model.

Let $N(t)$ represent the total human population at time $t$. The model subdivides the human population into susceptible humans $S_H(t)$, infected children aged 0–5 years $I_I(t)$, infected non-pregnant women (above 5 years) $I_W(t)$, infected adult males (above 5 years) $I_M(t)$, and infected pregnant women $I_P(t)$. Hence, the total population of human beings is stated as

$$N_H(t) = S_H(t) + I_P(t) + I_W(t) + I_I(t) + I_M(t). \tag{1}$$

Similarly, the vector (mosquito) population at time $t$, denoted by $N_V(t)$, is divided into two compartments. The division consists of susceptible mosquitoes $S_V(t)$ and infected mosquitoes $I_V(t)$. Hence,

$$N_v(t) = S_V(t) + I_V(t). \tag{2}$$

In this model, immunity in recovered humans is assumed to be temporary; therefore, recovered individuals return to the susceptible class. The current human life expectancy in Nigeria, according to the World Health Organization (WHO), is 55.75 years [26]. Following the method adopted by Duve *et al.* [15], the human natural mortality rate $\mu_H$ was calculated from life expectancy through the relationship: $\mu_H = \frac{1}{(L\times 365)}$, $L = 55.75$ years for Nigeria, then

$$\mu_H = \frac{1}{55.75 \times 365} \approx 4.914\ 10^{-5} \text{ per day}. \tag{3}$$

For human recruitment, adopting the method of Olaniyi and Obabiyi [24], $Z_H$ is obtained as

$$Z_H = \frac{1000}{55.75} \approx 17.935. \tag{4}$$

We employed the approach of scaling factors and followed the fact that adult men have a 30% higher exposure [31], a 10% lower recovery rate [32], and a 20% higher malaria-induced death rate

**Table 1. Model parameters and their sources.**

| Parameters | Description | Values | Sources |
|---|---|---|---|
| $Z_H$ | Recruitment into human population | 17.935 | (4) |
| $Z_V$ | Recruitment into mosquitoes | 17380.95 | [25] |
| $\theta_{VH}$ | Fraction of bites that successfully infect humans | 0.1 | [24] |
| $\theta_{HV}$ | Fraction of bites that successfully infect mosquitoes | 0.09 | [24] |
| $\phi_P$ | Number of bites on pregnant women per mosquito | 0.01405 | [18] |
| $\phi_W$ | Number of bites on non-pregnant women per mosquito | 0.96144 | [29] |
| $\phi_I$ | Number of bites on children under 5 per mosquito | 0.32437 | [18] |
| $\phi_M$ | Number of bites on adult men per mosquito | 0.39 | (5) |
| $\mu_H$ | Natural mortality rates for humans | 0.00004914 | 3 |
| $\mu_V$ | Natural mortality rates for mosquitoes | 0.04 | [15] |
| $a_0$ | Transition rate from $I_I$ to $I_W$ class | 0.0156 | (8) |
| $a_1$ | Transition rate from $I_I$ to $I_M$ class | 0.100 | (9) |
| $a_2$ | Transition rate from $I_W$ to $I_P$ class | 0.100 | [18] |
| $\pi_P$ | Malaria induced death rates for pregnant women | 0.5337 | [18] |
| $\pi_W$ | Malaria induced death rates for non-pregnant women | 0.1982 | [28] |
| $\pi_I$ | Malaria induced death rates for children under 5 | 0.01924 | [18] |
| $\pi_M$ | Malaria induced death rates for adult men | 0.00012 | (7) |
| $\Lambda_P$ | Recovery rates of pregnant women | 0.1615 | [18] |
| $\Lambda_W$ | Recovery rates of non-pregnant women | 0.16157 | [30] |
| $\Lambda_I$ | Recovery rates of children under 5 | 0.1391 | [18] |
| $\Lambda_M$ | Recovery rates of adult men | 0.045 | (6) |

**Table 2. Comparison of results from three methods at $t = 1$.**

| Element | Eigenvalue method | Series expansion | Laplace transform |
|---|---|---|---|
| $(1,1)$ | 0.5012 | 0.50016 | 0.5002 |
| $(2,2)$ | 0.6345 | 0.63461 | 0.6345 |
| $(3,3)$ | 0.7623 | 0.76037 | 0.7603 |
| $(4,4)$ | 0.9567 | 0.95601 | 0.9560 |
| $(5,5)$ | 0.9654 | 0.96172 | 0.9617 |
| $(2,5)$ | 0.0897 | 0.07699 | 0.0770 |
| $(5,2)$ | 0.0987 | 0.08251 | 0.0825 |

