The effects of rural/urban movement on Dengue transmission dynamics

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Abstract

Five serotypes of Dengue (DENV1-DENV5), a vector-borne disease transmitted by two species of mosquitoes, *Aedes aegypti* and *Aedes albopictus*, are prevalent in various tropical and subtropical regions of the world, posing a serious health threat to humans. Dengue is no longer restricted to tropical regions due to increasing levels of mobility via travel, migration, or displacement due to conflict. We use a system of nonlinear ordinary differential equations to explore the effects of rural/urban movement on Dengue transmission dynamics. The model incorporates movement between rural and urban regions. The population of hosts is subdivided into susceptible, exposed, infectious, and recovered classes. Vectors, assumed to remain in a single region, are divided into rural (*Ae. albopictus*) and urban (*Ae. aegypti*) populations. The vector populations are subdivided into susceptible, exposed, and infectious classes. We compute the basic reproductive number (R_0) for the system with and without movement and use this key dimensionless parameter to study the effects of rural/urban host movement on Dengue dynamics.

1 Introduction

Dengue fever is a mosquito-borne viral infection that is endemic in tropical regions worldwide and may be periodically epidemic in subtropical or temperate regions [11]. It occurs in over 100 countries, putting over 2.5 billion people at risk of infection, though it is most common in Central America and Southeast Asia [3, 5]. Classical Dengue Fever (CDF), the most common form of the disease, usually occurs in people over 15 years of age [13]. Its symptoms can include fever, headache, nausea, and vomiting with a duration of 3-7 days [13]. CDF is usually mild or even asymptomatic [24].

The more severe case, Dengue Hemorrhagic Fever (DHF), usually occurs in children under 15 years of age (though it may be observed in adults). DHF is associated with Dengue Shock Syndrome (DSS), and can lead to circulatory failure and death [13,24]. The factors that cause Dengue patients to contract DHF and DSS are not fully understood; however, current hypotheses suggest that this more severe form occurs in patients who have previously recovered from a different serotype of Dengue [11]. DHF and DSS tend to affect two categories of Dengue patients: those who are experiencing a secondary infection and infants whose mothers were previously infected by Dengue [13].

It is estimated that 50-100 million people contract Dengue yearly, while 250,000-500,000 people suffer from DHF [29]. There are approximately 22,000 deaths annually, mostly among young children [3].

Dengue is caused by five antigenically distinguishable serotypes (DENV1 - DENV5) of the genus *Flavivirus* [3,16,24]. Once an individual has been infected by one serotype, they are permanently immune to that serotype but only temporarily immune to the others [13]. The fifth serotype was very recently discovered, and is the only new serotype found in the last fifty years. Its discovery has made efforts to control and vaccinate for Dengue even more difficult [22].

There is not yet any specific treatment or effective vaccine for Dengue, though in some cases, fluid replacement therapy may be used [3, 16]. Several candidates for a Dengue vaccine have been clinically tested, but no vaccine has yet been applied on a broad scale [9, 29]. It is speculated that no vaccine could completely protect against all serotypes of the disease [16]. Currently, the main focus is on prevention by controlling mosquito populations and breeding sites [3].

DENV is transmitted by two species of mosquitoes: *Aedes aegypti* and *Aedes albopictus*. *Ae. aegypti* is the principal vector of Dengue viruses and has adapted to live closely with humans. This has made *Ae. aegypti* one of the most efficient mosquitoes for transmitting arboviruses, as they feed primarily on humans for blood meals, mostly during the day or in shaded areas at night [23]. These mosquitoes are highly resilient in both the adult and juvenile stages; their eggs can survive without water for up to six months [15]. They are common in urban areas, where they live close to houses and use water-holding containers, preferably in dark colors, for their reproduction. Their bite is often painless and they move quickly, though they do not fly very far [32].

Ae. albopictus, also called the Asian Tiger mosquito, is even more dangerous to humans

and domesticated animals. In addition to feeding on humans for blood meals, it will also feed on dogs, cats, squirrels, and deer. They are very aggressive all day long and have a rapid bite [32]. They prefer to use natural locations, such as plants, to lay their eggs [30]. They transmit not only the Dengue virus, but other vector-borne diseases such as West Nile, Eastern equine encephalitis, and Japanese encephalitis viruses as well [14,30].

Demographics, social change, infrastructure, and other factors related to urbanization contribute to the spread of dengue to new populations of mosquitoes [27]. Dengue emerges in tropical and subtropical areas as they are urbanized [31]. The extreme population growth that accompanies urbanization causes overcrowding, poor sanitation, and an increased need for water storage. This environment encourages the breeding of *Ae. aegypti* (which thrive in urban areas). Thus, the potential of DENV to spread from city to city via human displacement is increased [12,31]. These factors have contributed to the rapid evolution of DENV, causing a serious public health problem [19].

2 Mathematical model

We explore the dynamics of Dengue fever among humans and female mosquitoes of the species *Ae. aegypti* and *Ae. albopictus*. The model is divided into rural and urban populations, between which only host movement can occur. These populations are further subdivided into susceptible, exposed, infectious, and recovered classes. The system is compartmentally symmetric, so the principles and patterns for the rural population also apply to the urban population, and vice versa. However, there are differences between some parameter values due to the different species of vectors, which, although they transmit the same disease, have different capabilities.

Table 1: State variables

State Variable	**Description**
S_{HR}	Population of susceptible hosts in a rural area
E_{HR}	Population of exposed hosts in a rural area
I_{HR}	Population of infectious hosts in a rural area
R_{HR}	Population of recovered hosts in a rural area
S_{HU}	Population of susceptible hosts in an urban area
E_{HU}	Population of exposed hosts in an urban area
I_{HU}	Population of infectious hosts in an urban area
R_{HU}	Population of recovered hosts in an urban area
S_{VR}	Population of susceptible vectors in a rural area
E_{VR}	Population of exposed vectors in a rural area
I_{VR}	Population of infectious vectors in a rural area
S_{VU}	Population of susceptible vectors in an urban area
E_{VU}	Population of exposed vectors in an urban area
I_{VU}	Population of infectious vectors in an urban area

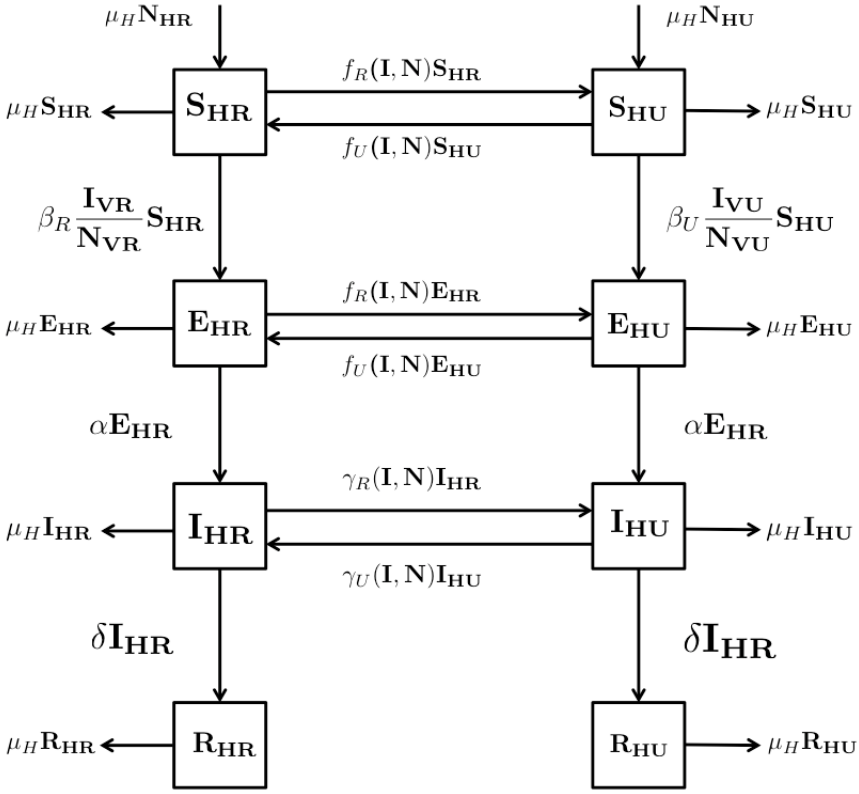

Figure 1: Transmission dynamics for rural (R) and urban (U) host populations.

