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MATHEMATICAL MODELING OF THE SPREAD OF VECTOR BORNE DISEASES WITH INFLUENCE OF VERTICAL TRANSMISSION AND PREVENTIVE STRATEGIES

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ABSTRACT

This work is aimed at formulating a mathematical model of the spread of vector-borne diseases with influence of vertical transmission and preventive strategies. Vector borne diseases are caused by viruses, bacteria, and parasites typically conveyed by mosquitoes**.** Certain illnesses transmitted by vectors include West Nile Virus, Malaria, Zika virus, Dengue fever, Rift valley fever, and Viral encephalitis induced by pathogens like bacteria, viruses, and parasites. The positive solutions of the model are presented and the theory of basic reproduction number (R_0) was used to study the model dynamical behaviour. When(R_0) reduces; the diseases are wiped out of the population with time and vice versa. The disease free and endemic equilibria states of the model were determined and investigated to be locally and globally stable.We incorporated the use of Insecticide – Treated Nets (ITN), Indoor Residual Sprayings (IRS) and condom usage as preventive measures in the presence of treatment. Numerical simulations show that complete intervention measures, that is, the use of ITN, IRS and condom usage while placing the infected on treatment have valuable impact on the spread of vector-borne diseases.

Keywords: Vector-borne, vertical, prevention, transmission, spread, strategies

INTRODUCTION

Vector-borne ailments, alternatively termed vector-borne diseases, arise from viruses, bacteria, and parasites typically conveyed by mosquitoes. Approximately 700 million individuals contract an illness transmitted by mosquitoes annually, leading to a mortality toll exceeding one million. (Caraballo, 2014). A mosquito's feeding phase often goes unnoticed, and the bite becomes evident due to the immune response it triggers. (Atokolo, et al, 2022, Atokolo et al, 2020). When a human is bitten by a mosquito, it introduces saliva and anti-coagulants. Initially, there is no response to the first bite, but subsequent bites trigger the individual's immune system to produce antibodies, leading to inflammation and itching within 24 hours. (WHO, 2015, 2016). Certain illnesses transmitted by vectors include West Nile Virus, Malaria, Zika virus, Dengue fever, Rift valley fever, and Viral encephalitis induced by pathogens like bacteria, viruses, and parasites (Abdulah & June, 2018). Arthropods, including ticks, mosquitoes, biting flies, and lice, are parasitic creatures that feed on blood and are referred to as vectors. (Abdulah & June, 2018, Dasti, 2016). The vectors acquire pathogens from an infected host and convey them to a human host, given that humans serve as the primary host, or to animals. Nevertheless, potential modes of direct transmission include transplantation-related, transfusion-related, and needle-stick-related transmissions. (Abdulah & June, 2018, Dasti, 2016).

Certain diseases transmitted by vectors, such as Zika, can be spread through sexual contact. This aligns with clinical research indicating elevated levels of the virus in the semen and saliva of individuals even weeks after their recovery. (Danbaba & Garba, 2018). It's also observed that vector-borne illnesses can be transmitted vertically, meaning the transfer of infection from infected parents to their offspring. (Atkinson et al, 2016, Musso et al, 2015, Foy & Tesh, 2011). Manifestations of diseases transmitted by vectors are distinctive according to the particular viral ailment and differ in intensity, contingent upon the affected individual. The majority of individuals contracting the West Nile virus typically remain asymptomatic. Nevertheless, certain individuals may experience instances of profound exhaustion, debility, migraines, bodily discomfort, joint, and muscle pain. Fever, skin rash, headaches, muscular discomfort, and conjunctivitis are prevalent indicators linked to infections caused by the Zika virus and malaria. (WHO, 2020). Certain diseases transmitted by vectors, such as malaria, chikungunya, and yellow fever, can be managed using Non-Steroidal Anti-inflammatory Drugs (NSAIDs), with supportive care provided to patients infected with the Zika virus. (WHO, 2018, WHO, 2020). The World Health Organization states that approximately 700 million individuals suffer from mosquito-transmitted diseases, resulting in around one million fatalities annually. (Caraballo, 2014). The World Health Organization revealed that malaria is identified as the primary cause of untimely fatalities. Approximately half of the global population, totaling around 3.3 billion individuals across 97 nations, faces the risk of contracting malaria. (Abdulah & June, 2018). Dengue affects as much as 40% of the global population, causing 50-100 million infections annually. (Gao, et al 2016) Proceeded by the remaining vector-transmitted illness.Aligned with the mentioned progress, employing a mathematical model is crucial for managing diseases transmitted by vectors. Our suggestion involves implementing Insecticide-Treated Mosquito Nets (ITN) and Indoor Residual Spraying (IRS) to regulate vector populations. The utilization of ITN minimizes interactions between vectors and residents at home, enabling the elimination of mosquitoes lingering in the vicinity of treated homes through Indoor Residual Spraying (Iornem eta l, 2023).

Several mathematical models have been introduced to examine the dynamics and spread of diseases transmitted by vectors.

Brawer & Castilo (2001), Chowell et al (2007), Yakob & In 2013, Clements introduced compartmental SEIR models for

human populations coupled with SEI compartmental models for mosquito populations. These models were employed to understand the transmission of vector-borne illnesses such as Chikungunya and dengue.

(Hills, 2016, Andraud et al, 2012, Chikaki & Ishikawa, 2009) have introduced various infectious disease models, including one addressing the transmission of the Zika virus through sexual contact with travelers and another focusing on sequential infections with all four serotypes in a dengue model.

Danbaba and Garba (2018) showcased the application of the sterile insect technique for examining the transmission dynamics and management of the zika virus. In a related context, Atokolo et al (2022) utilized the Laplace Adomian Decomposition method to offer an estimated solution to a model focused on sterile insect technology for zika virus control.

This present paper investigates the influence of condom utilization in lessening the transmission of vector-borne diseases among humans. In addition, ITN and IRS were utilized to diminish interactions between humans and vectors. Our model integrates these three preventative measures alongside treatment for the initial time, aiming to effectively manage the dissemination of these diseases.

MATERIALS AND METHODS

Model formulation

We develop a mathematical model that shows the spread of mosquito-borne diseases with influence of vertical transmission and preventive measures.

Human population is divided into Susceptible (S_H), Infected (I_H), Infected but under treatment (I_{HT}) and Recovered human (R_H). The human population is given by:

$N_H(t) = S_H(t) + I_H(t) + I_{HT}(t) + R_H(t)$.

Similarly, the mosquito's population is divided into susceptible mosquitoes (S_M), and infected mosquitoes (I_M). Thus, the total mosquito population is given by:

$N_M(t) = S_M(t) + I_M(t)$.

The flow diagram and the mathematical model that represent the spread of mosquito – borne diseases with influence of vertical transmission and preventive measures are presented below:

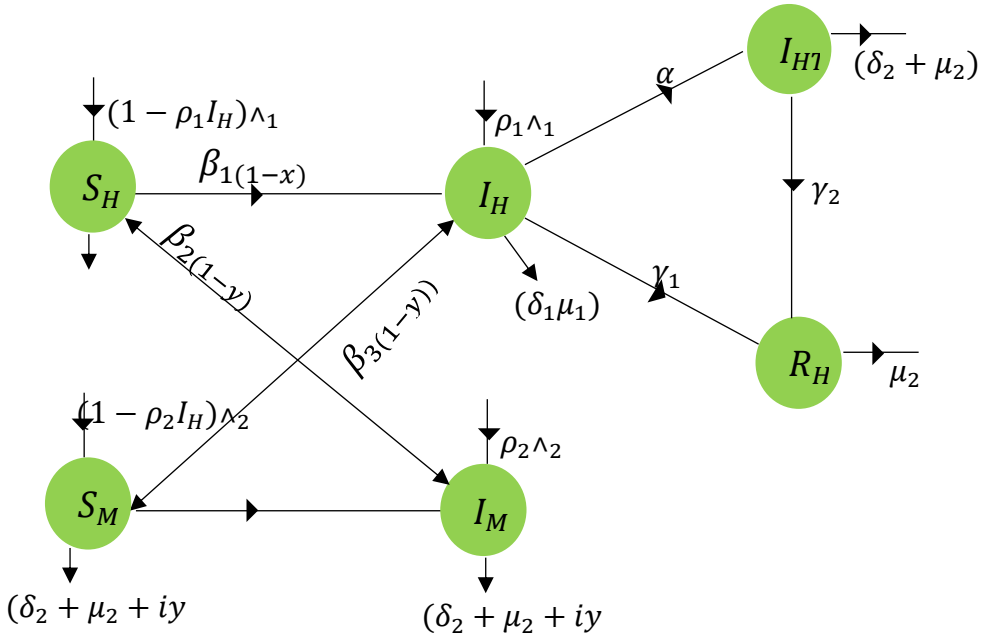

Figure 1: Flow diagram that shows interactions and transfer of the disease in both human and mosquito population.

$$\begin{aligned}
\frac{dS_H}{d_t} &= (1-\rho_1 I_H)\wedge_1 - \beta_1(1-x)S_H I_H - \beta_2(1-y)S_H I_M - \mu_1 S_H \\
\frac{dI_H}{d_t} &= \rho_1\wedge_1 I_H + \beta_1(1-x)S_H I_H - \beta_2(1-y)S_H I_M - (\alpha + \gamma_1 + \delta_1 + \mu_1)I_H \\
\frac{dI_{HT}}{d_t} &= \alpha I_H - (\gamma_2 + \delta_2 + \mu_1)I_{HT} \\
\frac{dR_H}{d_t} &= \gamma_1 I_H + \gamma_2 I_{HT} - \mu_1 R_H \\
\frac{dS_M}{d_t} &= (1-\rho_2 I_M)\wedge_2 - \beta_3(1-y)S_M I_H - (\delta_2 + \mu_2 + iy + jz)S_H \\
\frac{dI_M}{d_t} &= \rho_2 I_M\wedge_2 + \beta_3(1-y)S_M I_H - (\delta_2 + \mu_2 + iy + jz)I_H
\end{aligned} \tag{1}$$

With the initial conditions

$$S_H(0) \geq 0,\ I_H(0) \geq 0,\ I_{HT}(0) \geq 0,\ R_H(0) \geq 0, S_M(0) \geq 0,\ I_M(0) \geq 0 \tag{2}$$

The human population experiences continuous recruitment at a steady birth rate $(\wedge_1)$, with a fraction (ρ_1) being born infected from infected parents. The transmission rates (β_1), (β_2), (β_3) depict the interactions between susceptible humans, infected humans, and infected mosquitoes. Susceptible mosquitoes become infected (β_3) after biting an infected human. The natural death rate of humans is (μ_1), while infectious humans undergo treatment (α), recover naturally (γ_1), or succumb to the disease (δ_1).