**Table 3. Sensitivity analysis result.**

| Parameters | Index | Parameters | Index |
|---|---|---|---|
| $Z_H$ | $-0.5$ | $\Lambda_W$ | $-0.1772$ |
| $Z_V$ | $+0.5$ | $\Lambda_I$ | $-0.01046$ |
| $\theta_{VH}$ | $+0.5$ | $\Lambda_M$ | $-0.7083$ |
| $\theta_{HV}$ | $+0.5$ | $a_0$ | $+0.00897$ |
| $\mu_H$ | $+0.4994$ | $a_1$ | $+0.01001$ |
| $\mu_V$ | $-1.0000$ | $a_2$ | $+0.000131$ |
| $\phi_P$ | $+0.000633$ | $\pi_P$ | $-0.000454$ |
| $\phi_W$ | $+0.01524$ | $\pi_W$ | $-0.000354$ |
| $\phi_I$ | $+0.01219$ | $\pi_I$ | $-0.001447$ |
| $\phi_M$ | $+0.01486$ | $\pi_M$ | $-0.001889$ |
| $\Lambda_P$ | $-0.000137$ | | |

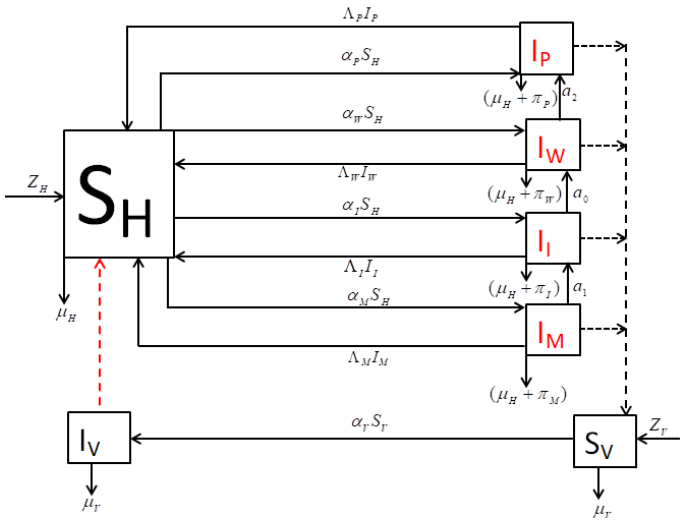


**Figure 1.** Compartmental diagram of malaria infection.

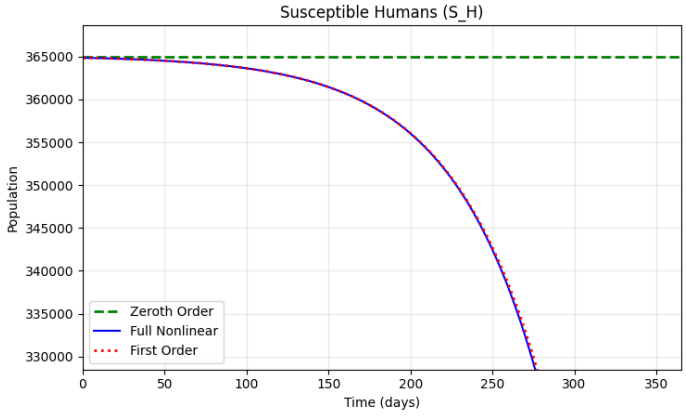


**Figure 2.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $S_H$ class.

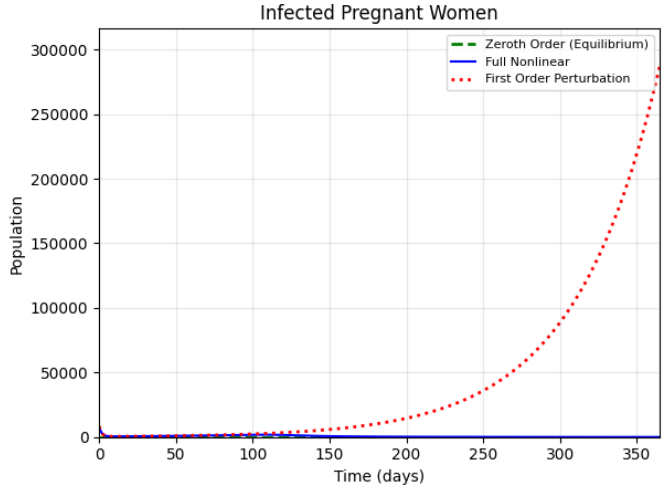


**Figure 3.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $I_P$ class.

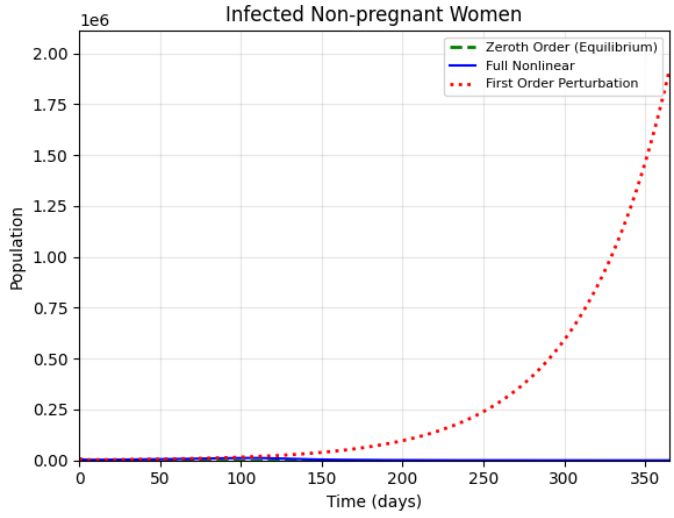


**Figure 4.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $I_W$ class.

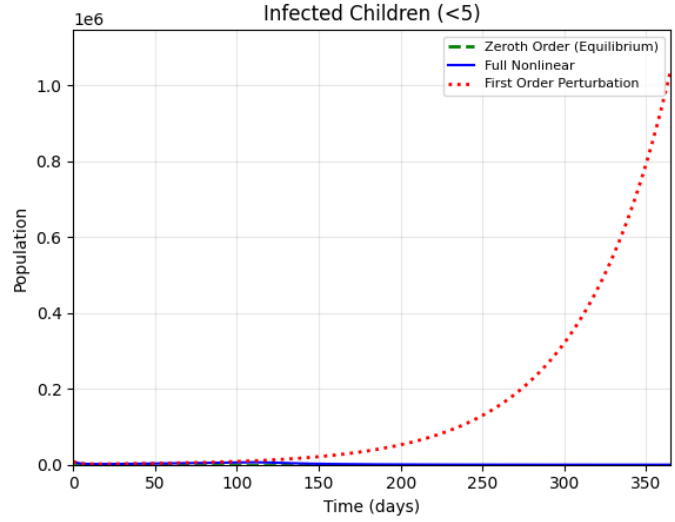


