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VERTICALLY TRANSMITTED VECTOR-BORNE DISEASES AND THE EFFECTS OF EXTREME TEMPERATURE

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Abstract: An existing compartmental model of vector-borne diseases is considered to incorporate vertical transmissions in the vector and the host populations. The effects of extrinsic incubation rate of the disease causing pathogen in mosquitoes on the epidemic as well as the endemic nature of the disease are assessed for different values of model parameters. Our numerical simulations indicate that if measures such as personal protection and mosquito control are intensified, then the negative effects of weather-enhanced parameters could be significantly diminished. The effectiveness of these measures is also shown to reduce epidemic levels due to vertical transmissions in the vector as well as the host populations. The existence of a backward bifurcation and its reactions to changes of parameters reacting to temperature increase and the global stability of the disease-free equilibrium, under some conditions are analytically established.

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Key Words: backward bifurcation, vertical transmission, extrinsic incubation rate

1. Introduction

In addition to horizontal transmission cycle (vector-host-vector), some mosquito

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species pass the disease causing pathogens to their progeny by transovarian transmission (also known as vertical transmission). While infected hosts are absent, disease carrying adult mosquitoes emerge following favorable weather conditions (such as hot weather and abundant rainfall). One possible reason for this is disease transmission to eggs which survive a dry season to evolve into adult stage as infectious [1, 19]. Furthermore, the configuration of weather conditions such as, favorable temperature and precipitation affects the biological dynamics of the vector as well as the disease causing pathogen. Transmission potential of vector-borne diseases are also affected by a small increase of temperature as it is documented in studies of malaria and dengue fever [28, 37]. The addition of vertical transmission to this could bring substantial challenges to the fight against disease burden.

Vertical transmission is observed in dengue virus transmitting mosquitoes of Aedes aegypti, Aedes albopitus and Culex species [1, 21, 33] and other mosquitoborne flavivirus [38]. Similarly, the survival of the Rift Valley fever (RVf) causing virus in infected eggs of Aedes aegypti is likely a key factor in the epidemic cycle following extreme climate and weather events (such as extreme temperature along with heavy rainfall and flood). It should also be noted that although Rift Valley fever is transmitted by two species of mosquitoes, Culex and Aedes aegypti, only the Aedes aegypti species transmit the pathogen to their progeny [15, 24]. Additionally, vertical transmissions of West Nile virus (WNv) in Culex and Aedes aegypti species of mosquitoes [3, 26], and yellow fever in Aedes aegypti [20] are known challenges.

Vertical transmissions of a few vector-borne diseases also occur in host populations and this could contribute to the emergence of the disease in non-endemic areas, owing it to human movement [18]. Notable examples include sleeping sickness (African trypanosomiasis) [2, 42] and Chagas disease [8, 29, 34]. In rare cases, mother-to-child transmission of some vector-borne diseases occur, for example, malaria (related to delivery) [36] and dengue fever due to blood transfusion, breast feeding [12] or dengue related complications during pregnancy [17]. However, while vertical transmission plays part of the role for emergence and reemergence of these diseases [16, 36], their prevalence in the host and the mosquito populations is enhanced when, among other factors, personal protection and mosquito control are poor. Part of our work will look into these and the role of extreme weather conditions on the dynamics of vector-borne diseases.

The effects of extreme weather conditions on the dynamics of vector-borne diseases in general are vital challenges to the fight against many vector-borne diseases [13, 41]. Extreme temperatures such as very hot and dry followed by

heavy rain could cause emergence of vector-borne diseases where initial epidemic could lead to endemic state. The pre-adult stages in aquatic habitat of *Anophelis* and *Aedes aegypti* synchronize with rising temperature that sustains mosquito survival. This increases the recruitment of adult mosquitoes of these species which are known to transmit diseases (such as WNv, RVf, dengue and malaria). Recently, a number of cases have been studied to address the connections between extreme weather conditions and vector-borne diseases. Among them, WNv [19, 30], dengue virus [1], malaria [37] and RVf [15, 24] are some examples worth mentioning.

Extreme weather could reduce the average time interval for the pathogen to complete its life cycle in the vector (also known as extrinsic incubation period). This gives the adult female mosquito ample time to transmit the disease to susceptible hosts before it dies. Furthermore, high temperature also increases the incubation rate of mosquitoes (shorten the development time from egg to adult stage) in the mean time causing the mosquito density to increase. Another parameter affected by high temperature is the biting rate of the vector, which could increase the rate of effective contacts with the host. In perspective, rising temperature and precipitation facilitate conditions that could trigger emergence or reemergence and outbreak of vector-borne diseases, especially for vertically transmitted vector-borne diseases. Our work assesses the endemic and epidemic effects of key parameters such as extrinsic and intrinsic incubation rate, disease-induced death rate of hosts, vertical transmission and personal protection.

A number of mathematical models, specifically systems of differential equations of vector-borne diseases have been developed to assess the potential impacts of different measures to mitigate disease burden (see for example, dengue fever [1, 16, 25, 23], Rift Valley fever [15, 24], West Nile virus [5, 7, 19, 31, 32] and sleeping sickness [2]). However, very few models have incorporated vertical transmission in mosquitoes, some examples are: the connection of epidemic seasonal changes and vertical transmission of RVf in some mosquito vectors [15], the vaccination of livestock and the problem of larvacide-based interventions [24], emergence of seasonal outbreak of WNv from the endemic state [19], and maturation delay of mosquitoes and its impact on outbreak of WNv [22]. Additionally, the efficiency of vertical transmission to cause outbreaks of dengue virus is addressed in [1].

This paper is organized as follows. The model studied in [6] is extended to incorporate vertical transmission and density dependent recruitment rate in the host population and an invariant subset of the positive octant is provided in Section (2). In Section (3), an epidemiological threshold is formulated and the asymptotic dynamics of the model, specifically, the existence of

a backward bifurcation and the effects of key model parameters, such as, the disease-induced death rate, mosquito-to-human ratio and disease transmission rates is established. Simulation results about the effects of increased extrinsic incubation rate on the bifurcation and on the endemic level are also presented in Section (3). Other simulations about the epidemic levels of the disease and their reactions to vertical transmission in the vector and the host, and disease transmission rates are provided in Section (5). Then the global stability of the disease-free equilibrium point is established in Section (6) which is followed by concluding remarks in Section (7).

2. The Main Model

A model for a vertically-transmitted vector-borne disease is formulated extending what is given in [6]. To this end, the host population is grouped into four compartments: susceptible, exposed (no symptom), infectious and treated (or immune) which are denoted by x_1 , x_2 , x_3 and x_4 , respectively and the total population size of the host is $N = x_1 + x_2 + x_3 + x_4$. On the other hand, the vector population is grouped into three compartments, susceptible, exposed and infectious with sizes y_1 , y_2 and y_3 , respectively with the total population of the vector given by $P = y_1 + y_2 + y_3$.

In [6], it is assumed that vectors are exposed after biting only an infectious host, but in this work, a more realistic approach is considered: vectors are assumed to be exposed after they bite (with average contact rate ϕ per day) an infectious host or a host who is exposed to the disease but asymptomatic and could transmit the disease. This means, infection in the vector population is the sum of two incidence functions namely, $\frac{\phi\theta_1x_3}{N}$ which describes infection by biting infectious hosts, and $\frac{\phi\theta_2x_2}{N}$ infections due to biting exposed hosts who carry the infection in their blood. Thus, the incidence function for vectors is

$$\frac{\phi\theta_1x_3}{N} + \frac{\phi\theta_2x_2}{N}.$$

The exposed vectors become infectious after an incubation period of $1/\varepsilon$. Note that θ_1 and θ_2 are transmission efficacy of the disease from infectious and exposed hosts, respectively. Once infected, vectors carry the disease throughout their life time. In the host population, the exposed group, x_2 , increases as a result of effective bites from infectious vectors (with incidence function $\frac{\phi \beta y_3 x_1}{N}$), those who carry it from birth (from infected parent $\Lambda \zeta_1 x_3$). The exposed group is diminished due to disease progression, but the incubation period $\frac{1}{d}$ for inoculated host could vary even remarkably and we take average values. Because

Parameter	Description (rates are per day)			
μ	host death rate (when density is ignored)			
ζ_1	vertical disease transmission rate (in the host)			
ζ_2	vertical disease transmission rate (in the vector)			
δ_0	average number of new adult female mosquitoes			
δ_1	a factor for density dependent maturation of mosquitoes to			
	adulthood			
Λ	per capita birth rate of host			
r	host recovery rate			
β	the probability that the disease is transmitted			
	from an infected vector to a host per contact			
ρ	host recruitment rate (assumed susceptible)			
ϕ	the number of contacts between a host and a vector			
α	disease-induced host death rate			
ψ	fading rate of treatment to make hosts susceptible to the			
	disease			
γ	density independent death rate of vectors			
ε	incubation rate of the disease in a vector			
d	incubation rate of the disease in a host			
θ_1	transmission efficacy of the disease from infectious host to			
	vector			
θ_2	transmission efficacy of the disease from exposed host to			
	vector			

Table 1: Model parameter and their descriptions.

of a delayed time in hosts to stay as exposed but asymptomatic, incubation periods are also important parameters in models of vector-borne diseases.