The total host (H) population, N_H, is constant, while the host subpopulations, N_{HR} and N_{HU}, are not constant when movement occurs. The vector (V) subpopulations, N_{VR} and N_{VU}, are constant. We assume that vectors do not move between populations, due to their limited flight range.

The susceptible classes, S_{HR} and S_{HU}, are increased by a constant birth rate $\mu_H N_H$ and a factor of movement $f_R(\boldsymbol{I}, \boldsymbol{N})S_{HR}$ or $f_U(\boldsymbol{I}, \boldsymbol{N})S_{HU}$. This class is decreased by individuals who die of natural causes or move away, and individuals who become exposed at the constant rates β_R and β_U, which describe the transmission rates of DENV from the proportion of infectious vectors ($\frac{I_{VR}}{N_{VR}}$ and $\frac{I_{VU}}{N_{VU}}$) to susceptible hosts.

Once a susceptible host is bitten by an infectious vector, they may become exposed to Dengue. The rates of transmission of Dengue from infectious vectors to susceptible hosts differ between rural and urban areas because the *Ae. albopictus*, which is found in rural areas, and *Ae. aegypti*, which is found in urban areas, have different biological properties that affect the overall rate of Dengue transmission. The exposed classes, E_{HR} and E_{HU},

consist of individuals who are in the latency period as a result of DENV infection. Individuals in these classes cannot infect susceptible vectors. Research suggests that latency periods last between three to five days [21]. While in the exposed classes, individuals are asymptomatic and are not aware that they have the virus, so they are assumed to move as if they were uninfected. The exiting rate α is defined as the rate at which individuals complete the incubation period, become infectious, and begin to experience symptoms.

Individuals in the infectious classes, I_{HR} and I_{HU}, can transmit the disease to susceptible vectors. Once an individual progresses to the infectious class, their rate of movement may be affected by the symptoms they experience, so the movement rates, $\gamma_R(\boldsymbol{I}, \boldsymbol{N})$ and $\gamma_U(\boldsymbol{I}, \boldsymbol{N})$, is proportional to the movement rates of the uninfectious classes. We assume that some proportion $p \in [0, 1]$, of the infectious individuals remain mobile enough to move normally, while $(1 - p)$ are too ill to move as they ordinarily would. The infectious period of Dengue fever lasts an average of six days before individuals enter the recovered classes, R_{HR} and R_{HU}, at the recovery rate δ [10].

The recovered classes consist of individuals who are permanently immune to the particular serotype of Dengue. Since they can neither infect nor be infected by vectors, the rural/urban movement between these classes is omitted from the model for the sake of simplicity.

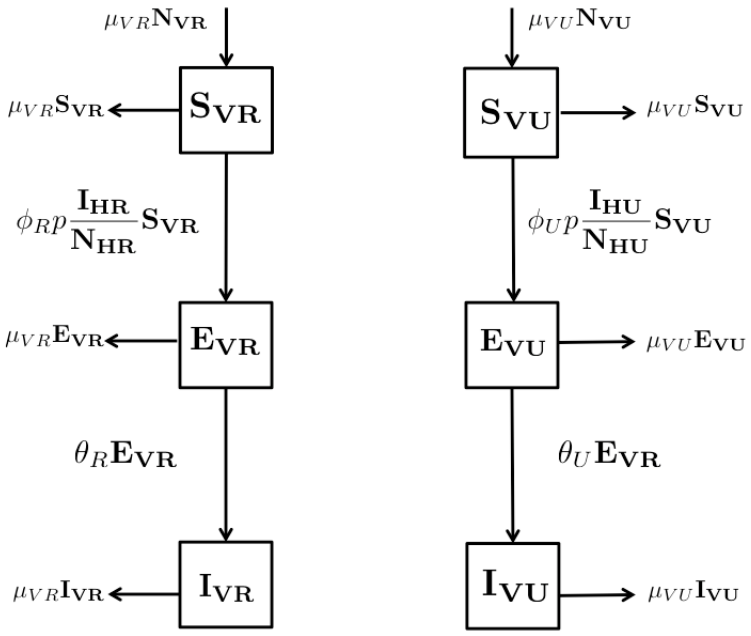

Figure 2: Transmission dynamics for rural and urban vector populations.

The vector's subpopulations are compartmentally symmetric, as patterns and ideas hold true for both the urban and rural species, though the experimentally determined values of these parameters differ between the species. μ_{VR} and μ_{VU} are the rates at which

vectors mature into adults, become susceptible to DENV, and enter the classes S_{VR} and S_{VU}, respectively. The rates of entry into the exposed classes, E_{VR} and E_{VU}, are ϕ_R and ϕ_U, respectively. These are the transmission rates as a susceptible vector bites an infectious host, so this rate also depends on the proportion of infectious hosts ($\frac{I_{HR}}{N_{HR}}$ and $\frac{I_{HU}}{N_{HU}}$). We assume that only the proportion p of infectious hosts who are mobile are available to be bitten by the vectors. That is, the proportion $(1-p)$ of infectious hosts who are too ill to be mobile will be isolated, bedridden, or hospitalized, and vectors will be unable to bite them. After the blood meal from an infectious host is consumed, the vector enters a latency stage, in which they are exposed to DENV but cannot transmit the disease. The exposed classes are decreased by natural death and progression (at rates θ_R and θ_U) to the infectious classes, I_{VR} and I_{VU}. Extrinsic incubation lasts ten days, on average, before the vector becomes infectious. Since the vector's lifespan is approximately two to three weeks, it is important to consider the exposed class because some mosquitoes will die before they can progress to the infectious class. Due to their relatively short lifespan, vectors remain infectious for life and never recover.

The system of nonlinear ordinary differential equations for our model is given by

$$
\begin{aligned}
S'_{HR} &= \mu_H N_{HR} - \beta_R \frac{I_{VR}}{N_{VR}} S_{HR} - f_R(\boldsymbol{I},\boldsymbol{N}) S_{HR} + f_U(\boldsymbol{I},\boldsymbol{N}) S_{HU} - \mu_H S_{HR},\\
E'_{HR} &= \beta_R \frac{I_{VR}}{N_{VR}} S_{HR} - \alpha E_{HR} - f_R(\boldsymbol{I},\boldsymbol{N}) E_{HR} + f_U(\boldsymbol{I},\boldsymbol{N}) E_{HU} - \mu_H E_{HR},\\
I'_{HR} &= \alpha E_{HR} - \delta I_{HR} - \gamma_R(\boldsymbol{I},\boldsymbol{N}) I_{HR} + \gamma_U(\boldsymbol{I},\boldsymbol{N}) I_{HU} - \mu_H I_{HR},\\
R'_{HR} &= \delta I_{HR} - \mu_H R_{HR},\\
S'_{HU} &= \mu_H N_{HU} - \beta_U \frac{I_{VU}}{N_{VU}} S_{HU} + f_R(\boldsymbol{I},\boldsymbol{N}) S_{HR} - f_U(\boldsymbol{I},\boldsymbol{N}) S_{HU} - \mu_H S_{HU},\\
E'_{HU} &= \beta_U \frac{I_{VU}}{N_{VU}} S_{HU} - \alpha E_{HU} + f_R(\boldsymbol{I},\boldsymbol{N}) E_{HR} - f_U(\boldsymbol{I},\boldsymbol{N}) E_{HU} - \mu_H E_{HU},\\
I'_{HU} &= \alpha E_{HU} - \delta I_{HU} + \gamma_R(\boldsymbol{I},\boldsymbol{N}) I_{HR} - \gamma_U(\boldsymbol{I},\boldsymbol{N}) I_{HU} - \mu_H I_{HU},\\
R'_{HU} &= \delta I_{HU} - \mu_H R_{HU},
\end{aligned}
\tag{1}
$$

$$
\begin{aligned}
S'_{VR} &= \mu_{VR}N_{VR} - \phi_R p\frac{I_{HR}}{N_{HR}}S_{VR} - \mu_{VR}S_{VR}, \qquad (2)\\
E'_{VR} &= \phi_R p\frac{I_{HR}}{N_{HR}}S_{VR} - \theta_R E_{VR} - \mu_{VR}E_{VR},\\
I'_{VR} &= \theta_R E_{VR} - \mu_{VR}I_{VR},\\
S'_{VU} &= \mu_{VU}N_{VU} - \phi_U p\frac{I_{HU}}{N_{HU}}S_{VU} - \mu_{VU}S_{VU},\\
E'_{VU} &= \phi_U p\frac{I_{HU}}{N_{HU}}S_{VU} - \theta_U E_{VU} - \mu_{VU}E_{VU},\\
I'_{VU} &= \theta_U E_{VU} - \mu_{VU}I_{VU},
\end{aligned}
$$

where

$$
\begin{aligned}
N_{HR} &= S_{HR} + E_{HR} + I_{HR} + R_{HR}, \qquad (3)\\
N_{HU} &= S_{HU} + E_{HU} + I_{HU} + R_{HU},\\
N_{VR} &= S_{VR} + E_{VR} + I_{VR},\\
N_{VU} &= S_{VU} + E_{VU} + I_{VU}.
\end{aligned}
$$

