



# Sensitivity of Malaria-HBV Co-Infection Dynamics to Parameter Changes: A Mathematical Analysis

<sup>1</sup>Gana Abubakar, <sup>2</sup>Ashafa S. U., <sup>3</sup>Liman Asabe Suleiman

<sup>1</sup>Department of Mathematics, Yobe State University Damaturu, Nigeria.

<sup>2 & 3</sup>Department of Mathematics and Statistics, Federal Polytechnic Nasarawa

Corresponding Author's Email: [umarashafa@gmail.com](mailto:umarashafa@gmail.com)

Received on: January, 2025

Revised and Accepted on: February, 2025

Published on: March, 2025

## ABSTRACT

Malaria and Hepatitis B Virus (HBV) co-infection pose significant health challenges, particularly in tropical developing countries. This study develops a mathematical model describing the co-infection dynamics of Malaria and HBV diseases in human populations. The model is divided into eleven compartments, and its qualitative analysis reveals the equilibrium points, positivity of solutions, and invariant regions. The disease-free equilibrium point and reproduction numbers are determined, indicating local asymptotic stability. Sensitivity analysis identifies key parameters influencing the co-infection model, including HBV recovery rate, malaria transmission probability in humans, and probability of transmission of malaria in mosquitoes. Numerical simulations demonstrate that increasing HBV recovery rate and decreasing malaria transmission probabilities can effectively control the spread of the infection. Specifically, increasing HBV recovery rate reduces HBV-only and Malaria-HBV co-infected populations, while decreasing malaria transmission probabilities in humans and mosquitoes reduces Malaria-only, HBV-only, and Malaria-HBV co-infected populations. These findings provide valuable insights for policymakers and stakeholders to develop effective strategies for controlling the spread of Malaria-HBV co-infection.

**Keywords:** Co-Infection, Malaria, Hepatitis B Virus, Mathematical Modeling, infectious diseases.

## 1.0 INTRODUCTION

Throughout history, infectious diseases have had a profound impact on human societies, with their effects still being felt in many developing countries today (Petrunina, Semenov, & Novikov, 2021). Malaria, a preventable and treatable disease, continues to have a devastating impact on human life, particularly in Africa. In Nigeria, malaria remains a significant public health concern, with approximately 68 million reported cases and 194,000 fatalities attributed to the disease in 2021. The country carries a disproportionate burden of the global malaria epidemic, accounting for nearly 27% of worldwide malaria cases (WHO, 2023). Despite the alarming statistics, concerted global efforts to combat malaria have led to notable successes, with over 25 countries making significant progress towards eliminating the disease.

The African region, particularly sub-Saharan Africa, has been hardest hit by malaria, accounting for 80% of global malaria deaths reported in 2017, with projections suggesting a continued rise in mortality rates. Nigeria has been a major contributor to the regional malaria burden, accounting for 27% of global malaria cases and ranking among the top 32 African countries responsible for 93% of malaria deaths worldwide. The malaria situation has worsened in recent years, with cases increasing from 227 million in 2019 to 241 million in 2020, and a staggering 95% of

cases and 96% of deaths concentrated in Africa (Ayub, Ibrahim, & Abdulkareem, 2024).

Hepatitis B, a liver disease with potentially fatal consequences, poses a considerable threat to global health. Recent estimates suggest that approximately 254 million individuals worldwide are living with chronic hepatitis B infection, with a significant 1.2 million new cases reported annually. The hepatitis B virus (HBV) is estimated to cause around 1.1 million fatalities each year, mainly due to acute and chronic complications (WHO, 2024). Transmission of HBV occurs through contact with infected bodily fluids, including blood, semen, and other fluids, and can be facilitated by high-risk behaviors such as unprotected sex, contaminated blood transfusions, unsterile tattoos, and shared needles (ECDC, 2022).

Malaria and hepatitis B are two major public health issues that have a disproportionate impact on low- and middle-income countries, especially in sub-Saharan Africa. Notably, the liver plays a crucial role in hosting both infections, as Plasmodium parasites and the hepatitis B virus (HBV) both infect hepatocytes, creating a reservoir for the diseases to take hold (Ashafa, Oduwale, & Babangambo, 2022).

The co-occurrence of malaria and hepatitis B infections is becoming increasingly alarming, especially in regions where both diseases are prevalent. Evidence

suggests that co-infection can result in heightened morbidity and mortality rates, as well as accelerated liver deterioration and disease progression (Ashafa, Oduwole, & Umar, 2022). Additionally, research has probed the epidemiological patterns, clinical features, and outcomes of individuals co-infected with both diseases, underscoring the urgent need for effective preventive and therapeutic measures (Kotepui & Kotepui, 2020).

Mathematical modeling has been employed to study the dynamics of malaria and hepatitis B co-infection. For instance, (Ashafa, Oduwole, & Babangambo, 2022) developed a mathematical model to describe the co-infection dynamics, consisting of nine compartments. However, their model did not account for the exposed class, which is a critical component in understanding the transmission dynamics of both diseases. The exposed class represents individuals who have been infected but are not yet infectious, and its inclusion can significantly impact the model's predictions and insights.

Similarly, other studies have also overlooked the exposed class in their modeling frameworks. For example, a study by (Chinebu, et al., 2023) developed an intracellular mathematical model to describe the dynamics of hepatitis B virus (HBV) and malaria co-infection which takes into account the immune response and treatment, but did not consider the exposed class in their analysis. Another study (Kenneth, Vivian, Onolunosen, & Husseni, 2023) examined the co-infection dynamics in Wukari, Taraba State, North East, Nigeria, but also did not account for the exposed class.

This study aims to address this knowledge gap by developing an upgraded mathematical model that includes the exposed class. The model will be used to investigate the co-infection dynamics of malaria and hepatitis B, with a focus on the impact of the exposed class for each disease on disease transmission and progression.

## 2.0 MATERIALS AND METHOD

### 2.1 Material

#### 2.1.1 Model Formulation

The model is divided into sub compartments. The total population of human ( $N_H$ ) is divided into following sub compartments; susceptible humans ( $S_H$ ), Malaria exposed humans ( $E_M$ ), HBV exposed humans ( $E_H$ ), individuals infected with Malaria only ( $I_M$ ), individuals infected with HBV only ( $I_H$ ), individuals infected with both Malaria and HBV ( $I_{MH}$ ), and those recovered from Malaria, HBV, and from both Malaria and HBV, respectively, ( $R_M$ ), ( $R_H$ ), and ( $R_{MH}$ ). Total vector

population ( $N_V$ ) which is divided into the following sub compartments; susceptible mosquitos ( $S_V$ ) and infected mosquitos ( $I_V$ ). These two different classes of population therefore give us the following equations:

$$N_H = S_H + E_M + E_H + I_M + I_H + I_{MH} + R_M + R_H + R_{MH} \quad \dots (1)$$

$$N_V = S_V + I_V \quad \dots (2)$$

For the human population, the susceptible increase by recruitment rate of  $\Lambda_h$  and also from recovered Malaria compartment with rate of  $\gamma_1$ , HBV recovered compartment with rate of  $\gamma_2$  and from compartment of recovered co-infectious with rate of  $\gamma_3$ . An infectious female anopheles mosquito from  $I_V$ , usually attacks a susceptible human in  $S_H$  through piercing and sucking and this introduces an enzyme from the saliva into human's bloodstream. In the process, the parasite is injected into the blood and this moves the susceptible human to the malaria exposed class  $E_M$  with a rate  $\alpha_1$ . This class ( $E_M$ ) reduces due to the progression of malaria exposed humans to malaria infected humans at a rate of  $t_1$  as well as due to death from natural causes. In a similar manner the population of the HBV exposed humans,  $E_H$ , grows as a result of infection of individuals in susceptible humans at the rate  $\alpha_2$ . This class reduces due to the progression of HBV exposed humans to HBV infected humans at a rate of  $t_2$  as well as due to death from natural causes. Forces of infection of Malaria and HBV are  $\alpha_1$  and  $\alpha_2$  respectively where  $\alpha_1 = \frac{\alpha_{mh}b_m I_V}{N_H}$  and  $\alpha_2 = \alpha_H(I_H + I_{MH})$ .