The size of infectious hosts, x_3 , is reduced due to recovery at a rate of r and also as a result of disease-induced death rate, α , and natural death which is assumed to have the same rate μ in each class. The susceptible host population is increased as a result of new recruits (by birth) and from addition of treated hosts who lost immunity (at a rate of ψ). Earlier models of different vector-borne diseases in malaria [14], dengue fever [23], sleeping sickness [2] and West Nile virus [5] have all made the assumption of susceptibility (losing immunity) after recovery. Lasting protective immunity against any one of the vector-borne diseases which are known to have a wide spread effects is not known yet. The

foregoing modeling assumptions extend the model given in [6] to

$$\frac{dx_1}{dt} = \rho + \Lambda(x_1 + x_2 + x_4) + \Lambda(1 - \zeta_1)x_3 + \psi x_4 - \frac{\beta \phi y_3 x_1}{N} - \mu x_1
\frac{dx_2}{dt} = \frac{\beta \phi y_3 x_1}{N} + \Lambda \zeta_1 x_3 - dx_2 - \mu x_2
\frac{dx_3}{dt} = dx_2 - (r + \alpha + \mu)x_3
\frac{dx_4}{dt} = rx_3 - (\psi + \mu)x_4
\frac{dy_1}{dt} = \delta_0 + \delta_1(y_1 + y_2) + \delta_1(1 - \zeta_2)y_3 - \frac{\phi \theta_1 x_3 y_1}{N} - \frac{\phi \theta_2 x_2 y_1}{N} - \gamma y_1
\frac{dy_2}{dt} = \frac{\phi \theta_1 x_3 y_1}{N} + \frac{\phi \theta_2 x_2 y_1}{N} + \delta_1 \zeta_2 y_3 - \varepsilon y_2 - \gamma y_2
\frac{dy_3}{dt} = \varepsilon y_2 - \gamma y_3,$$
(2.1)

with initial conditions $x_i(0) \ge 0$, $i = 1, \dots, 4$ and $y_i(0) \ge 0$, i = 1, 2, 3. If $\zeta_1 = \zeta_2 = \theta_2 = 0$, and if the birth rates in both populations are constant, this model reduces to the model studied in [6]. A list of parameters along with their definitions is given in Table 1.

Adding the first four equations in (2.1) yields the dynamics of the host population

$$\frac{dN}{dt} = \rho + \Lambda N - \alpha x_3 - \mu N \le \rho + \Lambda N - \mu N. \tag{2.2}$$

Note that $\frac{dz}{dt} = \rho + \Lambda z - \mu z$ is a one-dimensional autonomous equation with attracting set $[0, N^*]$, where $N^* = \frac{\rho}{\mu - \Lambda}$ is a positive equilibrium point of $\frac{dz}{dt} = \rho + \Lambda z - \mu z$, $\mu > \Lambda$. It should be noted that in the absence of disease-induced death rate, this describes the dynamics of the total population, with equilibrium N^* . Moreover, from differential inequality [27], (2.2) implies that $N(t) \leq z(t)$ for $N(0) \leq z(0)$. Thus, for initial values $0 \leq N(0) \leq N^*$ the host population size N(t) remains in $[0, N^*]$. Furthermore, if $N(0) > N^*$ then N(t) approaches N^* . Similarly, adding the last three equations in (2.1), yields the dynamics of the vector population

$$\frac{dP}{dt} = \delta_0 + \delta_1 P - \gamma P \tag{2.3}$$

with a positive equilibrium $P^* = \frac{\delta_0}{\gamma - \delta_1}$, $\gamma > \delta_1$. It is clear that for initial values $0 \le P(0) < P^*$ the vector population P(t) remains in $[0, P^*]$.

Define vectors X and Y by $X = (x_1, x_2, x_3, x_4)$ and $Y = (y_1, y_2, y_3)$. Based on the above discussion, the set

$$\Omega = \{(X, Y) \in R_+^4 \times R_+^3, \sum_{i=1}^4 x_i \in [0, N^*],$$

$$\sum_{i=1}^3 y_i \in [0, P^*]\} = [0, \frac{\rho}{\mu - \Lambda}] \times [0, \frac{\delta_0}{\gamma - \delta_1}].$$
(2.4)

is forward invariant under system (2.1) and furthermore, it is attractor. Moreover, for $N(0) \geq \frac{\rho}{\mu - \Lambda}$, $N(t) \rightarrow \frac{\rho}{\mu - \Lambda}$. Similarly, $P(0) \geq \frac{\delta_0}{\gamma - \delta_1}$ implies that $P(t) \rightarrow \frac{\delta_0}{\gamma - \delta_1}$. Putting these results together, we have the following theorem.

Theorem 2.1. Ω is positively invariant under system (2.1).

We study the model given by (2.1) restricting the state variables to Ω .

3. Epidemiology Threshold

The disease-free equilibrium point (DFE) of (2.1) is

$$E_0 = (x_1^*, 0, 0, 0, y_1^*, 0, 0), \tag{3.1}$$

where
$$x_1^* = \frac{\rho}{\mu - \Lambda}$$
, $y_1^* = \frac{\delta_0}{\gamma - \delta_1}$, $\gamma > \delta_1$, $\mu > \Lambda$.

The basic reproduction number, \mathcal{R}_0 , is defined as the number of secondary infections that one infectious individual infects during the time period it survives as infectious in a susceptible population, and where the disease is vertically transmitted. The epidemiological threshold \mathcal{R}_0 is vital to draw many conclusions which are linked to model parameters. In this paper one of our goals are to investigate the dynamic effects of model parameters which are influenced by extreme weather changes along with vertical transmission and those connected to disease control measures. The threshold R_0 will serve this purpose. This threshold could have different forms depending on the specific approaches implemented to derive it (see for example, [24], [39]), however, its definition and use remain the same: it is an index used in control methods, it further indicates conditions which could make the disease endemic. We calculate \mathcal{R}_0 by using the approaches in [39]. To do this, we define a vector valued function \widetilde{F}_1 for rate of new infection cases in the infected and recovered groups of populations in x_2, x_3, x_4, y_2, y_3 , which is

$$\widetilde{F} = (\frac{\beta \phi y_3 x_1}{N} + \Lambda \zeta_1 x_3, 0, 0, \frac{\phi \theta_1 x_3 y_1}{N} + \frac{\phi \theta_2 x_2 y_1}{N} + \delta_2 \zeta_2 y_3, 0).$$

We also define another function \widetilde{V} for the transmission terms between the disease infected compartments listed above and the exit terms (by mortality or emigration), that is,

 $\widetilde{V} = (k_1x_2, -dx_2 + k_3x_3, -rx_3 + k_2x_4, k_4y_2, -ky_2 + \gamma y_3),$ where the parameters k_i , i = 1, ..., 4 which are used for clarity, are given by

$$k_1 = d + \mu, \quad k_2 = \psi + \mu, \quad k_3 = r + \alpha + \mu, \quad k_4 = \varepsilon + \gamma.$$
 (3.2)

Next, we evaluate F and V, which are the Jacobian matrices of \widetilde{F} and \widetilde{V} , respectively evaluated as functions of the vector $(x_2, x_3, x_4, y_2, y_3)$ at the disease-free equilibrium E_0 given by (3.1),

Clearly,

$$V^{-1} = \begin{pmatrix} k_1^{-1} & 0 & 0 & 0 & 0 \\ d(k_1k_3)^{-1} & k_3^{-1} & 0 & 0 & 0 \\ rd(k_1k_2k_3)^{-1} & r(k_1k_2)^{-1} & k_2^{-1} & 0 & 0 \\ 0 & 0 & 0 & k_4^{-1} & 0 \\ 0 & 0 & 0 & \varepsilon(k_4\gamma)^{-1} & \gamma^{-1} \end{pmatrix}$$

and as it is given in [39], the basic reproduction number $R_0(\zeta)$ is defined as the spectral radius of