With this model, our main goal is to analyze the effect of human movement on the transmission dynamics of Dengue. For the sake of comparison, we first analyze the system for a case in which there is no movement between the rural and urban areas.

In this case, we analyze the complete system as two independent systems where the rural population is affected by *Ae. albopictus* and the urban population is affected by *Ae. aegypti*. These particular vectors are selected for these areas because they are observed to prefer those respective habitats. Since these two models are compartmentally symmetric, we first analyze the zero-movement model as consisting of only one host population and one vector population, each with a constant population.

In the second case we consider constant movement. In this model, we consider different scenarios for the rural and urban populations due to the fact that the species of vectors that are native to each area have different qualities. We assume that the urban and rural host populations are not constant (due to movement between them), but that the total host population is constant. We define movement rates as a constant that designates the base rate of movement. In this case, the movement rates between the susceptible classes are equal to the movement rates between the exposed classes, since an exposed individual is asymptomiatic and will not show a change in behavior. However, the movement rates between the infectious classes are a reduced rate. We assume that some proportion $(1-p)$ of the infectious population is too ill to move normally, and so they are removed from the movement rates. We assume that the same constant p applies in both the rural and urban populations, as the severity of illness will not change with respect to location.

The final case introduces non-constant functions to describe the movement rates. These functions are defined as $f_R(\boldsymbol{I}, \boldsymbol{N})$ and $f_U(\boldsymbol{I}, \boldsymbol{N})$, where f_R is the rate of rural to urban movement, f_U is the rate of urban to rural movement, $\boldsymbol{I} = (I_{HR}, I_{HU})$, and $\boldsymbol{N} =$

(N_{HR}, N_{HU}). We consider the change in movement rate to depend on the relative proportions of infectious populations. We assume that when there are no infectious hosts, the movement rates are equal to the constant movement rates C_R and C_U, while the presence of infectious individuals will modify these base rates. We assume that when there is an increased proportion of infectious hosts in the rural area, movement towards the rural area is less likely and movement away from the rural area is more likely; the equivalent is true for an increased proportion of infectious hosts in the urban area. We analyze functions that modify the base movement rates and describe the rates in terms of the proportions of infectious hosts to the total population in each area. We use this proportion, rather than the raw number of infectious individuals, because the rural and urban populations are likely to be very different in total size.

3 Descriptions and Values for Variables and Parameters

The following parameter values are based on experimental data from previous research where referenced.

Table 2: Parameters

Parameter	**Description**	**Units**
μ_H	Host natural mortality rate	Time^{-1}
μ_{VR}	Vector mortality rate in rural areas	Time^{-1}
μ_{VU}	Vector mortality rate in urban areas	Time^{-1}
β_R	Transmission rate for rural hosts	Time^{-1}
β_U	Transmission rate for urban hosts	Time^{-1}
ϕ_R	Transmission rate for rural vectors	Time^{-1}
ϕ_U	Transmission rate for urban vectors	Time^{-1}
α	Rate of advancement from exposed to infectious hosts	Time^{-1}
θ_R	Rate of advancement from rural exposed to infectious vectors	Time^{-1}
θ_U	Rate of advancement from urban exposed to infectious vectors	Time^{-1}
δ	Host recovery rate	Time^{-1}
C_R	Rate of rural $\rightarrow$ urban movement in disease-free case	Time^{-1}
C_U	Rate of urban $\rightarrow$ rural movement in disease-free case	Time^{-1}
p	Proportion of infectious hosts that are mobile	Dimensionless

Table 3: Parameter values

Parameter	Mean	Minimum	Maximum	Reference
μ_H	$^1/_{75*365}$ days			[7]
μ_{VR}	1/21	1/42	1/14	[2,6,20]
μ_{VU}	1/14	1/42	1/8	[8,18,26]
α	1/5	1/7	1/4	[17,25]
θ_R	1/10	1/14	$^1/_7$	[2]
θ_U	1/10	1/14	1/7	[4,17]
δ	1/6	1/12	1/4	[1,10]
p	0.9	0	1	
C_R	0.42	0	1	
C_U	0.24	0	1	

4 Mathematical Analysis

4.1 Zero Movement

4.1.1 System Equations

In the first and most simple case, we examine the system without movement, that is, where

$$f_R(\boldsymbol{I}, \boldsymbol{N}) = f_U(\boldsymbol{I}, \boldsymbol{N}) = 0.$$

No movement between rural and urban populations implies the existence of two independent compartmentally symmetric systems. This allows us to analyze a model for the transmission of Dengue in a single area and with a particular kind of mosquito.

We denote by N_H and N_V the total host and vector population sizes, respectively, with $N_H = S_H + E_H + I_H + R_H$ and $N_V = S_V + E_V + I_V$. We assume constant sizes for N_H

and N_V. The model for the single-patch model is given by

$$\begin{aligned} S'_H &= \mu_H N_H - \beta \frac{I_V}{N_V} S_H - \mu_H S_H \\ E'_H &= \beta \frac{I_V}{N_V} S_H - \alpha E_H - \mu_H E_H \\ I'_H &= \alpha E_H - \delta I_H - \mu_H I_H \\ R'_H &= \delta I_H - \mu_H R_H \\ S'_V &= \mu_V N_V - \phi p \frac{I_H}{N_H} S_V - \mu_V S_V \\ E'_V &= \phi p \frac{I_H}{N_H} S_V - \theta E_V - \mu_V E_V \\ I'_V &= \theta E_V - \mu_V I_V, \end{aligned} \tag{4}$$

where

$$N_H = S_H + E_H + I_H + R_H \quad \text{and} \quad N_V = S_V + E_V + I_V.$$

4.1.2 Variables and Parameters

Since the systems with zero movement are compartmentally symmetric, they use the parameters for a single system regardless of rural or urban location. All the parameters in this system are nonnegative and they allow that if the initial values

$$(S_H(0), E_H(0), I_H(0), R_H(0), S_V(0), E_V(0), I_V(0)) \in \mathbb{R}^7_+$$

then the solutions remain in this region for $t \geq 0$.

4.1.3 Equilibria and Stability

We use the next generation operator method to calculate the basic reproductive number for the case in which there is no movement. By calculating the spectral radius FV^{-1} using the methods outlined by van der Drische and Watmough [28], the basic reproductive number is given by

$$\widetilde{R}_0 = \rho(FV^{-1}) = \sqrt{\frac{\beta}{\delta + \mu_H} \cdot \frac{\phi p}{\mu_V} \cdot \frac{\theta}{\mu_V + \theta} \cdot \frac{\alpha}{\alpha + \mu_H}}.$$

The first term, $\frac{\beta}{\delta+\mu_H}$, is the probability of transmission when a vector bites a susceptible host. Secondly, $\frac{\phi p}{\mu_V}$ is the transmission rate to the infectious vector class when a susceptible vector bites an infectious host. $\frac{\theta}{\mu_V+\theta}$ denotes the probability that an exposed vector will

survive the extrinsic incubation period and become infectious. Lastly, $\frac{\alpha}{\alpha+\mu_H}$ expresses the probability that an exposed host survives the incubation period and becomes infectious.