Since the liver condition in humans infected by malaria increase susceptibility of humans to HBV infection, it means that, the rate of infection of humans in  $I_M$  is higher than those in  $S_H$ , so we present a parameter  $\psi_1 > 1$  to alter the HBV infection rate of  $I_M$  from  $\alpha_2$  to  $\psi_1 \alpha_2$ . Similarly, HBV infection in humans increase susceptibility of humans to Malaria infection, it means that, the rate of infection of humans in  $I_H$  is higher than those in  $S_H$ , so we present a parameter  $\psi_2 > 1$  to alter the malaria infection rate of  $I_H$  from  $\alpha_1$  to  $\psi_2 \alpha_1$ . Malaria – only recovered compartment is increased by the rate,  $\beta_1$ . HBV – only compartment is increased by the rate,  $\beta_2$  while the co-infection recovered compartments increase with a rate of  $\delta$ . In the recovered co-infectious compartment, either the individuals recovered only from Malaria, HBV, or from the two diseases with probability  $\delta e, \delta f(1 - e)$  or  $\delta(1 - f)(1 - e)$ , respectively, with  $0 \leq e \leq 1$  and  $0 \leq f \leq 1$ . The natural death rate is represented by  $\mu_h$  and the rate Malaria – causing death and rate of HBV – causing death are represented by  $d_1$  and  $d_2$ , respectively. For the mosquito population, the compartment that are susceptible increase by a constant recruitment rate of  $\Lambda_v$  and decrease by the mosquito

death rate represented by  $\mu_v$ . While new infected mosquitoes are generated at a rate of  $\xi_v = \frac{\alpha_{hm}b_m}{N_H}(I_M + I_{MH})$ , and are removed from the compartment through the natural death rate of mosquito. Parameters used in

this model are assumed to be nonnegative. Based on the details given above, the co-infection dynamics are mathematically represented using the following system of Ordinary Differential Equations (ODEs).

$$\frac{dS_H}{dt} = \Lambda_h + \gamma_1 R_M + \gamma_2 R_H + \gamma_3 R_{MH} - (\alpha_1 + \alpha_2 + \mu_h) S_H \quad \dots (3)$$

$$\frac{dE_M}{dt} = \alpha_1 S_H - (\mu_h + t_1) E_M \quad \dots (4)$$

$$\frac{dE_H}{dt} = \alpha_2 S_H - (\mu_h + t_2) E_H \quad \dots (5)$$

$$\frac{dI_M}{dt} = t_1 E_M - (\psi_1 \alpha_2 + \mu_h + d_1 + \beta_1) I_M \quad \dots (6)$$

$$\frac{dI_H}{dt} = t_2 E_H - (\psi_2 \alpha_1 + \mu_h + d_2 + \beta_2) I_H \quad \dots (7)$$

$$\frac{dI_{MH}}{dt} = \psi_1 \alpha_2 I_M + \psi_2 \alpha_1 I_H - (\mu_h + d_1 + d_2 + \delta) I_{MH} \quad \dots (8)$$

$$\frac{dR_M}{dt} = \beta_1 I_M + \delta e I_{MH} - (\mu_h + \gamma_1) R_M \quad \dots (9)$$

$$\frac{dR_H}{dt} = \beta_2 I_H + (\delta f(1 - e)) I_{MH} - (\mu_h + \gamma_2) R_H \quad \dots (10)$$

$$\frac{dR_{MH}}{dt} = (\delta(1 - f)(1 - e)) I_{MH} - (\mu_h + \gamma_3) R_{MH} \quad \dots (11)$$

$$\frac{dS_V}{dt} = \Lambda_v - (\mu_v + \xi_v) S_V \quad \dots (12)$$

$$\frac{dI_V}{dt} = \xi_v S_V - \mu_v I_V \quad \dots (13)$$

initial conditions of model (equations 3 –13) are

$$S_H(0) \geq 0 \quad \dots (14)$$

$$E_M(0) \geq 0 \quad \dots (15)$$

$$E_H(0) \geq 0 \quad \dots (16)$$

$$I_M(0) \geq 0 \quad \dots (17)$$

$$I_H(0) \geq 0 \quad \dots (18)$$

$$I_{MH}(0) \geq 0 \quad \dots (19)$$

$$R_M(0) \geq 0 \quad \dots (20)$$

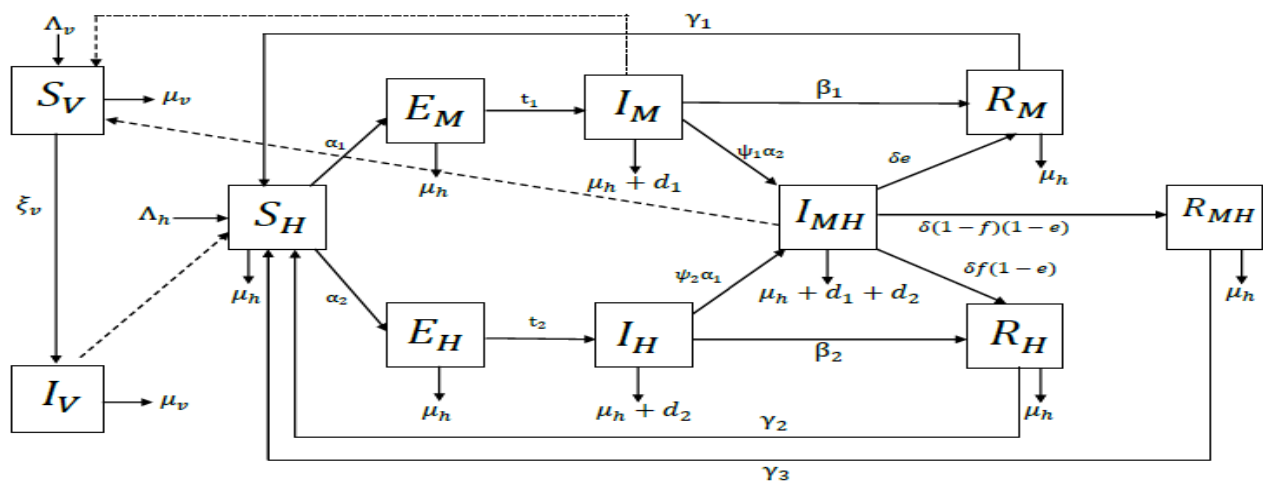
$$R_H(0) \geq 0 \quad \dots (21)$$

$$R_{MH}(0) \geq 0 \quad \dots (22)$$

$$S_V(0) \geq 0 \quad \dots (23)$$

$$I_V(0) \geq 0 \quad \dots (24)$$

From the above system of differential equations, the following diagram is obtained



**Figure 1: Co-infection model schematic diagram**

### 2.1.2 Invariant Region Malaria HBV Co-infection Model

We demonstrate that the solution to the mathematical model (equations 3-13) is both epidemiologically and mathematically valid, and that a positive solution exists within a well-defined invariant region. To establish the boundaries of this invariant region, where the model's solution remains confined, we considered the total population dynamics.

$$N_H = S_H + E_M + E_H + I_M + I_H + I_{MH} + R_M + R_H + R_{MH}; N_V = S_V + I_V \quad \dots (24)$$

Now,

$$\begin{aligned} \frac{dN_H}{dt} &= \frac{dS_H}{dt} + \frac{dE_M}{dt} + \frac{dE_H}{dt} + \frac{dI_M}{dt} + \frac{dI_H}{dt} + \frac{dI_{MH}}{dt} + \frac{dR_M}{dt} + \frac{dR_H}{dt} + \frac{dR_{MH}}{dt} \\ &= \Lambda_h - \mu_h N_H - d_1(I_M + I_{MH}) - d_2(I_H + I_{MH}) \end{aligned} \quad \dots (25)$$

Similarly,

$$\frac{dN_V}{dt} = \Lambda_v - \mu_v N_V \quad \dots (26)$$

Assuming  $d_1 = d_2 = 0$  and letting  $\begin{pmatrix} N_H \\ N_V \end{pmatrix}, \Lambda = \begin{pmatrix} \Lambda_h \\ \Lambda_v \end{pmatrix}$  and  $\mu_a = \begin{pmatrix} \mu_h & 0 \\ 0 & \mu_v \end{pmatrix}$ , we obtain

$$\frac{dN}{dt} \leq \Lambda - \mu_a N \quad \dots (27)$$

Solving equation (27) we obtain

$$\Omega = \left\{ (S_H, E_M, E_H, I_M, I_H, I_{MH}, R_M, R_H, R_{MH}, S_V, I_V) \in \mathbb{R}^{11}, 0 \leq N \leq \frac{\Lambda}{\mu_a} \right\} \quad \dots (28)$$

which is the model invariant region for the solution to the model.