Infected humans under treatment recover at the rate (γ_2) or face natural and disease-induced mortality at rates $(\mu_1)$and (δ_2). The assumption is that recovered individuals gain permanent immunity, precluding re-infection. Mosquito population experiences a constant recruitment rate $(\wedge_2)$, with a fraction (ρ_2) born infected from infected mosquitoes. Susceptible mosquitoes become infected (β_3)after biting an infected human.

Susceptible mosquitoes can die while seeking a blood meal (δ_2) or experience natural death (μ_2). Infectious mosquitoes face similar mortality rates (δ_2) and (μ_2). To integrate preventive measures, Insecticide-Treated mosquito Nets (ITN) impact susceptible humans' transition to the exposed class via a parameter $(1-y)$ as $y=1$, susceptibility occurs solely through sexual transmission, eliminating mosquito-mediated transmission.

Similarly, condom usage during sexual activities impacts the transition from susceptible to exposed humans through a parameter $(1-x)$. With $x=1$, susceptibility results only from mosquito bites.

On the other hand, when $y=0$,the net exert no influence, and illnesses can propagate via both vector and sexual transmissions. Similarly, when $x=0$, condoms are ineffective, allowing the disease to spread through both vector and sexual transmissions. The utilization of insecticide-treated bed nets (ITN) diminishes transmission between vulnerable individuals and mosquitoes, while employing condoms during sexual activities lessens or eradicates transmission among humans, contingent on the level of adherence to their use.

We also integrate Indoor Spraying using residual Insecticides (IRS) along with Insecticide-Treated mosquito Nets (ITN) as preventative measures in the vector model. The ITN is introduced to minimize interactions between an infected individual and a susceptible mosquito through a specified parameter. $(1-y).(z)$ represents indoor spraying with residual insecticides (IRS).

Lastly, we introduce parameters for the removal of mosquitoes denoted by (i) and (j) associated respectively with ITN(y) and IRS (z) where ITN $(y)$and IRS (z)range from $0\ to\ 1$ following the method used by Padmanabhan *et al* (2017) in (21).

Basic Properties

To be mathematically and epidemiological meaningful it is necessary that all the solutions of the proposed model (1) with the stated initial conditions (2) remain positive and bounded for all time.

Theorem 1

The solution of system of equations (1) exists uniquely. It is also positive and bounded in a positively invariant set, which remains for all time $t \geq 0$.

Proof

From system (1), the right hand side of each equation is continuous in the convex domain $C = (t, S_H(t), I_H(t), I_{HT}(t), R_H(t), S_M(t), I_M(t))$ of (6 + 1) – dimensional space $R_+^{6+1}$with continuous partial derivatives.

The model (1) has a unique solution in R_+^6which exists for a given finite time $t\varepsilon[0,\infty]$ with the stated initial conditions in (2).

From the human population, we have;

$$N_H(t) = S_H(t) + I_H(t) + I_{HT}(t) + R_H(t)$$

Similarly, from the vector population, we have;

$N_M(t) = S_M(t) + I_M(t)$,

We can therefore write from equation (1) that:

$$\left.\begin{aligned} \frac{dN_H}{d_t} &= \wedge_1 - \mu_1 N_H - \delta_1 I_H - \delta_2 I_{HT} \\ \frac{dN_M}{d_t} &= \wedge_2 - (\delta_2 + \mu_2 + iy + jZ) H_M \end{aligned}\right\} \quad (3)$$

Then; when there is no disease equation (3) becomes

$$\left.\begin{aligned} \frac{dN_H}{d_t} &\leq \wedge_1 - \mu_1 N_H \ \ and \\ \frac{dN_M}{d_t} &\leq \wedge_2 - (\delta_2 + \mu_2) N_M \\ \Rightarrow N_H \leq N_H(0)\rho^{-\mu_1(t)} + \tfrac{\wedge_1}{\mu_1}\left(\rho^{-\mu_1(t)}\right) &\text{ and} \\ N_M \leq N_M(0)\rho^{-(\delta_2+\mu_2)(t)} + \frac{\wedge_2}{(\delta_2+\mu_2)}(1 + \rho^{-(\delta_2+\mu_2)(t)}) & \end{aligned}\right\} \quad (4)$$

This means,

$\lim_{t\to\infty} SupN_H \leq \frac{\wedge_1}{\mu_1}and\lim_{t\to\infty} SupN_M \leq \frac{\wedge_2}{(\delta_2+\mu_2)}$

The stated initial conditions in (2) show that $N_H(0) \geq 0$ and $N_M(0) \geq 0$. We therefore present the feasible region for model (1) as;

$$\emptyset = \left\{(S_H, I_H, I_{HT}, R_H, S_M, I_M)\epsilon R_+^6,\ N_H \leq \frac{\wedge_1}{\mu_1},\ N_M \leq \frac{\wedge_2}{(\delta_2+\mu_2)}\right\}$$

This shows that the total population for both human and mosquito classes are bounded for all finite time$t \geq 0$.

We have successfully shown using the above theorem that the formulated model is mathematically and epidemiologically well posed in an invariant positively set $\emptyset$.

All the model parameters and variables remain positive at all time as all the solutions of model (1) start and remain in the positively set $\emptyset$ for all time $t \geq 0$.

DETERMINATION OF THE MODEL EQUILIBRIUM POINTS

Disease free equilibrium

The steady state solution of system (1) in the absence of disease is known as the disease free equilibrium point.
To determine the equilibrium point of model (1), we set the right – hand side of all the equations equal to zero. Since the population is free of disease, $I_H = I_{HT} = R_H \; and \; I_M = 0$
Similarly, since there will be no infected births from infected parents
, $\rho_1 = 0 \; and \; \rho_2 = 0$
By direct calculation, we therefore present the disease free equilibrium point of the model as;

$$E_1 = (S_H, I_H, I_{HT}, R_H, S_M, I_M) = (\frac{\wedge_1}{\mu_1}, 0, 0, 0, \frac{\wedge_2}{(\delta_2 + \mu_2)}, 0)$$

Basic Reproduction Number

The Basic Reproduction Number is defined as the anticipated count of subsequent cases generated by an average infected person throughout their entire infectious period in a population entirely susceptible to the infection (Diekmann et al, 1990).
Mathematically, the basic reproduction number is presented as:

$$R_0 = \left(\frac{Infection}{Contact}\right).\left(\frac{Infection}{Time}\right).\left(\frac{Time}{Infection}\right)$$

Which can also be written as;

$$R_0 = ABC$$

Where:
A = the probability of infection given contact between susceptible and infected individuals.
B = the average rate of contact between susceptible and infected individuals and lastly
C = the duration of infectiveness.
This quantity is a threshold parameter that predicts whether a disease will spread or die out in a population. This threshold can be calculated using the next generation matrix presented in (Driessche &Watmough, 2002).
From our model, the infected states are $I_H, I_{HT}, and \; I_M$.
The uninfected states are $S_H, R_H$and S_M.
We now present matrices $F \; and \; V$ as the rate of production of new infections and the transition rates between states respectively as:

$$F = \begin{pmatrix} \alpha_1 \wedge_1 I_H + \beta_1(1-x)S_H I_H + \beta_2(1-y)S_H I_M \\ 0 \\ 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (\alpha + \gamma_1 + \delta_1 + \mu_1)I_H \\ (\gamma_2 + \delta_2 + \mu_1)I_{HT} - \alpha I_H \\ (\delta_2 + \mu_2 + iy + jz)I_M - \rho_2 \wedge_2 I_M - \beta_3(1-y)S_M I_H \end{pmatrix}$$

But at the disease free equilibrium state (E_1), we have that

$$S_H = N_H = \frac{\wedge_1}{\mu_1}, I_H = I_{TH} = R_H = 0, S_M = N_M = \frac{\wedge_2}{(\delta_2 + \mu_2)} \; and \; I_M = 0$$

The Jacobian matrices at the disease free equilibrium state (E_1) can now be expressed as;

$$F = \begin{pmatrix} \rho_1 \wedge_1 + \beta_1(1-x)N_H & 0 & \beta_2(1-y)N_H \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$F = V \begin{pmatrix} (\alpha + \gamma_1 + \delta_1 + \mu_1) & 0 & 0 \\ -\alpha & (\gamma_2 + \delta_2 + \mu_1) & 0 \\ -\beta_3(1-y)N_M & 0 & (\delta_2 + \mu_2 + iy + jz - \rho_2 \wedge_2) \end{pmatrix}$$

Using mat lab software (7.0), we compute the largest Eigen value ofFV^{-1} as the basic reproduction number which is given by:

$$R_0 = \frac{\rho_1 \wedge_1 + \beta_1(1-x)N_H}{\alpha + \gamma_1 + \delta_1 + \mu_1} + \frac{\beta_2(1-y)\beta_3(1-y)N_M N_H}{(\alpha + \gamma_1 + \delta_1 + \mu_1)(\delta_2 + \mu_2 + iy + jz - \rho_2 \wedge_2)} \quad (5)$$

When there is no influence of vertical transmission, then $\rho_1 = \rho_2 = 0$ then R_0as presented below is the basic reproduction for the model with only horizontal transmission(R_1).

$$R_1 = \frac{\beta_1(1-x)N_H}{\alpha + \gamma_1 + \delta_1 + \mu_1} + \frac{\beta_2(1-y)\beta_3(1-y)N_M N_H}{(\alpha + \gamma_1 + \delta_1 + \mu_1)(\delta_2 + \mu_2 + iy + jz)} \quad (6)$$

We can see that from equation (5), the number of new infections emanates from both horizontal and vertical transmission; this implies that the basic reproduction number increases with the presence of transmission from both parents in human and mosquito population to their respective off springs. The inverse relationship is seen in equation (6), as the removal of vertical transmission reduces the number of new cases of the vector – borne diseases represented in equation (6).
The effluence of the basic reproduction number has a major impact on the transmission dynamics of vector – borne diseases as this is evident from both the susceptible and infected human populations of system (1).

$$\left.\begin{aligned}\frac{dS_H}{d_t} &= \wedge_1 - (\alpha + \gamma_1 + \delta_1 + \mu_1)R_0 I_H - \mu_1 S_H \\ \frac{dI_H}{d_t} &= (\alpha + \gamma_1 + \delta_1 + \mu_1)(R_0 - 1)I_H\end{aligned}\right\} \quad (7)$$

In equation (7), if Ro is less than 1, it signifies that, on average, each infected individual spreads the infection to fewer than one person, resulting in a decline in the number of infected individuals and eventual eradication of the disease. Conversely, when Ro is greater than 1, it indicates that each infected person, on average, transmits the infection to more than one person, leading to a positive change in the number of infected individuals and the spread of the disease within the population. When Ro equals 1, it means that there is no net change in the number of infected individuals, as each infected person, on average, infects exactly one other person, resulting in the disease persisting in the population. This concept is illustrated in figure (15).