Proposition 4.1. *System 4 has a disease free equilibrium,* $DFE = (N_H, 0, 0, 0, N_V, 0, 0)$. *If* $\widetilde{R}_0 > 1$, *there is a unique endemic equilibrium* $END = (S_H^*, E_H^*, I_H^*, R_H^*, S_V^*, E_V^*, I_V^*)$ *in* $\mathbb{R}_+^7$.

Proof. From the first seven equations, the equilibrium points satisfy the following conditions:

$$
\begin{aligned}
S_H &= \frac{N_H N_V \mu_H}{N_V \mu_H + I_V^* \beta}, \qquad (5)\\
E_H &= \frac{N_H \beta \mu_H}{(\alpha+\mu_H)(N_V\mu_H + \beta I_V^*)} I_V^*,\\
I_H &= \frac{\alpha}{\alpha+\mu_H} \cdot \frac{\beta}{\delta+\mu_H} \cdot \frac{N_H}{N_V\mu_H + \beta I_V^*} I_V^*,\\
R_H &= \frac{\alpha}{\alpha+\mu_H} \cdot \frac{\beta}{\delta+\mu_H} \cdot \frac{\delta N_H}{N_V\mu_H + \beta I_V^*} I_V^*,\\
S_V &= \frac{\mu_V N_V}{\mu_V + \frac{\alpha}{\alpha+\mu_H} \cdot \frac{\beta}{\delta+\mu_H} \cdot \frac{\phi p \mu_H}{N_V\mu_H+\beta I_V^*} I_V^*},\\
E_V &= \frac{N_V \alpha \beta \mu_H \mu_V \phi p}{(\theta+\mu_V)((\alpha+\mu_H)(\delta+\mu_H)(N_V\mu_H+\beta I_V^*)\mu_V + \alpha\beta\mu_H\phi p I_V^*)} I_V^*.
\end{aligned}
$$

From the last equation of the system, the equilibrium points satisfy

$$
\begin{aligned}
&\left(N_V\mu_H\mu_V(\mu_H\mu_V(\delta+\mu_H)(\theta+\mu_V) + \alpha(\mu_V(\theta+\mu_V)(\delta+\mu_H) - \beta\theta\phi p)\right) I_V\\
&+ \left(\beta\mu_V(\theta+\mu_V)(\mu_H\mu_V(\delta+\mu_H) + \alpha(\mu_V(\delta+\mu_H) + \mu_H\phi p))\right) I_V^2 = 0. \qquad (6)
\end{aligned}
$$

From 6, if $I_V^* = 0$, then $I_H^* = 0$. Substituting these values in (5) proves that DFE exists.

For the endemic case, suppose now that $I_V^* \neq 0$. Then 6 has a positive solution

$$
I_V^* = \frac{N_V\mu_H\left(\beta\phi p\theta\alpha - (\delta+\mu_H)\mu_V(\theta+\mu_V)(\alpha+\mu_H)\right)}{\beta(\delta+\mu_H)\mu_V(\theta+\mu_V)(alpha+\mu_H) + \mu_H(theta+\mu_V)\beta\phi p\alpha}
$$

if $\widetilde{R}_0 > 1$. Thus, the END exists only if $\widetilde{R}_0^2 > 1$ and is given by

$$
\begin{aligned}
S_H^* &= \frac{N_H}{\widetilde{R}_0^2}\left(\frac{\beta+\mu_H\widetilde{R}_0^2(1+\frac{\mu_V}{\theta})}{\beta+\mu_H(1+\frac{\mu_V}{\theta})}\right),\\
E_H^* &= (\widetilde{R}_0^2-1)\cdot\frac{N_H}{\widetilde{R}_0^2}\cdot\frac{\mu_H}{\alpha+\mu_H}\cdot\frac{\beta}{\beta+\mu_H(1+\frac{\mu_V}{\theta})},\\
I_H^* &= (\widetilde{R}_0^2-1)\frac{N_H}{\widetilde{R}_0^2}\cdot\frac{\alpha}{\alpha+\mu_H}\cdot\frac{\beta}{\delta+\mu_H}\left(\frac{\mu_H}{\beta+\mu_H(1+\frac{\mu_V}{\theta})}\right),\\
R_H^* &= (\widetilde{R}_0^2-1)\frac{N_H}{\widetilde{R}_0^2}\cdot\frac{\alpha}{\alpha+\mu_H}\cdot\frac{\beta}{\delta+\mu_H}\left(\frac{\delta}{\beta+\mu_H(1+\frac{\mu_V}{\theta})}\right),\\
S_V^* &= \frac{\beta+\mu_H(1+\frac{\mu_V}{\theta})}{\beta+\mu_H\widetilde{R}_0^2(1+\frac{\mu_V}{\theta})},\\
E_V^* &= (\widetilde{R}_0^2-1)\frac{\mu_V}{\theta}\cdot\frac{N_V\mu_H}{\beta+\mu_H\widetilde{R}_0^2(1+\frac{\mu_V}{\theta})},\\
I_V^* &= (\widetilde{R}_0^2-1)\frac{N_H\mu_H}{\beta+\mu_H\widetilde{R}_0^2(1+\frac{\mu_V}{\theta})}.
\end{aligned}
$$

□

Theorem 4.2. *The disease free equilibrium DFE is locally asymptotically stable in $\mathbb{R}_+^7$ when $\widetilde{R}_0 < 1$, and unstable if $\widetilde{R}_0 > 1$.*

Proof. The local stability of DFE is determined by the Jacobian matrix of the system. The Jacobian matrix of (4) is

$$
\mathcal{J}_{\mathcal{DFE}} = \begin{pmatrix}
\mu_H & 0 & 0 & 0 & 0 & 0 & 0\\
0 & -\alpha-\mu_H & \alpha & 0 & 0 & 0 & 0\\
0 & 0 & -\delta-\mu_H & \delta & -\frac{N_V p\phi}{N_H} & \frac{N_V p\phi}{N_H} & 0\\
0 & 0 & 0 & -\mu_H & 0 & 0 & 0\\
0 & 0 & 0 & 0 & -\mu_V & 0 & 0\\
0 & 0 & 0 & 0 & 0 & -\theta-\mu V & \theta\\
-\frac{N_H\beta}{N_V} & \frac{N_H\beta}{N_V} & 0 & 0 & 0 & 0 & -\mu_V
\end{pmatrix},
$$

in which case the eigenvalues are given by

$$-\mu_H, -\mu_V, -\mu_H,$$

together with the solutions to the polynomial

$$(\lambda+\alpha+\mu_H)(\lambda+\delta+\mu_H)(\lambda+\mu_V)(\lambda+\theta+\mu_V) - p\alpha\beta\theta\phi = 0. \tag{7}$$

Rearranging equation (7), we obtain

$$\begin{aligned}\widetilde{R}_0^2 &= \left(\frac{\lambda+\delta+\mu_H}{\delta+\mu_H}\right)\left(\frac{\lambda+\mu_V}{\mu_V}\right)\left(\frac{\lambda+\theta+\mu_V}{\theta+\mu_V}\right)\left(\frac{\lambda+\alpha+\mu_H}{\alpha+\mu_H}\right)\\ &= \left(\frac{\lambda}{\delta+\mu_H}+1\right)\left(\frac{\lambda}{\mu_V}+1\right)\left(\frac{\lambda}{\theta+\mu_V}+1\right)\left(\frac{\lambda}{\alpha+\mu_H}+1\right).\end{aligned}$$

From this equation, we notice that if the roots of (7) have nonnegative real parts, then every term in $\widetilde{R}_0^2 \geq 1$ is greater than or equal to 1, which implies that $\widetilde{R}_0^2 \geq 1$. This implies, $\widetilde{R}_0^2 \geq 1 \Rightarrow \widetilde{R}_0 \geq 1$. Thus, if $\widetilde{R}_0^2 < 1$, the roots of equation (7) must have negative real parts. Hence, we have local stability at the DFE. □

Theorem 4.3. *The endemic equilibrium is locally stable in* $\mathbb{R}_+^7$ *when* $\widetilde{R}_0 > 1$.