### 2.1.3 Positivity of the Solution

#### Theorem 1

If

$$S_{H_0} > 0, E_{M_0} > 0, E_{H_0} > 0, I_{M_0} > 0, I_{H_0} > 0, I_M > 0, R_{M_0} > 0, R_{H_0} > 0, R_{MH_0} > 0, S_{V_0} > 0, I_{V_0} > 0,$$

then all the solution sets

$$(S_H(t), E_M(t), E_H(t), I_M(t), I_H(t), I_{MH}(t), R_M(t), R_H(t), R_{MH}(t), S_V(t), I_V(t))$$

are positive for future time.

**Proof:** To prove by contradiction, firstly, take  $t_a$  as

$$t_a = \sup\{t > 0: S_H(\tau) > 0, E_M(\tau) > 0, E_H(\tau) > 0, I_M(\tau) > 0, I_H(\tau) > 0, I_{MH}(\tau) > 0, R_M(\tau) > 0, R_H(\tau) > 0, R_{MH}(\tau) > 0, S_V(\tau) > 0, I_V(\tau) > 0 \text{ for all } \tau \in [0, t]\} \quad \dots (29)$$

Now consider

$$S_{H_0} \geq 0, E_{M_0} \geq 0, E_{H_0} \geq 0, I_{M_0} \geq 0, I_{H_0} \geq 0, I_{MH_0} \geq 0, R_{M_0} \geq 0, R_{H_0} \geq 0, R_{MH_0} \geq 0, S_{V_0} \geq 0, I_{V_0} \geq 0;$$

thus,  $t_a > 0$ .

If  $t_a < \infty$ , then necessarily,  $S_H$  or  $E_M$  or  $E_H$  or  $I_M$  or  $I_H$  or  $I_{MH}$  or  $R_M$  or  $R_H$  or  $R_{MH}$  or  $S_V$  or  $I_V$  is equal to zero at  $t_a$ . From equation (3– 13), we have

$$\frac{dS_H(t)}{dt} = \Lambda_h + \gamma_1 R_M + \gamma_2 R_H + \gamma_3 R_{MH} - (\alpha_1 + \alpha_2 + \mu_h) S_H(t) \quad \dots (30)$$

Equation 30 can be solved using variation formula at  $t_a$ :

$$S_H(t_a) = S_H(0) \exp \left[ - \int_0^{t_a} (\alpha_1 + \alpha_2 + \mu_h)(s) ds \right] + \int_0^{t_a} (\Lambda_h + \gamma_1 R_M + \gamma_2 R_H + \gamma_3 R_{MH}) \exp \left[ - \int_s^{t_a} (\alpha_1 + \alpha_2 + \mu_h)(\tau) d\tau \right] ds \quad \dots (31)$$

Consequently, all variables are non negative in  $[0, t_a]$ ; then  $S_H(t_a) > 0$ .

In a similar manner, we can show

$$E_M(t_a) > 0, E_H(t_a) > 0, I_M(t_a) > 0, I_H(t_a) > 0, I_{MH}(t_a) > 0, R_M(t_a) > 0, R_H(t_a) > 0, R_{MH}(t_a) > 0, S_V(t_a) > 0 \text{ and } I_V(t_a) > 0 \text{ which is a contradiction. Hence } t_a = \infty. \quad \blacksquare$$

### 2.1.4 Existence of Disease Free Equilibrium (DFE) Point for the Co-infection Model.

At Disease Free Equilibrium point, ( $E_0$ ), none of the diseases exists. The disease free equilibrium point, no Malaria HBV co-infection exist. At Disease-free Equilibrium  $E_0$ ;  $E_M = E_H = I_M = I_H = I_{MH} = I_V = 0$ . Therefore, eliminating  $E_M, E_H, I_M, I_H, I_{MH}$  and  $I_V$  from equation (3 – 13) and equating right hand side of the equation to zero, we get

$$\Lambda_h + \gamma_1 R_M + \gamma_2 R_H + \gamma_3 R_{MH} - \mu_h S_H = 0 \quad \dots (32)$$

$$-(\mu_h + \gamma_1) R_M = 0 \quad \dots (33)$$

$$-(\mu_h + \gamma_2) R_H = 0 \quad \dots (34)$$

$$-(\mu_h + \gamma_3) R_{MH} = 0 \quad \dots (35)$$

$$\Lambda_v - \mu_v S_V = 0 \quad \dots (36)$$

From equations (32 –36) we get  $R_M = R_H = R_{MH} = 0$ , and the DFE point is

$$E_0 = (S_H, E_M, E_H, I_M, I_H, I_{MH}, R_M, R_H, R_{MH}, S_V, I_V) = \left( \frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0 \right) \dots (37)$$

### 3.0 RESULTS

#### 3.1 Computation of the Basic Reproduction Number

This section focuses on computing the basic reproduction number, denoted as  $\mathcal{R}_0$ , utilizing the next-generation method, as described by Sukhamoy and Susmita (2020).  $\mathcal{R}_0$  represents the average number of secondary infections generated by an infectious individual within a fully susceptible population. This critical threshold plays a pivotal role in understanding the spread and control of diseases. An  $\mathcal{R}_0$  value less than 1 indicates the potential for disease elimination, whereas an  $\mathcal{R}_0$  value greater than 1 suggests the disease will become endemic. To calculate  $\mathcal{R}_0$ , the next-generation operator method is employed, which involves dividing the compartmental model into infected and non-infected compartments. The model equations are then rearranged, starting with the infected compartments, and the resulting relationship is used to derive  $\mathcal{R}_0$ .

$$\mathcal{R}_0 = \rho(FV^{-1}) = \max\{\lambda_i\}_{i=1,2,\dots} \dots (38)$$

where

$\rho(FV^{-1})$  Denotes spectral radius in the next generation matrix  $FV^{-1}$  with F and V denoting the matrices at fresh infections and transition or infection transfer respectively.

##### 3.1.1 Basic Reproduction Number for the Malaria HBV Co-infection Model

This section deals with the basic reproduction number computation usually denoted by  $\mathcal{R}_0$  for the Malaria HBV Co-infection Model by the next generation method as applied in (Sukhamoy & Susmita, 2020). From the model equations, using their approach we first rearrange the model equations beginning with the infective classes to obtain the following equations

$$\frac{dE_M}{dt} = \frac{\alpha_{mh}b_mI_V}{N_H}S_H - (\mu_h + t_1)E_M \dots (39)$$

$$\frac{dE_H}{dt} = \alpha_H(I_H + I_{MH})S_H - (\mu_h + t_2)E_H \dots (40)$$

$$\frac{dI_M}{dt} = t_1E_M - (\psi_1\alpha_H(I_H + I_{MH}) + \mu_h + d_1 + \beta_1)I_M \dots (41)$$

$$\frac{dI_H}{dt} = t_2E_H - \left(\psi_2 \frac{\alpha_{mh}b_mI_V}{N_H} + \mu_h + d_2 + \beta_2\right)I_H \dots (42)$$

$$\frac{dI_{MH}}{dt} = \psi_1\alpha_H(I_H + I_{MH})I_M + \psi_2 \frac{\alpha_{mh}b_mI_V}{N_H}I_H - (\mu_h + d_1 + d_2 + \delta)I_{MH} \dots (43)$$

$$\frac{dI_V}{dt} = \frac{\alpha_{hm}b_m(I_M + I_{HM})}{N_H}S_V - \mu_vI_V \dots (44)$$

Using their approach, we have;

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\alpha_{mh}b_mS_H}{N_H} \\ 0 & 0 & 0 & \alpha_H S_H & \alpha_H S_H & 0 \\ t_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & t_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \psi_1\alpha_H(I_H + I_{MH}) & \psi_1\alpha_H I_M + \psi_2 \frac{\alpha_{mh}b_mI_V}{N_H} & \psi_1\alpha_H I_M & \psi_2 \frac{\alpha_{mh}b_mI_H}{N_H} \\ 0 & 0 & \frac{\alpha_{hm}b_mS_V}{N_H} & 0 & \frac{\alpha_{hm}b_mS_V}{N_H} & 0 \end{pmatrix} \dots (45)$$

$$V = \begin{pmatrix} \mu_h + t_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \mu_h + t_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \psi_1 \alpha_H (I_H + I_{MH}) + \mu_h + d_1 + \beta_1 & 0 & \psi_1 \alpha_H I_M & 0 \\ 0 & 0 & 0 & \psi_2 \frac{\alpha_{mh} b_m I_V}{N_H} + \mu_h + d_2 + \beta_2 & 0 & \psi_2 \frac{\alpha_{mh} b_m I_H}{N_H} \\ 0 & 0 & 0 & 0 & \mu_h + d_1 + d_2 + \delta & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_v \end{pmatrix} \dots (46)$$

