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THEORETICAL STUDY OF THE MATHEMATICAL MODEL ON THE POPULATION DYNAMICS OF DENGUE

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Abstract

We present a deterministic nonlinear mathematical model describing the population dynamics of Dengue which provides public health insights to the impact of dual infectivity of an Aedes aegypti with two strains of dengue, where both strains are co-circulating with cross-immunity (CI) and antibody-dependent enhancement (ADE) in the course of the dynamics of the disease. The model is rigorously analyzed qualitatively and thresholds for eradication are established.

Keywords: Basic reproduction number, disease-free equilibrium, epidemic, dengue disease, infective, co-circulating, cross-immunity (CI), antibody-dependent enhancement (ADE)

1.0 Introduction

Dengue virus (DENV), is an eruptive febrile illness transmitted by arthropods, it's the most common mosquito borne viral disease in human [1]. Dengue virus is also well known to affect the antigen presenting immune cells, a type of dendritic cell in the skin [2, 3]. However, the disease could also attack other vital body organs e.g bone marrow and Lymph nodes, macrophages in both the spleen, liver and monocytes in the blood [4]. Dengue virus which causes Dengue fever, is a vector borne flavivirus, a genus that also includes the West Nile virus (WNV) and yellow fever virus (YFV) [5]. There are four distinct sero-types of the virus in circulation among humans: DENV-1, DENV-2, DENV-3, DENV-4 and DENV-5, which is the potential fifth serotype, was recently isolated from a patient in Borneo [6]. However, it has not been cleared if this virus is capable of sustained transmission between humans [7].

Dengue virus is sustained in a life-cycle that involves vectors feed on blood and vertebrate hosts [8]. Report shows that this particular viruses are sustained in Africa and Southeast Asia forests by transmission from a genus Aedes female mosquitoes to humans through mosquito bites [9]. The vector obtains the virus from an infected human by taking a blood meal during the initial 2 to 10 days febrile phase of illness [10].

Dengue fever starts suddenly after the period of incubation of 3 to 7 days. The first is called the febrile phase, the second phase is called the critical phase and the third is called the recovery phase [11]. When invaded with a pathogen like the dengue virus, the immune system defends the body against the organism (virus) [12]. The immune system is made up of two parts; the first part that gives general protection and provides the body with the immediate defense is called the innate immune system. The innate immune response recognizes and responds rapidly to the pathogen, but it doesn't proffer long-term immunity for an infected person against an invading pathogen [4]. The second part of the immune system is responsible in producing cells that efficient to target microorganism and cells that are infected and it's called the adaptive immune system [4]. The adaptive immune system cells produce antibody- secreting B cells and cytotoxic T cells called immunoglobulin, or Ig. These antibodies secreted by the B cells recognize and bind to foreign molecules [4]. The adaptive immune system provides a person with long-term against a pathogen but takes longer time to respond unlike the innate immune response [4].

When a person recovers from a first dengue infection, the person is been protected from other three dengue serotypes for about two to three months but the protection is not for long [4]. After a short interval, the person becomes susceptible and can be infected with any of the serotypes. Individuals that are been exposed to dengue virus the second time will have high risk of severe dengue infection due to antibody-dependent enhancement [13].

Anti-body dependent enhancement (ADE), occurs when non-neutralizing antiviral proteins facilitate virus entry into host cell which can lead to increased infectivity in the cells. Documented evidence had it that memory cells provide immunity from reinfection with the dengue serotypes that cause the first infection, persons who recover for the first dengue serotype that cause the infection becomes permanently immune to that particular stain and be- come temporary immune to the other strain [14]. Individuals who recover from one dengue strain enjoy cross-immunity against other strains [15].

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Further research reveals that the first infection produces antibodies that spread the dengue viral infection and the amount of virus in the bloodstream. This occurrence can also manifest in children who acquire antibodies against dengue virus from their mothers while in the womb. Surprisingly, the existing antibodies and the antibodies produced by the memory B cells assist the virus to infect host cells more instead of destroying the virus [12]. In conclusion, the consequence of antibody dependent enhancement is that the body's immune system response actually makes the clinical manifestation of dengue worse and raises the risk of severe dengue illnesses [16].
Dengue fever without signs are discomfort and pain, skin rash, joint pain, headache, abdominal pain, nausea and vomiting, positive tourniguet test and leukopenia. Warning signs of dengue are persistent vomiting, severe abdominal pain, clinical accumulation of fluids, agitation, bleeding from mucosae letharaggy, and hepatomegaly. Severe stage of Dengue is respiratory insufficiency, accumulation of fluids, shock (dengue shock syndrome (DSS)) with severe bleeding, liver, severe organ compromise, altered conscious, heart and other organs [17].
During the febrile phase of the dengue fever, antipyretic treatment with pain relievers and oral fluid administration is recommended. Other medicine with aspirin should be avoided, a patient can be managed at home if he or she has access to a health care facility nearby with observation of daily full blood. Admission to hospital for close observation is highly needed when the case result in excessive vomiting, diarrhea, dehydration and bleeding [17].

1.0 Model Formulation

We present a new deterministic nonlinear mathematical model describing the spread dynamics of Dengue which asses and the impact of dually infected vectors on the population dynamics of Dengue taking into consideration the phenomenon of antibody dependent enhancement (ADE) and cross immunity (CI) in a given human population. The classical work, to be outlined, consider homogeneous mingling of the human and insect (mosquito) populace, so that each insect mouthful has equivalent coincidental of transferring the virus to a susceptible individuals in the populace. Hence the total populace of human at any time *t* is denoted by $N_{h0}(t)$, is sub-divided into jointly restrictive populaces of susceptible individuals $(S_{h0}(t))$ exposed humans with variant 1 $(E_{h1}(t))$, infectious humans with variant 1 $(I_{h1}(t))$, recuperated humans from variant 1 $(R_{h1}(t))$, populace of susceptible humans to variant 2 who recover from variant 1 $(S_{h12}(t))$, exposed humans with variant 2 $(E_{h2}(t))$, infectious humans with variant 2 $(I_{h2}(t))$, recuperated humans from variant 2 $(R_{h2}(t))$, population of susceptible humans to variant 1 who had earlier recuperated from variant 2 $(S_{h21}(t))$, Population of exposed humans to variant 2 who had earlier recuperated from variant 1 $(E_{h12}(t))$, population of infectious humans with variant 2 after recovering from variant 1 $(I_{h12}(t))$, population of exposed humans to variant 1 who had earlier recuperated from variant 2 $(E_{h21}(t))$, Population of infectious humans with variant 1 who had earlier recuperated from variant 2 $(I_{h21}(t))$ and population of recuperated humans from both variant $(M_h(t))$, so that

$$N_h(t) = S_{h0}(t) + E_{h1}(t) + I_{h1}(t) + R_{h1}(t) + E_{h2}(t) + I_{h2}(t) + R_{h2}(t) + S_{h12}(t) + E_{h12}(t) + I_{h12}(t) + S_{h21}(t) + E_{h21}(t) + I_{h21}(t) + M_h(t) \quad (1)$$

Likewise, the insect (mosquitoes) populace at time *t*, is split into susceptible vectors (mosquitoes) $(S_v(t))$, exposed vectors with variant 1 $(E_{v1}(t))$, infectious vectors with variant 1 $(I_{v2}(t))$, exposed vectors with variant 2 $(E_{v2}(t))$, infectious vectors with variant 2 $(I_{v2}(t))$, population of exposed vectors with variant 1 now exposed to variant 2 $(E_{v12}(t))$, infectious vectors with variant 1 now also infectious with variant 2 $(I_{v12}(t))$, exposed vectors with variant 2 now infected with variant 1 $(E_{v21}(t))$, infectious vectors with variant 2 now also infectious with variant 1 $(I_{v21}(t))$.
The susceptible human populace is assumed to be produced via enrollment at the frequency $(\Lambda_v(t))$. This populace decreases as susceptible individuals make contact with infected vectors, the case results into dengue infection with either of the variant at the rate $\lambda_{h1}, \lambda_{h2}, \lambda_{h3}$ and λ_{h1} (the force of infection with both variants), where

$$\lambda_{h1} = \frac{\beta_{vh1}(\eta_{v1}E_{v1} + I_{v1})}{N_h}$$

$$\lambda_{h2} = \frac{\beta_{vh2}(\eta_{v2}E_{v2} + I_{v2})}{N_h}$$

$$\lambda_{h3} = \frac{\beta^1{}_{vh}(\eta_{v12}E_{v12} + I_{v12})}{N_h}$$

$$\lambda_{h4} = \frac{\beta^2{}_{vh}(\eta_{v21}E_{v21} + I_{v21})}{N_h} \quad (2)$$

also β_{vh1} and β_{vh2} are the rate of Dengue transmission from vectors to humans with variant 1 and variant 2 respectively, β_{vh}^{1} and β_{vh}^{2} are the rate of Dengue transmission from vectors that are dually infected with variant 1 and variant 2. The modification parameters η_{v1} and η_{v2} model the reduced transmissibility of exposed Dengue vectors with variant 1 or variant 2 $(E_{v1}\ and\ E_{v2})$ compared to vectors with active Dengue $(I_{v1}\ and\ I_{v2})$, also η_{v12} and η_{v21} models the reduced transmissibility of exposed Dengue vectors that are dually infected $(E_{v12}\ and\ E_{v21})$ compared to vectors with active Dengue with both variant $(I_{v12}\ and\ I_{v21})$, hence the assumption $0<\eta_{v1}<\eta_{v2}<\eta_{v12}<\eta_{v21}<1$ [18]. σ_1 is the rate of increased infectiousness due to cross enhancement with variant 1, σ_2 is the rate of increased infectiousness due to cross enhancement with variant2