Theorem 3.1
The disease free equilibrium state *(E_1)* is locally asymptotically stable if $R_o < 1$, otherwise unstable.

Proof:
Linearizing model (1) at the disease free equilibrium point *(E_1)* gives the following Jacobian matrix:

$$J_1 = \begin{pmatrix} -\mu_1 & (-\rho_1 \wedge_1 -\beta_1(1-x)\frac{\wedge_1}{\mu_1}) & 0 & 0 & 0 & \beta_2(1-y)\frac{\wedge_1}{\mu_1} \\ 0 & (\rho_1 \wedge_1 -\beta_1(1-x)\frac{\wedge_1}{\mu_1} - (\alpha + \gamma_1 + \delta_1 + \mu_1)) & 0 & 0 & 0 & \beta_2(1-y)\frac{\wedge_1}{\mu_1} \\ 0 & \alpha & -(\gamma_2 + \delta_2 + \mu_2) & 0 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -\mu_1 & 0 & 0 \\ 0 & -\beta_3(1-y)\frac{\wedge_2}{(\delta_2 + \mu_2)} & 0 & 0 & (\delta_2 + \mu_2 + iy + jz) & -\rho_2 \wedge_2 \\ 0 & \beta_3(1-y)\frac{\wedge_2}{(\delta_2 + \mu_2)} & 0 & 0 & -(\delta_2 + \mu_2 + iy + jz) & -\rho_2 \wedge_2 \end{pmatrix}$$

The characteristic equation of the Jacobian matrix (J_1) is given as;

$$(M + \mu_1)(M + \mu_1)(M + \delta_2 + \mu_2 + iy + jz)(M + \gamma_2 + \delta_2 + \mu_1)$$
$$(K_0 M^2 + K_1 M + K_2) = 0 \quad (8)$$

Where

$$K_0 = \mu_1(\delta_2 + \mu_2 + iy + jz),$$
$$K_1 = \mu_1(\alpha + \gamma_1 + \delta_1 + \mu_1)(\delta_2 + \mu_2 + iy + jz) + \mu_1(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)(\delta_2 + \mu_2 + iy + jz)$$
$$- \beta_1(1-x)\wedge_1(\delta_2 + \mu_2 + iy + jz) - \wedge_1\rho_1\mu_1(\delta_2 + \mu_2 + iy + jz)$$
$$K_2 = \mu_1(\alpha + \gamma_1 + \delta_1 + \mu_1)(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)(\delta_2 + \mu_2 + iy + jz)(1 - R_0)$$

Four Eigen values, that is, $-\mu_1, -\mu_1,$

$$-(\delta_2 + \mu_2 + iy + jz) \; and \; -(\gamma_2 + \delta_2 + \mu_1)$$

Out of six, have a negative real part. The remaining two Eigen values are the roots of the equation.

$$K_0 M^2 + K_1 M + K_2 = 0$$

For $R_0 < 1$ and $(\alpha + \gamma_1 + \delta_1 + \mu_1) - (\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2) > \beta_1(1-x)N_H + \wedge_1\rho_1$

We have $K_1 > 0$ and $K_1 K_2 > 0$.

Using the Routh- Hurwitz criteria used by (Rao, 2009), these two Eigen values have negative real parts. We can also therefore conclude that the model (1) is locally asymptotically stable at the disease free equilibrium point since each Eigen value of the characteristic equation (8) has a negative real part when the basic reproduction number $(R_0) < 1$.

Endemic equilibrium point
The steady state solution of system (1) in the presence of disease is known as the endemic equilibrium point. We therefore represent this equilibrium state as:
$E_2 = (S_H^*, I_H^*, I_{HT}^*, R_H^*, S_M^*, I_M^*)$. Setting the right hand sides of model (1) to zero and solving the equations simultaneously at steady state, we have:

$$S_H^* = \frac{\wedge_1 - (\alpha + \gamma_1 + \delta_1 + \mu_1)I_H^*}{\mu_1}$$

$$I_{TH}^* = \frac{\alpha I_H^*}{(\gamma_2 + \delta_2 + \mu_1)}$$

$$R_H^* = \frac{((\gamma_2 + \delta_2 + \mu_1)\gamma_1 + \gamma_2\alpha)I_H^*}{\mu_1(\gamma_2 + \delta_2 + \mu_1)}$$

$$S_M^* = \frac{(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)I_M^*}{\beta_3(1-y)I_H^*}$$

$$I_M^* = \frac{(\delta_2 + \mu_2 + iy + jz)\beta_3(1-y)N_M I_H^*}{\beta_3(1-y)(\delta_2 + \mu_2 + iy + jz)I_H^* + (\delta_2 + \mu_2 + iy + jz)(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)}$$

Theorem 2
The model (1) equilibrium point E_2 is locally asymptotically stable if $R_0 > 1$, otherwise unstable.

Proof
Linearizing model (1) also at the endemic equilibrium point (E_2) gives the following Jacobian matrix.

$$J_2 = \begin{pmatrix} a_{11} & -(\rho_1\wedge_1 + \beta_1(1-x)s_H^*) & 0 & 0 & 0 & -\beta_2(1-y)S_H^* \\ a_{21} & a_{22} & 0 & 0 & 0 & \beta_2(1-y)S_H^* \\ 0 & \alpha & -(\gamma_2 + \delta_2 + \mu_1) & 0 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -\mu_1 & 0 & 0 \\ 0 & -\beta_2(1-y)S_H^* & 0 & 0 & a_{55} & -\rho_2\wedge_2 \\ 0 & \beta_3(1-y)S_M^* & 0 & 0 & \beta_3(1-y)I_H^* & a_{66} \end{pmatrix}$$

$$where\ a_{11} = (\beta_1(1-x)I_H^* + \beta_2(1-y)I_M^* + \mu_1), \quad a_{21} = (\beta_1(1-x)I_H^* + \beta_2(1-y)I_M^* + \mu_1) - \mu_1$$

$$a_{22} = (\rho_1\wedge_1 + \beta_1(1-x)S_H^*) - (\alpha + \gamma_1 + \delta_1 + \mu_1), a_{55} = (-\beta_3(1-y)I_H^* - \mu_2 - \delta_2 - iy - jz)$$
$$a_{66} = -(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)$$

Where we have$-(\gamma_2 + \delta_2 + \mu_1)$ and $-\mu_1$ as two of the Eigen Values of which the remaining Eigen Values can be obtain from the matrix below;

$$J_3 = \begin{pmatrix} b_{11} & b_{12} & b_{13} & b_{14} \\ b_{21} & b_{22} & b_{23} & b_{24} \\ b_{31} & b_{32} & b_{33} & b_{34} \\ b_{41} & b_{42} & b_{43} & b_{44} \end{pmatrix}$$

Where:

$b_{11} = -(\beta_1(1-x)I_H^* + \beta_2(1-y)I_M^* + \mu_1), b_{12} = -(\rho_1\wedge_1 + \beta_1(1-x)S_H^*), b_{13} = 0,\ b_{14} = -\beta_2(1-y)S_H^*, b_{21} = -(\beta_1(1-x)I_H^* + \beta_2(1-y)I_M^* + \mu_1) - \mu_1,\ b_{22} = (\rho_1\wedge_1 + \beta_1(1-x)S_H^*) - (\alpha + \gamma_1 + \delta_1 + \mu_1)$, $b_{23} = 0,\quad b_{24} = \beta_2(1-y)S_H^*, b_{31} = {}^{0,b}{}_{32} = -\beta_3(1-y)S_M^*, b_{33} = -[\beta_3(1-y)I_H^* + (\mu_2 + \delta_2 + iy + jz)]'$
$b_{34} = -\rho_2\wedge_2, b_{41} = 0, b_{42} = \beta_3(1-y)S_M^*,\ b_{43} = \beta_3(1-y)I_H^*,\ b_{44} = -(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)$

From the Jacobian Matrix(J_3), three out of the Eigen Values have a negative real part.
These are:
$-(\beta_1(1-x)I_H^* + \beta_2(1-y)I_M^* + \mu_1),\ -[\beta_3(1-y)I_H^* + (\mu_2 + \delta_2 + iy + jz)]$and
$-(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)$
The second Eigenvalue $\left(\rho_1\wedge_1 + \beta_1(1-x)\right)\frac{\wedge_1}{\mu} - (\alpha + \gamma_1 + \delta_1 + \mu_1)$
has a negative real part if and only if

$$\left(\rho_1\wedge_1 + \beta_1(1-x)\right)\frac{\wedge_1}{\mu} - (\alpha + \gamma_1 + \delta_1 + \mu_1) < 0$$

Rewriting this equation now in terms of R_0, we have;
$-\beta_1(1-x)\beta_3(1-y)(\delta_2 + \mu_2 + iy + jz)(I_H^*)^2 + [\beta_3(1-y)(\delta_2 + \mu_2 + iy + jz)\mu_1(\alpha + \gamma_1 + \delta_1 + \mu_1)(1-R_0)]I_H^* +$
$(\delta_2 + \mu_2 + iy + jz)(\delta_2 + \mu_2 + iy + jz - \rho_2\wedge_2)\mu_1(\alpha + \gamma_1 + \delta_1 + \mu_1)(1-R_0)$ (9)
The coefficients of equation (9) will all be negative if $R_0 > 1$. Thus all the Eigen Values have negative real parts, which implies that the endemic equilibrium point $(E_2)$is locally asymptotically stable if $R_0 > 1$.