Proof. The Jacobian matrix associated with this equilibrium is

$$J_{END} = \begin{pmatrix} -\frac{\widetilde{R}_0\mu_H\omega}{\psi} & \frac{(\widetilde{R}_0-1)\beta\theta\mu_H}{\psi} & 0 & 0 & 0 & 0 & 0\\ 0 & -\alpha-\mu_H & \alpha & 0 & 0 & 0 & 0\\ 0 & 0 & -\delta-\mu_H & \delta & -\frac{N_V p\omega\phi}{N_H(\psi)} & \frac{N_V p\omega\phi}{N_H\psi} & 0\\ 0 & 0 & 0 & -\mu_H & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & -\mu_V-\frac{\varepsilon}{\varphi\omega} & \frac{\varepsilon}{\varphi\omega} & 0\\ 0 & 0 & 0 & 0 & 0 & -\theta-\mu_V & \theta\\ -\frac{N_H\beta\psi}{N_V\widetilde{R}_0\omega} & \frac{N_H\beta\psi}{N_V\widetilde{R}_0\omega} & 0 & 0 & 0 & 0 & -\mu_V \end{pmatrix},$$

where

$$\begin{aligned}\omega &= \beta\theta+\mu_H(\theta+\mu_V),\\ \psi &= \beta\theta+\widetilde{R}_0^2\mu_H(\theta+\mu_V),\\ \varphi &= \widetilde{R}_0^2(\alpha+\mu_H)(\delta+\mu_H),\\ \varepsilon &= (\widetilde{R}_0^2-1)\alpha\ \beta\ \theta\ \phi\ p\ \mu_H,\end{aligned}$$

with eigenvalues given by $-\mu_H, -\mu_V$, and eigenvalues for which the real part is negative if $\widetilde{R}_0 > 1$. □

The model exhibits a forward bifurcation. The stable endemic equilibrium exists only when $\widetilde{R_0} > 1$.

4.2 Movement cases

We now consider the change in movement rates, which depend on the relative proportions of infectious populations. We assume that an increasing proportion of infectious hosts in a rural area will make movement towards the rural area less likely and movement away from

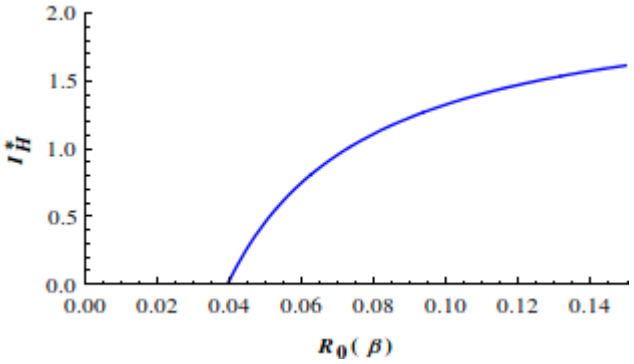

Figure 3: Bifurcation with parameters $\mu_H = 0.0000365297$, $\mu_V = 0.047619$, $\phi = 0.33$, $\alpha = 0.25$, $\theta = 0.1$, $\delta = 0.166667$, $p = 0.9$, and varied β.

the rural area more likely; the same is true for the urban area. It is important to note that we are considering the proportions of infectious populations, not the raw amount of infectious individuals, as the rural and urban populations are likely to be very different in total size.

$$f_R(\boldsymbol{I}, \boldsymbol{N}) = C_R \left(1 - \left(\frac{I_{HU}}{N_{HU}} - \frac{I_{HR}}{N_{HR}}\right)\right)$$
$$f_U(\boldsymbol{I}, \boldsymbol{N}) = C_U \left(1 - \left(\frac{I_{HR}}{N_{HR}} - \frac{I_{HU}}{N_{HU}}\right)\right)$$

At the DFE, this function models movement at the constant rates C_R and C_U. When the infectious class exists, the movement is modeled as a threatening factor. In other words, movement is altered by an individual's perception of the infectious proportions of the populations (assuming an accurate perception). Using the next generation operator method, which implies stability at the DFE, we calculate R_0.

$$R_0 = \frac{1}{2}\left(\widetilde{R}_{0_R}^2 + \widetilde{R}_{0_U}^2\right) + \sqrt{\frac{1}{4}\left(\frac{C_U+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}_{0_R}^2 - \frac{C_R+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}_{0_U}^2\right)^2 + \left(C_R\widetilde{R}_{0_R}^2\right)\left(C_U\widetilde{R}_{0_U}^2\right)}.$$

We are able to rewrite R_0 in terms of $\widetilde{R}_{0_R}$ and $\widetilde{R}_{0_U}$. This helps when trying to recognize the effect of adding movement to the system. The first term,

$$\frac{1}{2}(\widetilde{R}_{0_R}^2 + \widetilde{R}_{0_U}^2),$$

is the weighted average of the contribution of the two independent systems where there is no movement.

$$\frac{1}{4}\left(\frac{C_U+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}^2_{0_R} - \frac{C_R+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}^2_{0_U}\right)^2$$

demonstrates the proportions of subpopulations that are moving. Lastly,

$$\left(C_R\widetilde{R}^2_{0_R}\right)\left(C_U\widetilde{R}^2_{0_U}\right)$$

is the interaction of moving population.

5 Numerical Simulations

5.1 Deterministic model

In this part, several simulations are done on the deterministic model to show the role of movement between subdivided populations is introduced as a constant or a function dependent on the infectious proportion of the population. The section is organized as follows. The simulations done for the case without movement are illustrated first. Then, we include a constant rate of movement. Finally, simulations are presented for the case in which movement is determined by a function. We show the population dynamics presented for the rural and urban areas when $R_0^2 < 1$ and when $R_0^2 > 1$ for each case.

5.1.1 Case without movement

In this section, we discuss the meaning of a numerical simulation of the system when no movement occurs between the urban and rural populations. The systems are compartmentally symmetric (disregarding the numerical transmission rates) and are independent of one another. The dynamics of rural hosts and vectors are examined and later compared to the urban hosts and vectors. This interpretation will contribute to the conclusions drawn about the effects of rural/urban movement on DENV transmission dynamics.

Rural Host and Vector with $\widetilde{R}_0^2 < 1$

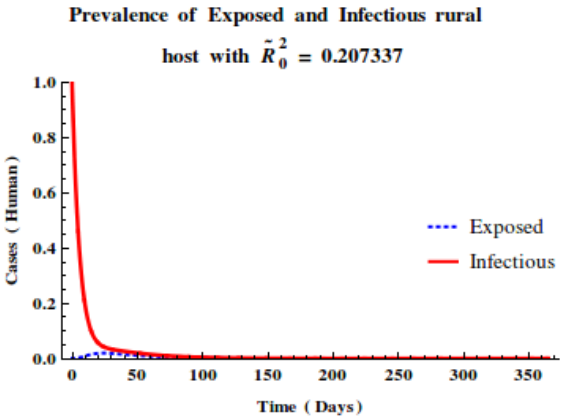

Figure 4: Infectious and exposed rural hosts where $\widetilde{R}_0^2 < 1$ and $\phi = .03$

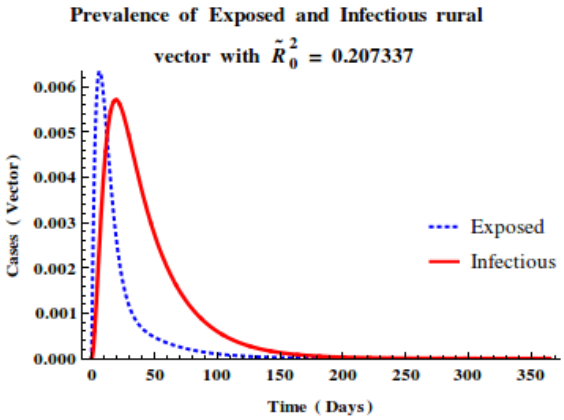

Figure 5: Infectious and exposed rural vectors where $\widetilde{R}_0^2 < 1$ and $\phi = .03$

When $\widetilde{R}_0^2 < 1$, the exposed and infectious classes of both hosts and vectors approach 0. The simulation begins with one infectious host in an otherwise completely susceptible population at 0 days. As time progresses, the infectious and exposed populations for hosts approach the DFE. Simultaneously, the vector class, which begins with 0 infectious vectors, initially increases but, as time progresses, also approaches the DFE. This illustrates the local stability of the DFE.