Likewise, the rate at which vectors becomes infected with variant 1 or 2 or both is given by $(\lambda_{v1}\ and\ \lambda_{v2})$ (the force of infection for vectors with both variants) respectively, where

$$\lambda_{v1}=\frac{\beta_{hv1}(\eta_{h1}E_{h1}+I_{h1})+\beta_{hv21}\sigma_1(\eta_{h21}E_{h21}+I_{h21})}{N_h}$$

$$\lambda_{v2}=\frac{\beta_{hv2}(\eta_{h2}E_{h2}+I_{h2})+\beta_{hv12}\sigma_2(\eta_{h12}E_{h12}+I_{h12})}{N_h} \qquad (3)$$

and β_{hv1} and β_{hv2} is the rate of Dengue transmission from humans to vectors with variant 1 or variant 2, β_{hv12} and β_{hv21} is of Dengue transmission from humans infected with variant 1 and variant 2. Also σ_1 and σ_2 are parameters that account for increased infectiousness of infected individuals with one strain but had recovered from the second variant. The modification parameters η_{h1} and η_{h2} represent the reduced transmissibility exposed humans with variant 1 or variant 2 compared to humans with active Dengue $(I_{h1}\ and\ I_{h2})$. Also $\eta_{h3}\ and\ \eta_{h4}$ models the reduced transmissibility of exposed Dengue humans that are infected with both variants $(E_{h12}\ and\ E_{h21})$ compared to humans with active Dengue with both variants $(I_{h12}\ and\ I_{h21})$, again we assume that $0<\eta_{h1}<\eta_{h2}<\eta_{h3}<\eta_{h4}<1$ Entities in each partition undergo expected mortality at the frequency μ_h.

The frequency of conversion of the susceptible human populace is generated by recruitment at the frequency Λ_h. This populace decrease as they come in contact with vectors infectious with variant 1 at the frequency λ_{h1}, with variant 2 at the frequency λ_{h2}, with the dual infected at the frequency λ_{h3} and λ_{h4} and finally by natural death at the frequency μ_h. Thus

$$\frac{dS_{h0}}{dt}=\Lambda_h-\lambda_{h1}S_{h0}-\lambda_{h2}S_{h0}-\lambda_{h3}S_{h0}-\lambda_{h4}S_{h0}-\mu_hS_{h0}, \qquad (4)$$

The parameter p_{v21} represents the proportion of new human contamination with variant 2 vectors initially infected with variant 2 and now infected with variant 1 while $(1-p_{v21})$ represent the remaining fraction of infections with variant 1 from such vectors. Similarly, p_{v12} illustrates the proportion of new humans with infections with variant 2 generated by vectors initially infected with variant 1 and now infected with variant 2, while $(1-p_{v12})$ illustrates the remaining fraction of infections with variant 1 from such vectors. The populace of infected individuals is generated by new infection at the frequency of λ_{h1}, the remaining fraction of new humans with infections with variant 2 at the rate $(1-p_{v12})\lambda_{h3}$, the remaining fraction of new human infections vectors initially infected with variant 2 and now infected with variant 1 at the frequency of $(1-p_{v21})\lambda_{h4}$ and reduces by progression of individuals (at the frequency $\gamma h1$) and natural death (at the frequency μh). Thus,

$$\frac{dE_{h1}}{dt}=\lambda_{hi}S_{h0}+(1-p_{v12})\lambda_{h3}S_{h0}+(1-p_{v21})\lambda_{h4}S_{h0}-(\gamma_{h1}+\mu_h)E_{h1}, \qquad (5)$$

The populace of infectious humans with variant 2 is generated by the advancement of infected humans with variant 2 (at the frequency γ_{h2}). This populace decreases due to recovery from variant 2 (at the frequency α_h), expected mortality (at the frequency μ_h) and dengue induced mortality (at the frequency δ_{h2}). This gives,

$$\frac{dI_{h1}}{dt} = \gamma_{h1}E_{h1} - (\alpha_{h1} + \mu_h + \delta_{h1})I_{h1}, \quad (6)$$

The recovered class from variant 2 is generated by humans from infectious class who recovered from variant 2 (at the frequency α_{h2}).

This populace is decreased by the waning cross-immunity to variant 1 due to recovery from variant 2 (at the frequency θ_1) and by natural mortality (at the frequency μ_h). Thus,

$$\frac{dR_{h1}}{dt} = \alpha_{h1} I_{h1} - (\theta_2 + \mu_h)R_{h1}. \quad (7)$$

The population of susceptible humans to variant 2 who recover from variant 1 increases by the inflow of individuals from (R_{h1}) class at the frequency θ_2. The populace is reduced due to infections with variant 2 at the frequency $\tau_{h2}\lambda_{h2}$, $\tau_{h2}r_{v12}^2\lambda_{h3}$, and $\tau_{h2}r_{v21}^2\lambda_{h4}$, respectively, where $\tau_{h2}(0 < \tau_{h2} < 1)$ represents cross-enhancement due to the previous infection with variant 1 and finally reduces by usual death (at the frequency μ_h). Thus,

$$\frac{dS_{h12}}{dt} = \theta_2 R_{h1} - \tau_2 S_{h12}\lambda_{h2} - \tau_2 r_{v12}^2 S_{h12}\lambda_{h3} - \tau_2 r_{v21}^2 S_{h12}\lambda_{h4} - \mu_h S_{h12}, \quad (8)$$

Population of infectious humans with variant 1 who lost temporary immunity to variant 1 after recovering from variant 2 (I_{h21}) isgenerated by individuals who progressed from exposed humans with variant 1 who lost temporary immunity to variant 1 after recovering from variant 2 class (E_{h21}) to infectious class (at the frequency γ_{h21}) and the populace is decreased by recovered individual with variant 2 (at frequency α_{h21}). The population further reduce as a result of dengue induced mortality (at frequency δ_{h21}) and usual mortality (at the frequency μ_h). Thus,

$$\frac{dE_{h2}}{dt} = \lambda_{hi}S_{h0} + p_{v12}\lambda_{h3}S_{h0} + p_{v21}\lambda_{h4}S_{h0} - (\gamma_{h2} + \mu_h)E_{h2} \quad (9)$$

The populace of infectious humans with variant 2 is generated by the advancement of infected humans with variant 2 (at the frequency γ_{h2}). This populace increases due to recovery from variant 2 (at the frequency α_{h2}), expected mortality (at the frequency μ_h) and dengue induced mortality (at the frequency δ_{h2}). This gives,

$$\frac{dI_{h2}}{dt} = \gamma_{h2}E_{h2} - (\alpha_{h2} + \mu_h + \delta_{h2})I_{h2}. \quad (10)$$

The recovered class from variant 2 is generated by humans from infectious class who recovered from variant 2 (at the frequency α_{h2}) this populace is decreased by the waning cross-immunity ti variant 1 due to recovery from variant 2(at the frequency θ_1) and by natural mortality (at the frequency μ_h).

Thus, $$\frac{dR_{h2}}{dt} = \alpha_{h2} I_{h2} - (\theta_1 + \mu_h)R_{h2}. \quad (11)$$

The population of susceptible humans to variant 1 who recover from variant 2 increases by the inflow of individuals from (R_{h2}) class at the frequency θ_1. The populace is reduced due to infections with variant 2 at the frequency $\tau_{h1}\lambda_{h1}$, $\tau_{h1}r_{v12}^1\lambda_{h3}$, and $\tau_{h1}r_{v21}^1\lambda_{h4}$, where $\tau_{h1}(0 < \tau_{h1} < 1)$ represent cross-enhancement due to the previous infection with variant 1 and finally reduces by usual death (at the frequency μ_h. Thus,

$$\frac{dS_{h21}}{dt} = \theta_1 R_{h2} - \tau_1 S_{h21}\lambda_{h1} - \tau_1 r_{v12}^1 S_{h21}\lambda_{h3} - \tau_1 r_{v21}^1 S_{h21}\lambda_{h4} - \mu_h S_{h21} \quad (12)$$

The population of infected humans to variant 2 who recover from variant 1 increases by the inflow of individuals from (S_{h12}) class at the frequency $\tau_{h2}\lambda_{h2}$, $\tau_{h2}r_{v12}^2\lambda_{h3}$ and $\tau_{h2}r_{v21}^2\lambda_{h4}$. This populace is reduced by progression of humans (at the frequency γ_{h12}) and natural death (at the frequency μ_h). Thus,

$$\frac{dE_{h12}}{dt} = \tau_2 S_{h12}\lambda_{h2} + \tau_2 r_{v12}^2 S_{h12}\lambda_{h3} + \tau_2 r_{v21}^2 S_{h12}\lambda_{h4} - (\gamma_{h12} + \mu_h)E_{h12} \quad (13)$$

The population of infectious humans with variant 2 who recover from variant 1 is increased by inflow of individual from

E_{h12} class at the frequency γ_{h12} and reduced recovery from variant 2 (at the frequency α_{h12}), dengue induced death (at the frequency δ_{h12}) and natural death (atthe frequency μ_h). This gives,

$$\frac{dI_{h12}}{dt} = \gamma_{h12}E_{h12} - (\alpha_{h12} + \mu_h + \delta_{h12})I_{h12} \quad (14)$$