**Figure 5.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $I_I$ class.

[33]. Hence, the mosquito biting rate for adult males is calculated as

$$\phi_M = 1.3 \times 0.3 = 0.39. \tag{5}$$

The recovery rate and malaria-induced death rate for adult men are calculated as follows:

$$\Lambda_M = 0.9 \times 0.05 = 0.045. \tag{6}$$

The malaria-induced death rate for adult men is calculated as

$$\pi_M = 1.2 \times 0.0001 = 0.00012. \tag{7}$$

The total aging rate for children under 5 is given as: $\frac{1}{T}$, where $T = 5$, then $\frac{1}{5} = 0.2$. Using the Nigeria's age $0-4$ sex ratio of 102 males per 100 females as stated by National Population Commission (NPC) and ICF, (2019) Ref. ([27]), then

$$a_0 = \frac{100}{202} \times 0.2 \approx 0.099, \tag{8}$$

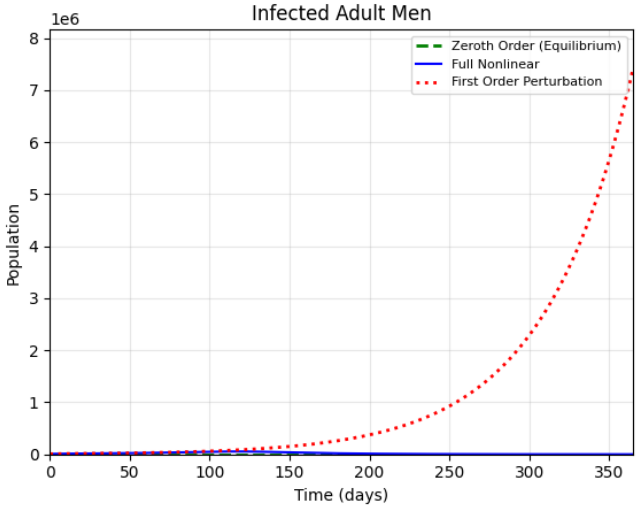


**Figure 6.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $I_M$ class.

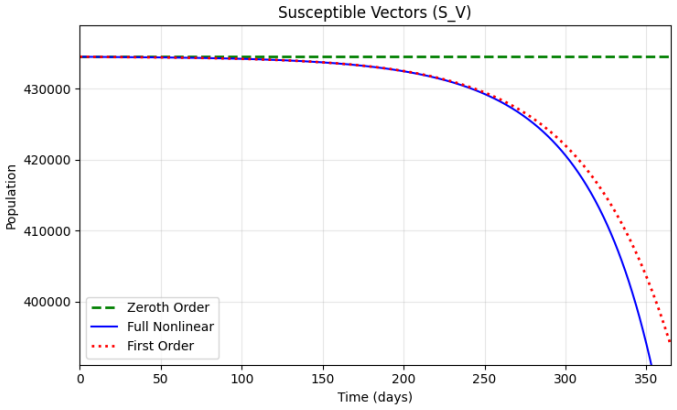


**Figure 7.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $S_V$ class.

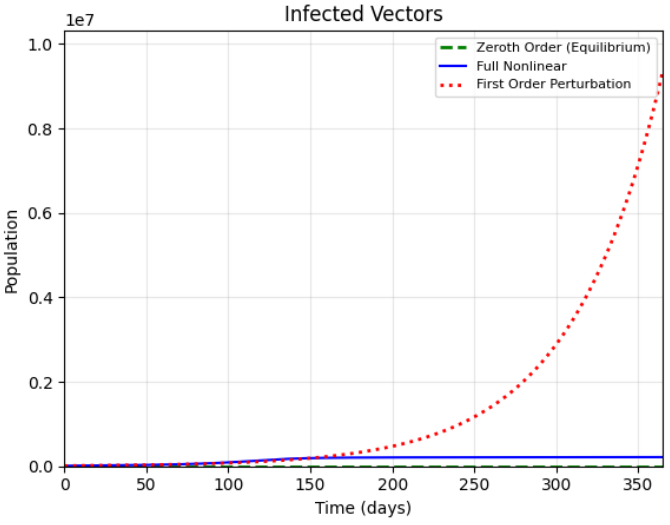


**Figure 8.** Comparison of the full nonlinear model, first-order perturbation, and zeroth-order solution for the $I_V$ class.

