

STABILITY ANALYSIS AND SEMI-ANALYTIC SOLUTION TO A SEIR-SEI MALARIA TRANSMISSION MODEL USING HE'S VARIATIONAL ITERATION METHOD

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Abstract:

We have considered a SEIR-SEI Vector-host mathematical model which captures malaria transmission dynamics, described and built on 7-dimensional nonlinear ordinary differential equations. We compute the basic reproduction number of the model; examine the positivity and boundedness of the model compartments in a region using well established methods viz: Cauchy's differential theorem, Birkhoff & Rota's theorem which verifies and reveals the well-posedness, and carrying capacity of the model respectively, the existence of the Disease-Free (DFE) and Endemic (EDE) equilibrium points were determined and examined

Using the Gaussian elimination method and the Routh-hurwitz criterion, we convey stability analyses at DFE and EDE points which indicates that the DFE (malaria-free) and the EDE (epidemic outbreak) point occurs when the basic reproduction number is less than unity (one) and greater than unity (one) respectively.

We obtain a solution to the model using the Variational iteration method (VIM) (an unprecedented method) to each population compartments and verify the efficacy, reliability and validity of the proposed method by comparing the respective solutions via tables and combined plots with the computer in-built Runge-kutta-Felhberg of fourth-fifths order (RKF-45).

We illustrate the combined plot profiles of each compartment in the model, showing the dynamic behavior of these compartments; then we speculate that VIM is efficient and capable to conduct analysis on Malaria models and other epidemiological models.

Keywords:

SEIR-SEI, Basic Reproduction number, Disease-Free equilibrium point (DFE), Endemic equilibrium point, Stability, Variational iteration method (VIM), Runge-Kutta-Fehlberg (RKF-45).

1. Introduction

Malaria is a mosquito-borne infectious disease that is life threatening to humans and other animals (Malaria fact sheet, 2014) [16]. This infectious disease is widely spread throughout the globe and predominantly present in tropical and sub-tropical regions of the earth including some parts of Europe.

The wide spread of this vector-borne disease (malaria) has urged numerous researchers and health organizations to study the epidemiology and transmission dynamics of the disease; so as to be able to implement an appropriate intervention strategy on its ubiquitous nature.

Because of its nature of being a fatal disease, this is why 25th of April is set aside as the world's annual malaria day for the global alertness against the disease. Malaria causes symptoms that typically include fever, tiredness, vomiting, and headaches (Caraballo, 2014). In severe cases it can cause yellow skin, seizures, coma, or death. (Caraballo, 2014) [15]. These symptoms usually begin ten to fifteen days after being bitten by an infected mosquito and if not properly treated, people may have recurrences of the disease months later (Malaria fact sheet W.H.O, 2014) [16].

Malaria is caused by single-celled microorganisms of the plasmodium group (Malaria Fact sheet W.H.O, 2014). The disease is most commonly spread by an infected female Anopheles mosquito. The mosquito bite introduces the parasites from the mosquito's saliva into the Host (Human). There are five different plasmodium species leading to malaria infection and disease among humans; These are: Plasmodium Falciparum (P. falciparum), Plasmodium vivax (P. vivax), Plasmodium Ovale (P. ovale), Plasmodium malariae (P. malariae), Plasmodium knowlesi (P. knowlesi) [8].

Most deaths are caused by *P. falciparum* as it is the most dangerous of all *Plasmodium* species [8, 13]. *P. vivax*, *P. ovale*, and *P. malariae* generally cause milder form of malaria while the *P. knowlesi* rarely cause disease in humans. This *P. falciparum* is mainly found in Africa as it is common and causing deaths worldwide. In addition, *Plasmodium knowlesi* is a type of malaria that infects macaques in Southeast Asia; also infect humans causing malaria that is transmitted from animal to human (zoonotic malaria) [8, 13-14].

WHO Malaria report (2013) shows that approximately 80% of malaria cases and 90% of deaths are estimated to occur in most countries of this sub-Saharan Africa [9]. In 2015, WHO estimates that 212 million clinical cases of malaria occurred and 429,000 people died of malaria, most of them were children in Africa [10]. The world Malaria Report in 2018 [38] shows an unprecedented period of success in global malaria control. An estimated 219 million cases of malaria occurred worldwide (95% confidence interval (CI): 203-262 million), compared with 239 million cases in 2010 (95% CI: 219-285 million) [11] and 217 million cases in 2016 (95% CI: 200-259 million) [12] with 92% cases in the African region, 5% in the South-East Asia region and 2% in the WHO Eastern Mediterranean region.

Very recently, in the common wealth malaria reports (April, 2019) [39]; a historic partnership of governments, civil society, the private sector and multilateral organizations, came together in London for a momentous malaria summit. Delivering US \$4.1 billion for the global malaria fight and two days later at the commonwealth heads of Government meeting (CHOGM), all 53 leaders committed to halve malaria in the commonwealth within five years.

The report here shows that the commonwealth countries: The Gambia, Belize, Bangladesh, India, Malaysia, Mozambique and Nigeria are already on a trajectory to achieve the target to halve malaria in 2023. See [39].

Due to the everyday attempt to control the epidemic and prevalent nature of malaria, several models have been developed by mathematicians; so as to understand the transmission dynamics of this infectious disease and implement a control strategy. Majority of these models are being described by differential equations of the nonlinear type. The first malaria model for malaria transmission and control was by Ronald Ross [4] which was later improved by Macdonald

(1957) [5] considering some biological assumptions. Since then, many models have been developed like Ngwa and Shu (1999) [18] Jia Li (2011) [3], Prashant Goswami et al (2012) [20], Olaniyi S and Obabiyi (2013) [2], Shah NH and Gupta. J (2013) [21], Hal-Feng Huo and Guang-ming Qiu(2014) [23], Altaf Khan et al (2015) [22], Oti eno (2016), Osman et al (2017) [1], Osman et al (2018) [24], Traore Bakare (2018) [7] to mention a few. Researchers and mathematicians have endeavored to proffer solution to these models including that of malaria via different methods so as to understand the transmission dynamics better in Nigar Ali et al. (2019) using Adomian Decomposition method [25], Abioye adesoye idowu et al (2018) using Differential transform method [28], Peter olumuyiwa james et al (2018) solved using Multi-step Homotopy analysis method [30]. Morufu oyedunsi olayiwola (2017) using the Variational iteration method solved a SEIRS epidemic model [26]. Yullita molliq Rangkuti (2014) obtained a numerical analytical solution of SIR model of Dengue fever disease in South sulawesi using HPM and VIM [19], Fazal Haq et al (2017) by Laplace Adomian decomposition method solved an epidemic model of a vector borne disease [17].

Of all the semi-analytical methods implemented to solve epidemic models including malaria, none have solved the malaria model using the variational iteration method and as a result, less attention has been paid using this method on malaria models. This method is unprecedented.

The main reason of this paper is to validate the efficiency of variational iteration method and also speculate its capability as alternative approach in solving and analyzing epidemiological models including malaria.

The huge advantage of this method over other methods include: the simplicity and straightforwardness, less computational stress or efforts of the method with no linearization of the nonlinear term, no computation of Adomian or He's polynomials, yet yielding highly accurate and rapidly convergent results devoid of errors when compared numerically and graphically.

In this research, we consider an existing SEIR model of Osman et al (2017), conduct a stability analysis, and then obtain semi-analytic solution via Variational iteration method (VIM).

The model presented here in this research is of two compartmental system of nonlinear ordinary differential equation involving the host which is the human and the Vector which is the mosquito. The human (host) is described by four differential equations and the mosquito by three differential equations.

The subsequent organization of this research work is structured as follows: Section 2 elucidates the compartmental model of the malaria transmission dynamics as well as the flow diagram of the model; Section 3 focuses on the mathematical analysis of the model which includes the analysis on the feasible region Γ of the model, so as to verify the epidemiological validity of the model; the disease-free equilibrium point (DFE), basic reproduction number, the endemic equilibrium point (EDE), stability of the DFE via Gaussian elimination method and the EDE with theorems, lemmas, and proofs were all computed here.

Semi-analytic solution was then proffered to the seven (7) compartments of the vector-host model using He's variational iteration method (VIM) in Section 4.

Lastly, numerical result comparison were made for the solved compartments via tables and combined plots of Runge-Kutta-Fehlberg 45 (RKF-45) and VIM, results were then interpreted and discussed before the final conclusion in section 5 and 6 respectively.

2. The Model

The model consists of two classes of population, the human population and the mosquito population. The human N population is subdivided into four compartments, the susceptible, the exposed, the infected, and the recovered. While the mosquito N population is subdivided into three compartments, the susceptible, the exposed, the infected as it is assumed that mosquitoes don't recover. We then have that the *SEIR* model for the humans (host) and the *SEI* model for the mosquito (vector). (**Table 1**)

2.1 Model Assumptions

The Population of the susceptible human $S_H(t)$ is increased by the recruitment of individuals at a rate Λ_H , and by the recovered individuals returning back to the compartment due to loss of immunity at a rate ρ , they acquire infection at a rate β_H , the population is then decreased by natural death of humans at a rate μ_H . (**Fig 1**) The population of the Exposed human $E_H(t)$ is generated by the infection of the susceptible individuals at a rate β_H , decreased by

humans whose infection has developed to the infectious compartment at a rate α_1 , and further decreased by natural death μ_H . (**Fig 2**)

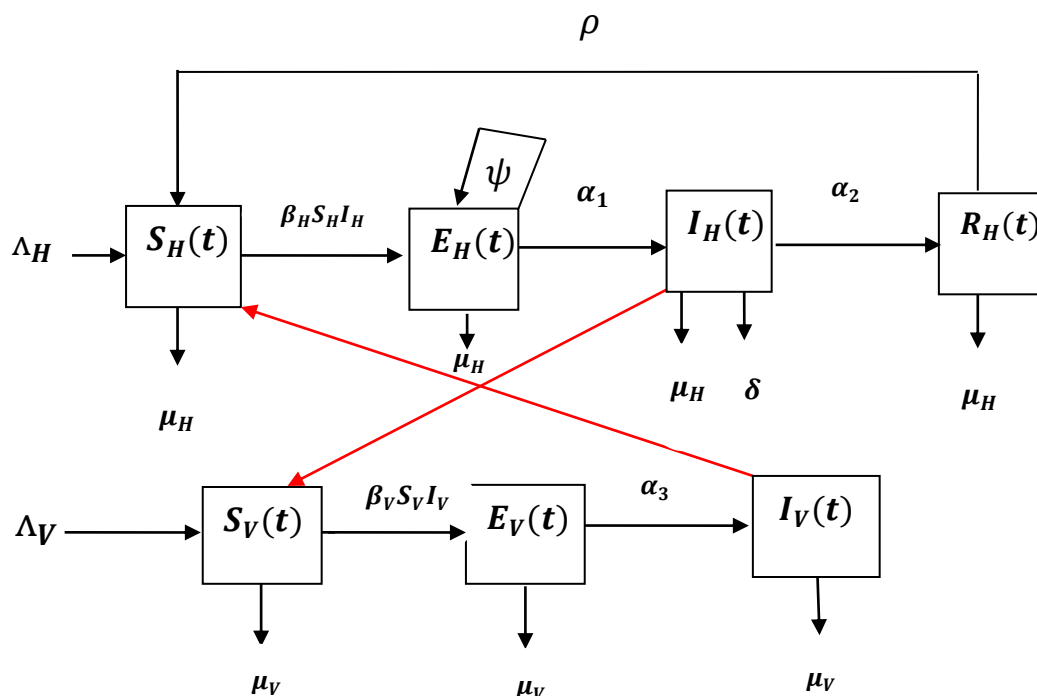
The population of the infected $I_H(t)$ is generated by humans who are infectious at a rate α_1 , increased by newborn baby with infection at rate ψ , then decreased by natural death μ_H , malaria induced death, and humans who have recovered at rates μ_H , δ , and α_2 respectively. (**Fig 3**)

The Recovered population $R_H(t)$ is generated by those who are infected but are being treated and recovering from malaria at a rate α_2 . It is then decreased by those who die naturally and lose their immunity at rates μ_H and ρ respectively. (**Fig 4**)

The susceptible mosquito population $S_V(t)$ is generated by the recruitment of mosquitoes into the compartment at a rate Λ_V , decreased by infection and death by natural cause with rates β_V and μ_V . (**Fig 5**)

The Exposed mosquito's population $E_V(t)$ is generated by susceptible mosquitoes exposed to the malaria pathogen infection at a rate β_V , decreased by mosquitoes that have developed into the infectious state, and by natural cause at rates α_3 , and μ_V . (**Fig 6**)