The population of infected humans to variant 1 who recover from variant 2 increase by the inflow of individuals from (S_{h21}) class at the frequency $\tau_1\lambda_{h1}$, $\tau_1 r^1_{v12}\lambda_{h3}$ and $\tau_1 r^1_{v21}\lambda_{h4}$. This populace is reduced by progression of humans (at the frequency γ_{h21}) and natural death (at the frequency μ_h). Thus,

$$\frac{dE_{h21}}{dt} = \tau_1 S_{h21}\lambda_{h1} + \tau_1 r^1_{vv12} S_{h21}\lambda_{h3} + \tau_1 r^1_{v21} S_{h21}\lambda_{h4} - (\gamma_{h21} + \mu_h)E_{h21} \quad (15)$$

The population of infectious humans with variant 1 who lost temporary immunity to variant 1 after recovering from variant 2 (I_{h21}) is generated by individuals who progressed from exposed humans with variant 1 who lost temporary immunity to variant 1 after recovering from variant 2 class (E_{h21}) to infectious class (at the frequency (γ_{h21})) and the populace is decreased by recovered individual with variant 2 (at frequency α_{h21}). The population further reduces as a result of dengue-induced mortality (at frequency E_{h21}) and usual mortality (at the frequency μh). Thus,

$$\frac{dI_{h21}}{dt} = \gamma_{h21}E_{h21} - (\alpha_{h21} + \mu_h + \delta_{h21})I_{h21} \quad (16)$$

The recovered class from both variant is generated by humans from I_{h12} and I_{h21} (at the frequency α_{h12} and α_{h21}). This populace is de-creased by usual death (at the frequency μ_h). Hence,

$$\frac{dM_h}{dt} = \alpha_{h12}I_{h12} + \alpha_{h21}I_{h21} - (\theta_1 + \mu_h)\mu_h M_h. \quad (17)$$

The populace of susceptible vectors is increased by birth (at the frequency Λv) and decrease by infection, with Infested humans with variant 1 or 2 (at the frequency λ_{v1} or λ_{v2}, separately). The vector populace undergoes natural mortality (at the frequency μ_v). Therefore, the degree of change of the susceptible mosquitoes is given by,

$$\frac{dS_v}{dt} = \lambda_{v1}S_v - \lambda_{v1}S_v - \lambda_{v2}S_v - \mu_v E_{v1} \quad (18)$$

The populace of infected vectors with variant 1 is increased via infection at the frequency $\lambda v1$, decreased by infected vector with variant 2 at the frequency $\pi^E_{hv1}\lambda_{v2}$ *where* π^E_{hv1} (is the parameter that account for dually infected vectors), progression (at the frequency γ_{v1}) and natural death at the frequency μ_v.

This gives:

$$\frac{dE_{v1}}{dt} = \lambda_{v1}S_v - \pi^E_{hv1}\lambda_{v2}E_{v1} - (\gamma_{v1} + \mu_v)E_{v1} \quad (19)$$

The populace of infectious vectors with variant 1 is produced by the progression of infested vectors with variant 1 (at the frequency γ_{v1}), decreased by infectious vector with variant 1 (at the frequency $\pi^I_{hv1}\lambda_{v2}$) where π^I_{hv1} is a parameter that account for dually infected vector infectious with variant 1, natural mortality (at the frequency μv) and mortality due to dengue disease (at the frequency δv,). This gives:

$$\frac{dI_{v1}}{dt} = \gamma_{v1}E_{v1} - \pi^I_{hv1}\lambda_{v2}I_{v1} - (\mu_v + \delta_v)I_{v1} \quad (20)$$

The populace of infected vectors with variant 2 is generated by the infected vector (at the frequency λ_{v2}), diminished by infected vectors (at the frequency $\pi^E_{hv2}\lambda_{v1}$) where π^E_{hv2} (is the parameter that account for dually infected vector with variant 2), normal mortality (at the frequency μ_v) and progression (at the frequency γ_{v2}). This gives:

$$\frac{dE_{v2}}{dt} = \lambda_{v2}S_v - \pi^E_{hv2}\lambda_{v1}E_{v2} - (\gamma_{v2} + \mu_v)E_{v2} \quad (21)$$

The infectious vectors populace is produced by the progression of vectors from infected vectors with variant 2 (at the frequency γ_{v2}) diminished by infected vector with variant 2 (at the frequency $\pi^I_{hv1}\lambda_{v2}$) normal mortality (at the frequency μ_v) mortality as a result of dengue virus (at the frequency δ_v). Thus

$$\frac{dI_{v2}}{dt} = \gamma_{v2}E_{v2} - \pi^{I}_{hv2}\lambda_{v1}I_{v2} - (\mu_v + \delta_v)I_{v2} \quad (22)$$

The populace of vectors infected with variant 1 now infected with variant 2 is generated by the exposed vector (at the frequency $\pi^{E}_{hv1}\lambda_{v2}$), increased by infectious vector with variant 1 (at the frequency $\pi^{I}_{hv1}\lambda_{v2}$) . This populace is diminished by natural mortality (at the frequency μ_v) and progression of vectors (at the frequency γ_{v12}). This gives:

$$\frac{dE_{v12}}{dt} = \pi^{E}_{hv1}\lambda_{v2}E_{v1} + \pi^{I}_{hv1}\lambda_{v2}I_{v1} - (\gamma_{v12} + \mu_v)E_{v12}, \quad (23)$$

The Populace of infectious vectors with variant 2 now also infectious with variant 1 is generated by the progression of vectors (at the frequency γ_{v12}), and decreases by natural mortality (at the frequency μ_v) and mortality due to dengue disease (at the frequency δ_v), This gives:

$$\frac{dI_{v12}}{dt} = \gamma_{v12}E_{v12} - (\mu_v + \delta_v)I_{v12}. \quad (24)$$

The populace of infected vectors with variant 2 now exposed to variant 1 is generated by the infected vectors with variant 2(at the frequency $\pi^{E}_{hv2}\lambda_{v1}$), increased by infectious vector with variant 2(at the frequency π^{I}_{hv2}). This populace diminishes by natural mortality (at the frequency μ_v) and progression of vectors (at the frequency γ_{v21}). This gives:

$$\frac{dE_{v21}}{dt} = \pi^{E}_{hv2}\lambda_{v1}E_{v2} + \pi^{I}_{hv2}\lambda_{v1}I_{v2} - (\gamma_{v21} + \mu_v)E_{v21}. \quad (25)$$

The population of infectious vectors with variant 2 now also infectious with variant 1 is produced by the progression of vectors from exposed vector with both variant (at the frequency γ_{v21}). This populace moderated by usual mortality (at the frequency μ_v) and mortality as a result of dengue disease (at the frequency δ_v). Thus:

$$\frac{dI_{v21}}{dt} = \gamma_{v21}E_{v21} - (\mu_v + \delta_v)I_{v21} \quad (26)$$

Hence, the dengue transmission model comprises of the resulting system of 23 of nonlinear ordinary differential equations is given in (27). Figure 1 gives the graphic illustration of the exchange of humans between compartments as well the mosquito dynamics as in (26). Table (1.1) and (1.2) respectively, explain the sub-populations and parameters employed in the mathematical formulations.