Thus, the spectral radius of FV^{-1} is

$$\mathcal{R}_0(\zeta_1, \zeta_2) = \rho(FV^{-1})$$

$$= \frac{1}{2} \left(\frac{\zeta_1 \Lambda d}{k_1 k_3} + \frac{\zeta_2 \delta_1 \varepsilon}{\gamma k_4} \right) + \sqrt{\left[\frac{1}{2} \left(\frac{\zeta_1 \Lambda d}{k_1 k_3} - \frac{\zeta_2 \delta_1 \varepsilon}{\gamma k_4} \right) \right]^2 + \mathcal{R}_h^2}, \tag{3.3}$$

where

$$\mathcal{R}_h = \phi \sqrt{\frac{\beta \varepsilon (\theta_1 d + \theta_2 k_3) y_1^*}{\gamma k_1 k_3 k_4 x_1^*}},$$
(3.4)

with $x_1^* = \frac{\rho}{\mu - \Lambda}$ and $y_1^* = \frac{\delta_0}{\gamma - \delta_1}$. Note that \mathcal{R}_h is the basic reproduction number for horizontal transmission of the disease where as, the terms with ζ_1 and ζ_2 are contributions from vertical transmissions of the disease in the host and the vector, respectively. It should be clear that in the absence of vertical transmission in the host population $(\zeta_1 = 0)$, \mathcal{R}_0 reduces to

$$\mathcal{R}_0 = \mathcal{R}_0(0, \zeta_2) = \rho(FV^{-1}) = \frac{\zeta_2 \delta_1 \varepsilon}{2\gamma k_4} + \sqrt{\left(\frac{\zeta_2 \delta_1 \varepsilon}{2\gamma k_4}\right)^2 + \mathcal{R}_h^2}.$$
 (3.5)

Our model could be applied to study a vector-borne disease where vertical transmission takes place in the host population like in Chaga's disease and Rift Valley fever through birth or exposure to infected blood or tissue. In more general cases, such as these, the threshold $\mathcal{R}_0(\zeta_1, \zeta_2)$ given by (3.3) could be implemented. However, for diseases like West Nile virus and dengue virus, vertical transmission in the host is less common. On the other hand, vertical transmission of diseases such as dengue virus, RVf and WNv is common in some mosquito species. Motivated by these reasons, we set $\zeta_1 = 0$ throughout the analytical part of our work. However, simulation results are presented to highlight the reaction of disease epidemic as vertical transmission of the disease in the host increases relative to vector-host contact rates. By Theorem 2 in [39] the disease-free equilibrium point, E_0 , of (2.1) is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. Thus, we have the following theorem.

Theorem 3.1. The disease-free equilibrium point E_0 , of system (2.1) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$, where \mathcal{R}_0 is defined by (3.3).

Theorem (3.1) highlights that when $\mathcal{R}_0 < 1$ the disease could be eliminated for small initial values. Bifurcation analysis is done on the model (2.1) to see how the dynamics of the disease reacts to changes in key parameters such as disease-induced death rate and those connected to mosquito dynamics, such as vertical transmission rates, extrinsic and intrinsic incubation rates and mosquito-to-host ratio. Basically, these parameters are influenced by climate conditions and the results in the coming two sections will address that.

3.1. Endemic Equilibrium Points

Essentially, the reproduction rates of the host and the vector are both density dependent. To this end, recall that the disease-free equilibrium is

$$E_0 = (x_1^*, 0, 0, 0, y_1^*, 0, 0),$$

where $x_1^* = \frac{\rho}{\mu - \Lambda}$, $\mu > \Lambda$, $y_1^* = \frac{\delta_0}{\gamma - \delta_1}$ and $\gamma > \delta_1$. Furthermore, since we focus on vertical transmission in the vector, we set $\zeta_1 = 0$. With these, the reproduction number $\mathcal{R}_0 = \mathcal{R}_0(\zeta_2)$ given by (3.5) reduces to

$$\mathcal{R}_0 = \frac{\zeta_2 \delta_1 \varepsilon}{2\gamma k_4} + \sqrt{\left(\frac{\zeta_2 \delta_1 \varepsilon}{2\gamma k_4}\right)^2 + \mathcal{R}_h^2},\tag{3.6}$$

where

$$\mathcal{R}_h = \phi \sqrt{\frac{\beta \delta_0 \varepsilon (\mu - \Lambda)(d\theta_1 + k_3 \theta_2)}{\rho \gamma (\gamma - \delta_1) k_1 k_3 k_4}}.$$
 (3.7)

Next, we define some constants to be used in the endemic equilibrium point, $E_1 = (x_1^*, x_2^*, x_3^*, x_4^*, y_1^*, y_2^*, y_3^*)$ - we drop * for clarity. We study the dynamics of system (2.1) as the threshold R_0 changes. The components of the endemic equilibrium points $E_1 = (x_1, x_2, x_3, x_4, y_1, y_2, y_3)$ satisfy the equations

$$x_1 = \frac{x_2 k_1}{\pi_1}, \quad x_3 = \frac{dx_2}{k_3}, \quad x_4 = \frac{dr x_2}{k_2 k_3} \quad \text{and} \quad x_2 = \frac{\rho k_2 k_3 \pi_1}{A \pi_1 + B},$$
 (3.8)

$$y_1 = (\frac{\gamma k_4 - \zeta_2 \delta_1 \varepsilon}{\gamma \pi_2}) y_2, \quad y_3 = \frac{\varepsilon \delta_0 \pi_2}{F \pi_2 + G}, \quad y_2 = \frac{\gamma \pi_2 \delta_0}{F \pi_2 + G},$$
 (3.9)

where

$$\pi_1 = \frac{\phi \beta y_3}{N}, \quad \pi_2 = \frac{\phi(\theta_1 x_3 + \theta_2 x_2)}{N},$$

$$F = (\gamma - \delta_1)k_4, \quad G = (\gamma - \delta_1)(\gamma k_4 - \zeta_2 \delta_1 \varepsilon), \tag{3.10}$$

with

$$A = (\mu - \Lambda)((\psi + \mu)(d + r + \alpha + \mu) + rd) + d\alpha(\psi + \mu) > 0,$$

$$B = k_1 k_2 k_3 (\mu - \Lambda),$$

$$D = (\psi + \mu)(r + \alpha + \mu) + d(\psi + \mu) + dr \text{ and}$$

$$N = x_1 + x_2 + x_3 + x_4 = \frac{\rho(k_1 k_2 k_3 + \pi_1 D)}{\pi_1 A + B}.$$
(3.11)

Note that $\gamma k_4 > \zeta_2 \delta_1 \varepsilon$ which could be verified from $\gamma > \delta_1$ and $k_4 > \varepsilon$. Thus, F, G and the equilibrium component $y_1 = (\frac{\gamma k_4 - \zeta_2 \delta_1 \varepsilon}{\gamma \pi_2}) y_2$ are all positive.

Using (3.8), (3.9) and (3.10) along with $N = x_1 + x_2 + x_3 + x_4$ (see Appendix A for the details) we get

$$a\pi_1^2 + b\pi_1 + c = 0. (3.12)$$

The coefficients of this quadratic equation are given by

$$a = \rho(\gamma - \delta_1)D(Ek_4 + D\mathcal{M}),$$

$$b = \rho(\gamma - \delta_1)k_1k_2k_3(Ek_4 + 2D\mathcal{M}) - \phi\beta\delta_0\varepsilon AE \quad \text{and}$$

$$c = (k_1k_2k_3)^2\rho\gamma k_4(\gamma - \delta_1)(1 - \frac{\delta_1\zeta_2\varepsilon}{\gamma k_4} - \mathcal{R}_h^2),$$
(3.13)

where

$$\mathcal{M} = \gamma(\varepsilon + \gamma) - \zeta_2 \delta_1 \varepsilon \quad \text{and}$$

$$E = \phi(\psi + \mu)(\theta_1 d + \theta_2 (r + \alpha + \mu)).$$
(3.14)

The conditions for the existence of endemic equilibrium (unique or multiple) points of the system (2.1) are given as follows.

Theorem 3.2. The system given by (2.1) has

- (i) a unique endemic equilibrium if c < 0 (i.e. if $\mathcal{R}_0 > 1$);
- (ii) a unique endemic equilibrium if b < 0 and c = 0 or $b^2 4ac = 0$;
- (iii) has two endemic equilibria if c > 0, b < 0 and $b^2 4ac > 0$;
- (iv) no endemic equilibria otherwise.