The Infected mosquito's population $I_V(t)$ is generated by exposed mosquito whose state has moved to the infectious state at the rate α_3 , and decreased by natural cause μ_V . (**Fig 7**)



$$\begin{aligned}\frac{dS_H(t)}{dt} &= \Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H \\ \frac{dE_H(t)}{dt} &= \beta_H S_H I_H - (\alpha_1 + \mu_H) E_H \\ \frac{dI_H(t)}{dt} &= \alpha_1 E_H - (\alpha_2 + \mu_H + \delta) I_H + \psi I_H \\ \frac{dR_H(t)}{dt} &= \alpha_2 I_H - (\mu_H + \rho) R_H \\ \frac{dS_V(t)}{dt} &= \Lambda_V - \beta_V S_V I_V - \mu_V S_V \\ \frac{dE_V(t)}{dt} &= \beta_V S_V I_V - (\alpha_3 + \mu_V) E_V \\ \frac{dI_V(t)}{dt} &= \alpha_3 E_V - \mu_V I_V\end{aligned}\tag{1}$$

Table 1: State Variables and parameter description of the SEIR-SEI model

State Variables	Description
$S_H(t)$	Susceptible Human at time t
$E_H(t)$	Exposed Human at time t
$I_H(t)$	Infected Human at time t
$R_H(t)$	Recovered Human at time t
$S_V(t)$	Susceptible mosquito at time t
$E_V(t)$	Exposed mosquito at time t
$I_V(t)$	Infected mosquito at time t
Parameters	Description
Λ_H	Recruitment rate of Humans
Λ_V	Recruitment rate of Mosquitoes

α_1	Development from exposure to being infectious (Humans)
α_2	Recovery rate of Humans
α_3	Development from exposure to being infectious (mosquito)
μ_H	Natural Death of Humans
μ_V	Natural Death of Mosquitoes
δ	Malaria induced death rate for Humans
q_H	Probability of transmission from an infectious mosquito to a susceptible Human
q_V	Probability of Transmission from an infectious Human to a susceptible mosquito
η_V	Mosquito Biting Rate
β_H	Infection rate of Humans($q_H \times \eta_V$)
β_V	Infection rate of Mosquito ($q_V \times \eta_V$)
ρ	Loss of Immunity for Humans
ψ	Newborn's birth with infection

3.0 MATHEMATICAL ANALYSIS OF THE MODEL

3.1 Positivity and Boundedness of Solution

Here, results are presented and verifications are made as to guarantee that the malaria model governed by the system (1) is epidemiologically and mathematically well-posed in a feasible region Γ ; given by: $\Gamma = \Gamma_H \times \Gamma_V \subset \Re_+^4 \times \Re_+^3$

Where,

$$\Gamma_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathfrak{R}_+^4 : S_H + E_H + I_H + R_H \leq \frac{\Lambda_H}{\mu_H}, S_H > 0, E_H \geq 0, I_H \geq 0, R_H \geq 0 \right\},$$

$$\Gamma_V = \left\{ (S_V, E_V, I_V) \in \mathfrak{R}_+^3 : S_V + E_V + I_V \leq \frac{\Lambda_V}{\mu_V}, S_V > 0, E_V \geq 0, I_V \geq 0 \right\}.$$

3.1.1 Theorem 1:

The feasible region of the system (1) given by

$$\Gamma = \left\{ (S_H, E_H, I_H, R_H, S_V, E_V, I_V) : S_H(t) + E_H(t) + I_H(t) + R_H(t) \leq \frac{\Lambda_H}{\mu_H}; S_V(t) + E_V(t) + I_V(t) \leq \frac{\Lambda_V}{\mu_V}; S_H > 0, \right. \\ \left. E_H \geq 0, I_H \geq 0, R_H \geq 0, S_V > 0, E_V \geq 0, I_V \geq 0 \right\}$$

is a positive invariant set and Bounded.

Proof: Let us consider the Host Population governed by the system

$$\begin{aligned} \frac{dS_H(t)}{dt} &= \Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H \\ \frac{dE_H(t)}{dt} &= \beta_H S_H I_H - (\alpha_1 + \mu_H) E_H \\ \frac{dI_H(t)}{dt} &= \alpha_1 E_H - (\alpha_2 + \mu_H + \delta) I_H + \psi I_H \\ \frac{dR_H(t)}{dt} &= \alpha_2 I_H - (\mu_H + \rho) R_H \end{aligned} \quad (2)$$

$N_H = S_H(t) + E_H(t) + I_H(t) + R_H(t)$ is the human net population.

Now from the derivatives of sums;

$$\frac{dN_H(t)}{dt} = \frac{dS_H(t)}{dt} + \frac{dE_H(t)}{dt} + \frac{dI_H(t)}{dt} + \frac{dR_H(t)}{dt} \quad (3)$$

This implies that,

$$\begin{aligned} \frac{dN_H(t)}{dt} &= (\Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H) + (\beta_H S_H I_H - (\alpha_1 + \mu_H) E_H) \\ &+ (\alpha_1 E_H - (\alpha_2 + \mu_H + \delta) I_H + \psi I_H) + (\alpha_2 I_H - (\mu_H + \rho) R_H) \end{aligned}$$

Then,

$$\begin{aligned} \frac{dN_H(t)}{dt} &= \Lambda_H - \mu_H (S_H + E_H + I_H + R_H) - (\delta - \psi) I_H \\ \therefore \frac{dN_H(t)}{dt} &= \Lambda_H - \mu_H N_H - (\delta - \psi) I_H \end{aligned}$$

This implies that $\frac{dN_H(t)}{dt} \leq \Lambda_H - \mu_H N_H$ when we remove the parameter $(\delta - \psi)I_H$.

$$\therefore \frac{dN_H(t)}{dt} + \mu_H N_H \leq \Lambda_H \quad (4)$$

By solving the first order linear differential inequality (4) using integrating factor method we have;

$$N_H(t) \leq \frac{\Lambda_H}{\mu_H} + p e^{-\mu_H t} \quad (5)$$

Where p is a constant of integration.

Then by applying Birkhoff and Rota's theorem [31] on the differential inequality (5), it follows that

$$\lim_{t \rightarrow \infty} N_H(t) \leq \frac{\Lambda_H}{\mu_H} \quad (6)$$

This is commonly known as the carrying capacity of the system and hence shows Boundedness.

It then follows that $N_H(t) = \left[\{S_H(t) + E_H(t) + I_H(t) + R_H(t)\} \in \Re_+^4 \leq \frac{\Lambda_H}{\mu_H} \right]$

This proves the boundedness of the solution inside the region Γ_H

Now for other classes of the population we have;

3.1.2 Other Compartments

We consider the rate of change of the population in the Susceptible Human compartment

$$\begin{aligned} \frac{dS_H(t)}{dt} &= \Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H \\ \Rightarrow \frac{dS_H(t)}{dt} + (\mu_H + \beta_H I_H) S_H &= \Lambda_H + \rho R_H \end{aligned} \quad (7)$$

Let $(\mu_H + \beta_H I_H) = \phi_H$,

Then, $\frac{dS_H(t)}{dt} + \phi_H S_H = \Lambda_H + \rho R_H$

We then can write that

$$\frac{dS_H(t)}{dt} + \phi_H S_H > 0$$

$$(9)$$

By separation of variables we obtain that;

$$\int \frac{dS_H(t)}{S_H} > \int -\phi_H dt \quad (10)$$

$$\Rightarrow \ln(S_H(t)) > -\phi_H t + c$$

$$\therefore S_H(t) > e^{-\phi_H t} \cdot e^c$$

Let $A = e^c$ then we have; $S_H(t) > Ae^{-\phi_H t}$

At the initial state when $t = 0$, $S_H(0) > A > 0$.

$\therefore S_H(t) = S_H(0)e^{-\phi_H t} = Ae^{-\phi_H t} > 0$ Holds and this implies that $S_H(t) > 0$ holds.

Indicating that $S_H(t)$ stays and remains positive.

Similarly, we consider the non-linear ODE for the exposed human

$$\frac{dE_H(t)}{dt} = \beta_H S_H I_H - (\alpha_1 + \mu_H) E_H$$

$$\therefore \frac{dE_H(t)}{dt} + (\alpha_1 + \mu_H) E_H = \beta_H S_H I_H \quad (11)$$

From $\beta_H S_H I_H$ on the right hand side of the equation (11), we have that $S_H > 0, S_H \neq 0$ from

our previous proof. Now for $I_H \geq 0$ we have that $\frac{dE_H(t)}{dt} + (\alpha_1 + \mu_H) E_H = 0$ when $I_H = 0$ and

$$\frac{dE_H(t)}{dt} + (\alpha_1 + \mu_H) E_H > 0 \text{ when } I_H > 0;$$

$$\therefore \frac{dE_H(t)}{dt} + (\alpha_1 + \mu_H) E_H \geq 0 \quad (12)$$

Solving the differential inequality (12) using separation of variable

We have

$$\int \frac{dE_H(t)}{E_H} \geq \int -(\alpha_1 + \mu_H) dt$$

Let $\xi_H = (\alpha_1 + \mu_H)$

$$\Rightarrow \int \frac{dE_H(t)}{E_H} \geq \int -\xi_H dt$$

$$\therefore \ln(E_H) \geq -\xi_H t + c$$

$$\Rightarrow E_H(t) \geq e^{-\xi_H t} \cdot e^c$$

When $t = 0$, we have

$$E_H(0) \geq Ae^0 = A$$

$$\therefore E_H(0) = A \geq 0$$

$$\Rightarrow E_H(t) = E_H(0)e^{-\xi_H t} \geq 0$$

Hence, $E_H(t) \geq 0$ Holds.

Similarly, we consider the nonlinear differential equations of other state variables $I_H(t)$ and $R_H(t)$ of the Infected and the recovered class; we let $\gamma_H = (\alpha_2 + \mu_H + \delta)$ and $\varepsilon_H = (\mu_H + \rho)$ respectively and solve the differential inequalities $\frac{dI_H}{dt} + \gamma_H I_H \geq 0$, $\frac{dR_H}{dt} + \varepsilon_H R_H \geq 0$ with the initial conditions.

We obtain the solutions to the ODEs and we have that $I_H(t) \geq 0$ and $R_H(t) \geq 0$ hold respectively.

3.1.3 Mosquito Model (Vector)

We consider the governing equation of the vector (SEI) model which is the Mosquito.

$$\begin{aligned} \frac{dS_V(t)}{dt} &= \Lambda_V - \beta_V S_V I_V - \mu_V S_V \\ \frac{dE_V(t)}{dt} &= \beta_V S_V I_V - (\alpha_3 + \mu_V) E_V \\ \frac{dI_V(t)}{dt} &= \alpha_3 E_V - \mu_V I_V \end{aligned} \tag{14}$$

The total Population density gives

$$N_V(t) = S_V(t) + E_V(t) + I_V(t) \tag{15}$$

From **Cauchy's differential theorem**,

$$\frac{dN_v(t)}{dt} = \frac{\partial N_v(t)}{\partial S_v} \cdot \frac{dS_v}{dt} + \frac{\partial N_v(t)}{\partial E_v} \cdot \frac{dE_v}{dt} + \frac{\partial N_v(t)}{\partial I_v} \cdot \frac{dI_v}{dt} \quad (16)$$

We have that

$$\frac{\partial N_v(t)}{\partial S_v} = \frac{\partial N_v(t)}{\partial E_v} = \frac{\partial N_v(t)}{\partial I_v} = 1, \quad (17)$$

$$\therefore \frac{dN_v(t)}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt}$$

$$\therefore \frac{dN_v(t)}{dt} = (\Lambda_v - \beta_v S_v I_v - \mu_v S_v) + (\beta_v S_v I_v - (\alpha_3 + \mu_v) E_v) + (\alpha_3 E_v - \mu_v I_v) \quad (18)$$

We then have,

$$\therefore \frac{dN_v(t)}{dt} + \mu_v N_v \leq \Lambda_v \quad (19)$$

By solving the differential inequality by method of integrating factor and apply Birkhoff and Rota's theorem [31]

$$\lim_{t \rightarrow \infty} N_v(t) \leq \frac{\Lambda_v}{\mu_v} \quad (20)$$

It then follows that $N_v(t) = S_v(t) + E_v(t) + I_v(t) \leq \frac{\Lambda_v}{\mu_v}$

This proves boundedness.

Similarly as the Host model, $S_v(t) > 0$, $E_v(t) \geq 0$, $I_v(t) \geq 0$ holds for the mosquito population.

This completely proves our **theorem 1**.

3.2 Disease-Free equilibrium points and the Reproduction Number

The points at which the differential equation is equal to zero are referred to as the equilibrium points or steady-state solutions.

The model consists of just two equilibrium points which is the disease-free and the Endemic equilibrium points

The point or time at which the disease wiped out and the entire population is susceptible is the **Disease-free equilibrium point** while the point at which the disease persists in the population (Epidemic outbreak) is the **Endemic equilibrium point**.