We now obtain  $F$  and  $V$  at the DFE point  $E_0$  and find  $FV^{-1}|_{E_0}$  as follows

$$FV^{-1}|_{E_0} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{\alpha_{mh} b_m}{\mu_v} \\ 0 & 0 & 0 & \frac{\alpha_H \Lambda_h}{(\mu_h + d_2 + \beta_2) \mu_h} & \frac{\alpha_H \Lambda_h}{(\mu_h + d_1 + d_2 + \delta) \mu_h} & 0 \\ \frac{t_1}{\mu_h + t_1} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{t_2}{\mu_h + t_2} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\alpha_{hm} b_m \mu_h \Lambda_v}{(\mu_h + d_1 + \beta_1) \Lambda_h \mu_v} & 0 & \frac{\alpha_{hm} b_m \mu_h \Lambda_v}{(\mu_h + d_1 + d_2 + \delta) \Lambda_h \mu_v} & 0 \end{pmatrix} \dots (47)$$

Therefore,  $\mathcal{R}_0 = \rho(FV^{-1})$  is spectral radius of  $FV^{-1}$ .

$$\mathcal{R}_0 = \max \left\{ \sqrt[3]{\frac{t_1 \alpha_{mh} \alpha_{hm} (b_m)^2 \mu_h \Lambda_v}{(\mu_h + t_1)(\mu_h + d_1 + \beta_1)(\mu_v)^2 \Lambda_h}}, \sqrt{\frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2) \mu_h}} \right\} \dots (48)$$

### 3.2 Model Stability Analysis

#### 3.2.1 DFE Point Stability Analysis for Malaria HBV Co-infection Model

The following theorem explains the local asymptotical stability of disease free case:

Theorem 2

If  $\mathcal{R}_0 < 1$ , then equilibrium point  $E_0$  of model (equations 3 – 13) is Locally Asymptotically Stable (L A S) and unstable if  $\mathcal{R}_0 > 1$ .

Proof. Assume  $\mathcal{R}_0 < 1$ ,

Evaluating Jacobian ( $J_0$ ) matrix of the model at the DFE point we get,

$$J_0 = \begin{pmatrix} j_{11} & 0 & 0 & 0 & -\frac{\alpha_H \Lambda_h}{\mu_h} & -\frac{\alpha_H \Lambda_h}{\mu_h} & \gamma_1 & \gamma_2 & \gamma_3 & 0 & -\alpha_{mh} b_m \\ 0 & j_{22} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{mh} b_m \\ 0 & 0 & j_{33} & 0 & \frac{\alpha_H \Lambda_h}{\mu_h} & \frac{\alpha_H \Lambda_h}{\mu_h} & 0 & 0 & 0 & 0 & 0 \\ 0 & t_1 & 0 & j_{44} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & t_2 & 0 & j_{55} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & j_{66} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_1 & 0 & \delta e & j_{77} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_2 & \delta f(1 - e) & 0 & j_{88} & 0 & 0 & \dots (50) \\ 0 & 0 & 0 & 0 & 0 & \delta(1 - f)(1 - e) & 0 & 0 & j_{99} & 0 & \dots (51) \\ 0 & 0 & 0 & 0 & -\frac{\alpha_{hm} b_m \mu_h \Lambda_v}{\Lambda_h \mu_v} & -\frac{\alpha_{hm} b_m \mu_h \Lambda_v}{\Lambda_h \mu_v} & 0 & 0 & 0 & j_{110} & \dots (52) \\ 0 & 0 & 0 & 0 & \frac{\alpha_{hm} b_m \mu_h \Lambda_v}{\Lambda_h \mu_v} & \frac{\alpha_{hm} b_m \mu_h \Lambda_v}{\Lambda_h \mu_v} & 0 & 0 & 0 & 0 & j_{111} \end{pmatrix} \dots (49)$$

where

$$j_{11} = -\mu_h$$

$$j_{22} = -(\mu_h + t_1)$$

$$j_{33} = -(\mu_h + t_2)$$

$$j_{44} = -(\mu_h + d_1 + \beta_1) \quad \dots (53)$$

$$j_{55} = -(\mu_h + d_2 + \beta_2) \quad \dots (54)$$

$$j_{66} = -(\mu_h + d_1 + d_2 + \delta) \quad \dots (55)$$

$$j_{77} = -(\mu_h + \gamma_1) \quad \dots (56)$$

$$j_{88} = -(\mu_h + \gamma_2) \quad \dots (57)$$

$$j_{99} = -(\mu_h + \gamma_3) \quad \dots (58)$$

$$j_{110} = -\mu_v \quad \dots (59)$$

$$j_{99} = -\mu_v \quad \dots (60)$$

For the DFE to be LAS, the eigenvalues of Jacobian matrix evaluated at the DFE point must have real parts that are negative. To find the eigenvalues, we need the characteristics equation which is,

$$(j_{110} - \lambda)(j_{111} - \lambda)(j_{77} - \lambda)(j_{44} - \lambda)(j_{88} - \lambda)(j_{22} - \lambda)(j_{111} - \lambda)(j_{99} - \lambda) \left( \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - (j_{33} - \lambda)(j_{55} - \lambda) \right) = 0 \quad \dots (61)$$

We will now show that all eigenvalues have real parts that are negative.

Eight eigenvalues are obtained from the seven linear factors of the characteristics equation, these are:

$$\lambda = j_{110} = -\mu_v < 0 \quad \text{since } \mu_v > 0 \quad \dots (62)$$

$$\lambda = j_{111} = -\mu_h < 0 \quad \text{since } \mu_h > 0 \quad \dots (63)$$

$$\lambda = j_{77} = -(\mu_h + \gamma_1) < 0 \quad \text{since } (\mu_h + \gamma_1) > 0 \quad \dots (64)$$

$$\lambda = j_{44} = -(\mu_h + d_1 + \beta_1) < 0 \quad \text{since } (\mu_h + d_1 + \beta_1) > 0 \quad \dots (65)$$

$$\lambda = j_{88} = -(\mu_h + \gamma_2) < 0 \quad \text{since } (\mu_h + \gamma_2) > 0 \quad \dots (66)$$

$$\lambda = j_{22} = -(\mu_h + t_1) < 0 \quad \text{since } (\mu_h + t_1) > 0 \quad \dots (67)$$

$$\lambda = j_{111} = -\mu_v < 0 \quad \text{since } \mu_v > 0 \quad \dots (68)$$

$$\lambda = j_{99} = -(\mu_h + \gamma_3) < 0 \quad \text{since } (\mu_h + \gamma_3) > 0 \quad \dots (69)$$

The remaining 2 eigenvalues can be gotten from the following quadratic equation

$$\frac{t_2 \alpha_H \Lambda_h}{\mu_h} - (j_{33} - \lambda)(j_{55} - \lambda) = 0 \quad \dots (70)$$

Rearranging equation 70 we get the following quadratic equation

$$a_2 \lambda^2 + a_1 \lambda + a_0 = 0 \quad \dots (71)$$

where

$$a_2 = -1; a_1 = (j_{33} + j_{55}); a_0 = \left( \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - j_{33} j_{55} \right) \quad \dots (72)$$

We apply the Routh – Hurwitz criteria.

The Routh – Hurwitz array for  $(a_2 \lambda^2 + a_1 \lambda + a_0)$  is

$$\begin{array}{c|cc} \lambda^2 & a_2 & a_0 \\ \lambda^1 & a_1 & 0 \\ \lambda^0 & a_0 & \end{array}$$

If the signs of  $a_2, a_1$  and  $a_0$  are the same, then we conclude that all eigenvalues for  $(a_2 \lambda^2 + a_1 \lambda + a_0)$  have negative real parts.