Rural Host and Vector with $\widetilde{R}_0^2 > 1$

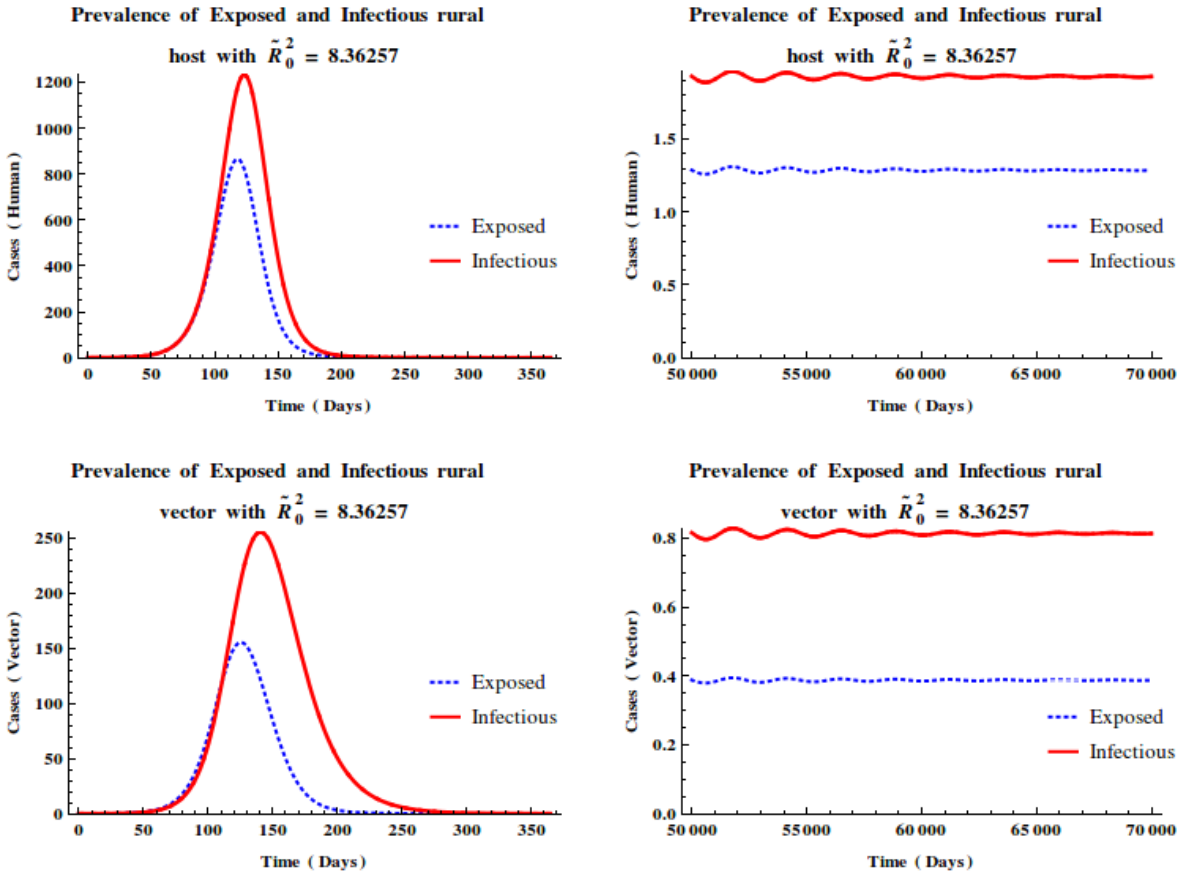

Figure 6: Top Left: Time interval (0, 365) of infectious and exposed in rural for $\widetilde{R}_0^2 > 1$ for host. Top Right: Time interval (50,000, 70,000) of infectious and exposed in rural for $\widetilde{R}_0^2 > 1$ for host. Bottom Left: Time interval (0, 365) of infectious and exposed in rural for $\widetilde{R}_0^2 > 1$ for vector. Bottom Right: Time interval (50,000, 70,000) of infectious and exposed in rural for $\widetilde{R}_0^2 > 1$ for vector.

When $\widetilde{R}_0^2 > 1$, the system reaches an epidemic relatively quickly. The differences in the time and magnitude of the exposed and infectious classes are a result of transmission and survival rates, since they are species specific. In the left side of the Figure 6 does not clearly demonstrate the effect of $R_0^2 > 1$, but the right side is a modified version for time at $50,000 - 70,000$ days. It shows the smaller outbreaks and how, as time progresses, the magnitudes of the outbreaks decrease. The simulation continues until the exposed and infectious hosts reach an endemic equilibrium. Also, Figure 6 demonstrate the incidences

of DENV as time progresses in a rural population where $\widetilde{R}_0^2 < 1$, as time progresses, the disease continues to exist with the magnitudes of the subsequent epidemics decreasing over time. For this scenario, the transmission rates for rural susceptible hosts and vectors, β_R and ϕ_R, are both set equal to 0.33. While the transmission rates for the susceptible urban host and vector, β_U and ϕ_U, are both set equal to 0.31. All other parameters are specific to the host and vector species.

Urban Host and Vector with $\widetilde{R}_0^2 < 1$

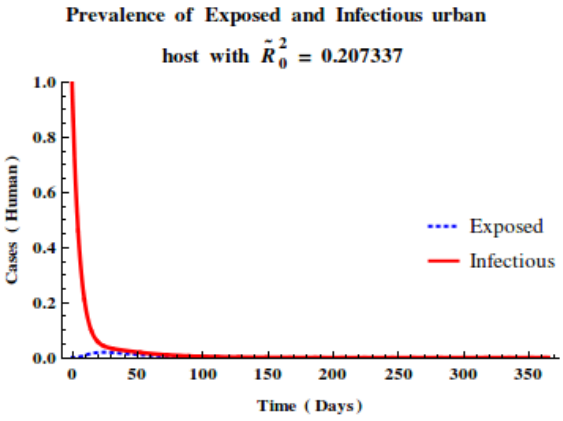

Figure 7: Time interval (0, 200) representation of infectious and exposed host class in the urban for $\widetilde{R}_0 < 1$

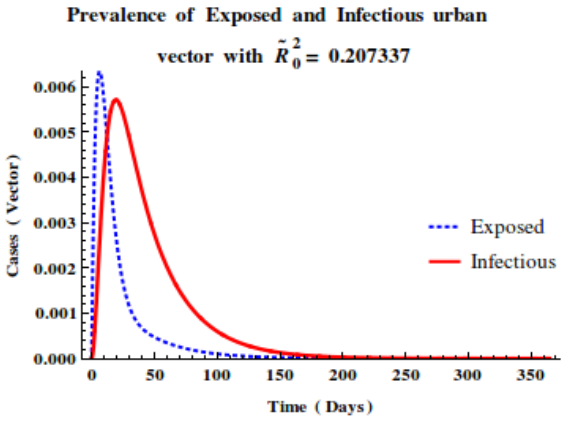

Figure 8: Time interval (0, 200) representation of infectious and exposed vector class in urban for $\widetilde{R}_0 < 1$

It can be seen in Figure 7 that the urban population of exposed and infectious hosts when $\widetilde{R}_0^2 < 1$ approaches the DFE. This follows the proof from the analytic analysis in the previous section when we show that the DFE is locally asymptotically stable when $\widetilde{R}_0 < 1$. The effective transmission rate β used is 0.03 for this scenario. The urban population of the vector when $\widetilde{R}_0^2 < 1$ approaches the DFE as well while the exposed class and infectious class have different timing and magnitudes for the epidemic.