Here, it should be clear that $\mathcal{R}_0 > 1$ implies that $\mathcal{R}_h^2 > 1 - \frac{\delta_1 \zeta_2 \varepsilon}{\gamma k_4}$ which then implies, based on the definition of c (see equation (3.13)), that c < 0 which is (i) of Theorem (3.2). Similarly, $\mathcal{R}_0 < 1$ implies that c > 0. Therefore, to have two endemic equilibrium points for $\mathcal{R}_0 < 1$, we need b < 0 and $b^2 - 4ac > 0$, which means two positive equilibria are possible when (iii) of this theorem holds. Furthermore, under case (iii) of Theorem (3.2), the quadratic (3.12) has two positive roots π_1 and π_1^* . From these two roots, we get two positive values for x_i , i = 1, ..., 4 and also two positive values of π_2 (see (3.10)). This yields two positive values of y_i , i = 1, 2, 3, thus, getting two endemic equilibria. Furthermore, (i) of Theorem (3.2) implies that the system given by (2.1) has a unique endemic equilibrium for $\mathcal{R}_0 > 1$. It is also clear that when $b^2 - 4ac < 0$, there is no endemic equilibrium and it is a common practice to get the critical value \mathcal{R}_c of the basic reproductive number \mathcal{R}_0 from $b^2 - 4ac = 0$. This means, when $\mathcal{R}_0 < \mathcal{R}_c$, (2.1) has only the disease-free equilibrium. The global stability of the disease-free equilibrium point when $\mathcal{R}_0 < \mathcal{R}_c$ is established in Section (6).

The foregoing approach uses a quadratic equation to establish conditions for the existence of endemic equilibrium points. Unfortunately, these conditions on a, b, c and $b^2 - 4ac$ are given in terms of complicated expressions of model parameters, which makes it difficult to assess the important role that key parameters play in the bifurcation dynamics. Among these parameters, the disease-induce death rate, contact and disease transmission rates, and those describing mosquito and virus dynamics along with mosquito-to-human ratio are worth mentioning. To look into the effects of some of these parameters, we use the center manifold theorem to establish the existence of backward bifurcation of endemic equilibrium points at $\mathcal{R}_0 = 1$. This is addressed in the following section.

4. Backward bifurcation

In this section we use analytical and numerical techniques to assess the directions of bifurcations and related dynamics as key parameters change. Backward bifurcation is a phenomenon where, two endemic equilibria, one stable and another one unstable co-exist along with the disease-free equilibrium for $\mathcal{R}_0 < 1$. The existence of a backward bifurcation indicates that reduction of the epidemiology threshold, \mathcal{R}_0 below unity is simply not a sufficient condition for disease control. Specifically, if the phenomenon of backward bifurcation emerges in mosquito-borne diseases, a combined effort (such as, mosquito control and strong personal protection) should be carried out to reduce the epidemiology threshold below a critical value to insure disease elimination despite initial size. In Section (6) we establish that once the epidemiology threshold \mathcal{R}_0 is reduced below the critical value \mathcal{R}_c , under some conditions, the disease could be eliminated for any initial size.

We establish the existence of backward bifurcation at $\mathcal{R}_0 = 1$ using the center manifold theorem (see [5] and [9]). Solving for β^* from $\mathcal{R}_0 = 1$ and evaluating the Jacobian matrix for system (2.1) at the disease-free equilibrium $E_0 = (x_1^*, 0, 0, 0, y_1^*, 0, 0)$ we get the singular matrix

$$J(\beta^*) = \begin{pmatrix} \Lambda - \mu & \Lambda & \Lambda & \Lambda + \psi & 0 & 0 & -\beta\phi \\ 0 & -k_1 & 0 & 0 & 0 & 0 & \beta\phi \\ 0 & d & -k_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & r & k_2 & 0 & 0 & 0 \\ 0 & -\frac{\phi\theta_2y_1}{x_1} & -\frac{\phi\theta_1y_1}{x_1} & 0 & \delta_1 - \gamma & \delta_1 & \delta_1(1 - \zeta_2) \\ 0 & \frac{\phi\theta_2y_1}{x_1} & \frac{\phi\theta_1y_1}{x_1} & 0 & 0 & -k_4 & \delta_1\zeta_2 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon & -\gamma \end{pmatrix},$$

where for simplicity we use $k_1 = d + \mu$, $k_2 = \psi + \mu$, $k_3 = r + \alpha + \mu$ and $k_4 = \varepsilon + \gamma$. The right and left eigenvectors of $J(\beta^*)$ corresponding to the zero eigenvalue are $w = (w_1, w_2, w_3, ..., w_7)^{\tau}$ (τ is transpose) and $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$, respectively, where

$$w_{1} = \frac{-\beta\phi}{\lambda - \mu} \left(\frac{k_{1}k_{2}k_{3} - \Lambda k_{2}(k_{3} + d) - rd(\Lambda + \psi)}{k_{1}k_{2}k_{3}} \right) w_{7}, \ w_{2} = \frac{\beta\phi}{k_{1}} w_{7},$$

$$w_{3} = \frac{\beta\phi d}{k_{1}k_{3}} w_{7}, \ w_{4} = \frac{\beta\phi rd}{k_{1}k_{2}k_{3}} w_{7}, \ w_{5} = -\frac{\gamma(\gamma - \delta_{1}) + \varepsilon(\delta_{1} + \gamma)}{\varepsilon(\gamma - \delta_{1})} w_{7},$$

$$w_{6} = \frac{\gamma}{\varepsilon} w_{7}, \ w_{7} = w_{7} = 1,$$

and

$$v_{2} = \frac{(\gamma k_{4} - \varepsilon \delta_{1} \zeta_{2})}{\varepsilon \beta \phi} v_{6}, \ v_{3} = \frac{\phi \theta_{1} y_{1}}{k_{3} x_{1}} v_{6}, \ v_{7} = \frac{k_{4}}{\varepsilon} v_{6},$$
$$v_{1} = v_{4} = v_{5} = 0, v_{6} = v_{6} = 1.$$

Clearly, $w_1 < 0$ and $w_5 < 0$. According to Theorem 4.1 in [9], the local bifurcation at $\mathcal{R}_0 = 1$ (equivalently, at $\beta = \beta^*$) is possible if $a_1 > 0$ and $b_1 > 0$, where

$$a_1 = \sum_{i,i,k=1}^{7} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j}$$
 and $b_1 = \sum_{i,k=1}^{7} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \varphi}$.

Note that each f_k , k=1,...,7 represents the right side of the k^{th} equation in the system (2.1). It should also be noted that $z-E_0$, where $z=(x_1,x_2,x_3,x_4,y_1,y_2,y_3)$ yields the first zero vector in $f_k(0,0)$ at the disease-free equilibrium E_0 and also, $\varphi=\beta-\beta^*$ is zero at β^* which is the scalar zero (the second component in $f_k(0,0)$). To this end,

$$a_{1} = v_{2} \sum_{i,j=1}^{7} w_{i} w_{j} \frac{\partial^{2} f_{2}(0,0)}{\partial x_{i} x_{j}} + v_{3} \sum_{i,j=1}^{7} w_{i} w_{j} \frac{\partial^{2} f_{3}(0,0)}{\partial x_{i} x_{j}} + v_{6} \sum_{i,j=1}^{7} w_{i} w_{j} \frac{\partial^{2} f_{6}(0,0)}{\partial x_{i} x_{j}} + v_{7} \sum_{i,j=1}^{7} w_{i} w_{j} \frac{\partial^{2} f_{7}(0,0)}{\partial x_{i} x_{j}}.$$

This yields

$$a_1 = a_{11} - (a_{22} + a_{33}), (4.1)$$

where

$$a_{11} = -v_6 w_1 \frac{\phi y_1^*}{(x_1^*)^2} (w_2 \theta_2 + w_3 \theta_1) + v_6 w_5 \frac{\phi}{x_1} (w_2 \theta_2 + w_3 \theta_1)$$

$$a_{22} = v_2 w_7 \frac{\beta \phi}{x_1^*} (w_2 + w_3 + w_4)$$

$$a_{33} = v_6 \frac{\phi y_1^*}{(x_1^*)^2} [2(w_2^2 \theta_2 + w_3^2 \theta_1) + w_4 (w_2 \theta_2 + w_3 \theta_1) + w_2 w_3 (\theta_1 + \theta_2)]$$

$$(4.2)$$

and

$$b_1 = \sum_{j,k=1}^{7} v_k w_j \frac{\partial^2 f_k(0,0)}{\partial x_j \partial \varphi} = v_2 w_7 \phi.$$

Clearly, $b_1 > 0$, however, $a_1 > 0$ if and only if

$$a_{11} > a_{22} + a_{33}. (4.3)$$

Thus, the following result is established based on [9].