**To prove:  $a_2 < 0$  and  $a_1 < 0$ :**

**Proof:**

$$a_2 = -1 < 0 \quad \dots (73)$$

$$a_1 = (j_{33} + j_{55}) = -(\mu_h + t_2) + -(\mu_h + d_2 + \beta_2) = -(2\mu_h + t_2 + d_2 + \beta_2) < 0 \quad \dots (74)$$

**To prove:  $a_0 < 0$**

**Proof:**

$$\begin{aligned} a_0 &= \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - j_{33} j_{55} = \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - (-(\mu_h + t_2)(-\mu_h + d_2 + \beta_2)) \\ &= \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - (\mu_h + t_2)(\mu_h + d_2 + \beta_2) \end{aligned} \quad \dots (75)$$

$$\mathcal{R}_0 = \max\{\mathcal{R}_0^M, \mathcal{R}_0^H\}$$

$$\text{where } \mathcal{R}_0^M = \sqrt[3]{\frac{t_1 \alpha_m h \alpha_{hm} (b_m)^2 \mu_h \Lambda_v}{(\mu_h + t_1)(\mu_h + d_1 + \beta_1)(\mu_v)^2 \Lambda_h}} \quad \text{and } \mathcal{R}_0^H = \sqrt{\frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2) \mu_h}}$$

$$\mathcal{R}_0 = \sqrt{\frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2) \mu_h}} < 1,$$

$$\begin{aligned} &\Rightarrow \sqrt{\frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2)\mu_h}} < 1 \\ &\Rightarrow \frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2)\mu_h} < 1 \\ &\Rightarrow \frac{t_2 \alpha_H \Lambda_h}{\mu_h} < (\mu_h + t_2)(\mu_h + d_2 + \beta_2) \\ &\Rightarrow \frac{t_2 \alpha_H \Lambda_h}{\mu_h} - (\mu_h + t_2)(\mu_h + d_2 + \beta_2) < 0 \\ &\Rightarrow a_0 < 0 \text{ from equation 75.} \end{aligned}$$

Hence, the eigenvalues of Jacobian matrix evaluated at the DFE point have real parts that are negative. This completes the proof that the DFE for the co-infection Model is LAS if  $\mathcal{R}_0 < 1$  and unstable whenever  $\mathcal{R}_0 > 1$  (since not every eigenvalues have real parts that are negative if  $\mathcal{R}_0 > 1$ ).

### 3.3 Sensitivity Analysis of Parameters

The sensitivity index of  $\mathcal{R}_0$  with respect to  $p$  is,

$$S_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0} \dots (76)$$

If  $S_p^{\mathcal{R}_0} < 0$  for  $p$ , then it means that  $\mathcal{R}_0$ , decreases when  $p$  is increased while if  $S_p^{\mathcal{R}_0} > 0$  for  $p$ , then it means that  $\mathcal{R}_0$  increases when the  $p$  is increased (Getachew, 2019).

The sensitivity indices for each parameter in  $\mathcal{R}_0$  is summarized in the following table

$$\begin{aligned} \mathcal{R}_0 &= \max\{\mathcal{R}_0^M, \mathcal{R}_0^H\} \\ &= \max\left\{\sqrt[3]{\frac{t_1 \alpha_{mh} \alpha_{hm} (b_m)^2 \mu_h \Lambda_v}{(\mu_h + t_1)(\mu_h + d_1 + \beta_1)(\mu_v)^2 \Lambda_h}}, \sqrt{\frac{t_2 \alpha_H \Lambda_h}{(\mu_h + t_2)(\mu_h + d_2 + \beta_2)\mu_h}}\right\} \dots (77) \end{aligned}$$

**Table 1: Sensitivity Indices of Parameter**

Parameter	Description	Indices	Indices
		$\mathcal{R}_0 = \mathcal{R}_0^M$	$\mathcal{R}_0 = \mathcal{R}_0^H$
$\Lambda_v$	Constant recruitment rate of mosquitoes	Positive	–
$\mu_v$	Natural death rate of mosquitoes	Negative	–
$d_1$	Malaria induced death rate in humans	Negative	–
$d_2$	HBV induced death rate in humans	–	Negative
$\beta_1$	Malaria recovery rate in humans	Negative	–
$\beta_2$	HBV recovery rate in humans	–	Negative
$\alpha_{hm}$	Probability of transmission of malaria in mosquitoes	Positive	–
$\alpha_{mh}$	Probability of transmission of malaria in humans	Positive	–
$\alpha_H$	Probability of transmission for HBV infection in humans	–	Positive
$t_1$	Malaria exposed humans infection rate	Positive	–
$t_2$	HBV infection exposed humans infection rate	–	Positive
$b_m$	Maximum number of mosquito bites	Positive	–

## 4.0 DISCUSSION

### 4.1 Numerical Simulation

This section presents the results of numerical simulations conducted on the Malaria-HBV co-infection model, focusing on the impact of various parameters on population dynamics. A table is provided, outlining the variable and parameter values, as well as the initial population sizes for each compartment, used in the simulations. For illustrative purposes, some parameter values and initial population sizes were assumed. The numerical solutions of the system of ordinary differential equations (ODEs) were obtained using the Maple 17 Range-Kutta (RK4) fourth-order scheme.

We will in the following section conduct numerical simulation to see the effects of some selected parameter

on the infected and recovered population. We selected some parameters as follows:

- i. HBV recovery rate in humans from  $I_H$  state to  $R_H$  state ( $\beta_2$ ): This has a negative sensitivity index sign on basic reproduction number ( $\mathcal{R}_0$ ) which means that  $\mathcal{R}_0$  will decrease when the value of  $\beta_2$  increases, this in turn will have an effect on the model especially the compartment with infectious humans (Dominic, Isaac, Shaibu, & Elvis, 2021). We will see how change in this parameter affects the model.
- ii. Transmission probability for malaria in humans ( $\alpha_{mh}$ ): This has a positive sensitivity index sign on basic reproduction number ( $\mathcal{R}_0$ ) which means that  $\mathcal{R}_0$  will decrease when the value of  $\alpha_{mh}$  decreases, this in turn will have an effect on the model especially the compartment with infectious

humans (Jones, Feng-Bin, & Naveen, 2015). We will see how change in this parameter affects the model.

iii. Probability of transmission of malaria in mosquitoes ( $\alpha_{hm}$ ): This has a positive sensitivity index sign on basic reproduction number ( $\mathcal{R}_0$ )

which means that  $\mathcal{R}_0$  will decrease when the value of  $\alpha_{hm}$  decreases, this in turn will have an effect on the model especially the compartment with infectious humans (Jones, Feng-Bin, & Naveen, 2015). We will see how change in this parameter affects the model.

**Table 2: Tables of parameter values**

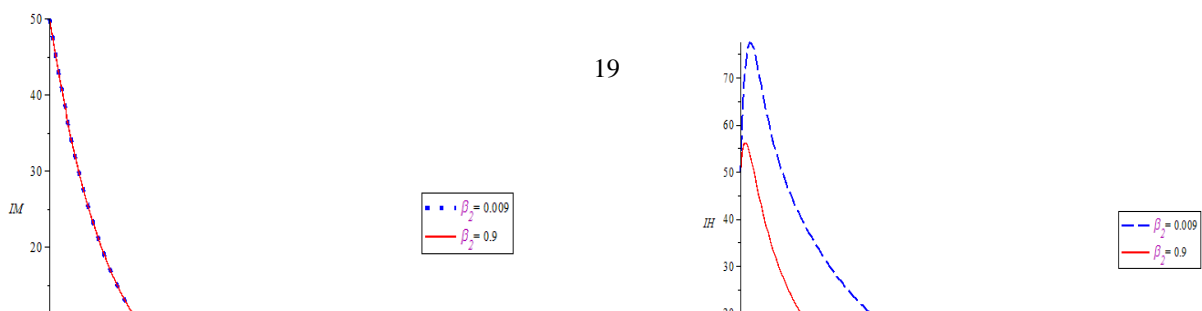
S/N	Parameter	Value (day <sup>-1</sup> )	Source
1	$\Lambda_h$	$9.6274 \times 10^{-5}$	(Onyango, Ogada, Thirika, & Lawi, 2018)
2	$\mu_h$	0.00004	(Jones, Feng-Bin, & Naveen, 2015)
3	$d_1$	0.6	(Jones, Feng-Bin, & Naveen, 2015)
4	$d_2$	0.0141	(Ebenezer, Rahat, & Fatma, 2020)
5	$\alpha_1$	0.02	(Segun, Michael, Michael, & Maba, 2020)
6	$\alpha_2$	0.169	(Magaji, Mubarak, & Dauda, 2019)
7	$\beta_1$	0.038	(Jones, Feng-Bin, & Naveen, 2015)
8	$\beta_2$	0.9738	(Hussam, et al., 2020)
9	$\gamma_1$	0.00156	(Onyango, Ogada, Thirika, & Lawi, 2018)
10	$\gamma_2$	0.01	(Hussam, et al., 2020)
11	$\gamma_3$	0.05	Assumed
12	$\delta$	0.45	Assumed
13	$e$	0.5	Assumed
14	$f$	0.5	Assumed
15	$\Lambda_v$	0.071	(Onyango, Ogada, Thirika, & Lawi, 2018)
16	$\mu_v$	0.1429	(Onyango, Ogada, Thirika, & Lawi, 2018)
17	$\xi_v$	0.312	Assumed
18	$\alpha_{hm}$	0.25	(Onyango, Ogada, Thirika, & Lawi, 2018)
19	$\alpha_{mh}$	0.8333	(Onyango, Ogada, Thirika, & Lawi, 2018)
20	$\alpha_H$	0.5	(Hussam, et al., 2020)
21	$b_m$	12	(Jones, Feng-Bin, & Naveen, 2015)
22	$\psi_1$	1.2	Assumed
23	$\psi_2$	1.2	Assumed
24	$t_1$	0.02	(Muhamad & Fajar, 2020)
25	$t_2$	<b>0.0550</b>	(Dominic, Isaac, Shaibu, & Elvis, 2021)