Urban Host and Vector with $\widetilde{R}_0^2 > 1$

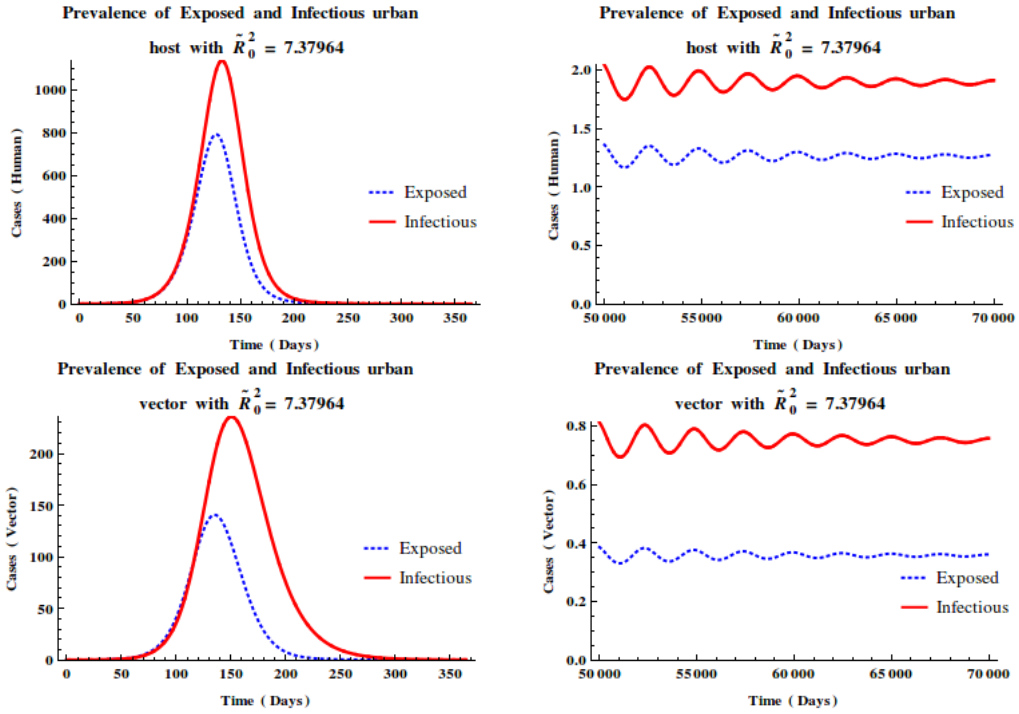

Figure 9: Top Left: Time interval (0, 365) of infectious and exposed in Urban for $\widetilde{R}_0^2 > 1$ for host. Top Right: Time interval (50,000, 70,000) of infectious and exposed in Urban for $\widetilde{R}_0^2 > 1$ for host. Bottom Left: Time interval (0, 365) of infectious and exposed in Urban for $\widetilde{R}_0^2 > 1$ for vector. Bottom Right: Time interval (50,000, 70,000) of infectious and exposed in Urban for $\widetilde{R}_0^2 > 1$ for vector

5.1.2 Case with constant movement

We consider the case when we have constant movement between the population. Also, we assume that these constant do not change in the course of the epidemic. Numerical results show that when $R_0 < 1$, the host and vector do not move through the system and remain susceptible. As in the urban area, the rural area also reaches a disease free equilibrium. Since DENV dies out, the system reaches a disease free equilibrium for this serotype, this is, the infectious and exposed states decrease and eventually go to zero.

In the rural area, the disease follows a similar pattern to that of the urban area. Over time, the disease reaches an endemic equilibrium for both the host and the vector. The number of individuals in the exposed and infectious classes decrease over time, and affect the rural area to smaller degree.

When $R_0 > 1$, the system eventually reaches an endemic equilibrium. The numerical simulations show future outbreaks, though they are smaller and occur with decreasing

magnitude over time. Similarly with hosts, when $R_0 > 1$, the system reaches an endemic state. The differences in the time and magnitude of the exposed and infectious classes are a result of transmission and survival rates. Due to their short lifespan, as compared with that of a host, the vector moves through the system much more quickly. Again, as time approaches infinity, future outbreaks will lessen in both frequency and intensity.
In the rural area, the disease follows a similar pattern to that of the urban area. Over time, the disease reaches an endemic equilibrium for both the host and the vector. The number of individuals in the exposed and infectious classes decrease over time, and affect the rural area to smaller degree.

5.1.3 Case with function dependent movement

In this section, we show numerical simulations of the system with a change in the movement rates. The movement between the populations is analyzed with respect the proportion of the respective population. The dynamics between rural and urban population are compared. We consider the movement functions defined by

$$f_R(\boldsymbol{I}, \boldsymbol{N}) = C_R \left(1 - \left(\frac{I_{HU}}{N_{HU}} - \frac{I_{HR}}{N_{HR}}\right)\right)$$

and

$$f_U(\boldsymbol{I}, \boldsymbol{N}) = C_U \left(1 - \left(\frac{I_{HR}}{N_{HR}} - \frac{I_{HU}}{N_{HU}}\right)\right).$$

Rural Host and Vector with $R_0^2 < 1$

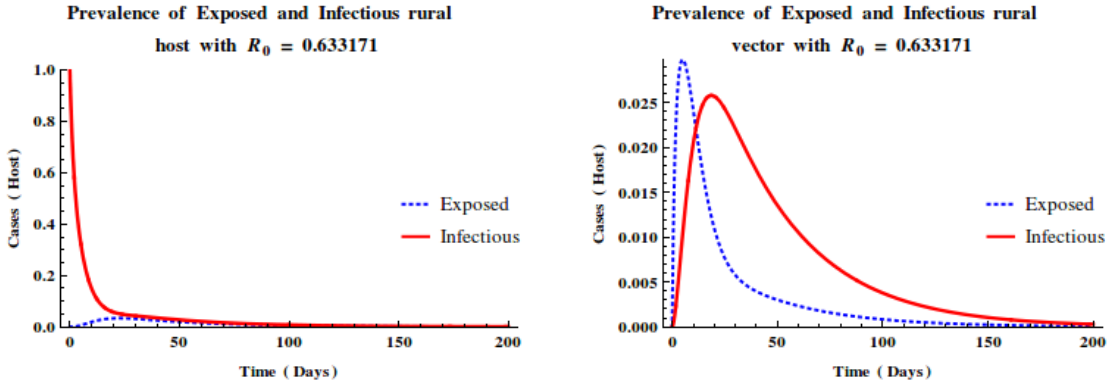

Figure 10: Time interval (0, 200) representation of infectious and exposed vectors in rural for $R_0 < 1$, $\phi_R = \phi_U = .03$ and $\beta_R = \beta_U = .09$

If $R_0 < 1$, the disease transmission reach 0 cases for host and vector population in the infectious and exposed states. The susceptible states (host and vector) reaches the total of population while the other cases go to 0, this is, we have stability in the DFE. We show two simulations with $R_0 < 1$, in two of them the DFE is reached relatively quickly.