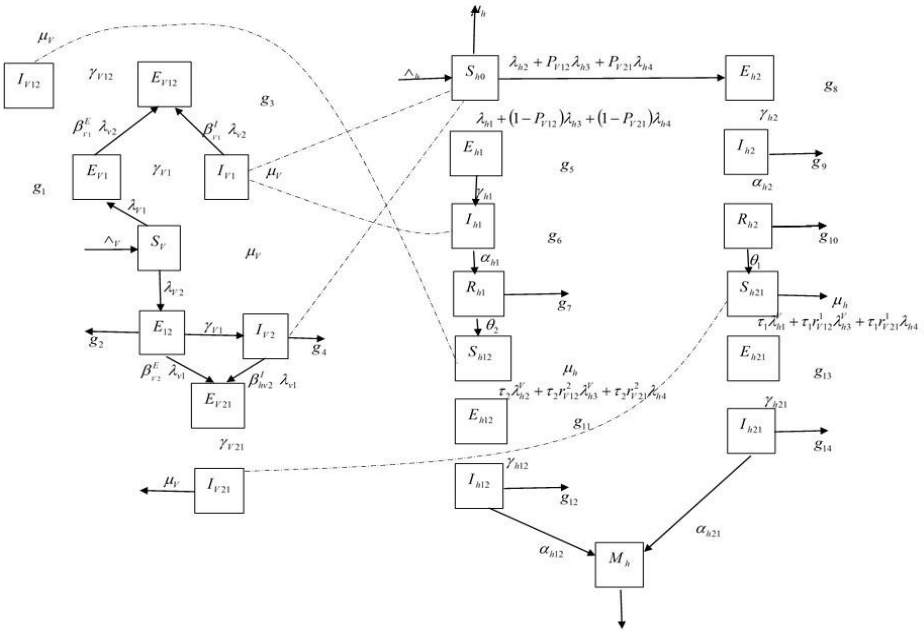

Figure 1: Schematic representation for the basic Dengue model

$$
\begin{aligned}
\frac{dS_{h0}}{dt} &= \Lambda_h - \lambda_{h1}S_{h0} - \lambda_{h2}S_{h0} - \lambda_{h3}S_{h0} - \lambda_{h4}S_{h0} - \mu_h S_{h0},\\
\frac{dE_{h1}}{dt} &= \lambda_{h1}S_{h0} + (1-p_{v12})\lambda_{h3}S_{h0} + (1-p_{v21})\lambda_{h4}S_{h0} - (\gamma_{h1}+\mu_h)E_{h1},\\
\frac{dI_{h1}}{dt} &= \gamma_{h1}E_{h1} - (\alpha_{h1}+\mu_h+\delta_{h1})I_{h1},\\
\frac{dR_{h1}}{dt} &= \alpha_{h1}I_{h1} - (\theta_2+\mu_h)R_{h1},\\
\frac{dS_{h12}}{dt} &= \theta_2 R_{h1} - \tau_2 S_{h12}\lambda_{h2} - \tau_2 r_{v12}^2 S_{h12}\lambda_{h3} - \tau_2 r_{v21}^2 S_{h12}\lambda_{h4} - \mu_h S_{h12},\\
\frac{dE_{h2}}{dt} &= \lambda_{h2}S_{h0} + p_{v12}\lambda_{h3}S_{h0} + p_{v21}\lambda_{h4}S_{h0} - (\gamma_{h2}+\mu_h)E_{h2},\\
\frac{dI_{h2}}{dt} &= \gamma_{h2}E_{h2} - (\alpha_{h2}+\mu_h+\delta_{h2})I_{h2},\\
\frac{dR_{h2}}{dt} &= \alpha_{h2}I_{h2} - (\theta_1+\mu_h)R_{h2},\\
\frac{dS_{h21}}{dt} &= \theta_1 R_{h2} - \tau_1 S_{h21}\lambda_{h1} - \tau_1 r_{v12}^1 S_{h21}\lambda_{h3} - \tau_1 r_{v21}^1 S_{h21}\lambda_{h4} - \mu_h S_{h21},\\
\frac{dE_{h12}}{dt} &= \tau_2 S_{h12}\lambda_{h2} + \tau_2 r_{v12}^2 S_{h12}\lambda_{h3} + \tau_2 r_{v21}^2 S_{h12}\lambda_{h4} - (\gamma_{h12}+\mu_h)E_{h12},\\
\frac{dI_{h12}}{dt} &= \gamma_{h12}E_{h12} - (\alpha_{h12}+\mu_h+\delta_{h12})I_{h12},\\
\frac{dE_{h21}}{dt} &= \tau_1 S_{h21}\lambda_{h1} + \tau_1 r_{vv12}^1 S_{h21}\lambda_{h3} + \tau_1 r_{v21}^1 S_{h21}\lambda_{h4} - (\gamma_{h21}+\mu_h)E_{h21},\\
\frac{dI_{h21}}{dt} &= \gamma_{h21}E_{h21} - (\alpha_{h21}+\mu_h+\delta_{h21})I_{h21},\\
\frac{dM_h}{dt} &= \alpha_{h12}I_{h12} + \alpha_{h21}I_{h21} - (\theta_1+\mu_h)\mu_h M_h,\\
\frac{dS_v}{dt} &= \lambda_{v1}S_v - \lambda_{v1}S_v - \lambda_{v2}S_v - \mu_v E_{v1},\\
\frac{dE_{v1}}{dt} &= \lambda_{v1}S_v - \pi_{hv1}^E\lambda_{v2}E_{v1} - (\gamma_{v1}+\mu_v)E_{v1},\\
\frac{dI_{v1}}{dt} &= \gamma_{v1}E_{v1} - \pi_{hv1}^I\lambda_{v2}I_{v1} - (\mu_v+\delta_v)I_{v1},\\
\frac{dE_{v2}}{dt} &= \lambda_{v2}S_v - \pi_{hv2}^E\lambda_{v1}E_{v2} - (\gamma_{v2}+\mu_v)E_{v2},\\
\frac{dI_{v2}}{dt} &= \gamma_{v2}E_{v2} - \pi_{hv2}^I\lambda_{v1}I_{v2} - (\mu_v+\delta_v)I_{v2},\\
\frac{dE_{v12}}{dt} &= \pi_{hv1}^E\lambda_{v2}E_{v1} + \pi_{hv1}^I\lambda_{v2}I_{v1} - (\gamma_{v12}+\mu_v)E_{v12},\\
\frac{dI_{v12}}{dt} &= \gamma_{v12}E_{v12} - (\mu_v+\delta_v)I_{v1},\\
\frac{dE_{v21}}{dt} &= \pi_{hv2}^E\lambda_{v1}E_{v2} + \pi_{hv2}^I\lambda_{v1}I_{v2} - (\gamma_{v21}+\mu_v)E_{v21},\\
\frac{dI_{v2}}{dt} &= \gamma_{v21}E_{v21} - (\mu_v+\delta_v)I_{v21},
\end{aligned}
\qquad (27)
$$

Table 1.1: Definitions of model variables

State Variables	Description
$S_{h0}(t)$	Populace of susceptible humans
$E_{h1}(t)$	Populace of humans exposed to variant 1
$I_{h1}(t)$	Populace of infectious individual with Variant 1
$R_{h1}(t)$	Populace of recovered humans from variant 1
$E_{h2}(t)$	Populace of humans exposed to variant 2
$I_{h2}(t)$	Populace of infectious humans with variant 2
$R_{h2}(t)$	Populace of recovered humans from variant 2
$S_{h12}(t)$	Populace of susceptible humans to variant 2 who recover from variant 1
$E_{h12}(t)$	Populace of exposed humans to variant 2 who had earlier recovered from variant 1
$I_{h12}(t)$	Populace of infectious humans with variant 2 after recovering from variant 1
$S_{h21}(t)$	Populace of susceptible humans to variant 1 who had earlier recovered from variant 2
$E_{h21}(t)$	Populace of exposed humans to strain 1 who had earlier recovered from variant 2
$I_{h21}(t)$	Populace of infectious humans with variant 1 who lost temporary 8immunity to variant 1 after recovering from variant 2
$M_h(t)$	Populace of recovered humans from both variant.
$S_v(t)$	Populace of susceptible vectors
$E_{v1}(t)$	Populace of exposed vectors to variant 1
$I_{v1}(t)$	Populace of infectious vectors with variant 1
$E_{v2}(t)$	Populace of exposed vectors to variant 2
$I_{v2}(t)$	Populace of infectious vectors with variant 2
$E_{v12}(t)$	Populace of exposed vectors with variant 1 now exposed to variant 2
$I_{v12}(t)$	Populace of infectious vectors with variant 1 now also infectious with variant 2
$E_{v21}(t)$	Populace of exposed vectors with variant 2 now infected with variant 1
$I_{v21}(t)$	Populace of infectious vectors with variant 2 now also infectious with variant 1 height

Table 1.2: Definition of model parameters

Parameter	Description
Δ_h	Human enrollment frequency
μ_h	natural death rate of humans
Δ_v	Vector recruitment frequency
μ_v	natural death frequency of vectors
β_{hv}	Spread probability from humans to vectors
β_{vh}	Spread probability from vectors to humans
π_{hv}	Adjustment parameter to dually infected vector
π_{hv}	Adjustment parameter to dually infected vector
η_{h1}	Adjustment parameters that accounts for the Lessen transmissibility of unprotected humans with variant 1
η_{h2}	Adjustment parameters that accounts for the Lessen transmissibility of unprotected humans with variant 2
η_{v1}	Adjustment parameters that accounts for the Lessen transmissibility of unprotected vectors with variant 1
η_{v2}	Adjustment parameters that accounts for the Lessen transmissibility of exposed vectors with variant 2
α_{h1}	Recovery rate of humans from variant 1
α_{h2}	Recovery rate of humans from variant 2
γ_{v1}	Progression rate of exposed vectors with variant 1
γ_{v2}	Progression rate of exposed vectors with variant 2
γ_{h1}	Progression rate of exposed humans with variant 1
γ_{h2}	Progression rate of humans with variant 2
δ_{h1}	Disease prompted mortality for humans with variant 1
δ_{h2}	Disease prompted mortality for humans with variant 2
δ_{h12}	Disease prompted mortality for humans with strain 2 who recovered from variant 1
δ_{h21}	Disease prompted mortality for humans with variant 1 who recovered from variant
θ_1	Cross immunity for variant 1
θ_2	Cross immunity for variant 2
τ_1	Cross enhancement due to previous infection with variant 1
τ_2	Cross enhancement due to previous infection with variant 2
r_{12}^1	Proportion of variant 1
r_{12}^2	Proportion of variant 2
r_{21}^1	Proportion of variant 1
r_{21}^2	Proportion of variant 2
σ_1	Increased infectiousness due to cross enhancement to variant 1
σ_2	Increased infectiousness due to cross enhancement to variant 2
p_{v12}	Fraction of new humans infections with variant 2
p_{v21}	Fraction of new humans infections with variant 1

2.0 MODEL ANALYSIS

We prove that all state variables of the model (27) are always positive for all time, t and that the orbits generated are positively invariant for all time.