Global stability analysis of the disease free equilibrium point (E_1)

We investigated and studied the global stability analysis of the disease free equilibrium point of our model (1). Using the direct Lyapunov method.

Theorem 3

When the basic reproduction number $(R_0) < 1$, then the disease free equilibrium state (E_1) of our model is globally asymptotically stable on $\emptyset$.

Proof

To show that (E_1) is globally asymptotically stable, we construct the following Lypunov function following the method used by (Abdulah & June, 2018).

With time derivative

$$Z(t) = I_H + \frac{\beta_2(1-y)\wedge_1 \dot{I}_M}{\mu_1(\delta_2+\mu_2+iy+jz-\rho_2\wedge_2)}$$
$$Z'(t) = \dot{I}_H + \frac{\beta_2(1-y)\wedge_1 I_M}{\mu_1(\delta_2+\mu_2+iy+jz-\rho_2\wedge_2)} \quad (10)$$

Then Z is c' on the interior of $\emptyset$, (E_1) is the global minimum of $Z\ on\ \emptyset$ and $Z(t) = 0$ at (E_1). Substituting the values from system (1) we obtain,

$$\begin{aligned} Z'(t) &= \rho_1\wedge_1 I_H + \beta_1(1-x)S_H I_H + \beta_2(1-y)S_H I_M - (\alpha+\gamma_1+\delta_1+\mu_1)I_H \\ &\quad + \frac{\beta_2(1-y)\wedge_1[\rho_2\wedge_2 I_M + \beta_3(1-y)S_M I_H - (\delta_2+\mu_2+iy+jz)I_M]}{\mu_1(\delta_2+\mu_2+iy+jz-\rho_2\wedge_2)} \\ &\leq \rho_1\wedge_1 I_H + \beta_1(1-x)N_H I_H + \beta_2(1-y)N_H I_M - (\alpha+\gamma_1+\delta_1+\mu_1)I_H \\ &\quad + \frac{\beta_2(1-y)\wedge_1[\rho_2\wedge_2 I_M + \beta_3(1-y)S_M I_H - (\delta_2+\mu_2+iy+jz)I_M]}{\mu_1(\delta_2+\mu_2+iy+jz-\rho_2\wedge_2)} \end{aligned}$$

$Since\ S_H \leq N_H$ and

$S_M \leq N_M$

$$Z'(t) = (R_0 - 1)I_H \quad (11)$$

Equation (11) implies that $Z'(t)$ is negative if $R_0 < 1$. This also implies that $Z'(t) = 0$ at the disease free equilibrium point (E_1).

By putting $I_H = I_{HT} = R_H = I_M = 0$

In the susceptible humans and susceptible vectors equations of our system (1) implies that

$S_H(t) \to \frac{\wedge_1}{\mu_1}$ and $S_M(t) \to \frac{\wedge_2}{(\delta_2+\mu_2)}$ as $t \to \infty$

Also by putting in the equations for infected but under treatment (I_{HT}) and recovered human (R_H) populations shows that

$$(I_{HT}(t), R_H(t)) \Rightarrow (0,0)\ as\ t \to \infty.$$

Therefore the largest compact invariant set in

$\{(S_H, I_H, I_{HT}, R_H, S_M, I_M) \in \emptyset : Z'(t) = 0\}$ Is the singleton disease – free equilibrium point.

From LaSalle's principle, the disease free equilibrium point is globally asymptotically stable in $\emptyset$.

Theorem 4

The endemic equilibrium point (E_2). is globally asymptotically stable if $R_0 > 1$.

Proof

To show that (E_2) is globally asymptotically stable, we construct the following Lypunov function following the method used by (Abdulah & June, 2018).

$$U(t) = \frac{1}{\beta_1(1-x)S_H^*}(S_H - S_H^* log S_H) + \frac{1}{\beta_3(1-y)}(S_M - S_M^* log S_M) + \frac{1}{\beta_1(1-x)S_H^*}I_H + \frac{1}{\beta_3(1-y)S_M^*}I_M \quad (12)$$

Taking the time derivate of $U(t)$, we have

$$U'(t) = \frac{1}{\beta_1(1-x)S_H^*}(S_H - S_H^*)\left[\frac{\wedge_1}{S_H} - \frac{\rho_1\wedge_1 I_H}{S_H} - \beta_1(1-x)I_H - \beta_2(1-y)I_M - \mu_1\right] + \frac{1}{\beta_3(1-y)S_M^*}(S_M - S_M^*)\left[\frac{\wedge_2}{S_M} - \frac{\rho_2\wedge_2 I_M}{S_M} - \beta_3 I_H - (\delta_2+\mu_2+iy+jz)\right] + \frac{1}{\beta_1(1-x)S_H^*}[\beta_1(1-x)S_H I_H + \beta_2(1-y)S_H I_M - (\alpha+\gamma_1+\delta_1+\mu_1-\rho_1\wedge_1)I_H] \quad (13)$$

We now consider the following:

$$\mu_1 = \frac{\wedge_1}{S_H} \Rightarrow \wedge_1 = \mu_1 S_H$$
$$(\delta_2+\mu_2+iy+jz) = \frac{\wedge_2}{S_M} \Rightarrow \wedge_2 = (\delta_2+\mu_2+iy+jz)S_M \quad (14)$$

$(\alpha+\gamma_1+\delta_1+\mu_1-\rho_1\wedge_1) = 2\beta_1(1-x)S_H$ and

$$(\delta_2+\mu_2+iy+jz-\rho_2\wedge_2) = \frac{\beta_2(1-y)\beta_3(1-y)S_H^*}{\beta_1(1-x)}$$

Now following the assumptions in (14), we rearranged equation (13) as;

$$u'(t) = \frac{\mu_1}{\beta_1(1-x)}\left(\frac{S_H}{S_H^*} + \frac{S_H^*}{S_H} - 2\right) - \frac{(\delta_2+\mu_2+iy+jz)}{\beta_3(1-y)}\left(\frac{S_M}{S_M^*} + \frac{S_M^*}{S_M} - 2\right) \quad (15)$$

From equation (15)

$$\frac{S_H}{S_H^*} + \frac{S_H^*}{S_H} \geq 2 \text{ and} \frac{S_M}{S_M^*} + \frac{S_M^*}{S_M} \geq 2 \quad (16)$$

This is because the arithmetic mean is greater than or equal to the geometric mean.

We can therefore say

$$u'(t) \leq 0 \; for \; all \; (S_H, I_H, I_{HT}, R_H, S_M, I_M) \in \emptyset \text{ and the equality } u'(t) = 0$$

holds for the endemic equilibrium point (E_2).

We therefore complete the proof as in the proof of theorem (4).

Model Simulation and Discussion of Results

We performed numerical simulations of our model to illustrate some of the theoretical results. Variables and parameters values used in the simulations are presented in table 1 below:

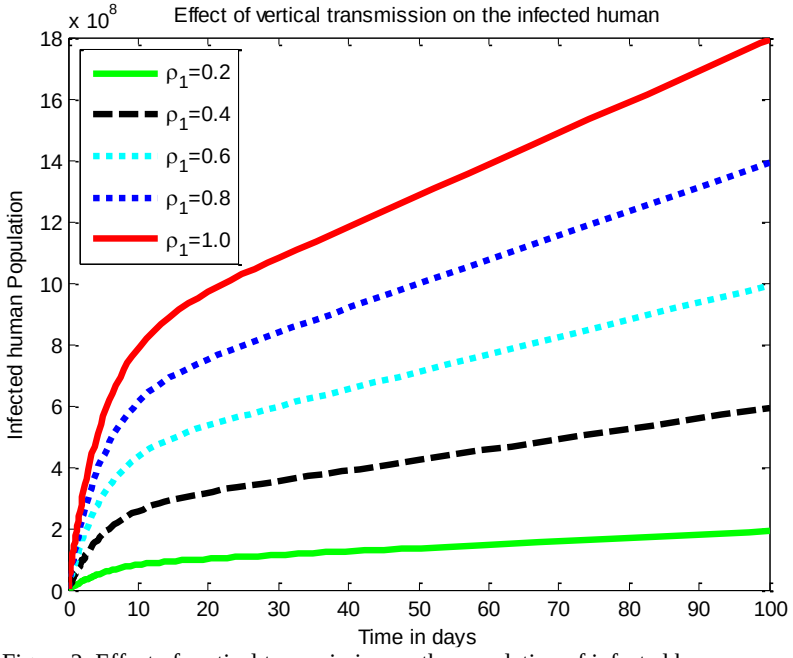

Figure 2: Effect of vertical transmission on the population of infected human population.

From figure 2, we discovered that as the number of infected births increases, the population of infected population increases, this is because the population will be filled with more infected persons and as such, the disease increases in the population.

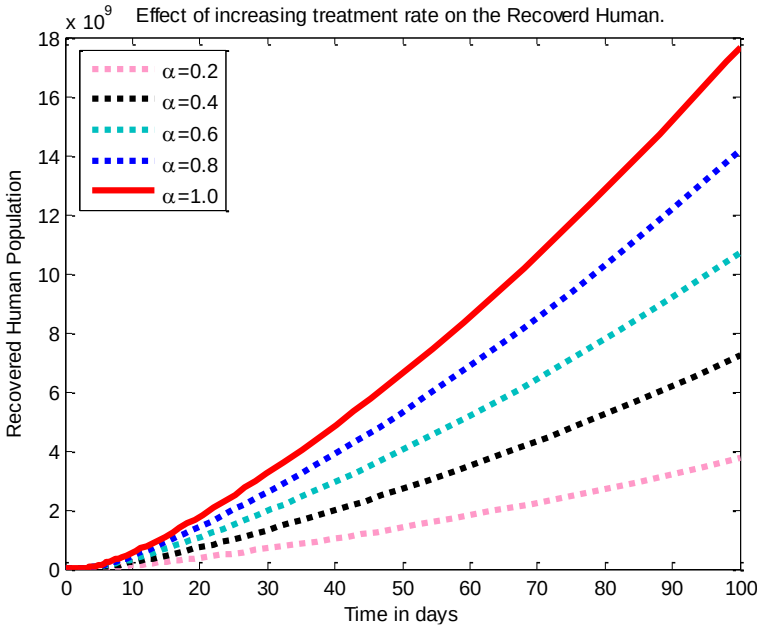

Figure 3: Effect of increasing treatment rate on the Recovered human.