At Equilibrium,

$$\frac{dS_H(t)}{dt} = \frac{dE_H(t)}{dt} = \frac{dI_H(t)}{dt} = \frac{dR_H(t)}{dt} = \frac{dS_V(t)}{dt} = \frac{dE_V(t)}{dt} = \frac{dI_V(t)}{dt} = 0 \quad (21)$$

By substituting (21) into the system of equations (1),

$$\begin{aligned} 0 &= \Lambda_H - \beta_H S_H^0 I_H^0 - \mu_H S_H^0 + \rho R_H^0 \\ 0 &= \beta_H S_H^0 I_H^0 - (\alpha_1 + \mu_H) E_H^0 \\ 0 &= \alpha_1 E_H^0 - (\alpha_2 + \mu_H + \delta) I_H^0 + \psi I_H^0 \\ 0 &= \alpha_2 I_H^0 - (\mu_H + \rho) R_H^0 \\ 0 &= \Lambda_V - \beta_V S_V^0 I_V^0 - \mu_V S_V^0 \\ 0 &= \beta_V S_V^0 I_V^0 - (\alpha_3 + \mu_V) E_V^0 \\ 0 &= \alpha_3 E_V^0 - \mu_V I_V^0 \end{aligned}$$

Then the DFE for the SEIR-SEI system is given by:

$$E^0 = (S_H^0, E_H^0, I_H^0, R_H^0, S_V^0, E_V^0, I_V^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0, 0 \right) \quad (22)$$

3.2.1 The Basic Reproduction Number R_0 of the SEIR-SEI Model of Malaria Transmission

An important concept of Epidemiological models is the basic reproduction number which is usually denoted by R_0 , this number is the average number of secondary infections in the $E_H(t)$ compartment, infected by an infectious individual in the $I_H(t)$ compartment in a completely susceptible population. The Reproduction number in this model would be calculated using the Next generation matrix method. Since our model is a vector-Host model, we define the Next generation as a square matrix 'G' in which the individual of type j which accounts for the infection using the reproduction number assuming that the population of type i is susceptible [21, 6].

The assumption that the population is susceptible implies that the reproduction number would be computed at DFE point. Since there are two classes of population, we have the 2×2 matrix

$$G = \begin{pmatrix} g_{11} & g_{12} \\ g_{21} & g_{22} \end{pmatrix} = \begin{pmatrix} 0 & R_{0V} \\ R_{0H} & 0 \end{pmatrix} \quad (23)$$

Let the reproduction number of the model be denoted by R_G .

From $|G - \lambda I| = 0$

where λ is an identity matrix.

$$\Rightarrow |G - \lambda I| = \lambda^2 - R_{0H}R_{0V} = 0 \quad (24)$$

$$\therefore \lambda = \sqrt{R_{0H}R_{0V}}$$

$$\Rightarrow R_G = \sqrt{R_{0H}R_{0V}} \quad (25)$$

From the human nonlinear system of ODEs;

$$\begin{aligned} \frac{dS_H(t)}{dt} &= \Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H \\ \frac{dE_H(t)}{dt} &= \beta_H S_H I_H - (\alpha_1 + \mu_H) E_H \\ \frac{dI_H(t)}{dt} &= \alpha_1 E_H - (\alpha_2 + \mu_H + \delta) I_H + \psi I_H \\ \frac{dR_H(t)}{dt} &= \alpha_2 I_H - (\mu_H + \rho) R_H \end{aligned}$$

Using the next generation matrix method,

Let

$$X = (E_H, I_H, S_H, R_H)^T \quad (26)$$

Then

$$X'_H = \frac{dX_H}{dt} = \begin{bmatrix} \frac{dE_H}{dt} \\ \frac{dI_H}{dt} \\ \frac{dS_H}{dt} \\ \frac{dR_H}{dt} \end{bmatrix} = \begin{bmatrix} \beta_H S_H I_H - (\alpha_1 + \mu_H) E_H \\ \alpha_1 E_H - (\alpha_2 + \mu_H + \delta) I_H + \psi I_H \\ \Lambda_H - \beta_H S_H I_H - \mu_H S_H + \rho R_H \\ \alpha_2 I_H - (\mu_H + \rho) R_H \end{bmatrix} \quad (27)$$

By splitting the matrix in the equation (27) we have;

$$\frac{dX_H}{dt} = \begin{bmatrix} \beta_H S_H I_H \\ 0 \\ 0 \\ 0 \end{bmatrix} - \begin{bmatrix} (\alpha_1 + \mu_H)E_H \\ -\alpha_1 E_H + (\alpha_2 + \mu_H + \delta)I_H - \psi I_H \\ -\Lambda_H + \beta_H S_H I_H + \mu_H S_H - \rho R_H \\ -\alpha_2 I_H + (\mu_H + \rho)R_H \end{bmatrix} \quad (28)$$

This is now in the form

$$\frac{dX_H}{dt} = F_i(X) - V_i(X) \quad (29)$$

$$F_i(X) = \begin{bmatrix} \beta_H S_H I_H \\ 0 \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{bmatrix}; V_i(X) = \begin{bmatrix} (\alpha_1 + \mu_H)E_H \\ -\alpha_1 E_H + (\alpha_2 + \mu_H + \delta)I_H - \psi I_H \\ -\Lambda_H + \beta_H S_H I_H + \mu_H S_H - \rho R_H \\ -\alpha_2 I_H + (\mu_H + \rho)R_H \end{bmatrix} = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{bmatrix}$$

Where $F_i(X)$ is the matrix of new infections and $V_i(X)$ is the matrix of other transfer terms [6]

The next step here is to linearize the matrix $F_i(X)$ and $V_i(X)$ by taking the jacobian of each term in the matrices at Disease free equilibrium point .

Let $J[F_i(X)] = F_H$ and $J[V_i(X)] = V_H$

$$\therefore F_H = \frac{\partial F_i(X)}{\partial X_j}, V_H = \frac{\partial V_i(X)}{\partial X_j} \quad \text{At DFE } F_H(E^0) = \frac{\partial F_i(E^0)}{\partial X_j}, V_H(E^0) = \frac{\partial V_i(E^0)}{\partial X_j}$$

$$\frac{\partial F_i(E^0)}{\partial X_j} = \begin{pmatrix} \frac{\partial F_1(E^0)}{\partial E_H} & \frac{\partial F_1(E^0)}{\partial I_H} & \frac{\partial F_1(E^0)}{\partial S_H} & \frac{\partial F_1(E^0)}{\partial R_H} \\ \frac{\partial F_2(E^0)}{\partial E_H} & \frac{\partial F_2(E^0)}{\partial I_H} & \frac{\partial F_2(E^0)}{\partial S_H} & \frac{\partial F_2(E^0)}{\partial R_H} \\ \frac{\partial F_3(E^0)}{\partial E_H} & \frac{\partial F_3(E^0)}{\partial I_H} & \frac{\partial F_3(E^0)}{\partial S_H} & \frac{\partial F_3(E^0)}{\partial R_H} \\ \frac{\partial F_4(E^0)}{\partial E_H} & \frac{\partial F_4(E^0)}{\partial I_H} & \frac{\partial F_4(E^0)}{\partial S_H} & \frac{\partial F_4(E^0)}{\partial R_H} \end{pmatrix}; \frac{\partial V_i(E^0)}{\partial X_j} = \begin{pmatrix} \frac{\partial V_1(E^0)}{\partial E_H} & \frac{\partial V_1(E^0)}{\partial I_H} & \frac{\partial V_1(E^0)}{\partial S_H} & \frac{\partial V_1(E^0)}{\partial R_H} \\ \frac{\partial V_2(E^0)}{\partial E_H} & \frac{\partial V_2(E^0)}{\partial I_H} & \frac{\partial V_2(E^0)}{\partial S_H} & \frac{\partial V_2(E^0)}{\partial R_H} \\ \frac{\partial V_3(E^0)}{\partial E_H} & \frac{\partial V_3(E^0)}{\partial I_H} & \frac{\partial V_3(E^0)}{\partial S_H} & \frac{\partial V_3(E^0)}{\partial R_H} \\ \frac{\partial V_4(E^0)}{\partial E_H} & \frac{\partial V_4(E^0)}{\partial I_H} & \frac{\partial V_4(E^0)}{\partial S_H} & \frac{\partial V_4(E^0)}{\partial R_H} \end{pmatrix}$$

For the Reproduction number, we only need terms in the Exposed and the infected compartments [27].

Then we have the matrix

$$F_H = \begin{pmatrix} 0 & \beta_H \frac{\Lambda_H}{\mu_H} \\ 0 & 0 \end{pmatrix}; V_H = \begin{pmatrix} (\alpha_1 + \mu_H) & 0 \\ -\alpha_1 & (\alpha_2 + \mu_H + \delta - \psi) \end{pmatrix}$$

R_{0H} Is the spectral radius or dominant Eigen value of $(F_H V_H^{-1})$ that is $|(F_H V_H^{-1}) - \lambda I| = 0$; I is an identity matrix.

By computing the spectral radius, the reproduction number is given as;

$$R_{0H} = \frac{\alpha_1 \beta_H \Lambda_H}{\mu_H (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi)} \quad (30)$$

Similarly, by considering the nonlinear system in the Mosquito's model

$$\begin{aligned} \frac{dS_V(t)}{dt} &= \Lambda_V - \beta_V S_V I_V - \mu_V S_V \\ \frac{dE_V(t)}{dt} &= \beta_V S_V I_V - (\alpha_3 + \mu_V) E_V \\ \frac{dI_V(t)}{dt} &= \alpha_3 E_V - \mu_V I_V \end{aligned}$$

Similarly, using the Next generation matrix approach on the vectors system of equations above we have the Mosquito's reproduction number as

$$R_{0V} = \frac{\alpha_3 \beta_V \Lambda_V}{\mu_V (\alpha_3 + \mu_V)} \quad (31)$$

From equation (25) we have that $R_G = \sqrt{R_{0H} R_{0V}}$ then by putting the equation (30) and (31) into (25) we have the general reproduction number of the SEIR-SEI system as:

$$R_G = \sqrt{\frac{\alpha_1 \alpha_3 \beta_H \beta_V \Lambda_H \Lambda_V}{\mu_H \mu_V^2 (\alpha_1 + \mu_H) (\alpha_3 + \mu_V) (\alpha_2 + \mu_H + \delta - \psi)}} \quad (32)$$

This gives the reproduction number of the complete system

By alternative notations, if we let

$$\begin{aligned} (\alpha_1 + \mu_H) &= \xi_H \\ (\alpha_3 + \mu_V) &= \xi_V \\ (\alpha_2 + \mu_H + \delta - \psi) &= \gamma_H \end{aligned} \quad (33)$$

Then,

$$R_G = \sqrt{\frac{\alpha_1 \alpha_3 \beta_H \beta_V \Lambda_H \Lambda_V}{\mu_H \mu_V^2 \xi_H \xi_V \gamma_H}} \quad (33)$$

$$\Rightarrow R_G^2 = \frac{\alpha_1 \alpha_3 \beta_H \beta_V \Lambda_H \Lambda_V}{\mu_H \mu_V^2 \xi_H \xi_V \gamma_H} \quad (34)$$

3.3 Existence of the Endemic Equilibrium Points

The SEI-SEI model of Malaria transmission possesses an endemic equilibrium point

$$E^* = (S_H^*, E_H^*, I_H^*, R_H^*, S_V^*, E_V^*, I_V^*) \quad (35)$$

At this point, there is persistence of the disease in the system and hence an epidemic outbreak.