Theorem 2.0 Let the initial figures for the dengue model (4) be given as

$S_v(0) > 0,\ E_{v1}(0) > 0,\ I_{v1}(0) > 0,\ E_{v2}(0) > 0,\ I_{v2}(0) > 0,\ E_{v12}(0) > 0,\ I_{v12}(0) > 0,\ E_{v21}(0) > 0,\ I_{v21}(0) > 0,\ S_{h0}(0) > 0,\ E_{h1}(0) > 0,$
$I_{h1}(0) > 0,\ R_{h1}(0) > 0,\ S_{h12}(0) > 0,\ E_{h2}(0) > 0,\ I_{h2}(0) > 0,\ R_{h2}(0) > 0,\ S_{h21}(0) > 0,\ E_{h12}(0) > 0,\ I_{h12}(0) > 0,\ E_{h21}(0) > 0,$
$I_{h21}(0) > 0,$ and $M_h(0) > 0$.

Then the trajectories

$(s_v(t), E_{v1}(t), I_{v1}(t), E_{v2}(t), I_{v2}(t), E_{v12}(t), I_{v12}(t), E_{v21}(t), I_{v21}(t),$
$s_{h0}(t), E_{h1}(t), I_{h1}(t), R_{h1}(t), s_{h12}(t), E_{h2}(t), I_{h2}(t), R_{h2}(t), s_{h21}(t), E_{h21}(t), I_{h21}(t), M_h(t))$

of the model with positive initial conditions, will remain positive for all time $t > 0$.

Proof: Following [19] and [20], the model (4) can be written in the form

$$\frac{dY_1}{dt} = F_1 + G_1 \qquad (5)$$

$$Y_1 = (S_{h0}(t), E_{h1}(t), I_{h1}(t), R_{h1}(t), S_{h12}(t), E_{h2}(t), I_{h2}(t), R_{h2}(t), S_{h21}(t), E_{h12}(t), I_{h12}(t), E_{h21}(t), I_{h21}(t), M_h(t),$$
$$S_v(t), E_{v1}(t), I_{v1}(t), E_{v2}(t), I_{v2}(t), E_{v12}(t), I_{v12}(t), E_{v21}(t), I_{v21}(t),)$$

where

$G_1 = (\Lambda_h\ 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)^T$,

and $f(Y_1)$, is a 23 x 23 matrix given as

$$f(Y_1) = \begin{pmatrix} f_{1(12x12)} & f_{2(12x11)} \\ f_{3(11x12)} & f_{4(11x11)} \end{pmatrix}$$

Where

$$f_1 = \begin{pmatrix} -(F_1+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ F_2 & g_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\gamma_{h1} & -g_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_{h1} & -g_7 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ F_3 & 0 & 0 & 0 & -g_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_{h2} & -g_9 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_{h2} & -g_{10} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta_2 & 0 & 0 & 0 & -(F_4+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \theta_1 & 0 & -(F_5+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & F_4 & 0 & -g_{11} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{h12} & -g_{12} & 0 & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & F_5 & 0 & 0 & -g_{12} & \end{pmatrix}$$

$$f_2 = \begin{pmatrix} 0 & 0 & 0 & -\beta_{vh1}\eta_{v1} & -\beta_{vh1} & -\beta_{vh2}\eta_{v2} & -\beta_{vh2} & -\beta^1_{vh}\eta_{v12} & -\beta^1_{vh} & -\beta^2_{vh}\eta_{v21} & -\beta^2_{vh} \\ 0 & 0 & 0 & \beta_{vh1}\eta_{v1} & \beta_{vh1} & 0 & 0 & F_{12} & F_{13} & F_{14} & F_{15} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_{vh2}\eta_{v2} & \beta_{vh2} & F_{16} & F_{17} & F_{18} & F_{19} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\tau_2\beta_{vh2}\eta_{v2} & -\tau_2\beta_{vh2} & -F_{20} & -F_{21} & -F_{22} & -F_{23} & & \\ 0 & 0 & 0 & -\tau_1\beta_{vh1}\eta_{v1} & -\tau_1\beta_{vh1} & 0 & 0 & -F_{24} & F_{25} & -F_{26} & -F_{27} \\ 0 & 0 & 0 & 0 & 0 & \tau_2\beta_{vh2}\eta_{v2} & \tau_2\beta_{vh2} & -F_{20} & F_{21} & F_{22} & F_{23} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\tau_1\beta_{vh1}\eta_{v1} & \tau_1\beta_{vh1} & 0 & 0 & F_{24} & F_{25} & F_{26} & F_{27} \end{pmatrix}$$

$$f_3 = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{h2} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{h12} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$f_4 = \begin{pmatrix} -g_{14} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha_{h21} & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(F_6+F_7+\mu_v) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & F_6 & -F_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_{h2} & -F_9 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & F_7 & 0 & 0 & -F_{10} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_{v2} & -F_{11} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \pi^E_{hv1}F_7 & \pi^I_{hv1}F_7 & 0 & 0 & g_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{v12} & -\mu_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \pi^E_{hv2}F_6 & \pi^I_{hv2}F_6 & 0 & 0 & -g_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{v21} & -\mu_v \end{pmatrix}$$

Where

$$F_1 = \frac{\beta_{vh1}(\eta_{h1}Y_{16}+Y_{17})+\beta_{vh2}(\eta_{v2}Y_{19}+Y_{19})+\beta^1_{vh}(\eta_{v12}Y_{20}+Y_{21})+\beta^2_{vh}(\eta_{v21}Y_{22}+Y_{23})}{N_h},$$

$$F_2 = \frac{\beta_{vh1}(\eta_{v1}Y_{16}+Y_{17})+(1-p_{v12})\beta^1_{vh}(\eta_{v12}Y_{20}+Y_{21})+(1-p_{v21})\beta^2_{vh}(\eta_{v21}Y_{22}+Y_{23})}{N_h},$$

$$F_3 = \frac{\beta_{vh2}(\eta_{v2}Y_{18}+Y_{19})+p_{v12}\beta^1_{vh}(\eta_{v12}Y_{20}+Y_{21})+p_{v21}\beta^2_{vh}(\eta_{v21}Y_{22}+Y_{23})}{N_h},$$

$$F_4 = \frac{\tau_2(\beta_{vh2}(\eta_{v2}Y_{18}+Y_{19})+r^2_{v12}\beta^1_{vh}(\eta_{v12}Y_{20}+Y_{21})+r^2_{v21}\beta^2_{vh}(\eta_{v21}Y_{22}+Y_{23}))}{N_h},$$

$$F_5 = \frac{\tau_1(\beta_{vh1}(\eta_{v1}Y_{16}+Y_{17})+r^1_{v12}\beta^1_{vh}(\eta_{v12}Y_{20}+Y_{21})+r^1_{v21}\beta^2_{vh}(\eta_{v21}Y_{22}+Y_{23}))}{N_h},$$

$$F_6 = \frac{\beta_{vh1}(\eta_{h1}Y_2+Y_3)+\beta_{hv21}\sigma_{h1}(\eta_{ha}Y_{12}+Y_{13})}{N_h}, \quad F_7 = \frac{\beta_{vh2}(\eta_{h2}Y_5+Y_6)+\beta_{hv12}\sigma_{h2}(\eta_{h3}Y_{10}+Y_{11})}{N_h},$$

$$g_1 = \gamma_{vi}+\mu_v,\ g_2 = \gamma_{v2}+\mu_v,\ g_3 = \gamma_{v12}+\mu_v,\ g_4 = \gamma_{v21}+\mu_v,\ g_5 = \gamma_{h1}+\mu_h,$$
$$g_6 = \alpha_{h1}+\mu_h+\delta_{h1},\ g_7 = \theta_2+\mu_h,\ g_8 = \gamma_{h2}+\mu_h,\ g_9 = \alpha_{h2}+\mu_h+\delta_{h2},\ g_{10} = \theta_1+\mu_h,$$
$$g_{11} = \gamma_{h12}+\mu_h+\delta_{h12}, \quad g_{13} = \gamma_{h21}+\mu_h, \quad g_{14} = \alpha_{h21}+\mu_h+\delta_{h21},$$

and

$$F_8 = \pi^E_{hv1}F_7+g_1,\ F_9 = \pi^I_{hv1}F_7+\mu_v,\ F_{10} = \pi^E_{hv2}F_6+g_2,\ F_{11} = \pi^I_{hv2}F_6+\mu_v,\ F_{12} = \beta^1_{vh}\eta_{v12}(1-p_{v12})$$
$$F_{13} = \beta^1_{vh}(1-p_{v12}), \quad F_{14} = \beta^2_{vh}\eta_{v21}(1-p_{v21}),\ F_{15} = \beta^2_{vh}(1-p_{v21}),\ F_{16} = \beta^1_{vh}\eta_{v21}p_{v21},$$
$$F_{17} = \beta^1_{vh}p_{v12},\ F_{18} = \beta^2_{vh}\eta_{v21}p_{v21},\ F_{19} = \beta^2_{vh}p_{v21},\ F_{20} = \tau_2 r^2_{v12}\beta^1_{vh}\eta_{v12},\ F_{21} = \tau_2 r^2_{v12}\beta^1_{vh},\ F_{22} = \tau_2 r^2_{v12}\beta^2_{vh}\eta_{v21},$$
$$F_{23} = \tau_2 r^2_{v12}\beta^2_{vh},\ F_{24} = \tau_1 r^1_{v12}\beta^1_{vh}\eta_{v12},\ F_{25} = \tau_1 r^1_{v12}\beta^1_{vh},\ F_{26} = \tau_1 r^1_{v12}\beta^2_{vh}\eta_{v21},\ F_{27} = \tau_1 r^1_{v12}\beta^2_{vh}.$$

also,

$$Y_1 = S_{h0},\ Y_2 = E_{h1},\ Y_3 = I_{h1},\ Y_4 = R_{h1},\ Y_5 = E_{h2},\ Y_6 = I_{h2},\ Y_7 = R_{h2},\ Y_8 = S_{h12},\ Y_9 = E_{h12},\ Y_{10} = I_{h12},\ Y_{11} = S_{h21},$$
$$Y_{12} = E_{h21},\ Y_{13} = I_{h21}\ Y_{14} = M_h \quad Y_{15} = S_v,\ Y_{16} = E_{v1},\ Y_{17} = I_{v1},\ Y_{18} = E_{v21},\ Y_{19} = I_{v2},\ Y_{20} = E_{v12},\ Y_{21} = I_{v12},$$
$$Y_{22} = E_{v21},\ Y_{23} = I_{v21}$$

Hence, exhausting the statement that $G_1 \geq 0$ and that the matrix $f(Y_1)$ is quasi-positive. It follows that (4) is positively-invariant in R^{23}_+. This shows that the orbits generated by the system (4) will always be positive for all non-negative initial data.