and

$$a_1 = \frac{102}{202} \times 0.2 \approx 0.101. \tag{9}$$

$$\begin{cases} \frac{dS_H}{dt} = Z_H + \Lambda_P I_P + \Lambda_W I_W + \Lambda_I I_I + \Lambda_M I_M \\ \qquad - \frac{(\phi_P + \phi_W + \phi_I + \phi_M)\theta_{VH} S_H I_V}{N_H} - \mu_H S_H, \\ \frac{dI_P}{dt} = \frac{\theta_{VH}\phi_P S_H I_V}{N_H} + a_2 I_W \\ \qquad - (\Lambda_P + \mu_H + \pi_P) I_P, \\ \frac{dI_W}{dt} = \frac{\theta_{VH}\phi_W S_H I_V}{N_H} + a_0 I_I \\ \qquad - (a_2 + \Lambda_W + \mu_H + \pi_W) I_W, \\ \frac{dI_I}{dt} = \frac{\theta_{VH}\phi_I S_H I_V}{N_H} \\ \qquad - (a_0 + a_1 + \Lambda_I + \mu_H + \pi_I) I_I, \\ \frac{dI_M}{dt} = \frac{\theta_{VH}\phi_M S_H I_V}{N_H} + a_1 I_I \\ \qquad - (\Lambda_M + \mu_H + \pi_M) I_M, \\ \frac{dS_V}{dt} = Z_V - \frac{\theta_{HV} S_V (\phi_P I_P + \phi_W I_W + \phi_I I_I + \phi_M I_M)}{N_H} \\ \qquad - \mu_V S_V, \\ \frac{dI_V}{dt} = \frac{\theta_{HV} S_V (\phi_P I_P + \phi_W I_W + \phi_I I_I + \phi_M I_M)}{N_H} \\ \qquad - \mu_V I_V, \end{cases} \tag{10}$$

where $S_H(0) > 0,\ I_P(0) \geq 0,\ I_W(0) \geq 0,\ I_I(0) \geq 0,\ I_M(0) \geq 0, S_V(0) > 0,\ I_V(0) \geq 0.$

### 2.1. MALARIA-FREE EQUILIBRIUM POINTS AND REPRODUCTION NUMBER

Malaria infection has disease-free equilibrium states, which are obtained by setting $I_P = I_W = I_I = I_M = 0$ and the remaining equations in Eq. (10) to zero. Let $\epsilon_0 = \{S_H^0, I_P^0, I_W^0, I_I^0, I_M^0, S_V^0, I_V^0\}$ denote the malaria-free state. Solving the equations for susceptible humans and susceptible mosquitoes gives

$$\epsilon_0 = \{\frac{Z_H}{\mu_H}, 0, 0, 0, 0, \frac{Z_V}{\mu_V}, 0\}.$$

The basic reproduction number $\mathcal{R}_0$ was computed by employing the next-generation matrix method established by van den Driessche and Watmough [20]. Let the infected compartments be defined as

$$\mathbb{X}_I = (I_P, I_W, I_I, I_M, I_V)^T.$$

Let $\mathcal{F}$ denote the appearance of new infection terms and $\mathcal{V}$ denote the transition terms in the model, defined as follows:

$$\mathcal{F}_i = \begin{pmatrix} \frac{\theta_{VH}\phi_P S_H I_V}{N_H} \\ \frac{\theta_{VH}\phi_W S_H I_V}{N_H} \\ \frac{\theta_{VH}\phi_I S_H I_V}{N_H} \\ \frac{\theta_{VH}\phi_M S_H I_V}{N_H} \\ q_0 \end{pmatrix} \qquad \mathcal{V}_i = \begin{pmatrix} (\Lambda_P + \mu_H + \pi_P) I_P - a_2 I_W \\ (a_2 + \Lambda_W + \mu_H + \pi_W) I_W - a_0 I_I \\ (a_0 + a_1 + \Lambda_I + \mu_H + \pi_I) I_I \\ (\Lambda_M + \mu_H + \pi_M) I_M - a_1 I_I \\ \mu_V I_V \end{pmatrix}$$

where

$$q_0 = \frac{\theta_{HV} S_V (\phi_P I_P + \phi_W I_W + \phi_I I_I + \phi_M I_M)}{N_H}.$$

Set $F$ and $V$ as the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ with respect to infected compartments evaluated at the disease-free equilibrium (DFE),

$$S_H^0 = \frac{Z_H}{\mu_H}, \quad I_P^0 = I_W^0 = I_I^0 = I_M^0 = I_V^0 = 0, \quad S_V^0 = \frac{Z_V}{\mu_V}$$