At equilibrium,

$$\frac{dS_H(t)}{dt} = \frac{dE_H(t)}{dt} = \frac{dI_H(t)}{dt} = \frac{dR_H(t)}{dt} = \frac{dS_V(t)}{dt} = \frac{dE_V(t)}{dt} = \frac{dI_V(t)}{dt} = 0$$

Then,

$$\begin{aligned} \Lambda_H - \beta_H S_H^* I_H^* - \mu_H S_H^* + \rho R_H^* &= 0 \\ \beta_H S_H^* I_H^* - (\alpha_1 + \mu_H) E_H^* &= 0 \\ \alpha_1 E_H^* - (\alpha_2 + \mu_H + \delta - \psi) I_H^* &= 0 \\ \alpha_1 I_H^* - (\mu_H + \rho) R_H^* &= 0 \\ \Lambda_V - \beta_V S_V^* I_V^* - \mu_V S_V^* &= 0 \\ \beta_V S_V^* I_V^* - (\alpha_1 + \mu_V) E_V^* &= 0 \\ \alpha_3 E_V^* - \mu_V I_V^* &= 0 \end{aligned} \quad (36)$$

We solve the system of equation (36) simultaneously for the corresponding endemic point

s. From $0 = \alpha_1 E_H^* - (\alpha_2 + \mu_H + \delta) I_H^* + \psi I_H^*$ in the system, we can write that $\alpha_1 E_H^* = (\alpha_2 + \mu_H + \delta - \psi) I_H^*$ Thus we have

$$E_H^* = \frac{(\alpha_2 + \mu_H + \delta - \psi) I_H^*}{\alpha_1} \quad (37)$$

Put (37) into $\frac{dS_H(t)}{dt}$ we have the relation,

$$\beta_H S_H I_H - \frac{(\alpha_1 + \mu_H)(\alpha_2 + \mu_H + \delta - \psi) I_H}{\alpha_1} = 0 \quad (38)$$

This implies that

$$\begin{aligned} I_H \left[\beta_H S_H - \frac{(\alpha_1 + \mu_H)(\alpha_2 + \mu_H + \delta - \psi)}{\alpha_1} \right] &= 0 \text{ Where } I_H \neq 0 \\ \Rightarrow \beta_H S_H - \frac{(\alpha_1 + \mu_H)(\alpha_2 + \mu_H + \delta - \psi)}{\alpha_1} &= 0; \\ S_H^* &= \frac{(\alpha_1 + \mu_H)(\alpha_2 + \mu_H + \delta - \psi)}{\beta_H \alpha_1} \end{aligned} \quad (39)$$

Again from $\frac{dR_H(t)}{dt}$, we have

$$R_H^* = \frac{\alpha_2 I_H^*}{(\mu_H + \rho)} \quad (40)$$

By substituting (39) and (40) into $\frac{dS_H(t)}{dt}$ and solving accordingly we have;

$$I_H^* = \frac{\alpha_1 \beta_H \Lambda_H (\mu_H + \rho) - \mu_H (\mu_H + \rho) (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi)}{\beta_H (\mu_H + \rho) (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi) - \beta_H \rho \alpha_1 \alpha_2} \quad (41)$$

Similarly by solving the system (36) appropriately, we obtain the endemic point

$$E_H^* = \frac{(\alpha_2 + \mu_H + \delta - \psi) [\alpha_1 \beta_H \Lambda_H (\mu_H + \rho) - \mu_H (\mu_H + \rho) (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi)]}{\alpha_1 \beta_H [(\mu_H + \rho) (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi) - \rho \alpha_1 \alpha_2]} \quad (42)$$

$$R_H^* = \frac{\alpha_2 \alpha_1 \beta_H \Lambda_H - \mu_H \alpha_2 (\alpha_1 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi)}{\beta_H [(\mu_H + \rho) (\alpha_2 + \mu_H) (\alpha_2 + \mu_H + \delta - \psi) - \alpha_1 \alpha_2 \rho]} \quad (43)$$

$$S_V^* = \frac{\mu_V (\alpha_3 + \mu_V)}{\beta_V \alpha_3} \quad (44)$$

$$E_V^* = \frac{\alpha_3 \beta_V \Lambda_V - \mu_V^2 (\alpha_3 + \mu_V)}{\alpha_3 \beta_V (\alpha_3 + \mu_V)} \quad (45)$$

$$I_V^* = \frac{\Lambda_V \beta_V \alpha_3 - \mu_V^2 (\alpha_3 + \mu_V)}{\mu_V \beta_V (\alpha_3 + \mu_V)} \quad (46)$$

3.4 Stability of the Disease-Free Equilibrium

We now check for the stability of the model at DFE by taking the jacobian of the seven dimensional ODES in equation (1) and obtaining its corresponding Eigen values.

The SEIR-SEI is stable if all of the Eigen values obtained from the linearized system are negative real values.

We have the jacobian of the model to be given as:

$$J(S_H, E_H, I_H, R_H, S_V, E_V, I_V) = \begin{pmatrix} -\beta_H I_H - \mu_H & 0 & -\beta_H S_H & \rho & 0 & 0 & 0 \\ \beta_H I_H & -(\alpha_1 + \mu_H) & \beta_H S_H & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & -(\alpha_2 + \mu_H + \delta - \psi) & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_2 & -(\mu_H + \rho) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_V I_V - \mu_V & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_V I_V & -(\alpha_3 + \mu_V) & \beta_V S_V \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 & -\mu_V \end{pmatrix} \quad (47)$$

At Disease-Free equilibrium point,

$$J(E^0) = \begin{pmatrix} -\mu_H & 0 & -\beta_H \frac{\Lambda_H}{\mu_H} & \rho & 0 & 0 & 0 \\ 0 & -(\alpha_1 + \mu_H) & \beta_H \frac{\Lambda_H}{\mu_H} & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & -(\alpha_2 + \mu_H + \delta - \psi) & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_2 & -(\mu_H + \rho) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_V & 0 & -\beta_V \frac{\Lambda_V}{\mu_V} \\ 0 & 0 & 0 & 0 & 0 & -(\alpha_3 + \mu_V) & \beta_V \frac{\Lambda_V}{\mu_V} \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 & -\mu_V \end{pmatrix} \quad (48)$$

By inserting our alternative notation

Let

$$\begin{aligned}
(\alpha_1 + \mu_H) &= \xi_H; & K_1 &= \beta_H \frac{\Lambda_H}{\mu_H}; \\
(\alpha_3 + \mu_V) &= \xi_V; & K_2 &= \beta_V \frac{\Lambda_V}{\mu_V}; \\
(\alpha_2 + \mu_H + \delta - \psi) &= \gamma_H; \\
(\mu_H + \rho) &= \varepsilon_H;
\end{aligned} \tag{49}$$

We have,

$$J(E^0) = \begin{pmatrix} -\mu_H & 0 & -K_1 & \rho & 0 & 0 & 0 \\ 0 & -\xi_H & K_1 & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & -\gamma_H & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_2 & -\varepsilon_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_V & 0 & -K_2 \\ 0 & 0 & 0 & 0 & 0 & -\xi_V & K_2 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 & -\mu_V \end{pmatrix} \tag{50}$$

For the Eigen-values of the matrix,

$$|J(E^0) - \lambda I| = \begin{vmatrix} -\mu_H - \lambda & 0 & -K_1 & \rho & 0 & 0 & 0 \\ 0 & -\xi_H - \lambda & K_1 & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & -\gamma_H - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_2 & -\varepsilon_H - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_V - \lambda & 0 & -K_2 \\ 0 & 0 & 0 & 0 & 0 & -\xi_V - \lambda & K_2 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 & -\mu_V - \lambda \end{vmatrix} \tag{51}$$

Now by applying some matrix techniques on equation (51), it is clear that the first column of (51) contains a diagonal term ‘ $-\mu_H - \lambda$ ’ only. Hence, $\lambda_1 = -\mu_H$ and we eliminate the first row and column in (51) to have a new matrix $J_0(E^0)$

$$J_0(E^0) = \begin{pmatrix} -\xi_H - \lambda & K_1 & 0 & 0 & 0 & 0 \\ \alpha_1 & -\gamma_H - \lambda & 0 & 0 & 0 & 0 \\ 0 & \alpha_2 & -\varepsilon_H - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu_V - \lambda & 0 & -K_2 \\ 0 & 0 & 0 & 0 & -\xi_V - \lambda & K_2 \\ 0 & 0 & 0 & 0 & \alpha_3 & -\mu_V - \lambda \end{pmatrix} \tag{52}$$

It also clear from (52) that the third and fourth column contains only diagonal terms ‘ $-\mu_v - \lambda$ ’ and ‘ $-\varepsilon_H - \lambda$ ’ which produces two Eigen values $\lambda_2 = -\mu_v$ and $\lambda_3 = -\varepsilon_H$. Hence we eliminate the third and fourth rows and columns so as to have a new matrix $J_1(E^0)$.

$$\therefore J_1(E^0) = \begin{pmatrix} -\xi_H - \lambda & K_1 & 0 & 0 \\ \alpha_1 & -\gamma_H - \lambda & 0 & 0 \\ 0 & 0 & -\xi_v - \lambda & K_2 \\ 0 & 0 & \alpha_3 & -\mu_v - \lambda \end{pmatrix} \quad (53)$$

$$J_1(E^0) = \begin{pmatrix} -\xi_H - \lambda & K_1 & 0 & 0 \\ \alpha_1 & -\gamma_H - \lambda & 0 & 0 \\ 0 & 0 & -\xi_v - \lambda & K_2 \\ 0 & 0 & \alpha_3 & -\mu_v - \lambda \end{pmatrix} = \begin{pmatrix} -\xi_H & K_1 & 0 & 0 \\ \alpha_1 & -\gamma_H & 0 & 0 \\ 0 & 0 & -\xi_v & K_2 \\ 0 & 0 & \alpha_3 & -\mu_v \end{pmatrix} - \begin{pmatrix} \lambda & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 \\ 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & \lambda \end{pmatrix} \quad (54)$$

Let

$$J_{1A}(E^0) = \begin{pmatrix} -\xi_H & K_1 & 0 & 0 \\ \alpha_1 & -\gamma_H & 0 & 0 \\ 0 & 0 & -\xi_v & K_2 \\ 0 & 0 & \alpha_3 & -\mu_v \end{pmatrix} \quad (55)$$

$$\therefore J_1(E^0) = |J_{1A}(E^0) - \lambda I| \quad (56)$$

By performing the following row transformation (Gaussian elimination method) on the matrix (55) with the operations,

$$R_4^* = R_4 + \frac{\alpha_3}{\xi_v} R_3;$$

$$R_2^* = R_2 + \frac{\alpha_1}{\xi_H} R_1;$$

We have the new Jacobian matrix

$$J_{1A}(E^0) = \begin{pmatrix} -\xi_H & K_1 & 0 & 0 \\ 0 & -\gamma_H + \frac{\alpha_1 K_1}{\xi_H} & 0 & 0 \\ 0 & 0 & -\xi_v & K_2 \\ 0 & 0 & 0 & -\mu_v + \frac{\alpha_3 K_2}{\xi_v} \end{pmatrix} \quad (57)$$

For the Eigen values,

$$\therefore J_1(E^0) = \begin{pmatrix} -\xi_H - \lambda & K_1 & 0 & 0 \\ 0 & \left(-\gamma_H + \frac{\alpha_1 K_1}{\xi_H}\right) - \lambda & 0 & 0 \\ 0 & 0 & -\xi_V - \lambda & K_2 \\ 0 & 0 & 0 & \left(-\mu_V + \frac{\alpha_3 K_2}{\xi_V}\right) - \lambda \end{pmatrix} \quad (58)$$

By applying the same matrix techniques which was applied on (51) accordingly and replacing alternative notations, we have the 7 Eigen values for the model as:

$$\begin{aligned} \lambda_1 &= -\mu_H, \lambda_2 = -\mu_V, \lambda_3 = -(\mu_H + \rho), \lambda_4 = -(\alpha_1 + \mu_H), \lambda_5 = -(\alpha_3 + \mu_V), \\ \lambda_6 &= -\left(\gamma_H - \frac{\alpha_1 \beta_H \Lambda_H}{\xi_H \mu_H}\right), \lambda_7 = -\left(\mu_V - \frac{\alpha_3 \beta_V \Lambda_V}{\xi_V \mu_V}\right) \end{aligned} \quad (59)$$

Clearly all our Eigen values are negative real values, and then the Disease-free equilibrium (DFE) is stable.

3.4.1 Theorem 2:

The Disease Free equilibrium (DFE) is locally asymptotically stable if $R_G < 1$.

Proof:

From the Eigen-values λ_6 and λ_7

$$\lambda_6 = -\left(\gamma_H - \frac{\alpha_1 \beta_H \Lambda_H}{\xi_H \mu_H}\right) = -\gamma_H \left(1 - \frac{\alpha_1 \beta_H \Lambda_H}{\xi_H \mu_H \gamma_H}\right) \quad (60)$$

$$\lambda_7 = -\left(\mu_V - \frac{\alpha_3 \beta_V \Lambda_V}{\xi_V \mu_V}\right) = -\mu_V \left(1 - \frac{\alpha_3 \beta_V \Lambda_V}{\xi_V \mu_V^2}\right) \quad (61)$$

Since $\frac{\alpha_1 \beta_H \Lambda_H}{\xi_H \mu_H \gamma_H} = R_{0H}; \frac{\alpha_3 \beta_V \Lambda_V}{\xi_V \mu_V^2} = R_{0V}$

Then,

$$\lambda_6 = -\gamma_H \left(1 - \frac{\alpha_1 \beta_H \Lambda_H}{\xi_H \mu_H \gamma_H}\right) = -\gamma_H (1 - R_{0H}) \quad (62)$$

$$\lambda_7 = -\mu_V \left(1 - \frac{\alpha_3 \beta_V \Lambda_V}{\xi_V \mu_V^2} \right) = -\mu_V (1 - R_{0V}) \quad (63)$$

Equation (62) and (63) above holds if and only if $R_{0H} < 1$ and $R_{0V} < 1$ holds respectively.

Thus, the DFE is stable if $R_{0H} < 1, R_{0V} < 1$.

From equation (26), $R_G = \sqrt{R_{0H} R_{0V}}$, which implies that $R_G^2 = R_{0H} R_{0V}$.

Then if

$$\begin{aligned} R_{0H} < 1, R_{0V} < 1 \\ \therefore R_G^2 = R_{0H} R_{0V} < 1 \\ \Rightarrow R_G < \sqrt{1} \end{aligned}$$

Showing that $R_G < 1$ holds ■

This proves theorem 2.