Furthermore, by the comparison theorem [21].

$$N^{/}_h(t) = \Lambda_h - \mu_h(S_{ho}+E_{h1}+I_{h1}+R_{h1}+E_{h2}+I_{h2}+R_{h2}+S_{h12}+E_{h12}+I_{h12}+S_{h21}+E_{h21}+I_{h21}+M_h)$$
$$-\delta_{h1}I_{v1}-\delta_{h2}I_{v2}-\delta_{h12}I_{v12},-\delta_{h21}I_{v21} \leq \Lambda_h - \mu_h N_h \quad \textit{and}$$
$$N^{/}_v(t) = \Lambda_v - \mu_v(S_v+E_{v1}+I_{v1}+E_{v2}+I_{v2}+R_{h2}+E_{v12}+I_{v12}+E_{v21}+I_{v21}) = \Lambda_v - \mu_v N_v$$

Can be written as

$$N_h(t) \leq N_h(0)\exp(-\mu_h(t))+\frac{\Lambda_h}{\mu_h}[1-\exp(-\mu_h(t))], \quad \textit{so that}$$

$$Lim_{t\to\infty}\sup N_h(t) \leq \frac{\Lambda_h}{\mu_h}$$

$$N_v(t) = N_v(0)\exp(-\mu_v(t))+\frac{\Lambda_v}{\mu_v}[1-\exp(-\mu_v(t))], \quad \textit{so that}$$

$$Lim_{t\to\infty}\sup N_v(t) \leq \frac{\Lambda_v}{\mu_h}$$

3.0 Local Asymptotic stability (LAS) of Disease-free Equilibrium (DFE)

The model system (4) has a DFE given by,

$$\xi_0 = (S^0_{h0}, E^0_{h1}, I^0_{h1}, R^0_{h1}, S^0_{h12}, E^0_{h2}, I^0_{h2}, R^0_{h2}, S^0_{h21}, E^0_{h12}, I^0_{h12}, E^0_{h21}, I^0_{h21}, M^0_h, S^0_v, E^0_{v1}, I^0_{v1},$$
$$I^0_{v2}, E^0_{v2}, I^0_{v12}, E^0_{v12}, E^0_{v21}, I^0_{v21}) = (\frac{\Lambda_h}{\mu_h},0,0,0,0,0,0,0,0,0,0,0,0,0,0,\frac{\Lambda_v}{\mu_v},0,0,0,0,0,0,0,0). \qquad (28)$$

The LAS of the DFE is investigated using the next generation matrix operator method [21]. The matrix, F is the matrix of new infections while the matrix V is the matrix of the other transfer terms given by

$$F = \begin{pmatrix} 0_{(8x8)} & F_{1(8x8)} \\ F_{2(8x8)} & 0_{(8x8)} \end{pmatrix}, \quad \textit{and} \quad V = \begin{pmatrix} V_{1(8x8)} & 0_{(8x8)} \\ 0_{(8x8)} & V_{2(8x8)} \end{pmatrix} \qquad (29)$$

With

$$F_1=\begin{pmatrix} \beta_{hv1}\eta_{h1}\frac{S_v}{N_h} & \beta_{hv1}\frac{S_v}{N_h} & 0 & 0 & 0 & 0 & \beta_{hv21}\sigma_{h1}\eta_{h4}\frac{S_v}{N_h} & \beta_{hv21}\sigma_{h1}\frac{S_v}{N_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{hv2}\eta_{h2}\frac{S_v}{N_h} & \beta_{hv2}\frac{S_v}{N_h} & \beta_{hv12}\sigma_{h2}\eta_{h3}\frac{S_v}{N_h} & \beta_{hv12}\sigma_{h2}\frac{S_v}{N_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$F_2=\begin{pmatrix} \beta_{vh1}\eta_{v1} & \beta_{vh1} & 0 & 0 & (1-p_{vh12})\beta^1_{vh}\eta_{v12} & (1-p_{vh12})\beta^1_{vh} & (1-p_{vh21})\beta^2_{vh}\eta_{v21} & (1-p_{vh21})\beta^2_{vh} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{vh2}\eta_{v2} & \beta_{vh2} & \beta_{hv2}\eta_{h2}\frac{S_v}{N_h} & \beta_{hv2}\frac{S_v}{N_h} & \beta_{hv12}\sigma_{h2}\eta_{h3}\frac{S_v}{N_h} & \beta_{hv12}\sigma_{h2}\frac{S_v}{N_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V_1=\begin{pmatrix} g_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_{v1} & \mu_v & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma_{v2} & \mu_v & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & g_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_{v12} & \mu_v & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & g_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\gamma_{v21} & \mu_v \end{pmatrix}$$

$$V_2=\begin{pmatrix} g_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_{h1} & g_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma_{h2} & g_9 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & g_{11} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_{h12} & g_{12} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & g_{13} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\gamma_{h21} & g_{14} \end{pmatrix}$$

The effective reproduction number of the model (4) $\Re_D=\rho(FV^{-1})=\max[\Re_{01},\Re_{02}]$,

With ρ being the spectral radius of FV^{-1}, is given by

$$\Re_{01}=\sqrt{\frac{\beta_{hv1}\beta_{vh1}\Lambda_v\mu_h(\gamma_{h1}+g_6\eta_{h1})(\gamma_{v1}+\eta_{v1}\mu_v)}{\Lambda_h\mu_v^2 g_1 g_5 g_6}},$$

$$\Re_{02}=\sqrt{\frac{\beta_{hv2}\beta_{vh2}\Lambda_v\mu_h(\gamma_{h2}+g_6\eta_{h2})(\gamma_{v2}+\eta_{v2}\mu_v)}{\Lambda_h\mu_v^2 g_1 g_5 g_6}}, \qquad (30)$$

where $g_1=\gamma_{v1}+\mu_v$, $g_2=\gamma_{v2}+\mu_v$, $g_5=\gamma_{h1}+\mu_h$, $g_6=\alpha_{h1}+\mu_h+\delta_{h1}$,
$g_8=\gamma_{h2}+\mu_h$, $g_9=\alpha_{h2}+\mu_h+\delta_{h2}$.

Using Theorem 2 in [21] we claim the following result:

Lemma 3.1 The disease free state (DFE) of the model (4) is locally asymptotically stable (LAS) in

D if $\Re_D < 1$, *and unstable if* $\Re_D > 1$.

4.0 Backward Bifurcation Analysis

Theorem 4.0: The model (27) exhibit backward bifurcation phenomenon at $\Re_D = 1$ whenever a bifurcation coefficient, denoted by a is positive.

Proof:

The existence of backward bifurcation is explored using the Center manifold Theory [22]

Let

$S_v = x_1,\ E_{v1} = x_2,\ I_{v1} = x_3,\ E_{v2} = x_4,\ I_{v2} = x_5,\ E_{v12} = x_6\ I_{v12} = x_7\ E_{v21} = x_8,\ I_{v21} = x_9, S_{h0} = x_{10},$

$E_{h1} = x_{11}, I_{h1} = x_{12}, R_{h1} = x_{13}, E_{h2} = x_{14}, I_{h2} = x_{15}, R_{h2} = x_{16}, S_{h12} = x_{17}, E_{h12} = x_{18}, I_{h12} = x_{19}, S_{h21}, = x_{20},$