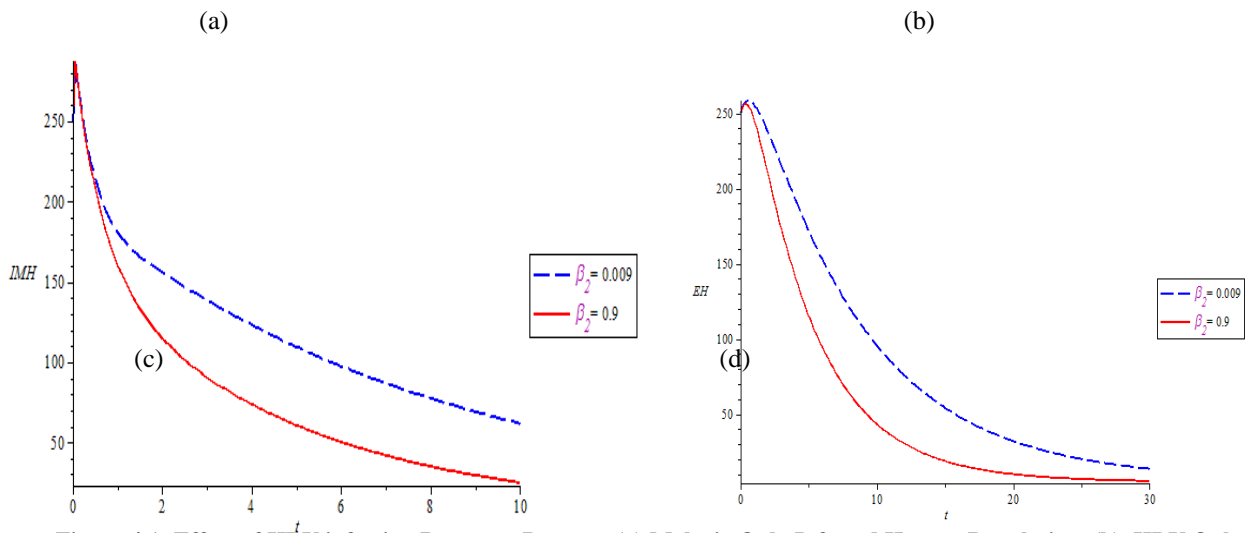
**Table 3: Tables of compartment initial population**

S/N	Compartment	Initial Population
1	$S_H$	200
2	$E_M$	60
3	$E_H$	60
4	$I_M$	250
5	$I_H$	250
6	$I_{MH}$	250
7	$R_M$	50
8	$R_H$	50
9	$R_{MH}$	40
10	$S_V$	260
11	$I_V$	120

**4.1.1 Effect of HBV infection recovery rate on Infectious Compartments:**

Figure 4.1 (a) shows that when the recovery rate of HBV infection increases, the number of Malaria only infectious population shows no significant change. We can see from the figure that as the HBV infection recovery rate is increased (from  $\beta_2 = 0.009$  to  $\beta_2 = 0.9$ ), the Malaria only infected Humans was not affected significantly. This means that stakeholders cannot use the recovery rate as a control for Malaria only infected population.





**Figure 4.1: Effect of HBV infection Recovery Rate on: (a) Malaria Only Infected Human Population, (b) HBV Only Infected Human Population, (c) Malaria HBV Co-infected Human Population, (d) HBV infection Exposed Population**

Figure 4.1 (b) shows when the HBV infection recovery rate is increased, the number of HBV Only infection population also decreases. We can see from the figure that as the HBV infection recovery rate is increased (from  $\beta_2 = 0.009$  to  $\beta_2 = 0.9$ ), the HBV only infected Humans decreases as expected. This means that stakeholders can use the recovery rate as a control for infected population – by increasing the HBV infection recovery rate which can be done using various means such as increasing HBV treatment etc. Therefore, different policies can be made which will concentrate on the maximization of the HBV infection recovery rate by way of treatment of those infected or by immunity boosting of individuals to the HBV disease.

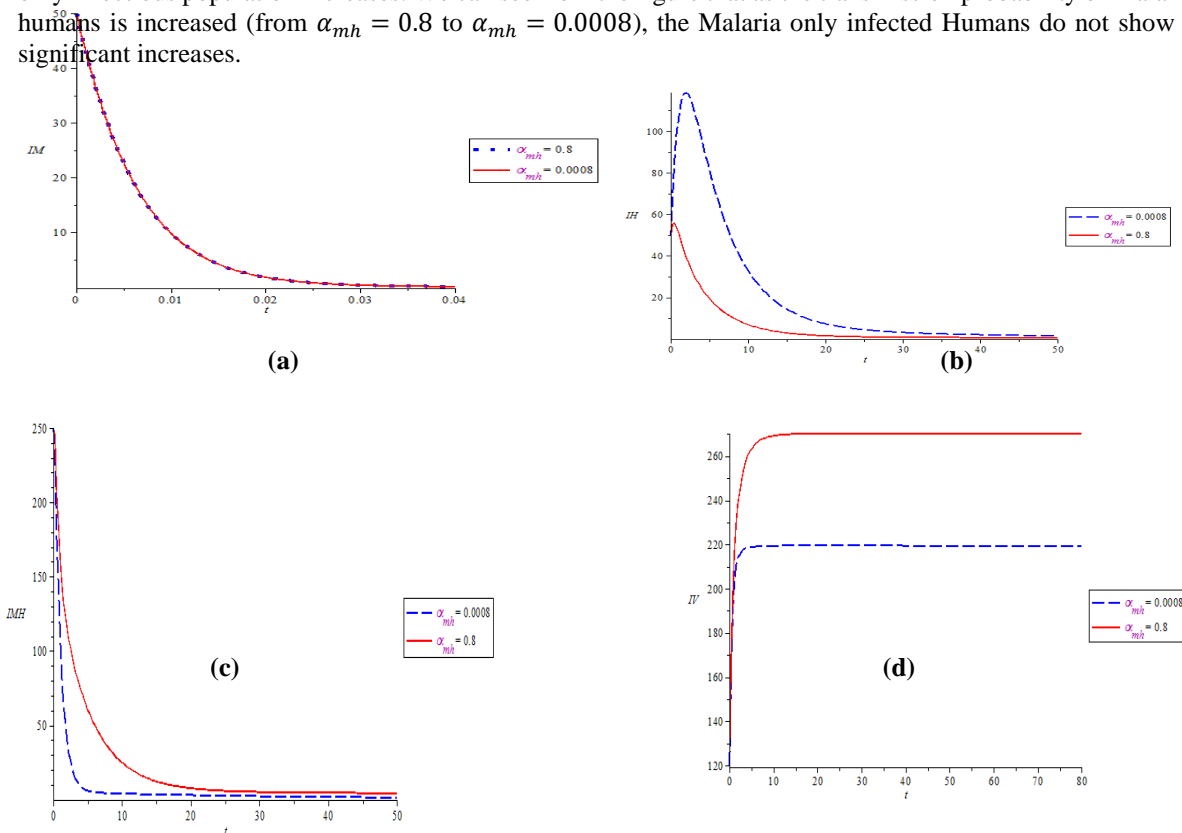
Figure 4.1 (c) reveals that when HBV infection recovery rate is increased; number of Malaria HBV co-infection population also decreases. We can see from the figure that as the HBV infection recovery rate is increased (from  $\beta_2 = 0.009$  to  $\beta_2 = 0.9$ ), the Malaria HBV co-infected Humans decreases as expected. This means that stakeholders can use the recovery rate as a control for the co-infected population – by increasing the HBV infection recovery rate which can be done

using various means such as increasing HBV infection treatment etc. Therefore, different policies can be made which will concentrate on the maximization of the HBV infection recovery rate by way of treatment of those infected or by immunity boosting of individuals to the HBV disease.