3.5 Stability of the Endemic Equilibrium Point

We evaluate $|J(E_H^*) - \lambda I| = 0, |J(E_V^*) - \lambda I| = 0$ for the Host and Vector respectively.

From the Human 4-dimensional differential equations we have the Jacobian as:

$$J(S_H, E_H, I_H, R_H) = \begin{pmatrix} -\beta_H I_H - \mu_H & 0 & -\beta_H S_H & -\rho \\ \beta_H I_H & -(\alpha_1 + \mu_H) & \beta_H S_H & 0 \\ 0 & \alpha_1 & -(\alpha_2 + \mu_H + \delta - \psi) & 0 \\ 0 & 0 & \alpha_2 & -(\mu_H + \rho) \end{pmatrix}$$

At Endemic points

$$J(S_H^*, E_H^*, I_H^*, R_H^*) = \begin{pmatrix} -\beta_H I_H^* - \mu_H & 0 & -\beta_H S_H^* & -\rho \\ \beta_H I_H^* & -(\alpha_1 + \mu_H) & \beta_H S_H^* & 0 \\ 0 & \alpha_1 & -(\alpha_2 + \mu_H + \delta - \psi) & 0 \\ 0 & 0 & \alpha_2 & -(\mu_H + \rho) \end{pmatrix} \quad (64)$$

By substituting equation (35) and alternative notations in (49)

$$|J(E_H^*) - \lambda I| = \begin{vmatrix} \left[\left(\frac{\mu_H \varepsilon_H \xi_H \gamma_H - \alpha_1 \beta_H \Lambda_H \varepsilon_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} - \mu_H \right) - \lambda \right] & 0 & -\frac{\xi_H \gamma_H}{\alpha_1} & -\rho \\ \left(\frac{\alpha_1 \beta_H \Lambda_H \varepsilon_H - \mu_H \varepsilon_H \xi_H \gamma_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right) & -\xi_H - \lambda & \frac{\xi_H \gamma_H}{\alpha_1} & 0 \\ 0 & \alpha_1 & -\gamma_H - \lambda & 0 \\ 0 & 0 & \alpha_2 & -\varepsilon_H - \lambda \end{vmatrix} = 0$$

The Characteristic equation for the above matrix gives

$$|J(E_H^*) - \lambda I| = Q_4 \lambda^4 + Q_3 \lambda^3 + Q_2 \lambda^2 + Q_1 \lambda + Q_0 \quad (65)$$

Where

$$Q_4 = 1;$$

$$Q_3 = \left[(\varepsilon_H + \xi_H + \gamma_H) - \left(\frac{\mu_H \alpha_1 \alpha_2 \rho - \alpha_1 \beta_H \Lambda_H \varepsilon_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right) \right];$$

$$Q_2 = \left[(\xi_H \gamma_H + \xi_H \varepsilon_H + \gamma_H \varepsilon_H + \xi_H \gamma_H) - (\varepsilon_H + \xi_H + \gamma_H) \left(\frac{\mu_H \alpha_1 \alpha_2 \rho - \alpha_1 \beta_H \Lambda_H \varepsilon_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right) \right];$$

$$Q_1 = \left(\frac{\xi_H \gamma_H \alpha_1 \beta_H \Lambda_H \varepsilon_H - \mu_H \varepsilon_H \xi_H^2 \gamma_H^2}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right) - (\xi_H \gamma_H + \xi_H \varepsilon_H + \gamma_H \varepsilon_H + \xi_H \gamma_H) \left(\frac{\mu_H \alpha_1 \alpha_2 \rho - \alpha_1 \beta_H \Lambda_H \varepsilon_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right);$$

$$Q_0 = \left[\frac{\xi_H \gamma_H \alpha_1 \beta_H \Lambda_H \varepsilon_H^2 - \mu_H \varepsilon_H^2 \xi_H^2 \gamma_H^2}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} + \frac{\rho \alpha_1 \alpha_2 \beta_H \Lambda_H \varepsilon_H - \rho \mu_H \varepsilon_H \xi_H \gamma_H \alpha_1 \alpha_2}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right] \quad (66)$$

Using the Routh-Hurwitz criterion for quartic polynomials, the characteristic equation (65) yields negative Eigen-values (**stable**) if and only if: $Q_4 > 0, Q_3 > 0, Q_2 > 0, Q_1 > 0, Q_0 > 0$ holds and this clearly seen. Also from this criterion, the characteristic equation has negative eigen values

(Stable) if the inequalities:

$$Q_3Q_2 - Q_1 > 0, Q_3Q_2Q_1 - Q_1^2 - Q_3^2Q_0 > 0, Q_3Q_2Q_1Q_0 - Q_4Q_1^2Q_0 > 0 \text{ are}$$

satisfied. This is trivial as well and has been verified; hence the endemic equilibrium is stable and we can hence have the Lemma.

3.5.1 **Lemma 3:**

The endemic equilibrium E_H^* of the SEIR-SEI vector host malaria transmission model is locally asymptotically stable if the inequalities

$$Q_3Q_2 - Q_1 > 0, Q_3Q_2Q_1 - Q_1^2 - Q_3^2Q_0 > 0, Q_3Q_2Q_1Q_0 - Q_4Q_1^2Q_0 > 0 \text{ are satisfied}$$

3.5.2 **Lemma 4:**

The positive equilibrium E_H^* of Human is locally asymptotically stable if $R_G > 1$.

Proof:

Using the Routh- Hurwitz criterion in the Characteristic polynomial in equation (2), we have that

$$Q_4 > 0, Q_3 > 0, Q_2 > 0, Q_1 > 0, Q_0 > 0 \text{ must hold.}$$

Here,

$$Q_0 = \left[\frac{\xi_H \gamma_H \alpha_1 \beta_H \Lambda_H \varepsilon_H^2 - \mu_H \varepsilon_H^2 \xi_H^2 \gamma_H^2}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} + \frac{\rho \alpha_1 \alpha_2 \beta_H \Lambda_H \varepsilon_H - \rho \mu_H \varepsilon_H \xi_H \gamma_H \alpha_1 \alpha_2}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right]$$

By mathematical manipulations of Q_0 in terms of the human reproduction number, we have

$$Q_0 = \left[\frac{\mu_H \varepsilon_H^2 \xi_H^2 \gamma_H^2 + \alpha_1 \alpha_2 \rho \varepsilon_H \xi_H \gamma_H \mu_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right] \left(\frac{\alpha_1 \beta_H \Lambda_H}{\mu_H \xi_H \gamma_H} - 1 \right) \quad (67)$$

$$\therefore Q_0 = \left[\frac{\mu_H \varepsilon_H^2 \xi_H^2 \gamma_H^2 + \alpha_1 \alpha_2 \rho \varepsilon_H \xi_H \gamma_H \mu_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right] (R_{0H} - 1) > 0$$

$$\Rightarrow \left[\frac{\mu_H \varepsilon_H^2 \xi_H^2 \gamma_H^2 + \alpha_1 \alpha_2 \rho \varepsilon_H \xi_H \gamma_H \mu_H}{\varepsilon_H \xi_H \gamma_H - \alpha_1 \alpha_2 \rho} \right] (R_{0H} - 1) > 0 \quad (68)$$

The Inequality (68) will hold if and only if $R_{0H} > 1$. This Proves the Lemma 4 ■

Similarly for the Vector Population (Mosquitoes), considering the Vector system of differential equations

$$\begin{aligned}\frac{dS_V(t)}{dt} &= \Lambda_V - \beta_V S_V I_V - \mu_V S_V \\ \frac{dE_V(t)}{dt} &= \beta_V S_V I_V - (\alpha_3 + \mu_V) E_V \\ \frac{dI_V(t)}{dt} &= \alpha_3 E_V - \mu_V I_V\end{aligned}$$

By taking the Jacobian at the Endemic point $E_V^* = (S_V^*, E_V^*, I_V^*)$ we have;

$$|J(E_V^*) - \lambda I| = \begin{vmatrix} \left(\frac{\mu_V^2 \xi_V - \alpha_3 \beta_V \Lambda_V}{\mu_V \xi_V} - \mu_V \right) - \lambda & 0 & -\beta_V S_V^* \\ \frac{\alpha_3 \beta_V \Lambda_V - \mu_V^2 \xi_V}{\mu_V \xi_V} & -\xi_V - \mu & \beta_V S_V^* \\ 0 & \alpha_3 & -\mu_V - \lambda \end{vmatrix} \quad (69)$$

The Characteristic equation of the above matrix is given as:

$$|J(E_V^*) - \lambda I| = A_3 \lambda^3 + A_2 \lambda^2 + A_1 \lambda + A_0 \quad (70)$$

Where,

$$A_3 = 1; A_2 = \left(\frac{\alpha_3 \beta_V \Lambda_V}{\mu_V \xi_V} + (\xi_V + \mu_V) \right); A_1 = \left(\frac{\alpha_3 \beta_V \Lambda_V (\xi_V + \mu_V)}{\mu_V} \right); A_0 = \alpha_3 \beta_V \Lambda_V - \mu_V^2 \xi_V.$$

It has been clearly verified using Routh-Hurwitz criterion that the system has all its eigen value to be negative if and only if $A_2 > 0, A_0 > 0, A_2 A_1 > A_0$ holds. This is very trivial and hence the Endemic equilibrium is **stable**.

3.5.3 Lemma 5:

The positive endemic equilibrium E_V^* of the vector (mosquito) population is locally asymptotically stable if $R_{0V} > 1$.

Proof:

By considering the condition for Routh-Hurwitz stability $A_0 > 0$ we have that

$$A_0 = \alpha_3 \beta_V \Lambda_V - \mu_V^2 \xi_V > 0;$$

$$\begin{aligned} \therefore \alpha_3 \beta_V \Lambda_V - \mu_V^2 \xi_V &> 0 \\ \Rightarrow \mu_V^2 \xi_V \left(\frac{\alpha_3 \beta_V \Lambda_V}{\mu_V^2 \xi_V} - 1 \right) &> 0; R_{0V} = \frac{\alpha_3 \beta_V \Lambda_V}{\mu_V^2 \xi_V} \end{aligned}$$

$$\therefore \mu_V^2 \xi_V \left(\frac{\alpha_3 \beta_V \Lambda_V}{\mu_V^2 \xi_V} - 1 \right) = \mu_V^2 \xi_V (R_{0V} - 1) > 0$$

$$\Rightarrow \mu_V^2 \xi_V (R_{0V} - 1) > 0$$

The inequality (5) holds if and only if $R_{0V} > 1$.

This completes the proof and hence proves the Lemma 5 ■

3.5.4 Theorem 3:

The Endemic equilibrium E_{HV}^* of the Vector-Host system (Human and Mosquito) is locally asymptotically stable if $R_G > 1$.

Proof:

We have that $R_{0H} > 1, R_{0V} > 1$ for the Host (Human) and the Vector (Mosquito) respectively from Lemma 4 and Lemma 5. Then,

$$\begin{aligned} R_{0H} &> 1, R_{0V} > 1; \\ \Rightarrow R_{0H} R_{0V} &> 1; \\ \therefore R_G^2 = R_{0H} R_{0V} &> 1; \\ \Rightarrow R_G^2 &> 1; R_G > \sqrt{1}; \\ \therefore R_G &> 1. \end{aligned}$$

This proves the Theorem 3.

4.0 Semi-Analytical Solution to the Seir-Sei Vector Host Model of Malaria Transmission by Variational Iteration Method (VIM)

The implementation of semi-analytical algorithms or methods has spontaneously developed over the years in the field of numerical analysis and computational mathematics.

Numerous researchers have implemented some methods appropriately in providing exact solutions to ordinary and partial differential equations viz [40-43]. Very recently, Loyinmi Adedapo C. and Akinfe Timilehin K. (2020) implemented an algorithm using the Elzaki transform to provide exact solutions to the Burgers-Huxley equation of three distinct cases as a result of variation in the equation parameters [44]. Again in (2019), using a hybrid algorithm involving Elzaki transform and homotopy perturbation method (EHTPM), they proffered exact solution to the family of Fisher's reaction-diffusion equation which is well applicable in genetics, stochastic processes, nuclear reactor theory, and so on. See ref [49]

Nadeem M, Li F, Ahmad H (2019) solved the fourth-order parabolic partial differential equation with variable coefficients using modified Laplace variational iteration method [45] and so on [46-48]

The idea of Variational iteration method (VIM) was introduced by Ji-Huan He (1998) [29] who modified the general Lagrange multiplier proposed by Inokuti (1978) [32] to solve nonlinear problems. Abbasbandy and Shivanian (2009) [33], Abdou and Soliman (2005) [35], Mosmani and Abuasad (2006) [34] have implemented this method to solve effectively, easily and accurately, a large class of nonlinear problems with approximations which converges quickly to accurate solutions.