$E_{h21}, = x_{21}, I_{h21}, = x_{22}, M_h = x_{23},$

It follows, that the model (27) can be re-written as

The forces of infection λ_{h1}, λ_{h2}, λ_{h3}, λ_{h4}, λ_{v1} and λ_{v2} are given as follows

$$
\left.
\begin{aligned}
\frac{dx_1}{dt} &= F_1 \equiv \Lambda_v - \lambda_{v1}x_1 - \lambda_{v1}x_1 - \lambda_{v2}x_1 - \mu_v x_1,\\
\frac{dx_2}{dt} &= F_2 \equiv \lambda_{v1}x_1 - \pi^E_{hv1}\lambda_{v2}x_2 - (\gamma_{v1} + \mu_v)x_2,\\
\frac{dx_3}{dt} &= F_3 \equiv \gamma_{v1}x_2 - \pi^I_{hv1}\lambda_{v2}x_3 - (\mu_v + \delta_v)x_3,\\
\frac{dx_4}{dt} &= F_4 \equiv \lambda_{v2}x_1 - \pi^E_{hv2}\lambda_{v1}x_4 - (\gamma_{v2} + \mu_v)x_4,\\
\frac{dx_5}{dt} &= F_5 \equiv \gamma_{v2}x_4 - \pi^I_{hv2}\lambda_{v1}x_5 - (\mu_v + \delta_v)x_5,\\
\frac{dx_6}{dt} &= F_6 \equiv \pi^E_{hv1}\lambda_{v2}x_5 + \pi^I_{hv1}\lambda_{v2}x_6 - (\gamma_{v12} + \mu_v)x_6,\\
\frac{dx_7}{dt} &= F_7 \equiv \gamma_{v12}x_6 - (\mu_v + \delta_v)x_7,\\
\frac{dx_8}{dt} &= F_8 \equiv \pi^E_{hv2}\lambda_{v1}x_4 + \pi^I_{hv2}\lambda_{v1}x_5 - (\gamma_{v21} + \mu_v)x_8,\\
\frac{dx_9}{dt} &= F_9 \equiv \gamma_{v21}x_8 - (\mu_v + \delta_v)x_9,\\
\frac{dx_{10}}{dt} &= F_{10} \equiv \Lambda_h - \lambda_{hi}x_{10} - \lambda_{h2}x_{10} - \lambda_{h3}x_{10} - \lambda_{h4}x_{10} - \mu_h x_{10},\\
\frac{dx_{11}}{dt} &= F_{11} \equiv \lambda_{hi}x_{10} + (1 - p_{v12})\lambda_{h3}x_{10} + (1 - p_{v21})\lambda_{h4}x_{10} - (\gamma_{h1} + \mu_h)x_{11},\\
\frac{dx_{12}}{dt} &= F_{12} \equiv \gamma_{h1}x_{11} - (\alpha_{h1} + \mu_h + \delta_{h1})x_{12},\\
\frac{dx_{13}}{dt} &= F_{13} \equiv \alpha_{h1}x_{12} - (\theta_2 + \mu_h)x_{13},\\
\frac{dx_{14}}{dt} &= F_{14} \equiv \theta_2 x_{13} - \tau_2\lambda_{h2}x_{14} - \tau_2 r^2_{v12}\lambda_{h3}x_{14} - \tau_2 r^2_{v21}\lambda_{h4}x_{14} - \mu_h x_{14},\\
\frac{dx_{15}}{dt} &= F_{15} \equiv \lambda_{h2}x_{10} + p_{v12}\lambda_{h3}x_{10} + p_{v21}\lambda_{h4}x_{10} - (\gamma_{h2} + \mu_h)x_{15},\\
\frac{dx_{16}}{dt} &= F_{16} \equiv \gamma_{h2}x_{15} - (\alpha_{h2} + \mu_h + \delta_{h2})x_{16},\\
\frac{dx_{17}}{dt} &= F_{17} \equiv \alpha_{h2}x_{16} - (\theta_1 + \mu_h)x_{17},\\
\frac{dx_{18}}{dt} &= F_{18} \equiv \theta_1 x_{17} - \tau_1\lambda_{h1}x_{18} - \tau_1 r^1_{v12}\lambda_{h3}x_{18} - \tau_1 r^1_{v21}\lambda_{h4}x_{x18} - \mu_h x_{18},\\
\frac{dx_{19}}{dt} &= F_{19} \equiv \tau_2\lambda_{h2}x_{14} + \tau_2 r^2_{v12}\lambda_{h3}x_{14} + \tau_2 r^2_{v21}\lambda_{h4}x_{14} - (\gamma_{h12} + \mu_h)x_{19},\\
\frac{dx_{20}}{dt} &= F_{20} \equiv \gamma_{h12}x_{19} - (\alpha_{h12} + \mu_h + \delta_{h12})x_{20},\\
\frac{dx_{21}}{dt} &= F_{21} \equiv \tau_1\lambda_{h1}x_{18} + \tau_1 r^1_{vw12}\lambda_{h3}x_{18} + \tau_1 r^1_{v21}\lambda_{h4}x_{18} - (\gamma_{h21} + \mu_h)x_{21},\\
\frac{dx_{22}}{dt} &= F_{22} \equiv \gamma_{h21}x_{21} - (\alpha_{h21} + \mu_h + \delta_{h21})x_{22},\\
\frac{dx_{23}}{dt} &= F_{23} \equiv \alpha_{h12}x_{20} + \alpha_{h21}x_{22} - (\theta_1 + \mu_h)\mu_h x_{23},
\end{aligned}
\right\} \quad (31)
$$

$$\lambda_{h1}=\frac{\beta_{vh1}(\eta_{v1}x_2+x_3)}{N_h}\qquad \lambda_{h2}=\frac{\beta_{vh2}(\eta_{v2}x_4+x_5)}{N_h}$$

$$\lambda_{h3}=\frac{\beta^1{}_{vh}(\eta_{v12}x_6+x_7)}{N_h}$$

$$\lambda_{h4}=\frac{\beta^2{}_{vh}(\eta_{v21}x_8+x_9)}{N_h}$$

$$\lambda_{v1}=\frac{\beta_{hv1}(\eta_{h1}x_{11}+x_{12})+\beta_{hv21}\sigma_1(\eta_{h21}x_{19}+x_{20})}{N_h}$$

$$\lambda_{v2}=\frac{\beta_{hv2}(\eta_{h2}x_{15}+x_{16})+\beta_{hv12}\sigma_2(\eta_{h12}x_{21}+x_{22})}{N_h} \qquad (32)$$

Where $N_h=x_{10}+x_{11}+x_{12}+x_{13}+x_{14}+x_{15}+x_{16}+x_{17}+x_{18}+x_{19}+x_{20}+x_{21}+x_{22}+x_{23}$

Consider the case with $\beta_{hv1}=\beta_{hv2}=\beta^*$, a bifurcation parameter. Solving for $\beta_{hv1}=\beta^*$ from $\Re_0=1$ yields

$$\beta_{hv1}=\beta^*=\frac{\mu_v N_h^* g_1 g_5 g_6}{x_1^*\beta_{hv1}(\gamma_{h1}+g_6\eta_{h1})(\gamma_{v1}+\eta_{v1}\mu_v)}=\frac{\mu_v N_h^* g_2 g_7 g_9}{x_1^*\beta_{hv2}(\gamma_{h2}+g_9\eta_{h2})(\gamma_{v2}+\eta_{v2}\mu_v)} \qquad (33)$$

The Jacobian of the system (33) at the DFE with $\beta_{hv1}=\beta^*$, is given by:

$$J_{\beta^*}=J(\varepsilon_0)|_{\beta^*}=\begin{pmatrix} J_{1(12x12)} & J_{2(12x11)} \\ J_{3(11x12)} & J_{4(11x11)} \end{pmatrix} \qquad (34)$$

The matrix J_{β^*} has a simple zero eigenvalue and all other eigenvalues have negative real parts with J_1 *to* J_4 given as follows

$$J_1=\begin{pmatrix}
-\mu_v & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\beta^*_{hv1}\eta_{h1}\frac{x_1^*}{N_h^*} & -\beta^*_{hv1}\frac{x_1^*}{N_h^*} \\
0 & -g_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta^*_{hv1}\eta_{h1}\frac{x_1^*}{N_h^*} & \beta^*_{hv1}\frac{x_1^*}{N_h^*} \\
0 & \gamma_{v1} & -\mu_v & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -g_2 & -\mu_v & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \gamma_{v2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -g_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \gamma_{v12} & -\mu_v & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_v & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{v21} & -\mu_v & 0 & 0 & 0 \\
0 & -\beta_{vh1}\eta_{v1} & -\beta_{vh1} & -\beta_{vh2}\eta_{v2} & -\beta_{vh2} & -\beta^1_{vh}\eta_{v12} & -\beta^1_{vh} & -\beta^2_{vh}\eta_{v21} & -\beta^2_{vh} & -\mu_h & 0 & 0 \\
0 & \beta_{vh1}\eta_{v1} & \beta_{vh1} & 0 & 0 & P_1\beta^1_{vh}\eta_{v12} & P_1\beta^1_{vh} & P_2\beta^2_{vh}\eta_{v21} & P_2\beta^2_{vh} & 0 & -g_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{h1} & -g_6
\end{pmatrix}$$

With $P_1=1-p_{v12}$ *and* $P_2=1-p_{v21}$

$$J_3=\begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{h1} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \beta_{vh2}\eta_{v2} & \beta_{vh2}p_{v12} & \beta^1_{vh}\eta_{v12}p_{v12} & \beta^1_{vh}p_{v12} & \beta^2_{vh}\eta_{v21}p_{v21} & \beta^2_{vh}p_{v21} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}$$

$$J_4=\begin{pmatrix}
-g_7 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\theta_2 & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -g_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \gamma_{h2} & -g_9 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \alpha_{h2} & -g_{10} & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \theta_1 & -\mu_h & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -g_{11} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \gamma_{h12} & -g_{12} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -g_{13} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_{h21} & -g_{14} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{h12} & 0 & \alpha_{h21} & -\mu_h
\end{pmatrix}$$

4.0: The Jacobian J_{β^*} has a right eigenvector given by W= $(w_1, w_2, w_3, ..., w_{23})^T$