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \theta_{VH}\phi_P \\ 0 & 0 & 0 & 0 & \theta_{VH}\phi_W \\ 0 & 0 & 0 & 0 & \theta_{VH}\phi_I \\ 0 & 0 & 0 & 0 & \theta_{VH}\phi_M \\ \theta_{HV}\phi_P\frac{Z_V\mu_H}{Z_H\mu_V} & \theta_{HV}\phi_W\frac{Z_V\mu_H}{Z_H\mu_V} & \theta_{HV}\phi_I\frac{Z_V\mu_H}{Z_H\mu_V} & \theta_{HV}\phi_M\frac{Z_V\mu_H}{Z_H\mu_V} & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} r_P & -a_2 & 0 & 0 & 0 \\ 0 & r_W & -a_0 & 0 & 0 \\ 0 & 0 & r_I & 0 & 0 \\ 0 & 0 & -a_1 & r_M & 0 \\ 0 & 0 & 0 & 0 & \mu_V \end{pmatrix},$$

where $r_P = (\Lambda_P + \mu_H + \pi_P)$, $r_W = (a_2 + \Lambda_W + \mu_H + \pi_W)$, $r_I = (a_0 + a_1 + \Lambda_I + \mu_H + \pi_I)$, $r_M = (\Lambda_M + \mu_H + \pi_M)$. The inverse of the matrix $V$ is obtained by applying the formula for the inverse of a lower triangular matrix [21]. The elements of $V^{-1}$ are determined by evaluating the system of equations formed as $VV^{-1} = I$. From the next-generation matrix $K = FV^{-1}$, the basic reproduction number is calculated from the spectral radius:

$$\mathcal{R}_0 = \rho(FV^{-1}).$$

The basic reproduction number $\mathcal{R}_0$, which is the dominant eigenvalue of $FV^{-1}$, is obtained from the characteristic equation $(FV^{-1} - \lambda I) = 0$ and is solved to obtain the largest absolute eigenvalue:

$$\mathcal{R}_0 = \sqrt{KQ}\,,$$

where

$$K = \frac{\theta_{VH}\theta_{HV}Z_V\mu_H}{Z_H\mu_V^2} \quad \text{and}$$

$$Q = \frac{\phi_P^2}{r_P} + \frac{\phi_W^2}{r_W} + \frac{\phi_I^2}{r_I} + \frac{\phi_M^2}{r_M} + \frac{a_2\phi_P\phi_W}{r_W r_P} + \frac{a_0a_2\phi_I\phi_P}{r_P r_W r_I} + \frac{a_0\phi_W\phi_I}{r_I r_W} + \frac{a_1\phi_I\phi_M}{r_I r_M}.$$

The terms $\theta_{VH}, \theta_{HV}$ represent the product of human-to-mosquito and mosquito-to-human transmission probabilities, $\phi_P, \phi_W, \phi_I, \phi_M$ denote the relative infectiousness of pregnant women, non-pregnant women, children under 5 and adult male human compartments, respectively. The terms $\frac{1}{r_P}, \frac{1}{r_W}, \frac{1}{r_I}, \frac{1}{r_M}$ represent the average removal rates for pregnant women, non-pregnant women, children under 5 and adult male humans represent the average time spent in the corresponding infectious compartments, while $\frac{1}{\mu_V}$ is the average mosquito lifespan. Where $r_P = (\Lambda_P + \mu_H + \pi_P)$, $r_W = (a_2 + \Lambda_W + \mu_H + \pi_W)$, $r_I = (a_0 + a_1 + \Lambda_I + \mu_H + \pi_I)$, $r_M = (\Lambda_M + \mu_H + \pi_M)$ are removal rates from $I_P$, $I_W$, $I_I$, and $I_M$ classes, respectively.

## 3. PERTURBATION METHOD

Perturbation methods are applied to study the malaria nonlinear dynamical system in (10) [22, 23]. This analysis applies regular perturbation theory to the malaria infection model, with the model parameters following established methodologies in infectious disease modeling [20]. We then introduce a small parameter $\varepsilon \ll 1$ and expand each variable around the MFE: $S_H^0 = N_H$, $S_V^0 = N_V$ as;

$$\begin{aligned} S_H &= N_H + \varepsilon S_H^1 + \varepsilon^2 S_H^2 + \mathcal{O}(\varepsilon^3), \\ I_P &= 0 + \varepsilon I_P^1 + \varepsilon^2 I_P^2 + \mathcal{O}(\varepsilon^3), \\ I_W &= 0 + \varepsilon I_W^1 + \varepsilon^2 I_W^2 + \mathcal{O}(\varepsilon^3), \\ I_I &= 0 + \varepsilon I_I^1 + \varepsilon^2 I_I^2 + \mathcal{O}(\varepsilon^3), \\ I_M &= 0 + \varepsilon I_M^1 + \varepsilon^2 I_M^2 + \mathcal{O}(\varepsilon^3), \\ S_V &= N_V + \varepsilon S_V^1 + \varepsilon^2 S_V^2 + \mathcal{O}(\varepsilon^3), \\ I_V &= 0 + \varepsilon I_V^1 + \varepsilon^2 I_V^2 + \mathcal{O}(\varepsilon^3). \end{aligned}$$

The term $\varepsilon = \frac{\theta_{VH}\times\theta_{HV}\times\varphi}{\Lambda}$, where $\varphi = \text{average}(\phi_P + \phi_W + \phi_I + \phi_M)$ which reflects the role of the number of mosquito bites and $\Lambda = \text{average}(\Lambda_P + \Lambda_W + \Lambda_I + \Lambda_M)$.