The implementation of asymptotic techniques/methods is quite an interesting and demanding field of computational mathematics as there is no single best method or algorithm for a

problem/model. The suitability of an algorithm to a problem depends on the simplicity, computational stress and radius/rate of convergence of such algorithm or method.

Adedapo Loyinmi C. and Akinfe Timilehin K. and other authors have buttressed these facts with their convergence analyses [49]

4.1 Basic Idea of VIM

Consider a non-linear differential equation

$$Lu(t) + Nu(t) = g(t) \quad (70)$$

L is the linear operator, N is the nonlinear operator and $g(t)$ is a known analytic function. We construct a correction functional for the equation (1) which is given as:

$$u_{n+1}(t) = u_n(t) + \int_0^t \pi(x) [Lu_n(x) + N\tilde{u}_n(x) - g(x)] dx \quad (71)$$

Where π is a lagrangian multiplier which can be obtained optimally and expressed as:

$$\pi(x) = \frac{(-1)^n}{(n-1)!} (x-t)^{n-1} \quad (71)$$

Where n is the highest order of the differential equation.

4.2 Solution of the Model using Variational Iteration Method

We consider the model's system of equations in (1), Subject to the initial condition (State Variables) Adopted from [1]. We have,

$$S_H(0) = 0.83, E_H(0) = 0.08, I_H(0) = 0.07, R_H(0) = 0.02, S_V(0) = 0.7, \\ E_V(0) = 0.2, I_V(0) = 0.1$$

Table 2: Parameter description and Values of model

Parameter	Description Of Parameter	Value	Source
Λ_H	Recruitment Rate of Humans	1.2	Osman et al.(2017) [1]
Λ_V	Recruitment Rate of Mosquitoes	0.7	Osman et al. (2017) [1]
α_1	Rate of Development from E(t) to I(t) for Humans	0.05	S. Olaniyi et al. (2013) [2]
α_2	Recovery Rate of Humans	0.0035	Shah NH et al. (2013) [21]
α_3	Rate of Development from E(t) to I(t) for Mosquitoes	0.083	Shah NH et al. (2013) [21]
μ_H	Death Rate of Humans	0.0115	Estimated
μ_V	Death Rate of Mosquitoes	0.05	Macdonald G. (1957) [5]
η_V	Mosquito Biting rate	0.46	Jia Li (2015) [35]
q_H	Probability of transmission from an infectious mosquito to Human	0.022	Chitnis N et al. (2008) [37]
q_V	Probability of transmission from an infectious Human to Mosquito	0.24	Chitnis N et al. (2008) [37]
β_H	Infection Rate of Humans	0.00638	Osman et al. (2017) [1]
β_V	Infection Rate of Mosquitoes	0.0696	Osman et al. (2017) [1]
δ	Disease Induced Death Rate in Humans	0.0681	Assumed
ψ	Newborn's birth rate with infection	0.0003	Osman et al. (2017) [1]
ρ	Loss of immunity in Humans	0.00017	Ishikawa H. [36] et al. (2013)

4.2.1 VIM Application to Model Equations

We have the correction functional of the system of equation governing this malaria model as:

$$\begin{aligned}
S_{(n+1)H}(t) &= S_{nH}(t) + \int_0^t \pi_1(x) [S'_{nH} + \beta_H \tilde{S}_{nH} \tilde{I}_{nH} + \mu_H S_{nH} - \Lambda_H - \rho R_{nH}] dx \\
E_{(n+1)H}(t) &= E_{nH}(t) + \int_0^t \pi_2(x) [E'_{nH} + (\alpha_1 + \mu_H) E_{nH} - \beta_H \tilde{S}_{nH} \tilde{I}_{nH}] dx \\
I_{(n+1)H}(t) &= I_{nH}(t) + \int_0^t \pi_3(x) [I'_{nH} + (\alpha_2 + \mu_H + \delta - \psi) I_{nH} - \alpha_1 E_{nH}] dx \\
R_{(n+1)H}(t) &= R_{nH}(t) + \int_0^t \pi_4(x) [R'_{nH} + (\mu_H + \rho) R_{nH} - \alpha_2 I_{nH}] dx \\
S_{(n+1)V}(t) &= S_{nV}(t) + \int_0^t \pi_5(x) [S'_{nV} + \mu_V S_{nV} + \beta_{nV} \tilde{S}_{nV} \tilde{I}_{nV} - \Lambda_V] dx \\
E_{(n+1)V}(t) &= E_{nV}(t) + \int_0^t \pi_6(x) [E'_{nV} + (\alpha_3 + \mu_V) E_{nV} - \beta_{nV} \tilde{S}_{nV} \tilde{I}_{nV}] dx \\
I_{(n+1)V}(t) &= I_{nV}(t) + \int_0^t \pi_7(x) [I'_{nV} + \mu_V I_{nV} - \alpha_3 E_{nV}] dx
\end{aligned} \tag{72}$$

Subject to the initial conditions

$$\begin{aligned}
S_H(0) &= 0.83, E_H(0) = 0.08, I_H(0) = 0.07, R_H(0) = 0.02, S_V(0) = 0.7, E_V(0) = 0.2, I_V(0) = 0.1 \\
\pi_1(x) &= \pi_2(x) = \pi_3(x) = \pi_4(x) = \dots = \pi_7(x) = -1
\end{aligned}$$

By putting the model parameters and the value of the lagrangian multiplier in eq. (72); we obtain an iteration formula for the seven (7) compartments as:

$$\begin{aligned}
S_{(n+1)H}(t) &= S_{nH}(t) - \int_0^t [S'_{nH} + 0.00638 S_{nH} I_{nH} + 0.0115 S_{nH} - 1.2 - 0.00017 R_{nH}] dx \\
E_{(n+1)H}(t) &= E_{nH}(t) - \int_0^t [E'_{nH} + 0.0615 E_{nH} - 0.00638 S_{nH} I_{nH}] dx \\
I_{(n+1)H}(t) &= I_{nH}(t) - \int_0^t [I'_{nH} + 0.0801 I_{nH} - 0.05 E_{nH}] dx \\
R_{(n+1)H}(t) &= R_{nH}(t) - \int_0^t [R'_{nH} + 0.01167 R_{nH} - 0.0035 I_{nH}] dx \\
S_{(n+1)V}(t) &= S_{nV}(t) - \int_0^t [S'_{nV} + 0.05 S_{nV} + 0.696 S_{nV} I_{nV} - 0.7] dx \\
E_{(n+1)V}(t) &= E_{nV}(t) - \int_0^t [E'_{nV} + 0.133 E_{nV} - 0.0696 S_{nV} I_{nV}] dx \\
I_{(n+1)V}(t) &= I_{nV}(t) - \int_0^t [I'_{nV} + 0.05 I_{nV} - 0.083 E_{nV}] dx
\end{aligned}$$

We obtain the iterated values for each population/compartments as:

$$S_H(t) = 0.83 + 1.190087722t - 0.007104328345t^2 + 0.00003194682064t^3 \\ - 7.001923802 \times 10^{-7}t^4 + 8.456685994 \times 10^{-10}t^5 + 1.163198684 \times 10^{-12}t^6 \\ - 3.423963937 \times 10^{-15}t^7 + 5.342315808 \times 10^{-16}t^8 - 3.723617710 \times 10^{-18}t^9 \\ + 6.322821679 \times 10^{-21}t^{10} - 2.271139500 \times 10^{-24}t^{11} + \dots$$

$$E_H(t) = 0.08 - 0.004549322000t + 0.0004012165808t^2 - 0.00001258921184t^3 \\ + 0.1061638566 \times 10^{-5}t^4 - 6.061655945 \times 10^{-9}t^5 + 1.270625328 \times 10^{-11}t^6 \\ - 2.077566160 \times 10^{-15}t^7 - 5.342315808 \times 10^{-16}t^8 + 3.723617710 \times 10^{-18}t^9 \\ - 6.322821679 \times 10^{-21}t^{10} + 2.271139500 \times 10^{-24}t^{11} + \dots$$

$$I_H(t) = 0.07 - 0.001607000000t - 0.00004937270000t^3 - 1.370365576 \times 10^{-7}t^4 \\ + 1.788543167 \times 10^{-8}t^5 - 1.196734896 \times 10^{-10}t^6 + 2.494651094 \times 10^{-13}t^7 \\ - 6.806885860 \times 10^{-17}t^8 - 2.967953227 \times 10^{-18}t^9 + 1.861808855 \times 10^{-20}t^{10} + \dots$$

$$R_H(t) = 0.02 + 0.00001160000000t - 0.2879936000 \times 10^{-5}t^2 - 4.30259846 \times 10^{-8}t^3 \\ - 2.017061262 \times 10^{-9}t^4 - 3.199583573 \times 10^{-10}t^5 + 2.406919418 \times 10^{-18}t^8 \\ - 6.934725980 \times 10^{-15}t^7 + 3.028880838 \times 10^{-12}t^6$$

$$S_V(t) = 0.7 + 0.6601280000t - 0.01908302144t^2 + 2.040227226 \times 10^{-4}t^3 \\ + 1.303653606 \times 10^{-5}t^4 + 1.519901404 \times 10^{-6}t^5 + 2.043333541 \times 10^{-7}t^6 \\ + 6.022746410 \times 10^{-9}t^7 - 7.190253944 \times 10^{-10}t^8 + 1.796296797 \times 10^{-11}t^9 \\ - 2.018406198 \times 10^{-13}t^{10} - 2.474577295 \times 10^{-14}t^{11} + 9.727190967 \times 10^{-16}t^{12} + \dots$$

$$E_V(t) = 0.2 - 0.02172800000t + 0.004024733440t^2 + 0.0000435937856t^3 \\ - 7.18167845 \times 10^{-6}t^4 + 3.93765013 \times 10^{-7}t^5 - 2.744923850 \times 10^{-7}t^6 \\ - 1.432467516 \times 10^{-9}t^7 + 6.147220304 \times 10^{-10}t^8 - 2.319266431 \times 10^{-11}t^9 \\ + 2.479625495 \times 10^{-13}t^{10} + 2.651694009 \times 10^{-14}t^{11} + \dots$$

$$\begin{aligned}
I_V(t) = & 0.1 + 0.01160000t - 0.001191712000t^2 + 0.0001312128252t^3 \\
& - 2.97650627 \times 10^{-6}t^4 + 3.88118335 \times 10^{-7}t^5 + 6.846185837 \times 10^{-8}t^6 \\
& - 2.099291186 \times 10^{-9}t^7 - 7.998304105 \times 10^{-12}t^8 + 2.968697857 \times 10^{-13}t^9 \\
& + 2.512090497 \times 10^{-15}t^{10} - 7.265313407 \times 10^{-17}t^{11} + \dots
\end{aligned}$$

This gives the Semi-analytic solution of the SEIR-SEI model of malaria Transmission.

5.0 Numerical Results

The analytical Results for the SEIR-SEI model is illustrated and demonstrated in this section.

This results were achieved using **Computer Software** and by using the parameter values and state variable values in the table 2 whose source were mainly from prominent literatures as well as assumptions.

The results obtained by our proposed VIM were compared with the Computer In-built **Runge-kutta felhberg** of fourth-fifth order (**RKF-45**) with degree four interpolant. In which a table of values obtained from all compartments is presented.

Table 3 and 4 presents the results comparison of $S_H(t)$, $E_H(t)$, $I_H(t)$, $R_H(t)$, $S_V(t)$, $E_V(t)$, and $I_V(t)$ between **VIM** and **RKF-45**.