From figure 3, as the treatment rate increases, many infected individuals recover from the disease. This is obvious as many go for treatment, recovery rate will be high, therefore also leading to a reduction in the number of cases among human.

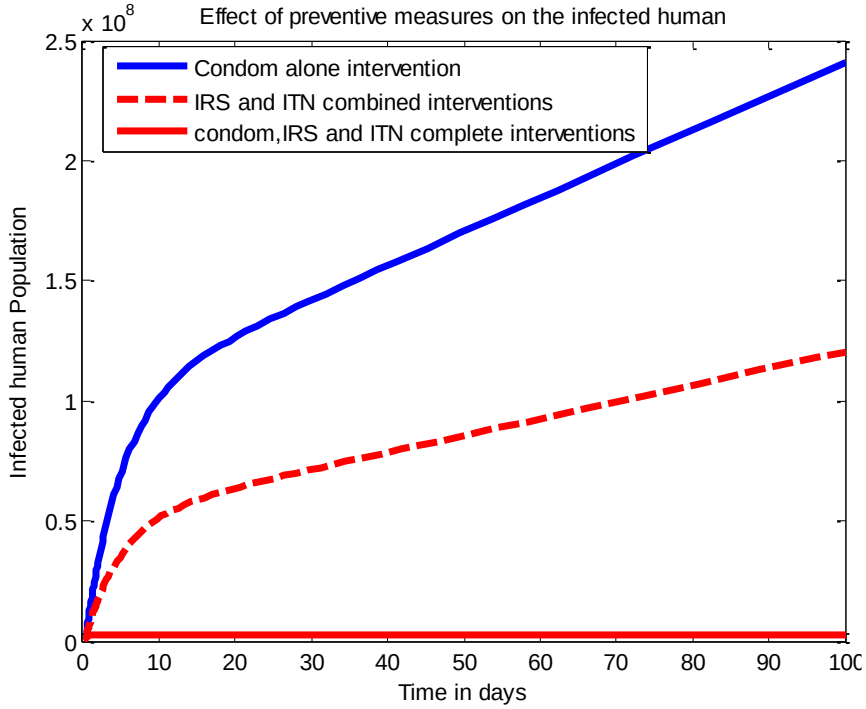

Figure 4: Effect of preventive measures on the infected human class.

From figure 4, it is evident that the population of the infected human population reduces significantly with the interventions of the three control measures, which are Condom, IRS and ITN interventions.

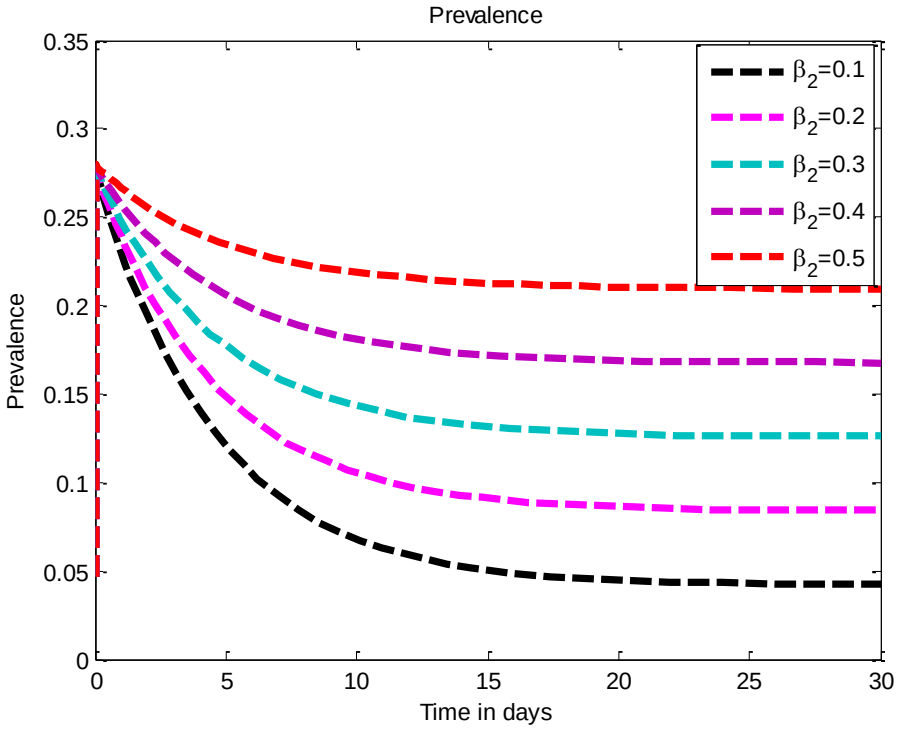

Figure 5: Prevalence (total number of infected individuals divided by the total population).

We discovered from figure 5 that, as the transmission rate between infected mosquitoes and susceptible human increases the prevalence rate increases.

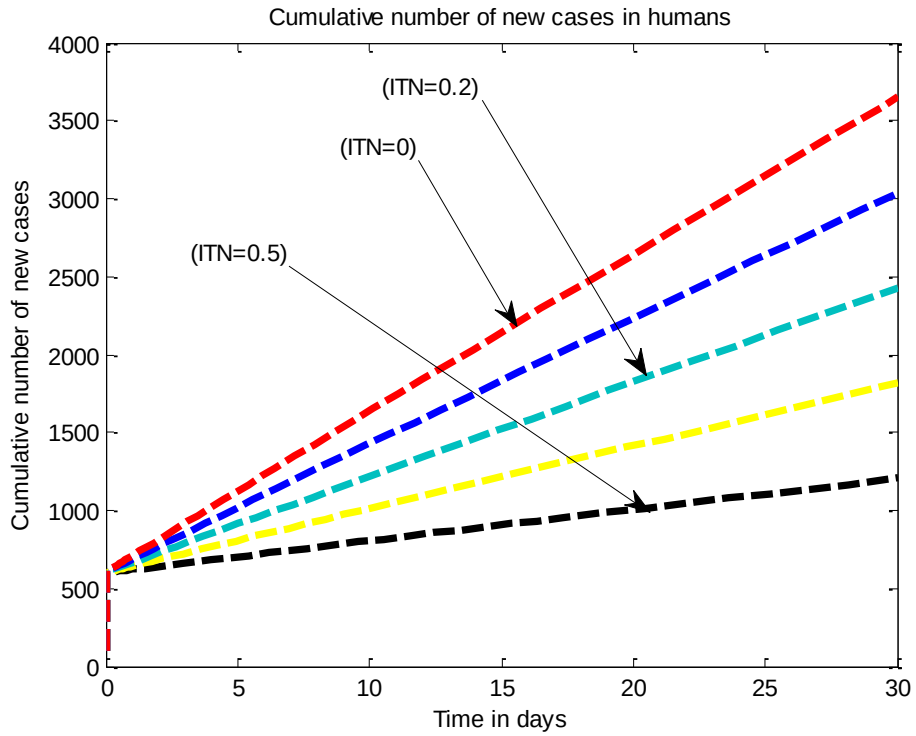

Figure 6: Cumulative number of cases with different value of ITN.

It is evident from figure 6 that, as the ITN intervention increases, the cumulative number of new cases also increases.

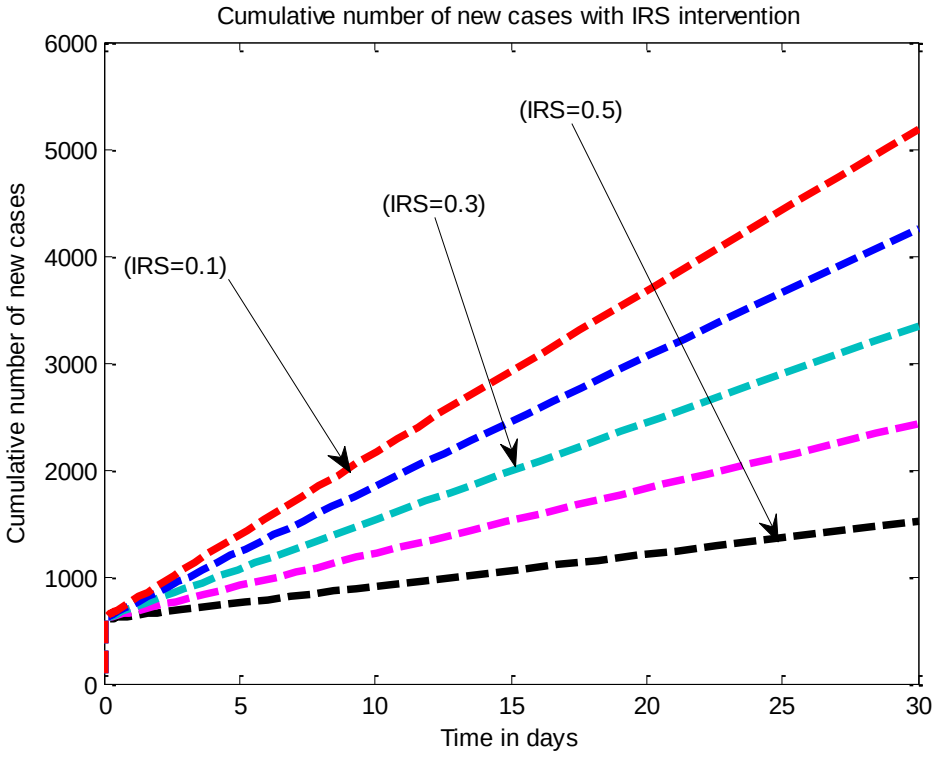

Figure 7: Cumulative number of new cases with different value of IRS.