### 3.1. ZEROTH-ORDER ($\varepsilon^0$) EQUATIONS

Here, we substitute the expansions into each equation and collect terms of order $\varepsilon^0$ (terms with no $\varepsilon$ factor).
For the $S_H$ equation:

$$\begin{aligned} \frac{d}{dt}(N_H + \varepsilon S_H^1 + \cdots) = Z_H &+ \Lambda_P(\varepsilon I_P^1 + \cdots) + \Lambda_W(\varepsilon I_W^1 + \cdots) \\ &+ \Lambda_I(\varepsilon I_I^1 + \cdots) + \Lambda_M(\varepsilon I_M^1 + \cdots) \\ &- \frac{(\xi_1)}{N_H} - \mu_H(N_H + \varepsilon S_H^1 + \cdots). \end{aligned}$$

where $\xi_1 = \phi_P + \phi_W + \phi_I + \phi_M)\theta_{VH}(N_H + \varepsilon S_H^1 + \cdots)(\varepsilon I_V^1 + \cdots)$. By identifying $\varepsilon^0$ terms and collect $\varepsilon^0$ terms to obtain

$$0 = Z_H - \mu_H N_H$$

For the $I_P$ equation:

$$\begin{aligned} \frac{d}{dt}(\varepsilon I_P^1 + \cdots) = &\frac{\theta_{VH}\phi_P(N_H + \varepsilon S_H^1 + \cdots)(\varepsilon I_V^1 + \cdots)}{N_H} \\ &+ a_2(\varepsilon I_W^1 + \cdots) - (\Lambda_P + \mu_H + \pi_P)(\varepsilon I_P^1 + \cdots). \end{aligned}$$

All terms have at least one factor of $\varepsilon$, so the $\varepsilon^0$ equation is

$$0 = 0$$

Similarly, for $I_W$, $I_I$, $I_M$, and $I_V$, all terms have at least one factor of $\varepsilon$, giving $0 = 0$.
For the $S_V$ equation:

$$\frac{d}{dt}(N_V + \varepsilon S_V^1 + \cdots) = Z_V - \frac{\xi_2}{N_H} - \mu_V(N_V + \varepsilon S_V^1 + \cdots),$$

where $\xi_2 = \theta_{HV}(N_V + \varepsilon S_V^1 + \cdots)(\phi_P(\varepsilon I_P^1 + \cdots) + \phi_W(\varepsilon I_W^1 + \cdots) + \phi_I(\varepsilon I_I^1 + \cdots) + \phi_M(\varepsilon I_M^1 + \cdots))$. We identify terms with $\varepsilon^0$ and collect $\varepsilon^0$ terms, to obtain

$$0 = Z_V - \mu_V N_V.$$

In summary, the zeroth-order solution for the model in (10) is given as: $N_H = \frac{Z_H}{\mu_H}$ $I_P = I_W = I_I = I_M = I_V = 0$, and $N_V = \frac{Z_V}{\mu_V}$, which confirms that the zeroth-order values are the equilibrium values.

### 3.2. FIRST-ORDER ($\varepsilon^1$) EQUATIONS

We substitute into the perturbation expansions and collect $\varepsilon^1$ terms for each compartment, identifying terms that have exactly one factor of $\varepsilon$.

For the $S_H$ equation:

$$\varepsilon\frac{dS_H^1}{dt} = \varepsilon\Lambda_P I_P^1 + \varepsilon\Lambda_W I_W^1 + \varepsilon\Lambda_I I_I^1 + \varepsilon\Lambda_M I_M^1 - \varepsilon(\phi_P + \phi_W + \phi_I + \phi_M)\theta_{VH} I_V^1 - \varepsilon\mu_H S_H^1.$$

Dividing through by $\varepsilon$ gives

$$\frac{dS_H^1}{dt} = \Lambda_P I_P^1 + \Lambda_W I_W^1 + \Lambda_I I_I^1 + \Lambda_M I_M^1 - (\phi_P + \phi_W + \phi_I + \phi_M)\theta_{VH} I_V^1 - \mu_H S_H^1.$$