5.1 Table 3: The Host (Human) Population Model

Comparison between Variational iteration Method and Runge-Kutta-Felhberg-45 (VIM vs RKF-45)

TIME	VIM	RKF-45	VIM	RKF-45	VIM	RKF-45
Day(s)	$S_H(t)$	$S_H(t)$	$E_H(t)$	$E_H(t)$	$I_H(t)$	$I_H(t)$
0	0.83000000	0.83000000	0.08000000	0.08000000	0.07000000	0.07000000
1	2.01301561	2.01301561	0.07583878	0.07583879	0.06835131	0.06835133
2	3.18201621	3.18201617	0.07240199	0.07240198	0.06664753	0.06664754
3	4.33719361	4.33719354	0.06961551	0.06961550	0.06492588	0.06492590
4	5.47873683	5.47873677	0.06741038	0.06741037	0.06321729	0.06321730
5	6.60683104	6.60683104	0.06572332	0.06572332	0.06154712	0.06154713
6	7.72165743	7.72165743	0.06449681	0.06449684	0.05993590	0.05993589
7	8.82339237	8.82339237	0.06367926	0.06367934	0.05839999	0.05839989
8	9.91220582	9.91220759	0.06322463	0.06322496	0.05695224	0.05695186
9	10.98826994	10.98826993	0.06309231	0.06309337	0.05560260	0.05560147
10	12.05174144	12.05174145	0.06324656	0.06324953	0.05435880	0.05435584

TIME	VIM	RKF-45
Day(s)	$R_H(t)$	$R_H(t)$
0	0.02000000	0.02000000
1	0.02000868	0.02000868
2	0.02001142	0.02001142
3	0.02000815	0.02000815
4	0.01999895	0.01999895
5	0.01998396	0.01998396
6	0.01996344	0.01996344
7	0.01993768	0.01993768
8	0.01990702	0.01990702
9	0.01987184	0.01987184
10	0.01983256	0.01983256

5.2 Table 4: The Vector (Mosquito) Population Model

Comparison between Variational iteration Method and Runge-Kutta-Felhberg-45 (VIM vs RKF-45)

TIME	VIM	RKF-45	VIM	RKF-45	VIM	RKF-45
Day(s)	$S_V(t)$	$S_V(t)$	$E_V(t)$	$E_V(t)$	$I_V(t)$	$I_V(t)$
0	0.70000000	0.70000000	0.20000000	0.20000000	0.10000000	0.10000000
1	1.34126043	1.34126044	0.18222063	0.18222068	0.11053636	0.11053636
2	1.94571554	1.94571556	0.17196734	0.17196734	0.11943083	0.11943067
3	2.51484441	2.51484462	0.16860461	0.16860489	0.12734893	0.12734679
4	3.05014134	3.05014133	0.17149462	0.17149495	0.13486396	0.13486393
5	3.55302285	3.55302267	0.18008280	0.18008282	0.14248463	0.14248433
6	4.02476836	4.02476839	0.19394581	0.19394865	0.15064626	0.15064609
7	4.46648246	4.46648299	0.21276299	0.21283345	0.16002117	0.15973839
8	4.87907270	4.87907287	0.23664713	0.23664911	0.17011741	0.17011742
9	5.26322652	5.26323337	0.26472804	0.26547877	0.18212468	0.18212189
10	5.61944102	5.61944155	0.28884690	0.29957234	0.19608738	0.19608754

Multiple plot profiles of all compartments in the model (Graphical illustrations)

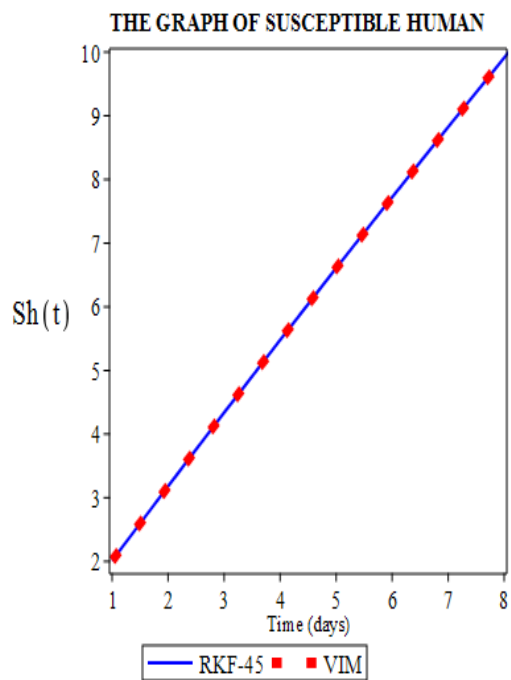


Fig 1 Solution comparison plot of the susceptible human population between variational iteration method and Runge-Kutta-Felhberg 45 (RKF-45).

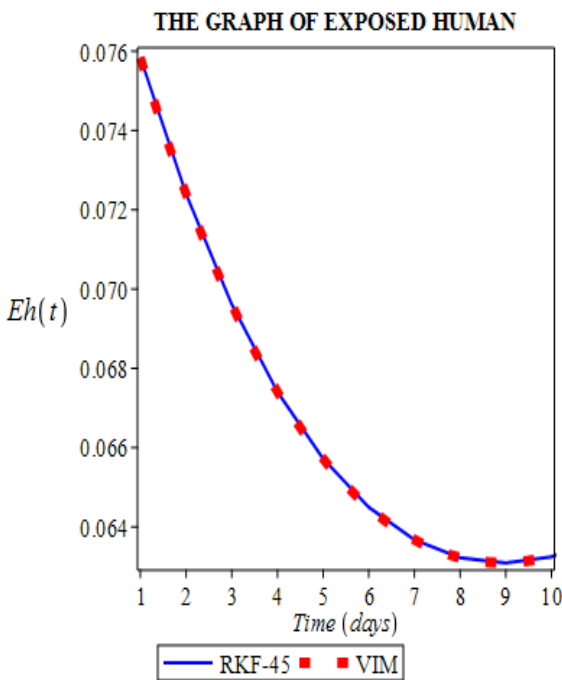


Fig 2 Solution comparison plot of the exposed population between variational Iteration method (VIM) and Runge-Kutta-Felhberg 45 (RKF-45).

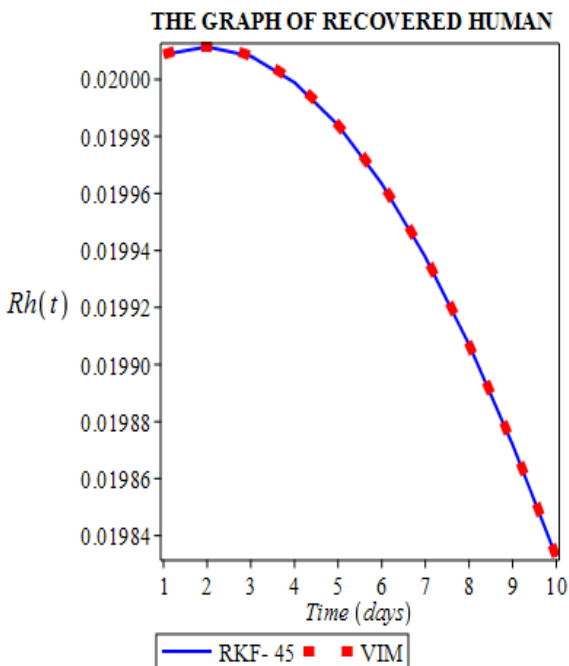
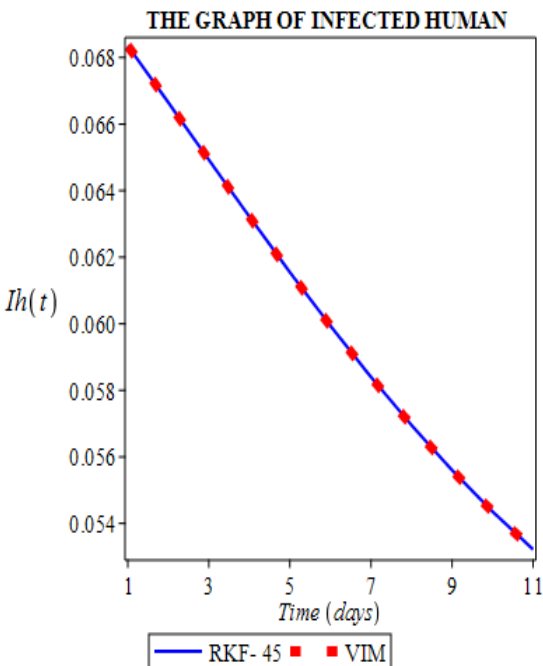


Fig 3 Solution comparison plot of the Infected human population between Variational iteration method (VIM) and Runge-Kutta-felhberg 45 (RKF-45)

Fig 4 Solution comparison plot of the recovered human population between variational iteration method and Runge-Kutta-felhberg 45 (RKF-45)

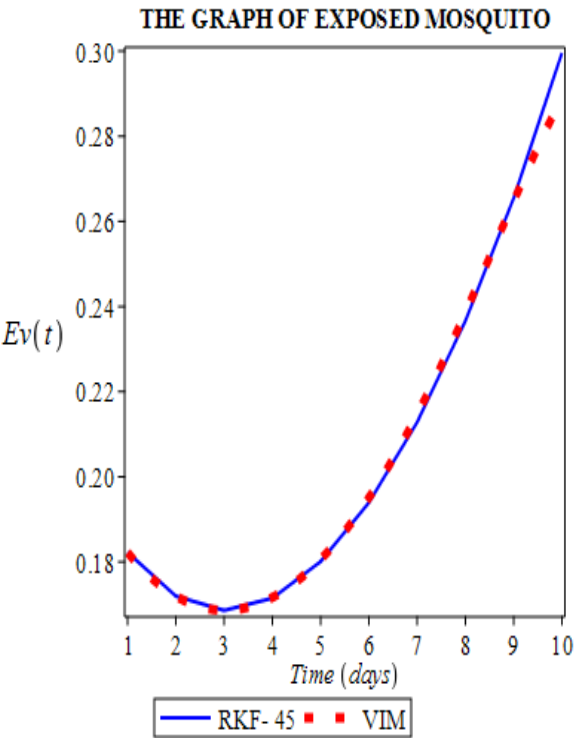
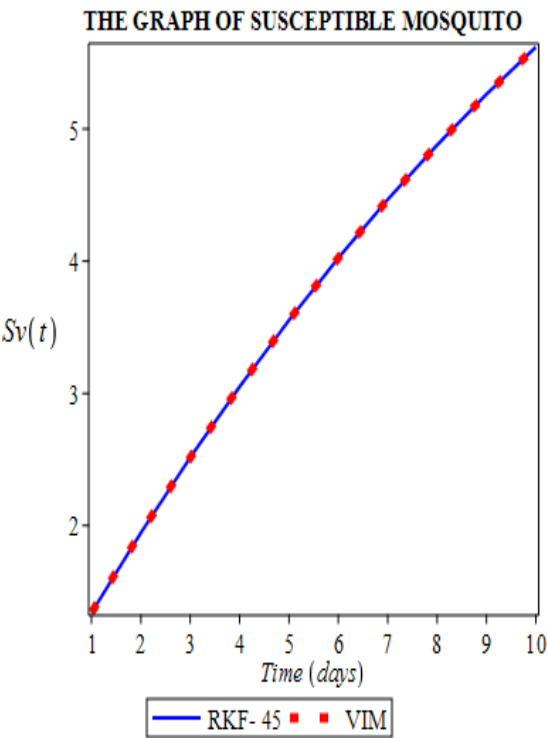


Fig 5 Solution comparison plot of the Susceptible mosquito between variational Iteration method (VIM) and Runge-Kutta-Felhberg 45 (RKF-45)

Fig 6 Solution comparsion plot of the exposed mosquitoes between variational iteration method and Runge-Kutta-felhberg

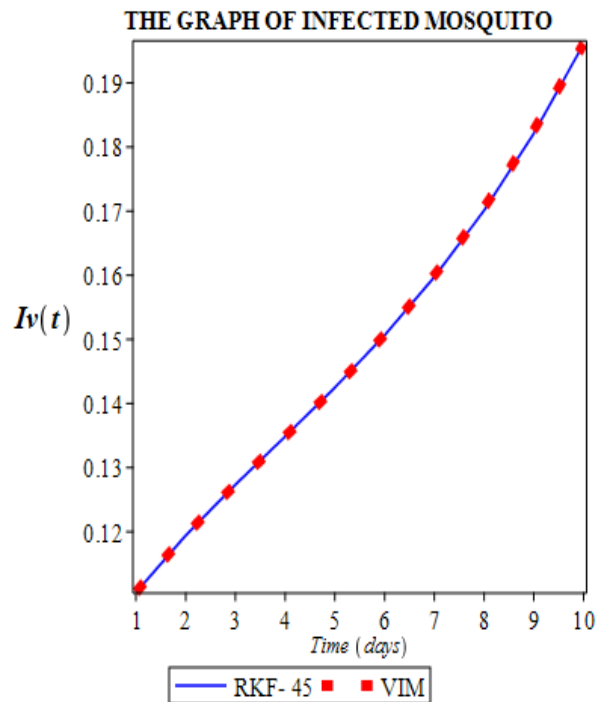


Fig 7 Solution comparison plot of the infected Mosquito between variational iteration method (VIM) And Runge-Kutta-Felhberg 45 (RKF-45)

5.3 Interpretation of Results

The Results obtained from the Numerical simulation and the stability analysis of the proposed SEIR-SEI Vector-host Malaria Transmission model shows that the Disease-free equilibrium is stable when a mosquito doesn't infect more than one individual. That is, when R_0 does not exceed unity ($R_0 < 1$); and moment the converse happens (when $R_0 > 1$) then there is an epidemic outbreak (endemic equilibrium).

Furthermore, the population of the susceptible human undergoes an **exponential growth** pattern, and the graph is perfectly linear.

The Population of the exposed and the infected human decays (decreases) in a **logistic** manner and hence a **logistic decay** which implies that there is a possibility of zero population (At DFE).

While the Recovered Human, Susceptible, Exposed and infected mosquito follows a **logistic growth** pattern.

All these imply that the populations in the system are prone to the infection since the population in the susceptible compartment is increasing.

As a result, serious attention should be focused on this compartment as regards an appropriate intervention strategy to combat the contact of malaria infection at a time when the infected and the exposed population is stable (when $R_0 < 1$).