Similar dynamics is seen in figure 7, as the IRS intervention increases, the cumulative number of new cases also increases. This implies that the IRS intervention strategy is able to reduce the number of human cases to some extent.

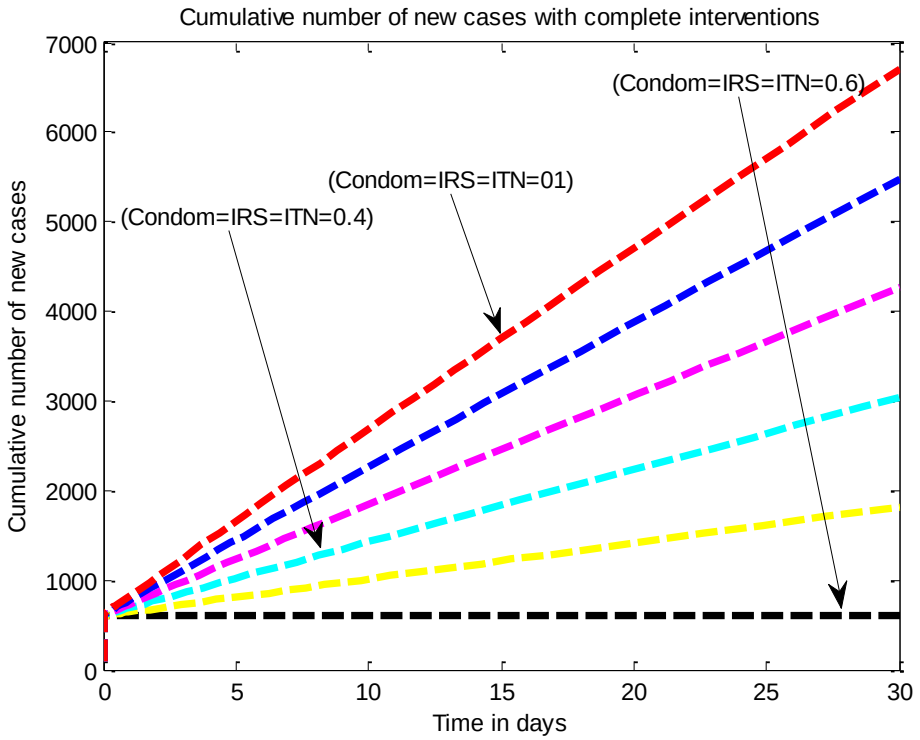

Figure 8: Cumulative number of cases with complete intervention.

From figure 8, we discovered that, the cumulative number of new cases reduces the more with the combine intervention strategies, which are the ITN and IRS. This shows the combined intervention of both ITN and IRS leads to a greater reduction in the number of new cases in human as compared to a separate intervention.

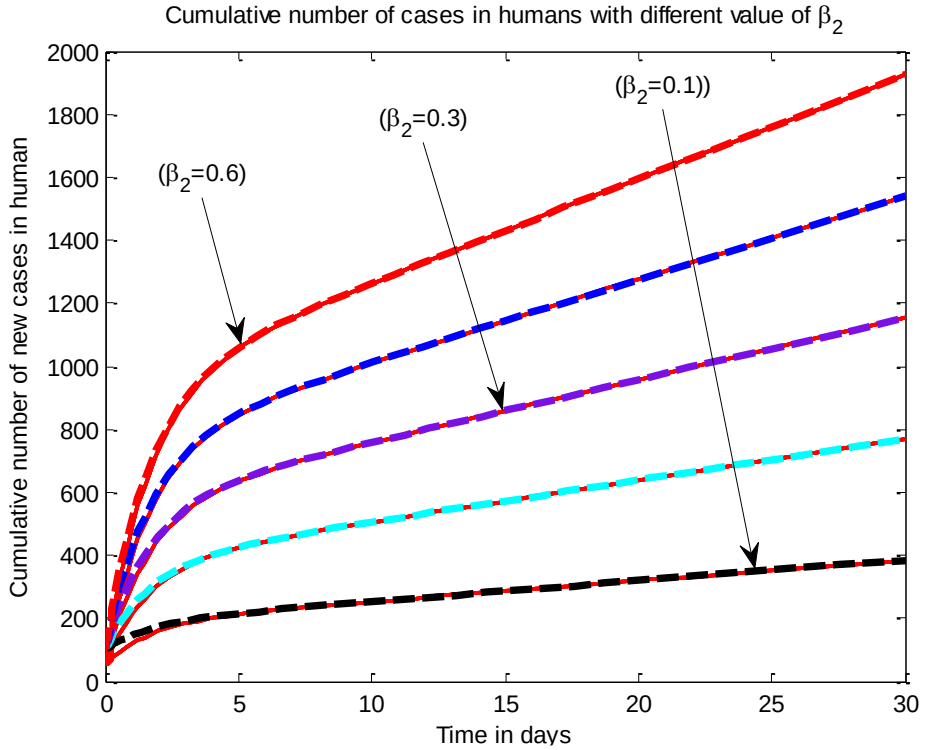

Figure 9: Cumulative number of cases in human population.

From figure 9, using different values of transmission rate between infected mosquitoes and susceptible human; we discovered that as the rate reduces, there is a reduction in the number of new cases of the disease.

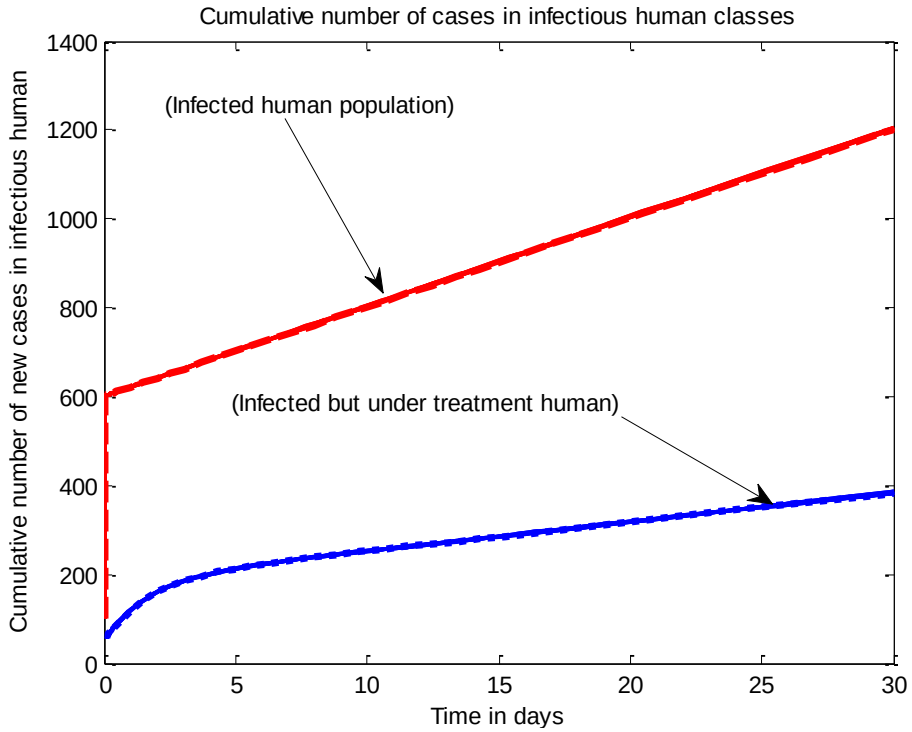

Figure 10: Cumulative number of new cases in the infectious classes.

Figure 10 shows that, the number of new cases in the infected human classes increases faster more than the infected but under treatment human class. This is because, the rate of disease progression is low in the infected but on treatment class as compared to the infected class.

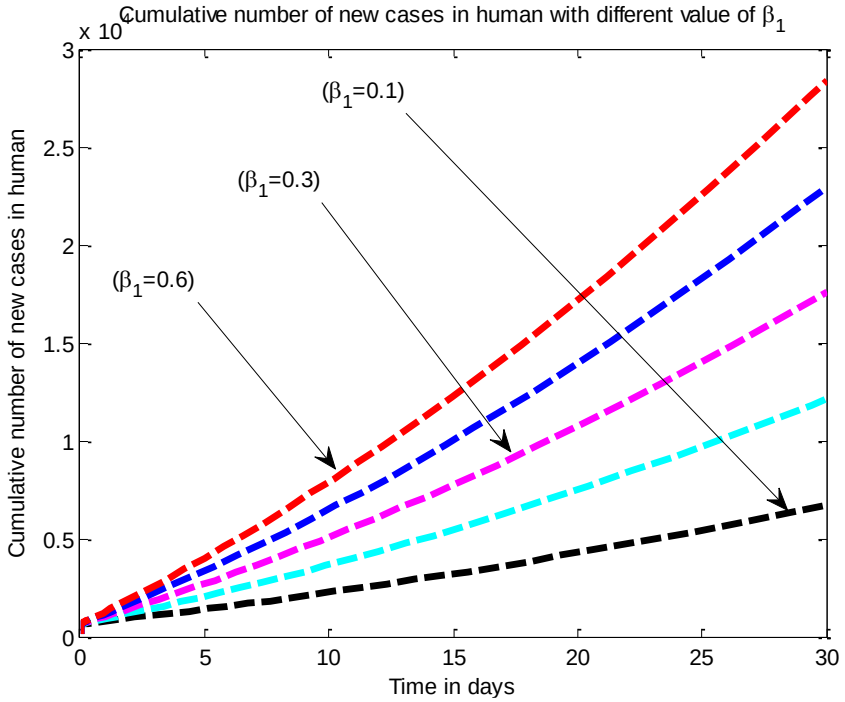

Figure 11: Cumulative number of cases in human population with different value of transmission rate between infected human and susceptible human.

From figure 11, we observed that, as the rate reduces, there is a reduction in the number of new cases of the disease. This rate reduces due to the efficacy in the use of condom among human.