Figure 4.1 (d) illustrates that when the HBV infection recovery rate is increased, the number of HBV infection Exposed population also decreases. We can see from the figure that as the HBV infection recovery rate is increased (from  $\beta_2 = 0.009$  to  $\beta_2 = 0.9$ ), the HBV infected Exposed Humans decreases as expected. This means that stakeholders can use the recovery rate as a control for HBV infection Exposed population – by increasing the HBV infection recovery rate which can be done using various means such as increasing HBV treatment etc. Therefore, different policies can be made which will concentrate on the maximization of the HBV infection recovery rate by way of treatment of those infected or by immunity boosting of individuals to the HBV disease.

#### 4.1.2 Effect of Malaria Transmission Probability in Humans Infectious Compartments

**Figure 4.2 (a)** shows when the Transmission probability for malaria in humans is increased, the number of Malaria only infectious population increases. We can see from the figure that as the transmission probability of Malaria in humans is increased (from  $\alpha_{mh} = 0.8$  to  $\alpha_{mh} = 0.0008$ ), the Malaria only infected Humans do not show any significant increases.



**Figure 4.2: Effect of Transmission Probability of Malaria in Humans on: (a) Population of Malaria Only Infected, (b) HBV Only Infected Human Population, (c) Malaria HBV Co-infection Human Population, (d) Infected Mosquito Population**

Figure 4.2 (b) shows that when the Transmission probability for malaria in humans increases, the number of HBV only infectious population increases. We can see from the figure that as the transmission probability of Malaria in humans is increased (from  $\alpha_{mh} = 0.8$  to  $\alpha_{mh} = 0.0008$ ), the HBV only infected Humans increases as expected. This means that stakeholders can use the malaria transmission probability in humans as a control for infected population – by decreasing the malaria transmission probability in humans which can be done using various means such as application of insecticides, using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in humans by way of using mosquito nets or by practicing environmental hygiene, sanitation etc.

Figure 4.2 (c) reveals that that when the Transmission probability for malaria in humans increases, the number of Malaria HBV co-infection population increases. We can see from the figure that as the transmission probability of Malaria in humans is increased (from  $\alpha_{mh} = 0.8$  to  $\alpha_{mh} = 0.0008$ ), the

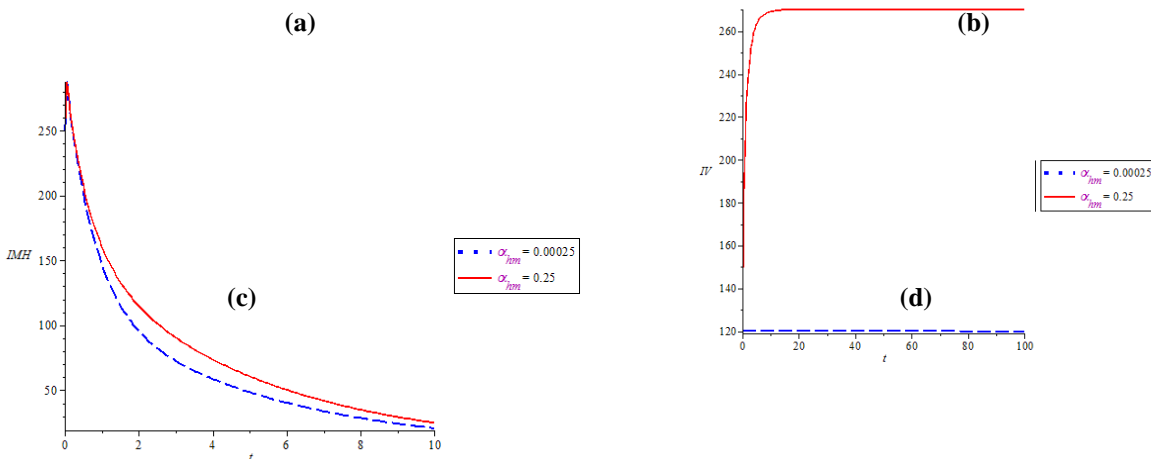
Malaria HBV co-infected Humans increases as expected. This means that stakeholders can use the malaria transmission probability in humans as a control for infected population – by decreasing the malaria transmission probability in humans which can be done using various means such as application of insecticides, using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in humans by way of using mosquito nets or by practicing environmental hygiene, sanitation etc.

Figure 4.2 (d) indicates that when the Transmission probability for malaria in humans increases, the number of Infected Mosquito remains the same. We can see from the figure that as the transmission probability of Malaria in humans is increased (from  $\alpha_{mh} = 0.8$  to  $\alpha_{mh} = 0.0008$ ), the Infected Mosquito population increases as expected. This means that stakeholders can use the malaria transmission probability in humans as a control for infected mosquito population – by decreasing the malaria transmission probability in humans which can be done using various means such as application of insecticides,

using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in humans by way of using mosquito nets or by practicing environmental hygiene, sanitation.

#### 4.1.3 Effect of Probability of transmission of malaria in mosquitoes on Infectious Compartments

**Figure 4.3 (a)** shows when the Transmission probability for malaria in mosquitoes is increased, the number of Malaria only infectious population increases. We can see from the figure that as the transmission probability of Malaria in mosquitoes is increased (from  $\alpha_{hm} = 0.25$  to  $\alpha_{hm} = 0.00025$ ), the Malaria only infected Humans do not show any significant increases.



**Figure 4.3: Effect of Transmission Probability of Malaria in Mosquitoes on: (a) Population of Malaria Only Infected, (b) HBV Only Infected Human Population, (c) Malaria HBV Co-infection Human Population, (d) Mosquito Infected Population**

Figure 4.3 (b) indicates that when the Transmission probability for malaria in mosquitoes increases, the number of HBV only infectious population increases. We can see from the figure that as the transmission probability of Malaria in mosquitoes is increased (from  $\alpha_{hm} = 0.25$  to  $\alpha_{hm} = 0.00025$ ), the HBV only infected Humans increases as expected. This means that stakeholders can use the malaria transmission probability in mosquitoes as a control for infected population – by decreasing the malaria transmission probability in mosquitoes which can be done using various means such as using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in mosquitoes by way reducing human – mosquito contacts.

Figure 4.3 (c) reveals that when the Transmission probability for malaria in mosquitoes increases, the number of Malaria HBV co-infectious population increases. We can see from the figure that as the transmission probability of Malaria in mosquitoes is increased (from  $\alpha_{hm} = 0.25$  to  $\alpha_{hm} = 0.00025$ ), the Malaria HBV co-infectious Humans increases as expected. This means that stakeholders can use the malaria transmission probability in mosquitoes as a

control for infected population – by decreasing the malaria transmission probability in mosquitoes which can be done using various means such as using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in mosquitoes by way reducing human – mosquito contacts.

Figure 4.3 (d) shows that when the Transmission probability for malaria in mosquitoes increases, the number of infected mosquitoes increases. We can see from the figure that as the transmission probability of Malaria in mosquitoes is increased (from  $\alpha_{hm} = 0.25$  to  $\alpha_{hm} = 0.00025$ ), the infected Mosquitoes increases as expected. This means that stakeholders can use the malaria transmission probability in mosquitoes as a control for infected population – by decreasing the malaria transmission probability in mosquitoes which can be done using various means such as using mosquito nets etc. Therefore different policies can be made which will concentrate on the minimization of the transmission probability of malaria in mosquitoes by way reducing human – mosquito contacts.

## 5.0 CONCLUSION

In this research work, we presented Malaria HBV co-infection was modeled using eleven-dimensional system of ODEs. We found the invariant region where the solution of the model is bounded as well as the positivity of the solution was proved. The model was analyzed for point of disease free equilibrium, ( $E_0$ ), and the next generation method was applied to find the explicit expression for the basic reproduction number,  $\mathcal{R}_0$ . It was proven that  $E_0$  was locally asymptotically stable if  $\mathcal{R}_0 < 1$  and unstable when  $\mathcal{R}_0 > 1$ .

Furthermore, a sensitivity analysis was performed for the identification of the parameter that influence the co-infection model Basic reproduction number of,  $\mathcal{R}_0$ , the most to see how change in the parameter affects the population of humans or mosquitos in the co-infection model.