Similarly, the summary of the first-order equations is given as

$$\begin{aligned}
\frac{dS_H^1}{dt} &= \Lambda_P I_P^1 + \Lambda_W I_W^1 + \Lambda_I I_I^1 + \Lambda_M I_M^1 - (\phi_P + \phi_W + \phi_I + \phi_M)\theta_{VH} I_V^1 - \mu_H S_H^1,\\
\frac{dI_P^1}{dt} &= \theta_{VH}\phi_P I_V^1 + a_2 I_W^1 - (\Lambda_P + \mu_H + \pi_P) I_P^1,\\
\frac{dI_W^1}{dt} &= \theta_{VH}\phi_W I_V^1 + a_0 I_I^1 - (a_2 + \Lambda_W + \mu_H + \pi_W) I_W^1,\\
\frac{dI_I^1}{dt} &= \theta_{VH}\phi_I I_V^1 - (a_0 + a_1 + \Lambda_I + \mu_H + \pi_I) I_I^1,\\
\frac{dI_M^1}{dt} &= \theta_{VH}\phi_M I_V^1 + a_1 I_I^1 - (\Lambda_M + \mu_H + \pi_M) I_M^1\\
\frac{dS_V^1}{dt} &= -\theta_{HV}\sigma(\phi_P I_P^1 + \phi_W I_W^1 + \phi_I I_I^1 + \phi_M I_M^1) - \mu_V S_V^1,\\
\frac{dI_V^1}{dt} &= \theta_{HV}\sigma(\phi_P I_P^1 + \phi_W I_W^1 + \phi_I I_I^1 + \phi_M I_M^1) - \mu_V I_V^1,
\end{aligned}$$

where $\sigma = \frac{N_V}{N_H}$. The first-order equations are linear and can be written in matrix form as $\frac{dX}{dt} = AX$ where $X = (S_H^1, I_P^1, I_W^1, I_I^1, I_M^1, S_V^1, I_V^1)^T$. Because of the complexity of the model, we focus on the infected compartments ($I_P^1$, $I_W^1$, $I_I^1$, $I_M^1$, $I_V^1$). Then the subsystem is given as:

$$\frac{dY}{dt} = MY, \quad Y = (I_P^1, I_W^1, I_I^1, I_M^1, I_V^1)^T,$$

$$M = \begin{bmatrix} -r_P & a_2 & 0 & 0 & \theta_{VH}\phi_P \\ 0 & -r_W & a_0 & 0 & \theta_{VH}\phi_W \\ 0 & 0 & -r_I & 0 & \theta_{VH}\phi_I \\ 0 & 0 & a_1 & -r_M & \theta_{VH}\phi_M \\ \frac{\theta_{HV}\phi_P N_V}{N_H} & \frac{\theta_{HV}\phi_W N_V}{N_H} & \frac{\theta_{HV}\phi_I N_V}{N_H} & \frac{\theta_{HV}\phi_M N_V}{N_H} & -\mu_V \end{bmatrix}.$$

For the computation of $Y(t) = e^{Mt}Y(0)$, three numerical approaches were applied to obtain the matrix exponential $e^{Mt}$: spectral decomposition $e^{Mt} = \sum_{i=1}^{5} e^{\lambda_i t}\frac{\prod_{j\neq i}(M-\lambda_j I)}{\prod_{j\neq i}(\lambda_i-\lambda_j)}$, where $\lambda_i$ are the eigenvalues of matrix $M$ satisfying the characteristic equation: $\det(M - \lambda I) = 0$, the Taylor-series definition $e^{Mt} = I + Mt + \frac{M^2t^2}{2!} + \frac{M^3t^3}{3!} + \frac{M^4t^4}{4!} + \cdots$ and the Laplace transform $Y(t) = \mathcal{L}^{-1}\{(sI - M)^{-1}\}Y(0)$ where $(sI - M)^{-1}$ is the resolvent matrix, and its inverse Laplace transform gives the matrix exponential.

### 3.3. SENSITIVITY OF THE MODEL PARAMETERS

The sensitivity index for the model is obtained by studying the influence of some parameters responsible for malaria transmission and its prevalence and determining how best to reduce malaria-related mortality and morbidity in humans [26, 27]. The normalized forward sensitivity index of any variable to a parameter is obtained by the ratio of the relative change in the variable to the relative change in the parameter, and the sensitivity index can be obtained using partial derivatives provided the variable is a differentiable function of the parameter [26]. The normalized forward sensitivity index of a variable, $\gamma$, that depends differentiably on a parameter, $P$, is stated as follows:

$$\gamma_p^{\mathcal{R}_0} = \frac{\partial\mathcal{R}_0}{\partial p}\cdot\frac{p}{\mathcal{R}_0}, \tag{11}$$

where $P$ represents the basic parameters.

Then the basic reproduction number $\mathcal{R}_0$ is redefined as: $\mathcal{R}_0 = \sqrt{K\cdot Q}$ , where

$$K = \frac{\theta_{VH}\theta_{HV}Z_V\mu_H}{Z_H(\mu_V)^2},$$

and

$$Q = \frac{\phi_P^2}{r_P} + \frac{\phi_W^2}{r_W} + \frac{\phi_I^2}{r_I} + \frac{\phi_M^2}{r_M} + \frac{a_2\phi_P\phi_W}{r_W r_P} + \frac{a_0 a_2\phi_I\phi_P}{r_P r_W r_I} + \frac{a_0\phi_W\phi_I}{r_I r_W} + \frac{a_1\phi_I\phi_M}{r_I r_M}.$$