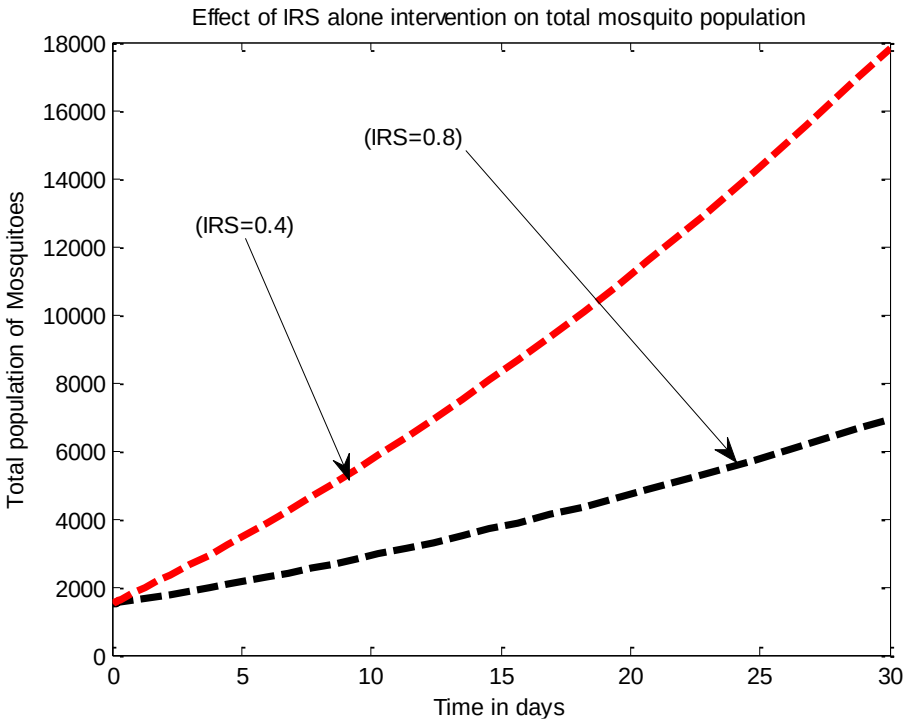

Figure 12: Effect of IRS intervention on total population of mosquitoes.

We discovered from figure 12 that an increase in the IRS intervention program leads to a reduction in the total population of mosquitoes. This is because, the IRS intervention is targeted at killing mosquitoes hence reduces the entire mosquitoes population.

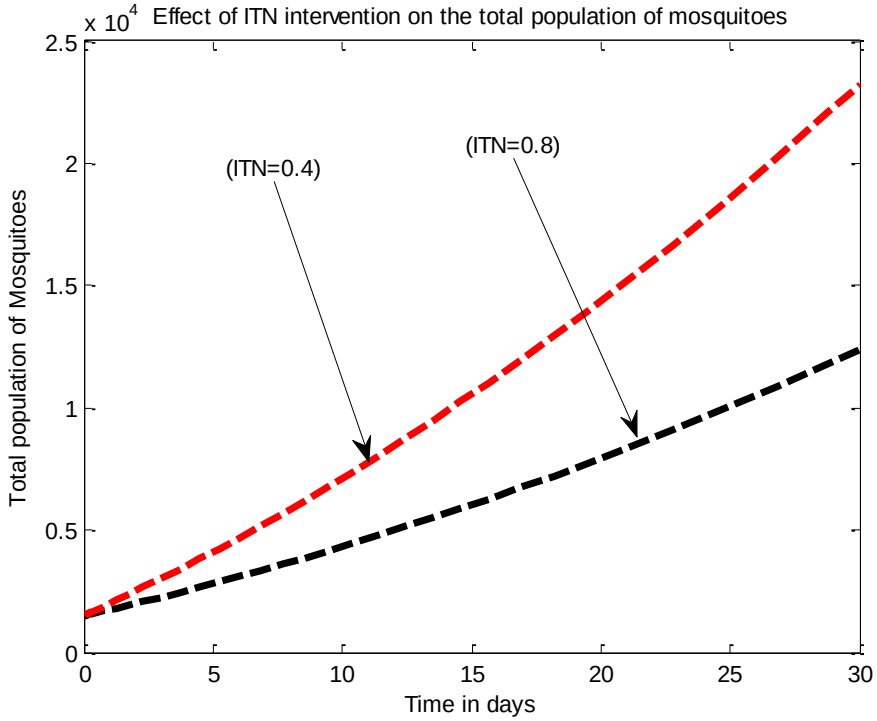

Figure 13: Effect of ITN intervention on total population of mosquitoes.

It was also discovered from figure 13 that, an increase in the ITN intervention program leads to a reduction in the total population of mosquitoes. The treated net kills mosquitoes and also reduces contact between mosquitoes and human.

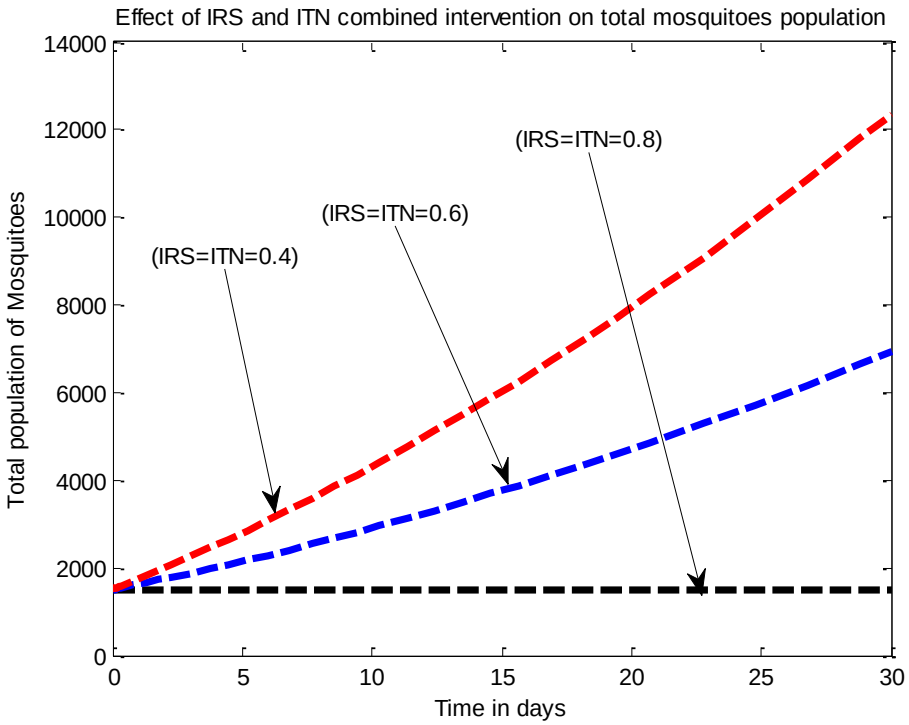

Figure 14: Effect of ITN and IRS combined intervention on total population of mosquitoes.

It was observed from figure 14 that, an increase in both intervention programs reduces the total population of mosquito's population significantly as compared to a single intervention measure.

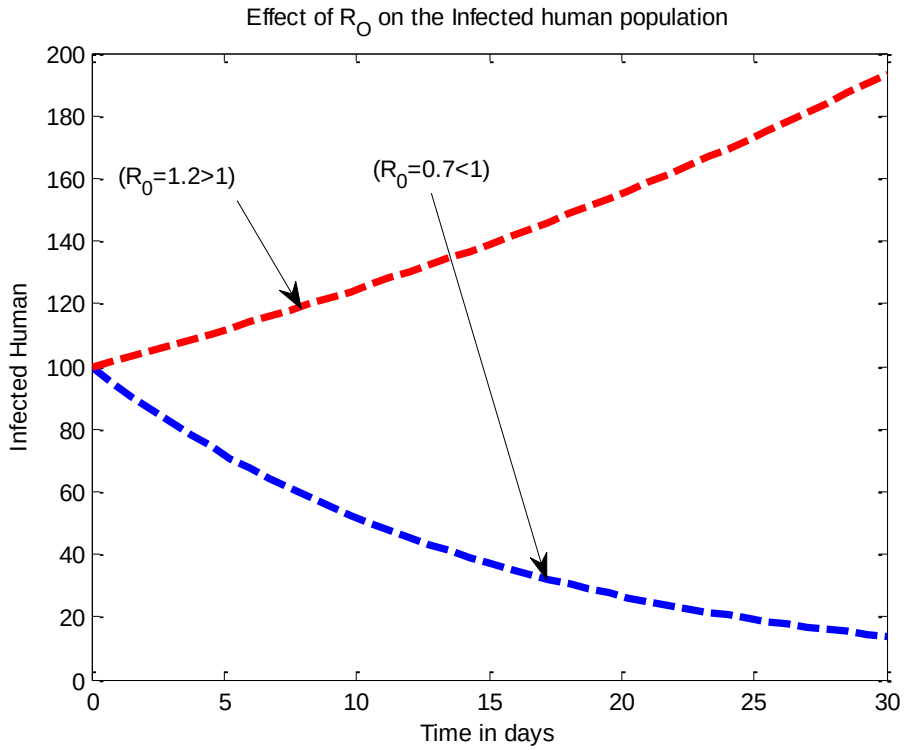

Figure 15: The impact of the basic reproduction number on the population of infected individuals.

It is evident from figure 15 that, when the basic reproduction number is below one, each infected individual spreads the infection to more than one person, resulting in a reduction of the infected population. Conversely, when the basic reproduction number exceeds one, each infected person transmits the infection to more than one person, maintaining a positive change in the number of infected individuals as the disease spreads through the population.