Theorem 4.1. The model given by system (2.1) exhibits a backward bifurcation at the critical point $\mathcal{R}_0 = 1$ when inequality (4.3) holds.

Rewriting a_1 , we get

$$a_{1} = v_{6} \frac{\phi y_{1}^{*}}{(x_{1}^{*})^{2}} [-w_{1}(w_{2}\theta_{2} + w_{3}\theta_{1}) - 2(w_{2}^{2}\theta_{2} + w_{3}^{2}\theta_{1}) - w_{4}(w_{2}\theta_{2} + w_{3}\theta_{1}) - w_{2}w_{3}(\theta_{1} + \theta_{2})] + v_{6} \frac{\phi}{x_{1}^{*}} w_{5}(w_{2}\theta_{2} + w_{3}\theta_{1}) - v_{2} \frac{\beta\phi}{x_{1}^{*}} (w_{2} + w_{3} + w_{4}).$$

$$(4.4)$$

The term with coefficient $v_6 \frac{\phi y_1^*}{(x_1^*)^2}$ in (4.4) reduces to

$$w_{2}^{2}v_{6}\frac{\phi y_{1}^{*}}{(x_{1}^{*})^{2}}\left[\frac{(k_{1}k_{2}k_{3}-\Lambda k_{2}(k_{3}+d)-rd(\Lambda+\psi))(k_{3}\theta_{2}+d\theta_{1})}{(\mu-\Lambda)k_{2}k_{3}^{2}} - \frac{rd}{k_{2}k_{3}^{2}}(k_{3}\theta_{2}+d\theta_{1}) - 2\frac{(k_{3}^{2}\theta_{2}+d^{2}\theta_{1})}{k_{3}^{2}} - \frac{d(\theta_{1}+\theta_{2})}{k_{3}}\right]$$

$$= w_{2}^{2}v_{6}\frac{\phi y_{1}^{*}}{(x_{1}^{*})^{2}}(\mathcal{O}_{1}-\mathcal{O}_{2}),$$

$$(4.5)$$

where

$$\mathcal{O}_1 = [d\alpha + (d+k_2)(\mu - \Lambda)](k_3\theta_2 + d\theta_1) \text{ and}
\mathcal{O}_2 = [2(k_3^2\theta_2 + d^2\theta_1) + k_3d(\theta_1 + \theta_2)](\mu - \Lambda).$$
(4.6)

The term in (4.5) is positive if and only if $\mathcal{O}_1 > \mathcal{O}_2$. Possible factors for this could be increased disease-induced death rate, α and host density (for example, increased Λ and reduced μ , while α is increased). Then,

$$a_{1} = w_{2}^{2} v_{6} \frac{\phi y_{1}^{*}}{(x_{1}^{*})^{2}} (\mathcal{O}_{1} - \mathcal{O}_{2}) - v_{6} \frac{\phi w_{2}}{x_{1}} (\frac{\gamma}{\varepsilon} + \frac{\gamma + \delta_{1}}{\gamma - \delta_{1}}) (\theta_{2} + \frac{d\theta_{1}}{k_{3}})$$

$$- w_{2} \left[\frac{\gamma(\gamma + \varepsilon) - \delta_{1} \zeta_{2} \varepsilon}{\varepsilon x_{1}} \right] (1 + \frac{d}{k_{3}} + \frac{rd}{k_{2} k_{3}})$$

$$= \frac{w_{2}^{2} \phi y_{1}^{*}}{(x_{1}^{*})^{2}} \left[v_{6} (\mathcal{O}_{1} - \mathcal{O}_{2}) - \frac{x_{1}^{*} k_{1}}{y_{1}^{*} \beta \phi} \left[v_{6} (\frac{\gamma}{\varepsilon} + \frac{\gamma + \delta_{1}}{\gamma - \delta_{1}}) (\theta_{2} + \frac{d\theta_{1}}{k_{3}}) + (\frac{\gamma(\gamma + \varepsilon) - \delta_{1} \zeta_{2} \varepsilon}{\phi \varepsilon}) (1 + \frac{d}{k_{3}} + \frac{rd}{k_{2} k_{3}}) \right] \right].$$

$$(4.7)$$

Increased mosquito density, specifically, high mosquito-to-human ratio along with increased effective contact rate, ϕ , could make $a_1 > 0$. Other contributers to this result include increased ζ_2 , ε and decreased γ . Essentially, all parameters focusing on mosquito dynamics are vital to make the threshold a_1 positive. Specifically, increased ζ_2 indicates higher intensity of vertical transmission of the disease from female mosquito to eggs. Furthermore, incubation rate, ε , of the disease causing pathogen in mosquitoes, among many factors, could be influenced by temperature. For instance, the extrinsic incubation period of a dengue virus strain in Aedes aegypti mosquito species reduces when the temperature increases (see [13, 41]). Additionally, to contain vertically transmitted mosquito-borne diseases, control efforts should focus on mosquito reduction which otherwise could be a challenge when combined with increased ζ_2 , ε and γ along with ϕ .

Numerical simulations based on dengue virus show the effect of increasing the extrinsic incubation rate from $\varepsilon = \frac{1}{12}$ to $\varepsilon = \frac{1}{8}$ on the bifurcation while the mosquito death rate is relatively low, $\gamma = 1/15$. This is depicted in Figure 1, where all other parameters are kept the same, except the extrinsic incubation rate.

However, if mosquito life span is reduced ($\gamma = 1/12$), the bifurcating curve shifts to the right even for $\varepsilon = 1/8$ which is a high incubation rate of the virus in mosquito (see Figure 2). The effects of a rising temperature and abundant precipitation on the dynamics of the mosquito and the virus population make significant contributions which could challenge disease control efforts.

Likewise, numerical simulations of the model (2.1) (data based on dengue virus) highlight the effects of some parameters on the direction of the bifurcation and disease prevalence. One of these parameters is the disease-induced death rate, α . A backward bifurcation where two stable equilibria co-exist (endemic

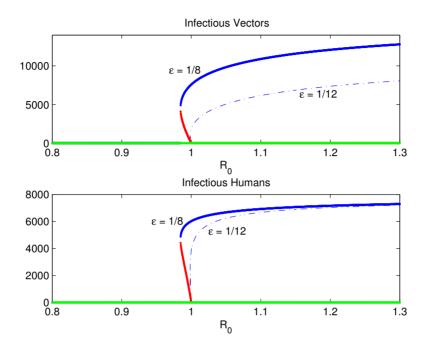


Figure 1: Bifurcation curves reflecting the effects of changes in extrinsic incubation rate and mosquito control: Unstable branch (red), stable branch (blue), green (extinction); $\varepsilon=1/8$ (continuous curve) and $\varepsilon=1/12$ (dashed curve). Other parameters are $\gamma=1/15$, $\beta=0.2, \zeta_2=0.67, \theta_1=0.0082, \theta_2=0.0289, \mu=1.01/(70\times365), \Lambda=0.379/(70\times365), \rho=205, \delta_0=1050, r=1/7, \delta_1=0.0399, d=1/10, <math>\psi=0.0014, \zeta_1=0, \alpha=0.0238, \phi=3$. The bifurcation is backward for high extrinsic incubation rate, ε but the direction changes to forward when ε is decreased.

and the disease-free) as shown in Figure 4 when the value of α changes from $\alpha = 0.0067$ to $\alpha = 0.0085$. This is in line with results given in the literature (see for example, in malaria [14] where the disease induced death rate changes from 3.419×10^{-5} to 3.454×10^{-4} for the direction of bifurcation to change), where backward bifurcation is connected to the severity of the disease, and in our model, this severity is measured by the values of α .