In view of the obtained results in this research work, some recommendations as follows are made:

- i. The use of basic reproduction number to keep the spread of co-infection under control. This can be done by either increasing or decreasing the parameter that have positive or negative sensitivity indices respectively for example, the rate of malaria transmission can be controlled by the use of mosquito nets thereby reducing the spread of malaria (Onyango, Ogada, Thirika, & Lawi, 2018).
- ii. There should be promotion of campaigns about the prevention and control of HBV infection & malaria infection as single infections and as co-infection. This recommends that all patients should continuously be encouraged to use all preventive methods like sanitation, use of mosquito treated nets and going for frequent medical checkups and quick treatment of malaria if infected. (Afolabi, Aremo, & Itansanmi, 2018).
- iii. Vector control procedures as well as vaccine production and administering should be applied in controlling the two infections. Different methods for controlling the mosquito population should be used and HBV vaccines should be encouraged to be taken by individuals tested for the HBV and found to be negative in order to prevent them from being infected by the virus and thereby spreading it to others (Gasim & Adam, 2015).
- iv. There should be frequent education of the people on the ways of combating the twin diseases. This is one of effective tools existing to fighting both infections of Malaria and HBV. The government concerned should, as a duty to people to offer correct amenities to efficiently undergo blood screening & other transfused

fluids of body to prevent any virus transmission, because inadequately screened blood can cause infection of individuals by malarial, HBV or other poisonous pathogens. Government should make vaccination programme available and accessible to all its citizen. There should be more aggressive approaches approved to fight against malaria like widespread ecological insecticide spraying, cleaning water ways and drainages and extensive effective circulation of Insecticide Treated Nets (ITNs) (Pauly, Terdzungwe, & Simon, 2011).

- v. Medical personnel especially those in hospitals and clinics, should recommend that any person infected with malaria should be tested for HBV infection. This should be done simultaneously with intensive education of patients on the infections (Gideon, et al., 2018).
- vi. Finally, malaria vaccine as well as HBV vaccine development should be intensified to aid the enhancing of the immune system in manufacturing antibodies against malaria and HBV infection (Gaetano, Vincenzina, & Lorenzo, 2019).

## REFERENCES

- Afolabi, O. J., Aremo, O. H., & Itansanmi, A. I. (2018). Malaria, hepatitis B and HIV /AIDS, and their co-infection among Patients Visiting Health Centres in Akure, Nigeria. *Journal of Biomedicine and Translational Research*, 22-26.
- Ashafa, S. U., Oduwale, H. K., & Babangambo, U. (2022). Model Parameter Effects On Infectious Compartments Of Mathematical Model Of Malaria – Hepatitis B Virus Co – Infection. *Journal of Science, Technology, Mathematics and Entrepreneurial Education (JSTMEE)*, 1(5), 268--297.
- Ashafa, S. U., Oduwale, H. K., & Umar, M. A. (2022, July ). An Optimal Control Analysis Of Malaria HBV Coinfection Model. *Abacus (Mathematics Science Series)*, 49(2), 126-149.
- Ayub, A. O., Ibrahim, A., & Abdulkareem, S. B. (2024). Poverty, HIV/Aids and Malaria: a Triple Burden of disease in Nigeria. *Kogi Journal Of Sociology*, 4(1), 100.
- Chinebu, T., Okonkwo, C., Oyedepo, T., Iyoke, O., Ukwungwu, M., Ikechukwu, N., et al. (2023). Numerical simulation of the intracellular dynamics of HBV/Malaria co-infection with immune control and treatment. *International Journal of Mathematical Analysis and Modelling*, 5(2), Retrieved from <https://tmsmb.org/journal/index.php/ijmam/article/view/55>.
- Dominic, O., Isaac, O. A., Shaibu, O., & Elvis, K. D. (2021). *Mathematical Modeling And Analysis Of*



- The Dynamics Of Hepatitis B With Optimal Control. *Commun. Math. Biol. Neurosci.*, 2021(43).
- Ebenezer, B., Rahat, Z., & Fatma, W. (2020). Mathematical Modeling Of Cancer And Hepatitis Co-Dynamics With Non-Local And Non-Singular Kernel. *Commun. Math. Biol. Neurosci.* 2020, 2020:91.
- ECDC. (2022). *Surveillance Report: Hepatitis B*. Stockholm: © European Centre for Disease Prevention and Control.
- Gaetano, S., Vincenzina, F., & Lorenzo, L. M. (2019). 201. *Le Infezioni in Medicina*, no. 1, 3-10.
- Gasim, G. I., & Adam, I. (2015). Hepatitis B Hepatitis C virus and Malaria co-infection. *Int J Vaccine Immunization*, 1(1).
- Getachew, T. T. (2019). Optimal Control Analysis of Pneumonia and Meningitis Coinfection. *Hindawi Computational and Mathematical Methods in Medicine*, Volume 2019 (Article ID 2658971).
- Gideon, K. H., Paul, A. A., Baba, S. M., Anthony, W., David, K., Abdul-Wahid, A., et al. (2018). Seroprevalence of Malaria and Hepatitis B Coinfection among Pregnant Women in Tamale Metropolis of Ghana: A Cross-Sectional Study. *Hindawi Canadian Journal of Infectious Diseases and Medical Microbiology.*, Volume 2018, Article ID 5610981.
- Hussam, A., Mohammad, A. S., Mahmoud, H. D., A., B., U., S., Muhammad, A. K. (2020). Optimal control analysis of hepatitis B virus with treatment and vaccination. *Elsevier: Results in Physics* 19 (2020) 103599.
- Jones, M. M., Feng-Bin, W., & Naveen, K. V. (2015). Modeling Malaria and Typhoid Fever Co - infection Dynamics. *Mathematical Biosciences*, 264 (2015) , 128–144.
- Jones, M. M., Feng-Bin, W., & Naveen, K. V. (2015). Modeling Malaria and Typhoid Fever Co - infection Dynamics. *Mathematical Biosciences*, 264 (2015), 128–144.
- Kenneth, I. E., Vivian, A. I., Onolunosen, A. A., & Husseni, I. (2023). The immunological evaluate of Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) co-infection with plasmodium in Wukari, Taraba State, North East, Nigeria. *International Journal of Medical and All Body Health Research*, 4(2), 43-47.
- Kotepui, K., & Kotepui, M. (2020). Prevalence of and Risk Factors for Plasmodium spp. co-infection with hepatitis B virus: A Systematic Review and Meta-analysis. *Malar J.*, 19(368).
- Magaji, A. S., Mubarak, N., & Dauda, M. K. (2019, March). Mathematical Modeling Of Hepatitis B In Kaduna Metropolis Using Euler Method. *Journal of Science, Technology, Mathematics and Education (JOSTMED)*, 15(1), 99-118.
- Muhamad, A. B., & Fajar, A.-K. (2020). Modelling Malaria Transmission in a Population with SEIRSp Method. *AIP Conference Proceedings* . 2264, 020002 (2020). AIP Publishing. 978-0-7354-2024-3/\$30.00.
- Onyango, L. O., Ogada, E. A., Thirika, A. M., & Lawi, G. O. (2018). Modeling Malaria And Rotavirus Co-Infection. *Neural, Parallel, and Scientific Computations*, No. 2 (2018)(26), 143-168.
- Paulyn, T. A., Terdzungwe, T. S., & Simon, H. T. (2011). Prevalence of Plasmodia and hepatitis B virus co-infection in blood donors at Bishop Murray Medical Centre, Makurdi, Benue State, Nigeria. *Asian Pacific Journal of Tropical Medicine.*, 224 - 226.
- Petrunina, Z. V., Semenov, A. B., & Novikov, D. V. (2021). Impact Of Epidemics On Socio-Economic, Migration And Cultural Transformations Of Society. *European Proceedings of Social and Behavioural Sciences*, 107(2021), 1297-1303, doi.org/10.15405/epsbs.2021.05.171.
- Segun, I. O., Michael, M. O., Michael, O. A., & Maba, B. M. (2020, July). Mathematical Modeling Of Malaria Disease With Control Strategy. *Communications in Mathematical Biology and Neuroscience* .
- Sukhamoy, D., & Susmita, S. (2020). Basic reproduction number and Bifurcation of Infectious Diseases with Carriers. *Global Journal of Pure Applied Mathematics.*, 16(2(2020)), pp. 187-197.
- WHO. (2023). *Report on malaria in Nigeria 2022*. Brazzaville: WHO Regional Office for Africa, WHO African Region, 2023.
- WHO. (2024, April 9). *Hepatitis B Fact Sheets*. Retrieved December 31, 2024, from <https://www.who.int/news-room/fact-sheets/detail/hepatitis-b>