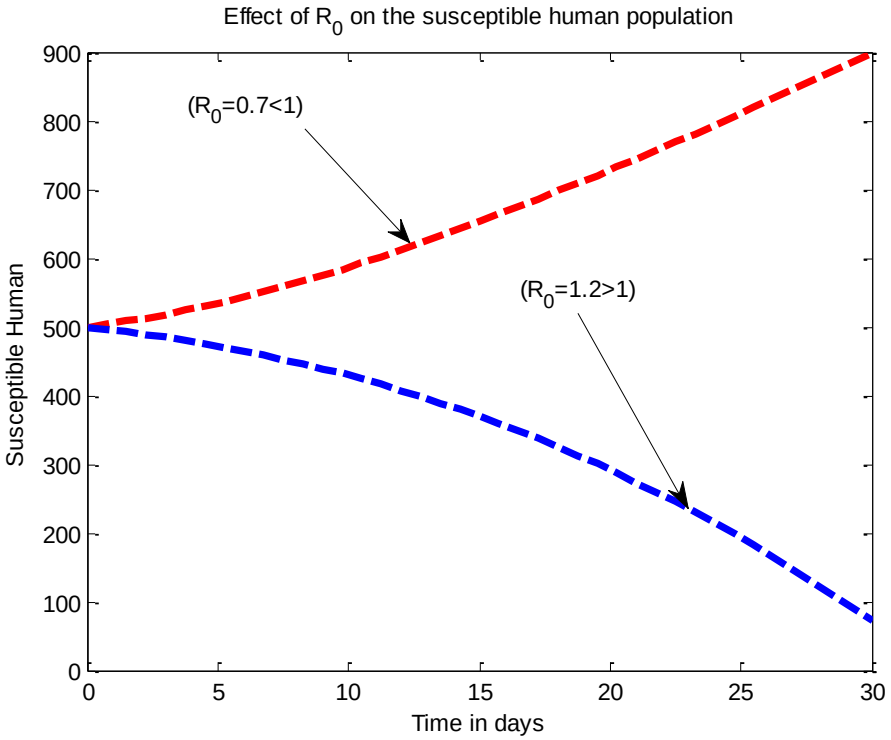

Figure 16: Effect of basic reproduction number on the susceptible human population.

From figure 16, it is evident that as the basic reproduction number decreases, the population of the susceptible increases, on the other hand, the population decreases with increase in the basic reproduction number. From figure 15, when the $R_O < 1$, we discovered that the infected population reduces, this implies that the population of the susceptible increases as depicted in figure 16 and otherwise.

Table 1: Numerical value of variables/parameters used for model simulation.

S/n	Variables(Parameters)	Description	Value	Source
1	S_H	Susceptible human	500	Assumed
2	I_H	Infected human	100	Assumed
3	I_{HT}	Infected human but under treatment	50	Assumed
4	R_H	Recovered human	20	Assumed
5	S_M	Susceptible Mosquitoes	1000	Assumed
6	I_M	Infected Mosquitoes	500	Assumed
7	ℓ_1	Proportion of human infected from birth	0.001 day^{-1}	Gao et al (2016)
8	ℓ_2	Proportion of mosquitoes infected from birth	0.002 day^{-1}	Lashari&Zaman (2011)
9	β_1	Transmission rate between susceptible human and infected human	0.0001 day^{-1}	Gao et al (2016)
10	β_2	Transmission rate between susceptible human and infected mosquitoes	0.0012 day^{-1}	Caraballo (2014)
11	β_3	Transmission rate between susceptible mosquitoes and infected human	0.001 day^{-1}	Lashari&Zaman (2011)
12	γ_1	Natural recovery rate	0.01 day^{-1}	Gao et al (2016)
13	γ_2	Recovery rate due to treatment	0.4 day^{-1}	Gao et al (2016)
14	δ_1	Disease induced death of human	0.01 day^{-1}	Caraballo (2014)
15	δ_2	Death of mosquitoes due to an attempt seeking for blood meal	0.4 day^{-1}	Caraballo (2014)
16	$\wedge_1$	Human recruitment rate	20 day^{-1}	Lashari&Zaman (2011)
17	$\wedge_2$	Mosquitoes recruitment rate	100 day^{-1}	Lashari&Zaman (2011)

18	μ_1	Natural death rate of human	0.000039 day^{-1}	Lashari&Zaman (2011)
19	μ_2	Natural death rate of mosquitoes	0.1 day^{-1}	Lashari&Zaman (2011)
20	α	Treatment rate	0.2 day^{-1}	Caraballo (2014)
21	i	Mosquitoes removal parameter associated with ITN intervention program	0.0027	Caraballo (2014)
22	j	Mosquitoes removal parameter associated with IRS intervention program	0.0027	Caraballo (2014)
23	x	Condom usage intervention	0.2	Assumed
24	y	ITN preventive measure	0.2	Assumed
25	z	IRS preventive measure	0.2	Assumed

CONCLUSION

We successfully formulated a mathematical model of the spread of vector borne diseases with the influence of vertical transmission and preventive strategies. We incorporated the use of Insecticide –Treated Nets (ITN), Indoor Residual Sprayings (IRS) and condom usage as preventive measures in the presence of treatment in other to gain insight into the transmission dynamics and control of vector borne disease. We discovered that, complete intervention measures, that is, the use of ITN, IRS and condom usage while placing the infected on treatment have valuable impact on the spread of vector-borne diseases.

REFERENCES

Abdullah, A., Jun, W (2018). New Mathematical Model of Vertical Transmission and cure of vector borne diseases and its numerical simulation. Advances in difference equations., 2018:66 https://doi-org/10.1186/s13662-018-1516-z.

Andraud, M., Hens, N., Marals, C., Beutels, P. (2012). Dynamic epidemiological models for dengue transmission; a systematic review of structure approaches. Plus one 7(11), 49085.

Atkinson, B., Hearn, P., Afrough, B., Lumley, S., Carter, D., Arons, E.J., Simpson, A.J.(2016). Brooks, T.J., Hewson, R; Detection of Zika Virus in Semen. Emerg. Infect. Dis. 22(5), 940

Atokolo, W. Aja, R. O.Aniaku, S.E., Onah, I.S., &Mbah, G.C.E (2022). Approximate Solution of the Fractional order 'sterile insect technology model via the Laplace – Adomian Decomposition Method for the Spread of Zika Virus Disease. International Journal of Mathematics and Mathematical Sciences. Volume 2022, Article ID 2297630, 2022.

Atokolo, W., &Mbah, G.C.E (2020). Modeling the control of zika virus vector population using the sterile insect technology. Journal of applied mathematics.

Brawer, F., & Castillo – Chavez, C.(2021). Mathematical Models in Populaiton Biology and Epidemiology. (Vol. 44, Pp: xxiv + 416). New York; Springer.

Caraballo, H (2014). Emergency Department Management of Mosquito – Borne illness, malaria, Dengue and West Nile Virus. Emergency Medicine Practice. 16(5), 1 – 23.

Chikaki, E., Ishikawa, H (2009). A Dengue Transmission Model in Thailand considering sequential infections with all four serotypes. J. Infect. Dev. Ctries. 3(9), 711 – 722.

Chowell, G., Diaz – Duenas, P., Miller, J.C., Alcazar – Velazco, A., Hyman, J.M., Fenimore, P.W., & Castillo – Chavez, C (2007). Estimation of the reproduction number of dengue fever from spatial epidemic data. Mathematical biosciences, 207, 571 – 589.

Danbaba, U.A. &Garba, S.M (2018). Analysis of Model for the Transmission Dynamics of Zika with Sterile Insect Technique. Texts in Biomathematics, Vol. 1, 81 – 99.

Dasti, J.I (2016).Zika Virus Infections; An overview of current Scenario. Asian Pac. J. Trop. Med. 9 (7). 621 – 625.

Diekmann, O., Heesterbeek, J.A.P., Metz, J.A.J (1990) On the Definition and the Computation of the Basic Reproduction Number Ro in Models for Infections Diseases in Heterogeneous Populations. J. Math. Biol. 28, 365.

Driessche, P.V.D., Watmough, J (2002). Reproduction Number and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission. Math. Biosciences 180, 29 – 48.

Foy, B.D., Kobylinski, K.C., Foy, J.L., Blitvich, B.J., Da Rosa, A.T., Haddow, A.D., Lanciotti, R.S., Tesh, R.B (2011). Probable non-vector borne transmission of zika virus. Emerg. Infect. Dis. 17(5), 880 – 882.

Gao, D., Lou, Y., He, D., Porco, T.C., Kuang, Y., Chowell, G., Ruan, S (2016). Prevention and Control of Zika as a mosquito – borne and sexually transmitted disease: A mathematically modelling analysis. Sci. Rep. 6. 28070.

Hills, S.L. (2016). Transmission of Zika Virus through Sexual Contact with Travellers to Areas of Ongoing Transmission – Continental United States, 2016. Morb. Mort.Wkly, Rep. 65. 215 – 216 .

Lasalle, J.P (1976). The Stability of Dynamical systems. SIAM, Philadelphia.

Lashari, A.A., Zaman, G (2011). Global Dynamics of Vector – Borne Disease with Horizontal Transmission in Lust Population Comput. Math. Appl. 61, 745 – 754.

Mosquito – Borne Diseases – American Mosquito Control Association. www.mosquito.org. Retrieved 2018 – 02 – 15.

Mosquito Control; can it stop Zika at source? World Health Organization (WHO, 2016). http://www. who.int/emergencies/ zika-virus/articles/mosquito-control/en/.

Musso, D., Roche, C., Robin, E., Nhan, T.,Teissier, A., Cao-Lormeau, V.M (2015). Potential Sexual Transmission of Zika Virus. Emerg. Infect. Dis. 21(2), 359 – 361.

Iornem T.V, Abdulkadir, S .S., Akinrefon, A. A., Ornguga I . G (2023). Modelling of Access to Mosquito Treated Net in Nigeria: Using Multilevel Logistic Regression Approach. FUDMA Journal of Sciences (FJS), https://doi.org/10.33003/fjs-2023-0703-1831, Vol.7, No. 3, 2023. Pp 144-149.

Padmanabhan, P., Seshaiyer, P., & Castillo-Chavez, C.(2017). Mathematical Modeling Analysis and Simulation of the Spread of Zika with Influence of Sexual Transmission and Preventive Measures. Letters in Biomathematics. Vol 4, No 1, 148 – 166.

Rao, V.S.H.(2009). Dynamic models and control of biological systems. Springer. Dordrecht.

WHO, (2015). Dengue and Severe Dengue, Fact Sheet n117, updated May 2015. http://www.who. int/mediacentre/factsheets/fs117/en.

Yakob, L., & Clements, A.C.A (2015) A Mathematical model of chikungunya dynamics and control. The major epidemic on Russian Island. Plus one, 8, e57448.