5.4 Discussion of Results

A SEIR-SEI Vector-Host model of malaria transmission built on 7-dimensional system of ordinary differential was analyzed and solved numerically using the Variational iteration method (VIM) with initial conditions and parameter values from prominent existing literature.

The stability analyses show that there is a possibility of the malaria infection going into extinction if one mosquito does not infect more than one individual (when $R_0 < 1$). Similarly, epidemic outbreak of the disease is visible and might occur when $R_0 > 1$.

The Semi-analytical solution to the Malaria model using VIM when compared favorably with the **Computer software** in-built Runge-Kutta-Felhberg of fourth-fifths order (**RKF-45**) shows a high level of agreement, convergence and similarities. The two methods follow the same pattern and behavior when plotted.

Having used our proposed method, it is now crystal clear that the variational iteration method is suitable, perfect and efficient in conducting and conveying analysis on Malaria models.

6.0 Conclusion

We have studied a nonlinear 7-dimensional ordinary differential equation that describes the transmission dynamics of malaria by carrying asymptotic stability analyses at the malaria-free and endemic equilibriums using the Gaussian elimination method and the routh-hurwitz criterion with three (3) theorems and three (3) lemmas. We also have solved the model using the variational iteration method (VIM). This method is unprecedented.

From all these, we have come to a conclusion that the variational is an efficient alternative for conducting analyses on malaria models and other epidemiological models and as a matter of fact can be implemented in the classroom to provide solutions to a wider class of ordinary differential equations and epidemiological models.

7.0 Declaration of Interest

Authors have declared that there is no conflict of interest relevant to this research

References

- [1] Mojeeb AL-Rahman EL-Nor Osman, Isaac Kwasi Adu, and Cuihong Yang, A Simple SEIR Mathematical Model of Malaria transmission, Asian Research journal of Mathematics 7(3) (2017), pp. 1-22.
- [2] Olaniyi S and Olaniyi OS, Mathematical model for malaria transmission dynamics on human and mosquito population with nonlinear forces of infectious disease, Int. Journal of Pure and Applied Mathematics, 88(1) (2013), pp. 125-150.
- [3] Jia Li, Modelling of transgenic mosquitoes and impact on malaria transmission, Journal of Biological Dynamics, 5:5 (2011), pp. 474-494, DOI: [10.1080/17513758.2010.523122](https://doi.org/10.1080/17513758.2010.523122)
- [4] Ross R. Report on the prevention of Malaria in Mauritius. London (1908). Available from <https://archive.org/details/onpreventi00rossreportrich> [Google scholar]
- [5] Macdonald G (1957), The Epidemiology and control of Malaria. Oxford University Press, London
- [6] Driessche PV and Watmough J, Reproduction numbers and sub-threshold endemic equilibria for Compartmental models of disease transmission, Mathematical Biosciences 180 (2002) pp. 29–48.
- [7] Traoré Bakary, Sangaré Boureima & Traoré Sado, A mathematical model of malaria transmission in a periodic environment, Journal of Biological Dynamics, 12:1 (2018), pp. 400-432, DOI: [10.1080/17513758.2018.1468935](https://doi.org/10.1080/17513758.2018.1468935)
- [8] Alejandra Ortiz-Ruiz, Maria postigo, Sara Gil-casanova, Daniel Cuadrado, Jose M. Bautista, Jose Miguel Rubio, Miguel Luengo-Oroz and Maria Linares, Plasmodium Species Differentiation by Non-expert on-line volunteers for Remote Malaria field Diagnosis, BMC Malaria Journal 17(54) (2018).
- [9] WHO World Malaria Report (2013), Available at https://www.who.int/malaria/publications/world_malaria_report_2013/en/
- [10] WHO World Malaria Report (2015), Available at <https://www.who.int/malaria/publications/world-malaria-report-2015/report/en/>

- [11] WHO World Malaria Report (2010) Available at https://www.who.int/malaria/world_malaria_report_2010/en/
- [12] WHO World Malaria Report (2016) Available at <https://www.who.int/malaria/publications/world-malaria-report-2016/report/en/>
- [13] Getachew Geleta and Tsige Ketema, Severe Malaria Associated with Plasmodium falciparum and P. vivax among Children in Pawe Hospital, Northwest Ethiopia, Malaria Research and Treatment, vol. 2016 (2016.), Article ID 1240962, 7 pages, <https://doi.org/10.1155/2016/1240962>.
- [14] Ruhani Yusof, Yee Ling Lau, Rohela Mahmud, Mun Yik Fong, Jenarun Jelip,... and Marlindawati Mohd Ali, High Proportion of Knowlesi Malaria in recent Malaria cases in Malaysia, BMC Malaria Journal 13(168) (2014).
- [15] Caraballo, H. and King, K., Emergency Department Management of Mosquito-Borne Illness: Malaria, Dengue, and West Nile Virus. Emergency Medicine Practice, 16 (2014), pp. 1-23.
- [16] Fact sheet on the World Malaria Report (2014) Available at https://www.who.int/malaria/media/world_malaria_report_2014/en/
- [17] Haq Fazal, Shah Kamal, Khan Asaf, Shahzad Muhammad and Rahman Ghaus, Numerical Solution of Fractional Order Epidemic Model of a Vector Born Disease by Laplace Adomian Decomposition Method. Punjab University Journal of Mathematics. 49(10) (2017).
- [18] Ngwa G.A, Shu W.S. A mathematical model for endemic with variable human and mosquito Populations, Available: www.ictp.trieste.it/pub.off.1999
- [19] Rangkuti, Yulita Molliq; Side, Syafruddin; Noorani, Mohd Salmi Md. Numerical Analytic Solution of SIR Model of Dengue Fever Disease in South Sulawesi using Homotopy Perturbation Method and Variational Iteration Method. Journal of Mathematical and Fundamental Sciences, 46(1) (2014), pp. 91-105. Available at: <http://journals.itb.ac.id/index.php/jmfs/article/view/819>.
doi:10.5614/j.math.fund.sci.2014.46.1.8.

- [20] Goswami P, Murty U.S, Mutheneni S.R, Kukkuthady A, Krishnan S.T, A Model of Malaria Epidemiology involving Weather, Exposure and Transmission applied to North East India. PloS One, 7(11) (2012).
- [21] Shah NH, Gupta J. SEIR model and simulation for vector borne diseases, Applied Mathematics. 4(2013), pp. 13-17.
- [22] Altaf Khan M, Abdul Wahid, Saeed Islam, Ilyas Khan, Sharidan Shafie, and Taza Gul, Stability analysis of an SEIR Model with non-linear saturated incidence and temporary immunity. Int. J. Adv. Appl. Math and Mech., 2(3) (2015):234-7-2529.
- [23] Hai-Feng Huo and Guang-Ming Qiu, Stability of a Mathematical Model of Malaria Transmission with Relapse, Abstract and Applied Analysis, vol. 2014 (2014), Article ID 289349, 9 pages. <https://doi.org/10.1155/2014/289349>.
- [24] Mojeeb AL-Rahman EL-Nor Osman, Appiagyei Ebenezer and Isaac Kwasi Adu, A Simple SEIR Mathematical Model of Malaria transmission, Journal of Advances in Mathematics and Computer science 28(6) (2018), pp. 1-17.
- [25] Nigar Ali & Saeed Ahmad & Sartaj Aziz & Gul Zaman,, The Adomian Decomposition Method For Solving HIV Infection Model Of Latently Infected Cells, Matrix Science Mathematic (MSMK), Zibeline International Publishing, vol. 3(1) (2019), pp. 5-8.
- [26] O, Olayiwola & Kayode Kolawole, Mutairu & Popoola, Amos, Variational Iteration Method for the Simulation of the Effect of Transmission Coefficient on the Susceptible-Exposed-Infected-Recovered-Susceptible (Seirs) Epidemic Model with Saturated Incidence Rate and Disease-Induced Death. 17 (2017). Pp. 357-364.
- [27] Abioye, Adesoye & Ibrahim, Mohammed Olanrewaju & Olumuyiwa, Peter & Sylvanus, Amadiogwu & Festus, Oguntolu. Differential Transform Method for Solving Mathematical Model of SEIR and SEI Spread of Malaria. 40 (2018). Pp. 197-219.

- [28] He, J. H, Variational iteration method a kind of nonlinear analytical technique: Some examples, *Int. J. Nonlinear Mech.*, Vol.34 (1999), Pp 669-708.
- [29] Olumuyiwa, P., Folasade, A.A., Abiodun, O.F., Sambo, B., & Emmanuel, A.C. Multi-step Homotopy Analysis Method for Solving Malaria Model, *Malaysian Journal of Applied Sciences*, 3(2) (2018).
- [30] G. Birkhoff and G. C. Rota, *Ordinary Differential Equations*, Ginn, Boston, 1982.
- [31] Inokuti, M., General Use of the Lagrange Multiplier in Nonlinear Mathematical Physics. In: Nemat Nasser, S., Ed., *Variational Method in the Mechanics of Solid*, Pergamon Press, Oxford, (1978) pp. 156-162.
- [32] Abbasbandy, E & Shivanian, E., Application of the variational iteration method for system of nonlinear Volterra's integro-differential equations, *Mathematical and Comp. Applic.*, Vol 14, Issue 2 (2009), Pp 147-158.
- [33] Momani, S, Abuasad, S (2006), Application of He's variational iteration method to Helmholtz equation, *Chaos, Solitons & Fractals.*, 27: 1119- 1123 strategies in microbial pathogenesis, *Nature*, 430, 250-256.
- [34] Abdou, M . A & Soliman, A. A, Variational iteration method for solving Burger's and coupled Burger's equation, *J. Comp. Appl. Math.*, Vol 181, Issue 2 (2005), 245-251.
- [35] Jia Li. New revised simple models for interactive Wild and sterile Mosquito Populations and their Dynamics. *Journal of Biological Dynamics*. 2015, 34:D25i92D;92D40.
- [36] Chitnis N, Hyman JM, Cushing JM. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology* 2008 70 pp. 1272-1296

- [37] Ishikawa H, et al. A mathematical model for the transmission of *Plasmodium vivax* malaria, Parasitology International 52 (2013) pp. 81-93.
- [38] WHO World Malaria Report (2018) Available at <https://www.who.int/malaria/publications/world-malaria-report-2018/en/>
- [39] 2019 Commonwealth Malaria Report Available at <https://www.malariamustdie.com/sites/default/files/2019-04/CommonwealthMalariaReport2019.pdf>
- [40] Ji-Huan He, Homotopy perturbation method for solving boundary value problems, Physics Letters A, 350(2): 87-88, (2006) <https://doi.org/10.1016/j.physleta.2005.10.005>.
Available at: (<http://www.sciencedirect.com/science/article/pii/S0375960105015434>)
- [41] Deniz, Sinan & Bildik, Necdet, A Note on Stability Analysis of Taylor Collocation Method, Celal Bayar Üniversitesi Fen Bilimleri Dergisi, 10.18466/cbayarfbe.302660, (2017)
- [42] Bildik, Necdet & Deniz, Sinan, The Use of Sumudu Decomposition Method for Solving Predator-Prey Systems. Mathematical Sciences Letters. 5. 285-289. 10.18576/msl/050310, (2016)
- [43] Alvaro Salas H., solving nonlinear partial differential equations by the sn-ns method, Abstract and Applied Analysis, Article ID 340824: 25 pages, (2012)
DOI: <https://doi.org/10.1155/2012/340824>.
- [44] Loyinmi, A.C., Akinfe, T.K. An algorithm for solving the Burgers-Huxley equation using Elzaki transform, SN Applied sciences, 2(7), (2020)
DOI: <https://doi.org/10.1007/s42452-019-1653-3>
- [45] Nadeem Muhammad, Li Fengquan and Ahmad Hijaz, Modified Laplace variational iteration method for solving fourth-order parabolic partial differential equation with variable coefficients. Computers and Mathematics with Applications. 10.1016/j.camwa.2019.03.053, (2019)
- [46] He, Ji-Huan., Homotopy perturbation method: A new nonlinear analytical technique. Applied Mathematics and Computation - AMC. 135. 73-79. 10.1016/S0096-3003(01)00312-5, (2003)

- [47] He, Ji-Huan., Application of homotopy perturbation method to nonlinear wave Equations. *Chaos, Solitons & Fractals*. 26. 695-700. 10.1016/j.chaos.2005.03.006, (2005)
- [48] Aziz Imra, Islam Siraj-ul-Islam, and Asif Muhammad, Haar wavelet collocation method for three-dimensional elliptic partial differential equations. *Computers and Mathematics with Applications*. 73. 10.1016/j.camwa.2017.02.034, (2017)
- [49] Loyinmi, A.C. & Akinfe, T.K.:Exact solution to the family of Fisher's reaction diffusion equation using Elzaki Homotopy transformation perturbation method. *Engineering Reports*. 2019; e12084. DOI: <https://doi.org/10.1002/eng2.12084>