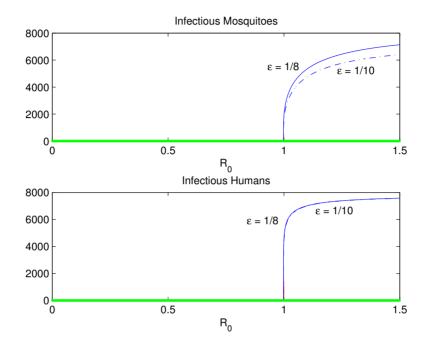
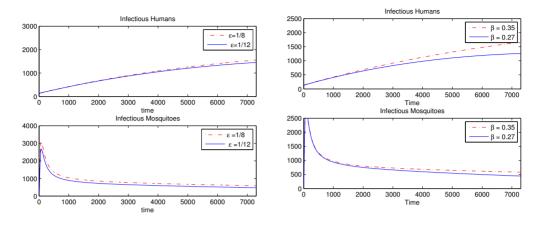


Figure 2: Bifurcation curves reflecting the importance of mosquito control to reduce the effects of extrinsic incubation rate : $\varepsilon=1/8$ (continuous curve) and $\varepsilon=1/10$ (dashed curve). Other parameters are $\gamma=1/12,~\beta=0.2,\zeta_2=0.67,\theta_1=0.0082,\theta_2=0.0289,$ $\mu=1.01/(70\times365),\Lambda=0.379/(70\times365),\rho=205,\delta_0=1050,r=1/7,\delta_1=0.0399,d=1/10,\psi=0.0014,\zeta_1=0,\alpha=0.0238,\phi=3.$ With increased mosquito death rate, the bifurcation remains forward for increased ε as well.

5. The effect of Weather Changes on Epidemic Levels

Increased temperature and precipitation are know to facilitate optimal conditions to enhance mosquito breading and survival rate which increase mosquito density. This is due to the fact that rise in temperature and enough precipitation increases breading ground, survival rate and shorten intrinsic incubation period. Moreover, a rise in temperature also increases mosquito biting rate and the rate by which the disease causing pathogen completes its cycle (extrinsic incubation rate). This together with vertical transmission of the disease in the



- (a) Effect of extrinsic incubation rate.
- (b) Effect of disease transmission from vector to humans.

Figure 3: Time plots of infectious population sizes as the extrinsic incubation rate increases (a) and as the disease transmission rate from mosquito to human increases (b). In each plot, $\gamma=1/17$ and all other parameters, except $\beta=0.2$ (in a) and $\varepsilon=1/10$ (in b), are as in Figure (2).

vector could facilitate conditions which contribute to emergence and reemergence of the disease as well as the rise in the epidemic level. It should be clear that other factors such as personal protections could contribute to disease control as well (see Figure 6), but once again our primary focus is on mosquito control reacting to conditions that increase mosquito density and virus replications in some mosquito species.

In this section, simulation results, are presented to illustrate the effects of weather conditions on the epidemic levels of the disease where parameters are estimated based on a dengue virus (see Table 2 in Appendix B). The simulation results show that higher extrinsic incubation rate in mosquitoes could increase the disease epidemic. Essentially, very high temperature raises the extrinsic incubation rate of the disease causing pathogen in some mosquito species. For example, a strain of dengue virus in Aedes aegypti accounts for high epidemic level which may slowly decay in time, or remain endemic. The elevated epidemic level due to increase in the extrinsic incubation rate could be reduced through effective mosquito control as it is given in Figure 5 for three different values $\varepsilon = 1/12, 1/10, 1/8$. Furthermore, as the simulation results in Figure 5

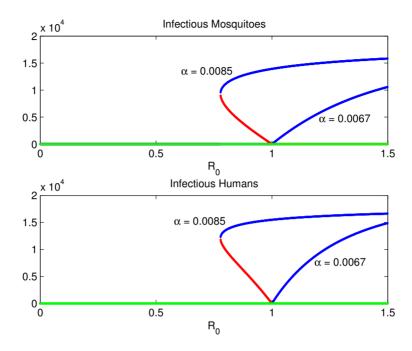


Figure 4: Bifurcation for two different values of α : $\alpha=0.0085$ and $\alpha=0.0067$. Unstable branch (red), stable branch (blue), green (extinction). Infectious vectors, y_3 and Infectious hosts, x_3 plotted against \mathcal{R}_0 , and other parameters are: $\theta_1=0.6383, \theta_2=0.0063, \mu=1/(70\times365), \Lambda=0.379/(70\times365), \rho=200, \gamma=1/17, \delta_0=1050, r=1/7, \delta_1=0.0399, \beta=0.7873, \phi=4, d=1/8, \varepsilon=1/10, \psi=0.0014, \zeta_2=0.67, \zeta_1=0.$

show, when mosquito survival period is reduced from 17 to 13 days (this means increased death rate from $\gamma = 1/17$ to $\gamma = 1/13$), even if the extrinsic incubation rate, ε , is increased, the epidemic could still be reduced. Indeed, a vector's survival through the extrinsic incubation period to become infectious makes a big difference in the epidemics level. Therefore, if there is any mechanism to reduce the survival period of mosquitoes, then the disease transmission cycle could be cut short, consequently, diminishing the epidemics. This could be among proactive intervention strategies following effective weather predictions.

Insecticide-based mosquito control products which focus on larva and young adults could contribute to insecticide resistance in mosquitoes. However, de-

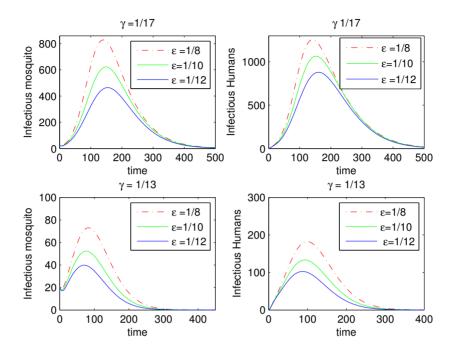


Figure 5: Reaction to change in ε and the effects of mosquito control. Population size of infectious mosquito for $\varepsilon=1/8,1/10,1/12$. Note that $\gamma=1/17$ in the upper two windows, but it is increased to $\gamma=1/13$ in the lower two windows. Other parameters are: $\theta_1=0.0083, \theta_2=0.00513, \mu=1/(70\times365), \Lambda=0.375/(70\times365), \rho=205, \delta_0=1050, \beta=0.2, r=1/7, \delta_1=0.0399, d=1/10, \psi=0.00233, \zeta_2=0.0087, \zeta_1=0, \alpha=0.0001, \phi=2$. Initial data: $x_0=[60;0;0;5;5000;10;20]$. The epidemic level increases for higher values of ε . Initially, there are no exposed and infectious hosts in the environment.

spite these side effects, when complex mosquito and virus dynamics challenge efforts to mitigate disease burden, insecticide application could be among preferred means to combat vertically transmitted vector-borne diseases (see [4] and references therein). This is especially the case when the problem covers a large area. More attention should be paid to mosquito control and this control should also react to weather conditions that could increase intrinsic and extrinsic incubation rates.

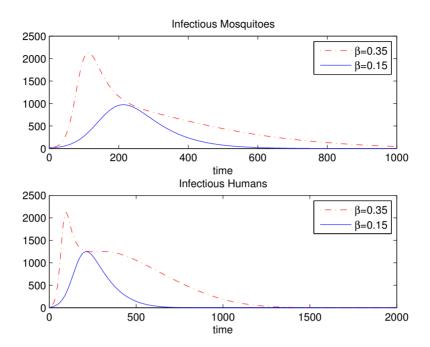


Figure 6: Reaction to change in disease transmission. Population size of infectious Mosquito (upper window) and Hosts (lower window). Parameters (other than β): $\theta_1 = 0.0083, \theta_2 = 0.00513, \mu = 1/(70 \times 365), \Lambda = 0.375/(70 \times 365), \rho = 205, \gamma = 1/17, \delta_0 = 1050, r = 1/7, \delta_1 = 1/20, d = 1/10, \varepsilon = 1/10, \psi = 0.00233, \zeta_2 = 0.0087, \zeta_1 = 0, \alpha = 0.0001, \phi = 2$. Initial data: $x_0 = [60; 0; 0; 5; 5000; 10; 20]$.

High rate of vertical transmission in mosquitoes could increase disease epidemics and its duration (the period that the epidemic lasts, see Figure 7). One reason for this is that it keeps the transmission loop within the mosquito population. This is sustained through mosquito-to-egg transmission which produces some infectious adults even if the weather is not favorable for mosquito density to increase. This could also cause the mosquito population to serve as a reservoir of the disease, which makes killing adult mosquitoes alone ineffective as disease control.