The sensitivity index of the model is computed as

$$\Upsilon_p^{\mathcal{R}_0} = \frac{p}{2}\left[\frac{1}{K}\frac{\partial K}{\partial p} + \frac{1}{Q}\frac{\partial Q}{\partial p}\right]. \tag{12}$$

It follows that for parameters in $K$, its index is calculated thus $\frac{p}{2K}\times\frac{\partial K}{\partial P}$, parameters in $Q$, its index is calculated as $\frac{p}{2Q}\times\frac{\partial Q}{\partial P}$ and parameters in both $K$ and $Q$ are obtained from the combined contribution. Hence, $\Upsilon_{Z_H}^{\mathcal{R}_0} = \frac{Z_H}{2K}\times -\frac{K}{Z_H} = -\frac{1}{2}$, $\Upsilon_{Z_V}^{\mathcal{R}_0} = \frac{Z_V}{2K}\times\frac{K}{2K} = +\frac{1}{2}$, $\Upsilon_{\theta_{VH}}^{\mathcal{R}_0} = \frac{\theta_{VH}}{2K}\times\frac{K}{\theta_{VH}} = +\frac{1}{2}$, $\Upsilon_{\theta_{HV}}^{\mathcal{R}_0} = \frac{\theta_{HV}}{2K}\times\frac{K}{\theta_{HV}} = +\frac{1}{2}$, $\Upsilon_{\mu_V}^{\mathcal{R}_0} = \frac{\mu_V}{2K}\times -\frac{2K}{\mu_V} = -1$, $\Upsilon_{\mu_H}^{\mathcal{R}_0} = \frac{1}{2} + \frac{\mu_H}{2Q}\sum_j\frac{\partial Q}{\partial r_j}$, $\Upsilon_{\phi_P}^{\mathcal{R}_0} = \frac{\phi_P}{2Q}\times\frac{2\phi_P}{r_P} + \frac{a_2\phi_W}{r_W r_P} + \frac{a_0 a_2\phi_I}{r_P r_W r_I}$. Similarly, the sensitivity indices of the other parameters are obtained.

## 4. RESULT

### 4.1. PERTURBATION ANALYSIS RESULT

$\varphi$ (average number of bites) is 0.4225 and $\Lambda$ (average recovery rate) is 0.1268; therefore, $\varepsilon = 0.029987$. The zeroth-order equilibrium populations show that $N_H$ (total humans) is 364978, with $S_H$ at equilibrium satisfying $\frac{Z_H}{\mu_H} = \frac{17.935}{0.00004919} = 364978$. Similarly, $N_V$ (total vectors) is 434524, with $S_V$ at equilibrium satisfying $\frac{Z_V}{\mu_V} = \frac{17380.95}{0.04} = 434524$, and $\mathcal{R}_0 = 1.2809 > 1$. A

Python program was used to carry out the malaria model perturbation analysis and simulations using the parameter values in Table 1. The graphs below showed the comparison of full nonlinear Model, First-Order Perturbation $X = X^0 + \varepsilon X^1$ (to examine the exponential growth/decay, $\mathcal{R}_0$), and Zeroth-Order Solutions $X^0$ (malaria equilibrium point) while Table 2 showed the result of the different numerical method used in solving the first order equation.

### 4.2. SENSITIVITY ANALYSIS RESULT

Table 3 gives the summarized results of the sensitivity indices, employing the values of the model parameters in Table 1. The sign of each value in the table indicates whether the index is positive $(+)$, meaning that increasing the parameter increases $\mathcal{R}_0$, or negative $(-)$, meaning that increasing the parameter decreases $\mathcal{R}_0$. The magnitude represents the relative influence of each parameter on malaria infection in each human class.

## 5. DISCUSSION OF RESULTS

A $10\%$ rise in mosquito mortality $\mu_V$ would result in a $10\%$ decrease in $\mathcal{R}_0$. This implies that the mosquito death rate is the model's most significant parameter, and the basic reproduction number would drop by precisely $10\%$ for every $10\%$ rise in mosquito mortality. Human recruitment $Z_H$ with the sensitivity index $-0.5$ is inversely proportional to $\mathcal{R}_0$, while $Z_V$, $\theta_{VH}$, and $\theta_{HV}$ are proportional to $\mathcal{R}_0$, respectively.

The fractions of mosquito bites $\phi$'s and progression rates $a$'s have very small sensitivity indices, respectively, while malaria-induced death rates $\pi$'s have negligible negative indices, while the adult male recovery rate $\Lambda_M$ is highly influential, as evident in its index value. Therefore, parameters influencing mosquito survival $\mu_V$ and recovery from the $I_M$ class $\Lambda_M$ are the most sensitive to the model, indicating that to curtail the spread of malaria, they should be the main focus of intervention.

The perturbation results show that, at zeroth order, the system is at the disease-free equilibrium, where malaria is absent from the system. The first-order perturbation shows exponential growth, indicating that the disease will grow rapidly when introduced into the population. This indicates that the malaria-free equilibrium (MFE) is unstable; even small perturbations lead to disease spread, as shown in Figures 2–8.

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## DATA AVAILABILITY

The data are available with the corresponding author upon request.

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