Where

$$w_1 = -\frac{1}{\mu_\upsilon}(w_2 g_2 + w_4 g_2),\ w_2 = w_2 > 0,\ w_3 = \frac{\gamma_{\upsilon 1} w_2}{\mu_\upsilon},\ w_4 = w_4 > 0,\ w_5 = \frac{\gamma_{\upsilon 2} w_4}{\mu_\upsilon},\ w_6 = 0,$$

$$w_7 = 0,\ w_8 = 0,\ w_9 = 0,\ w_{10} = -\frac{1}{\mu_h \mu_\upsilon}(\beta_{vh1} w_2(\eta_{v1}\mu_v + \gamma_{v1}) + \beta_{vh2} w_4(\eta_{v2}\mu_v + \gamma_{v2})),$$

$$w_{11} = \frac{(\beta_{vh1} w_2(\eta_{v1}\mu_v + \gamma_{v1})}{g_5 \mu_v}, w_{12} = \frac{(\beta_{vh1}\gamma_{h1} w_2(\eta_{v1}\mu_v + \gamma_{v1})}{g_5 g_6 \mu_v},\ w_{13} = \frac{(\beta_{vh1}\gamma_{h1}\alpha_{h1} w_2(\eta_{v1}\mu_v + \gamma_{v1})}{g_5 g_6 g_7 \mu_v}$$

$$w_{14} = \frac{(\beta_{vh1}\gamma_{h1}\alpha_{h1}\theta_2 w_2(\eta_{v2}\mu_v + \gamma_{v2})}{g_5 g_6 g_7 \mu_v \mu_h},\ w_{15} = \frac{(\beta_{vh2} w_4(\eta_{v2}\mu_v + \gamma_{v2})}{g_8 \mu_v},\ w_{16} = \frac{(\beta_{vh2}\gamma_{h2} w_4(\eta_{v2}\mu_v + \gamma_{v2})}{g_8 g_9 \mu_v},$$

$$w_{17} = \frac{(\beta_{vh2}\gamma_{h2}\alpha_{h2} w_4(\eta_{v2}\mu_v + \gamma_{v2})}{g_8 g_9 g_{10} \mu_v},\ w_{18} = \frac{(\beta_{vh2}\gamma_{h2}\alpha_{h2}\theta_1 w_4(\eta_{v2}\mu_v + \gamma_{v2})}{g_8 g_9 g_{10} \mu_v \mu_h},$$

$$w_{19} = w_{20} = w_{21} = w_{22} = w_{23} = 0 \qquad (35)$$

Similarly J_{β^*} has a left eigenvector $V = (v_1, v_2, v_3 ..., v_{23})$, satisfying V.W=1, with

$$a = -\frac{2v_2}{N_h^*} w_1 \beta_{hv1}^* (\eta_{h1} w_{11} + w_{12}) - \frac{2v_4}{N_h^*} w_1 \beta_{hv2} (\eta_{h2} w_{15} + w_{16}) - \frac{2v_{11}}{N_h^*} w_{10} \beta_{hv1}^* (\eta_{h1} w_2 + w_3)$$

$$- \frac{2v_2}{N_h^*} w_1 \beta_{hv2} (\eta_{h2} w_4 + w_5)$$

$$v_1 = 0,\ v_2 = v_2 > 0,\ v_1 = 0,\ v_3 = \frac{\beta_{vh1}\beta_{hv1}^* v_2 (g_6\eta_{h1} + \gamma_{h1})}{g_5 g_6 \mu_v N_h^*},\ v_4 = v_4 > 0,\ v_5 = \frac{\beta_{vh1}\beta_{hv1}^* v_4 (g_9\eta_{h2} + \gamma_{h2})}{g_8 g_9 \mu_v N_h^*},$$

$$v_6 = \frac{\beta_{vh}^1 [g_8 g_9 (1 - p_{v12})\beta_{hv1}^* x_1^* v_2 (g_6\eta_{h1} + \gamma_{h1}) + g_5 g_6 p_{v12}\beta_{hv1}^* x_1^* v_4 (g_9\eta_{h2} + \gamma_{h2}))(\gamma_{v12}\eta_{v21}\mu_v)]}{g_3 g_5 g_6 g_8 g_9 \mu_v N_h^*},$$

$$v_7 = \frac{\beta_{vh}^1 [g_8 g_9 (1 - p_{v12})\beta_{hv1}^* x_1^* v_2 (g_6\eta_{h1} + \gamma_{h1}) + g_5 g_6 p_{v12}\beta_{hv1}^* x_1^* v_4 (g_9\eta_{h2} + \gamma_{h2})]}{g_5 g_6 g_8 g_9 \mu_v N_h^*},$$

$$v_8 = \frac{\beta_{vh}^1 [g_8 g_9 (1 - p_{v21})\beta_{hv1}^* x_1^* v_2 (g_6\eta_{h1} + \gamma_{h1}) + g_5 g_6 p_{v21}\beta_{hv1}^* x_1^* v_4 (g_9\eta_{h2} + \gamma_{h2})]}{g_5 g_6 g_8 g_9 \mu_v N_h^*},$$

$$v_9 = \frac{\beta_{vh}^2 [g_8 g_9 (1 - p_{v21})\beta_{hv1}^* x_1^* v_2 (g_6\eta_{h1} + \gamma_{h1}) + g_5 g_6 p_{v21}\beta_{hv1}^* x_1^* v_4 (g_9\eta_{h2} + \gamma_{h2})]}{g_5 g_6 g_8 g_9 \mu_v N_h^*},\ v_{10} = 0,$$

$$v_{11} = \frac{\beta_{hv1}^* x_1^* v_2 (g_6\eta_{h1} + \gamma_{h1})}{g_5 g_6 N_h^*},\ v_{12} = \frac{\beta_{hv1}^* x_1^* v_2}{g_6 N_h^*},\ v_{13} = 0,\ v_{14} = 0,\ v_{15} = \frac{\beta_{hv1}^* x_1^* v_4 (g_9\eta_{h2} + \gamma_{h2})}{g_8 g_9 N_h^*},$$

$$v_{16} = \frac{\beta_{hv1}^* x_1^* v_4}{g_9 N_h^*},\ v_{17} = 0,\ v_{18} = 0,\ v_{19} = \frac{\beta_{hv12}\sigma_{h2} x_1^* v_4 (g_{12}\eta_{h3} + \gamma_{h12})}{g_{11} g_{12} N_h^*},\ v_{20} = \frac{\beta_{hv12}\sigma_{h2} x_1^* v_4}{g_{12} N_h^*},$$

$$v_{21} = \frac{\beta_{hv21}\sigma_{h1} x_1^* v_2 (g_{14}\eta_{h4} + \gamma_{h21})}{g_{13} g_{14} N_h^*},\ v_{22} = \frac{\beta_{hv21}\sigma_{h1} x_1^* v_2}{g_{14} N_h^*},\ v_{23} = 0, \qquad (36)$$

4.1 Computation of bifurcation coefficients a and b

Applying the Center Manifold Theory as stated in [22], we compute the associated non-zero partial derivatives of the right hand sides of the transformed system (31), (evaluated at the DFE with $\beta = \beta^*$) the associated bifurcation coefficients, a and b, are given by

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \quad b = \sum_{k,i,j=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \qquad (37)$$

Using the expression above in (37), the associated non-zero partial derivatives for **a** and **b** where derived and after several algebraic calculations, we considered the case of the system (27) without Dengue-induced death, increased susceptibility to variant i after recovery cross enhancement from variant j and dual infectivity of vectors (relative susceptibility of vectors initially infected with variant i to variant j). If we make the following substitution $\delta_{h1} = \delta_{h2} = \pi_{hv1}^E = \pi_{hv1}^I = \pi_{hv2}^E = \pi_{hv2}^I = \tau_1 = \tau_2 = 0,$ the expression for **a** reduces to

$$b = \frac{v_2 x_1^*}{N_h^*}(\eta_{h1} w_{11} + w_{12}) > 0 \qquad (38)$$

Clearly, $a < 0$ since $w_2, w_3, w_4, w_5, w_{11}, w_{12}, w_{15}, w_{16}, v_2, v_4, v_{11}$ *and* v_{15} are all greater than zero while w_1 *and* w_{10} are less than zero.

Hence, backward bifurcation does not occurs if and only if the disease-induced deaths for humans $(\delta_{h1}$ *and* $\delta_{h2})$, increased susceptibility to strain i after recovery (cross enhancement) from strain j $(\tau_1$ *and* $\tau_2)$ and dual infectivity of vectors (relative susceptibility of vectors initially infected with strain i to j) $\pi_{hv1}^E, \pi_{hv2}^E, \pi_{hv1}^I$ *and* π_{hv2}^I are absent.

4.2 CONCLUSION

The threshold measure $\Re_D$, is the effective reproduction number of the disease. It is the average number of secondary dengue infections produced by an infected individual in a completely susceptible populace. The epidemiological implication of Lemma 3.1 is that when $\Re_D$ is less than unity, dengue can be eradicated from the populaces if the initial sizes of the sub-population of the model are in the basin of attraction of the DFE, ξ_0 in a population with 2 variants co-circulating. Thus, a little number of dengue-infected individuals (with both variants) into the community will not produce large dengue outbreaks in the population where there are vectors dually-infected with both strains of the disease present in the population, and the disease will die out with time. Furthermore the last sections of our work confirmed that backward bifurcation phenomenon can be induced by cross-enhancement and dual infectivity of vectors.

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