The fight against a vertically transmitted (in mosquitoes) vector-borne disease could undoubtedly face more challenges when the vertical transmission also takes place in the host population, it is worse if vector-host contact is not ef-

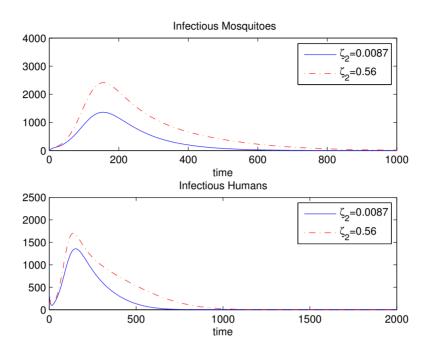


Figure 7: The total number of infectious host and vector reacting to increased vertical transmission in the vector. Note that $\zeta_2=0.56$ (dashed line) and $\zeta_2=0.0087$ (continuous line). The disease prevalence is increased reacting to increase in ζ_2 . Initial data: $x_0=[60;0;0;5;5000;10;20]$. Parameters (other than ζ_2): $\theta_1=0.0083, \theta_2=0.00513, \mu=1/(70\times365), \Lambda=0.375/(70\times365), \rho=205, \gamma=1/17, \delta_0=1050, r=1/7, \delta_1=1/20, \beta=0.15, d=1/10, \varepsilon=1/8, \psi=0.00233, \zeta_1=0, \alpha=0.0001, \phi=2.$

fectively controlled. As in the case of dengue fever, vertical transmission in the host population could also cause the epidemic in each population to increase. Simulation results on these effects are given in Figure 8 where $\phi=2$ in each case, and in Figure 9 where $\phi=3$ and $\zeta_1=0.56$; $\phi=2$ and $\zeta_2=0.0087$.

6. Global Stability of the Disease-Free Equilibrium

In this section we establish the global stability of the disease-free equilibrium when the epidemiology threshold is reduced below a critical value and for ζ_1 =

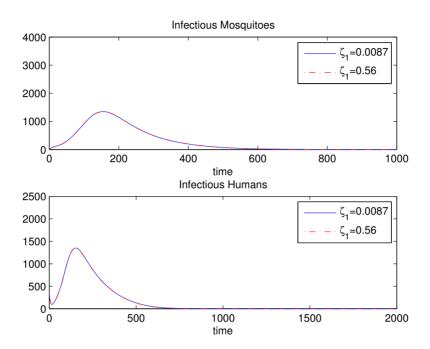


Figure 8: The total number of infectious host and vector reacting to increased vertical transmission in the host. Note that $\zeta_1=0.56$ (dashed line) and $\zeta_1=0.0087$ (continuous line). The reaction of the epidemic level to increase in ζ_1 when the contact rate is the same. Initial data: $x_0=[20;40;350;40;2000;40;10]$. Parameters (other than ζ_1): $\theta_1=0.0283, \theta_2=0.00513, \mu=1/(70\times365), \Lambda=0.375/(70\times365), \rho=205, \gamma=1/17, \delta_0=1050, r=1/7, \delta_1=1/20, \beta=0.15, d=1/10, \varepsilon=1/8, \psi=0.00233, \alpha=0.0001, \phi=2.$

 $\zeta_2 = 0$, which means, without vertical transmission in both populations. Clearly the set given by (2.4) is forward invariant and attractor. Thus, our focus is primarily to establish the global stability of the disease-free equilibrium in the forward invariant set Ω . In line with the previous sections, we will use \mathcal{R}_0 given by (3.5) where $\zeta_1 = 0 = \zeta_2$. When the bifurcation at $\mathcal{R}_0 = 1$ is forward, then from Theorem (3.2), the system (2.1) has no endemic equilibrium for $\mathcal{R}_0 < 1$. In the presence of a backward bifurcation, the critical value of \mathcal{R}_0 , denoted by \mathcal{R}_c and $0 < \mathcal{R}_c < 1$ is associated with the turning point of the bifurcating curve. Specifically, when $\zeta_2 = \zeta_1 = 0$, using (3.13) and $b^2 - 4ac = 0$, it is possible to

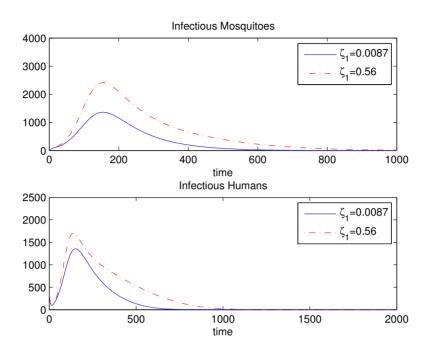


Figure 9: The total number of infectious host and vector reacting to increased vertical transmission in the host when the contact rate is also increased. Note that $\zeta_1 = 0.56, \phi = 3$ (dashed line) and $\zeta_1 = 0.0087, \phi = 2$ (continuous line). Initial data: $x_0 = [20; 40; 350; 40; 2000; 40; 10]$. Parameters (other than ζ_1 and ϕ): $\theta_1 = 0.0283, \theta_2 = 0.00513, \mu = 1/(70 \times 365), \Lambda = 0.375/(70 \times 365), \rho = 205, \gamma = 1/17, \delta_0 = 1050, r = 1/7, \delta_1 = 1/20, \beta = 0.15, d = 1/10, \varepsilon = 1/8, \psi = 0.00233, \alpha = 0.0001.$

see that

$$\mathcal{R}_c = \sqrt{1 - \frac{b^2}{4a(k_1 k_2 k_3)^2 \rho \gamma k_4 (\gamma - \delta_1)}}.$$
 (6.1)

For $\mathcal{R}_0 < \mathcal{R}_c$, the model given by (2.1) has no endemic equilibrium. Here are some examples of critical values from simulations of the system (2.1): $\mathcal{R}_c = 0.691$ for $\varepsilon = 1/8, \gamma = 1/17$, and then it shifts to $\mathcal{R}_c = 0.897$ for $\varepsilon = 1/12, \gamma = 1/15$ where in both cases we keep $\beta = 0.35$, $\phi = 5$. Then it shifts further to the right $\mathcal{R}_c = 0.9857$ when β and ϕ are reduced to $\beta = 0.2$ and $\phi = 3$ while $\varepsilon = 1/8, \gamma = 1/15$. In each of these examples, the values of the other parameters

are as in Figure 1.

Theorem 6.1. The Disease-free equilibrium E_0 which is given by (3.1) is globally asymptotically stable in Ω , the set defined by (2.4) when $\mathcal{R}_0 < \min\{\mathcal{R}_c, \sqrt{\Delta}\}$, where \mathcal{R}_c is given by (6.1) and $\Delta = \frac{(\mu - \Lambda)}{\rho}$.

Proof. Consider a Lyapunov function defined by

$$V = A_1 x_2 + A_2 x_3 + A_3 y_2 + A_4 y_3, (6.2)$$

where $A_1 = 1$, $A_2 = \frac{A_3\phi\theta_1\delta_0}{k_3(\gamma-\delta_1)}$, $A_3 = \frac{A_4\varepsilon}{k_4}$, $A_4 = \frac{\beta\phi}{\gamma}$ and $x_i, i = 2, 3$ and $y_i, i = 2, 3$ are components of a solution of (2.1) with initial value in Ω , the set given by (2.4).

Differentiating V with respect to t and using the right side of equation (2.1) we get

$$\dot{V} = A_{1}\dot{x}_{2} + A_{2}\dot{x}_{3} + A_{3}\dot{y}_{2} + A_{4}\dot{y}_{3}
= x_{2}(A_{2}d - A_{1}k_{1} + A_{3}\phi\theta_{2}\frac{y_{1}}{N}) + x_{3}(A_{3}\phi\theta_{1}\frac{y_{1}}{N} - A_{2}k_{3})
+ y_{2}(A_{4}\varepsilon - A_{3}k_{4}) + y_{3}(A_{1}\phi\beta\frac{x_{1}}{N} - A_{4}\gamma)
\leq x_{2}(A_{2}d - A_{1}k_{1} + A_{3}\phi\theta_{2}\frac{\delta_{0}}{\gamma - \delta_{1}}) + x_{3}(A_{3}\phi\theta_{1}\frac{\delta_{0}}{\gamma - \delta_{1}} - A_{2}k_{3})
+ y_{2}(A_{4}\varepsilon - A_{3}k_{4}) + y_{3}(A_{1}\phi\beta - A_{4}\gamma)
= x_{2}k_{1}[\mathcal{R}_{h}^{2}\frac{\rho}{\mu - \Lambda} - 1]
= x_{2}k_{1}\frac{\rho}{\mu - \Lambda}[\mathcal{R}_{h}^{2} - \Delta].$$
(6.3)

Then $\mathcal{R}_h^2 < \Delta$ implies $\dot{V} < 0$. Clearly, the coefficients of x_2, x_3, y_2 and y_3 are non positive which implies that $\dot{V}=0$ if and only if $x_i=0, i=2,3,4$ and $y_i=0, i=2,3$. Thus, $\dot{V}=0$ if and only if $\dot{x}_i=0, i=2,3,4$ and $\dot{y}_i=0, i=2,3$. Furthermore, if $\mathcal{R}_0 < \mathcal{R}_c$, the disease-free equilibrium $E_0=(x_1^*,0,0,0,y_1^*,0,0)$ is the only equilibrium of the vector field (2.1) in Ω . It then follows that the set $\{(x_1,x_2,x_3,x_4,y_1,y_2,y_3)\in\Omega:\dot{V}=0\}$ has $\mathcal{G}=\{(x_1^*,0,0,0,y_1^*,0,0)\}$ as a maximal invariant subset. Therefore, by Lyapunov-LaSalle theorem (Theorem 6.2 in [40]), for all solutions that start in Ω , the components (x_2,x_3,x_4,y_2,y_3) asymptotically approach (0,0,0,0,0). This clearly implies that x_1 and y_1 asymptotically approach $\frac{\rho}{\mu-\Lambda}$ and $\frac{\delta_0}{\gamma-\delta_1}$, respectively. Therefore, E_0 is globally asymptotically stable in Ω .

7. Discussion and Conclusions

We wrap up our study by making some concluding remarks about our findings. Analytical and numerical techniques are implemented to assess the influence of climate changes on the dynamics of vertically transmitted diseases. Specifically, the numerical results highlight changes in virus (due to increased incubation rate) and mosquito dynamics have effects on disease epidemic and could also cause the disease to be endemic. While mosquito and virus dynamics are not the only parameters reacting to climate changes, our numerical results show that the combination of these two parameters along with lack of personal protection and effective mosquito control could elevate the epidemic level which could also last over a longer period of time.

Part of our analytical results include derivation of the epidemiology threshold, \mathcal{R}_0 , the existence of a backward bifurcation and its connections to the disease-induced death rate, disease transmission and mosquito-to-human ratio. Other analytical results include the global stability of the disease-free equilibrium point when the epidemiology threshold is reduced below a critical value. Consequently, in the presence of a backward bifurcation, the reduction of the epidemiology threshold below one is not enough to eradicate the disease. This threshold should be reduced below a critical value, and the horizontal transmission threshold should also be kept below a critical value to accomplish the goal of eradication. This follows from the global stability of the disease-free equilibrium point, Theorem (6.1). Therefore, one of the most important measures to contain a vector-borne disease is to focus on parameters that reduce horizontal transmissions.

Numerical simulation results show the effect of increased extrinsic incubation rate on the bifurcation curves for different values of mosquito death rates. As it is depicted in Figure 1, when the mosquito death rate is $\gamma=1/15$, a backward bifurcation curve emerges for $\varepsilon=1/8$, and shifts to the right when the value of ε is reduced to 1/12. Furthermore, the equilibrium levels on the bifurcation curve corresponding to $\varepsilon=1/12$ are smaller (see for example, the time plot of infectious vector and host populations for $\varepsilon=1/8$ and 1/12 in Figure 3(a)). The endemic equilibrium level is also elevated due to changes in β from $\beta=0.27$ to $\beta=0.35$ in Figure 3(b). However, when mosquito death rate is increased to $\gamma=1/10$, even for $\varepsilon=1/8$, the backward bifurcation disappears (see Figure 2). The implication of these results is that reducing the life expectancy of mosquitoes could make a big difference in controlling disease burden. The disease-induced death rate of hosts is also another parameter which could influence the direction of bifurcation as it is depicted in Figure 4.

In part, due to transmission cycle and the short period it takes for the disease causing pathogen to complete its life cycle (when temperature increases) in female mosquitoes, increased extrinsic incubation period raises the disease prevalence in both populations. The combinations of these factors could enhance the challenges to fight mosquito-borne diseases such as dengue, RVF and WNV to mention some. On the other hand, if mosquito control (such as increasing γ) is in phase with weather conditions that raise the extrinsic and intrinsic incubation rates, then the epidemic level could be significantly reduced (compare the upper two windows, $\gamma = 1/17$ with the lower two windows, $\gamma = 1/13$ in Figure 5). Additionally, we see that the prevalence of vector-borne diseases reacts to a number of parameters, such as disease transmission rate, β , as it is given in Figure 6, where transmission control reduces the epidemic level and its duration. Furthermore, as it can be seen in Figure 7 the rate of vertical transmission in the vector population has greater influence in raising the epidemic level. However, the results in Figures 8 and 9 highlight that increased contact rate magnifies the impact of vertical transmission on the epidemic level and its durations. Although the data we used is based on a dengue virus, our results reveal that screening hosts and taking necessary control measures could prevent the spread of vertically transmitted diseases to more geographical locations.

When vertical transmission is present in mosquitoes, controlling adult mosquitoes alone may not reduce disease burden, since infected eggs hatch to be infectious adults. Therefore, following predictions of favorable weather changes, a proactive mosquito control measure should include larvacide. Our results provide some useful insights into appropriate measures that should be taken to mitigate the effects of the extrinsic incubation periods in vertically transmitted vector-borne diseases. Furthermore, a rise in extrinsic incubation rate elevates the epidemic level of the disease. Specifically, the results highlight the importance of accurate climate predictions which cause the extrinsic incubation rate of the disease causing pathogen in mosquitoes to increase. While mosquito control and personal protection are among the most effective measures to fight against vector-borne diseases, understanding the virus dynamics could also play a key role in this fight. Clearly, simulation results depend on the choice of parameters, thus more results and conclusions are possible. It is our hope that the analytical and numerical results of this study lay a groundwork to further assess the complex dynamics of vertically transmitted vector-borne diseases and the effects of extreme weather conditions.

8. Appendix A

We used the constants A, B, S, E which are given by (3.11) and (3.14) in Section 3.1.

The components of the endemic equilibrium $E_1 = (x_1, x_2, x_3, x_4, y_1, y_2, y_3)$ are given by (3.8)-(3.9) from which we get

$$N = x_1 + x_2 + x_3 + x_4 = \frac{k_1 x_2}{\pi_1} + x_2 + \frac{dx_2}{k_3} + \frac{dr x_2}{k_2 k_3} = (\frac{k_1}{\pi_1} + 1 + \frac{d}{k_3} + \frac{dr}{k_2 k_3})x_2$$
 (8.1)

$$=\frac{\rho(k_1k_2k_3+\pi_1D)}{\pi_1A+B}. (8.2)$$

From (3.8), (3.9) and (3.10) we get

$$\pi_1 = \frac{\phi \beta y_3}{N} = \frac{\phi \beta (B + A\pi_1)}{\rho(k_1 k_2 k_3 + \pi_1 D)} \frac{k \delta_0 \pi_2}{(\gamma - \delta_1)(k_4 \pi_2 + \mathcal{M})} \quad \text{and}$$
(8.3)

$$\pi_2 = \frac{E\pi_1}{k_1k_2k_3 + D\pi_1}. (8.4)$$

Using (8.4), we have $\frac{\pi_2}{k_4\pi_2 + \mathcal{M}(\zeta)} = \frac{E\pi_1}{k_1k_2k_3\mathcal{M} + \pi_1(Ek_4 + D\mathcal{M})}$. Thus, (8.3) yields

$$\pi_1 = \frac{\phi \beta (B + A\pi_1)}{\rho (k_1 k_2 k_3 + D\pi_1)} \frac{\delta_0 \varepsilon E \pi_1}{(\gamma - \delta_1) (k_1 k_2 k_3 \mathcal{M} + \pi_1 (E k_4 + D\mathcal{M}))}$$

which reduces to a quadratic equation in π_1

$$\pi_1^2 a + \pi_1 b + c = 0, (8.5)$$

with coefficients given by (3.13).

Par.	Range	Ref.	Par.	Range
γ	[1/17, 1/10]	[11]	θ_2	$(0,\theta_1)$
μ	$\left[\frac{1}{70(356)}, \frac{1}{45(356)}\right]$	estimate	θ_1	[0, 1)
ϕ	≥ 1	variable	α	(0, 0.001)
ε	[1/14, 1/7]	[13]	ψ	[0,1)
r	[0, 1/7]	[1]	β	(0,1)
ζ_2	[0,1)	estimate	ζ_1	[0,1)
d	[1/14, 1/3]	[10]	δ_1	[0,1)
δ_0	[700, 10, 000]	[5]		
Λ	$(0, \mu)$			

9. Appendix B

Table 2: Parameter estimation for dengue fever. All variables other than γ, ε, r and d are estimated in the given range of values. The values of parameters listed in column 4 are estimates. Rate is per day.

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