Rural Host and Vector with $R_0 > 1$

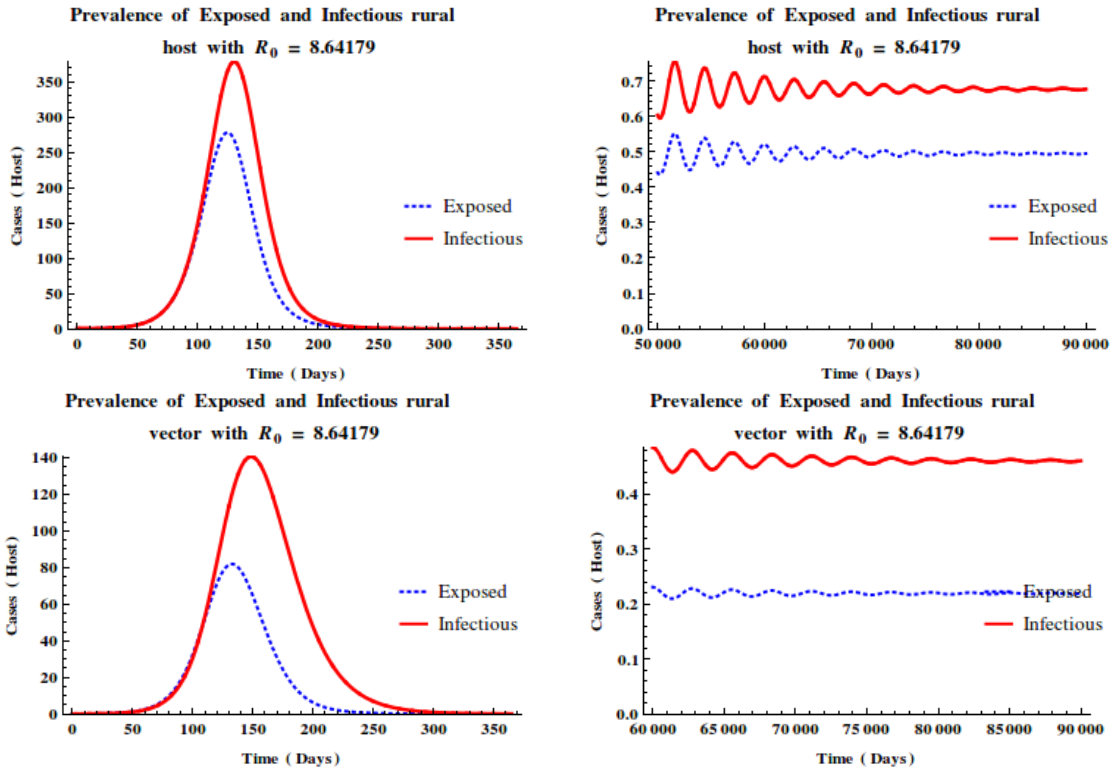

Figure 11: Top Left: Time interval (0, 365) of infectious and exposed in rural for $R_0 > 1$ for host. Top Right: Time interval (50,000, 90,000) of infectious and exposed in rural for $R_0 > 1$ for host. Bottom Left: Time interval (0, 365) of infectious and exposed in rural for $R_0 > 1$ for vector. Bottom Right: Time interval (50,000, 90,000) of Infectious and exposed in rural for $R_0 > 1$ for vector

When $R_0 > 1$ the epidemic is presented in a short period of time although the infectious does not reach the zero value in rural nor urban population, this is a consequence of transmission and survival rates in the vector. In both cases, the transmission rate for host and vector are equal in the each area, $\beta_R = \phi_R = .33$ and $\beta_U = \phi_U = .31$. We assume in this case a greater transmission rate for the rural population, which is affected by the *Ae. aegypti*. The different magnitudes and initial times are exposed in order to show the convergence of infectious and exposed states to the endemic equilibrium. In the right side

of the Figure 5.1.3 we show extension extensions in time of of the left side, in these plots we observe a oscillated convergence to the endemic point where the amplitude decrease when $t \to \infty$. Also, we note that the convergence in the rural vector is faster that the host population, this is result of the survival rates.

Urban Host and Vector with $R_0 < 1$

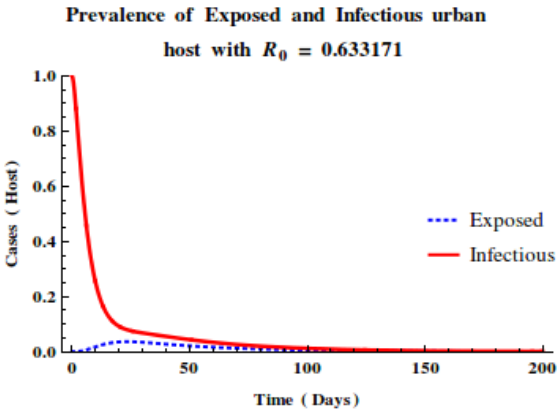

Figure 12: Time interval (0, 365) representation of infectious and exposed host class in the Urban for $R_0 < 1$

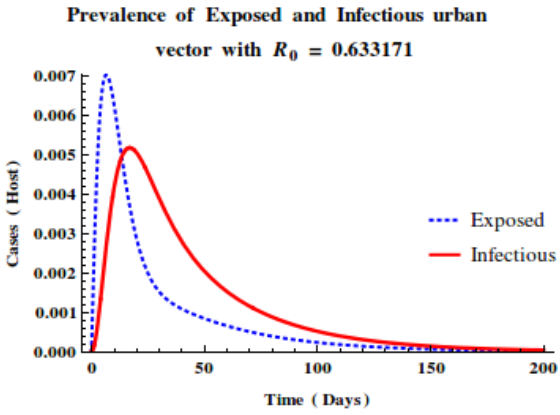

Figure 13: Time interval (0, 365) representation of infectious and exposed vector class in the Urban for $R_0 < 1$

In the figures above the urban populations (host and vector) are shown. Infectious and exposed states reach the disease free equilibrium as$t \to \infty$, which is stable for $R_0 < 1$. The transmission rate β_R, β_U, ϕ_R and ϕ_U change in each case, using $\beta_R = \phi_R = .09$ and $\beta_R = \phi_R = .15$ in the first graph, and $\beta_R = \phi_R = .09$ and $\beta_R = \phi_R = .15$ in the second one. We also show two different time scales in order to analyzed the convergence time with respect to transmission rates.

Urban Host and Vector with $R_0 > 1$

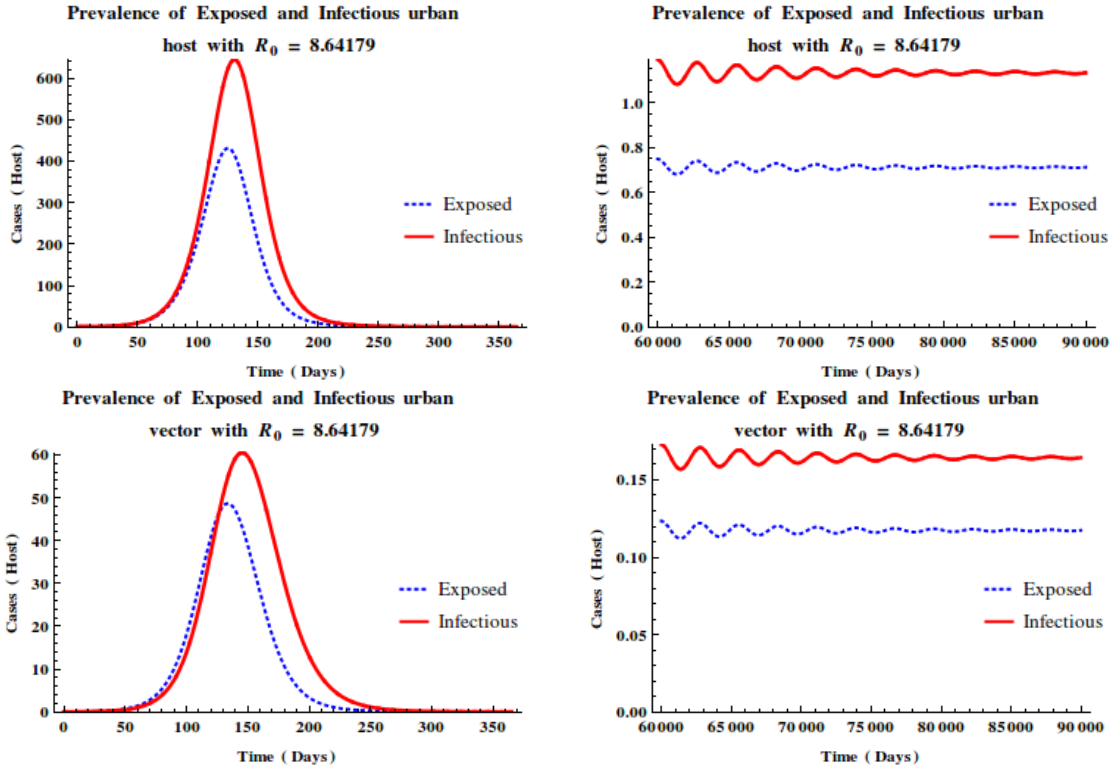

Figure 14: Top Left: Time interval (0, 365) of infectious and exposed in Urban for $R_0 > 1$ for host. Top Right: Time interval (40,000, 60,000) of infectious and exposed in Urban for $R_0 > 1$ for host. Bottom Left: Time interval (0, 365) of infectious and exposed in Urban for $R_0 > 1$ for vector. Bottom Right: Time interval (40,000, 60,000) of infectious and exposed in Urban for $R_0 > 1$ for vector

In the plots above the transmission rates are $\beta_R = \phi_R = .33$ and $\beta_R = \phi_R = .31$. In both cases the infectious and exposed states converge to the endemic equilibrium. Both reach the maximum value in the infectious states in a relatively short time.

The maximum number of infectious cases is small with respect to the initial conditions for the two populations.

6 Stochastic Simulations

6.1 Zero Movement

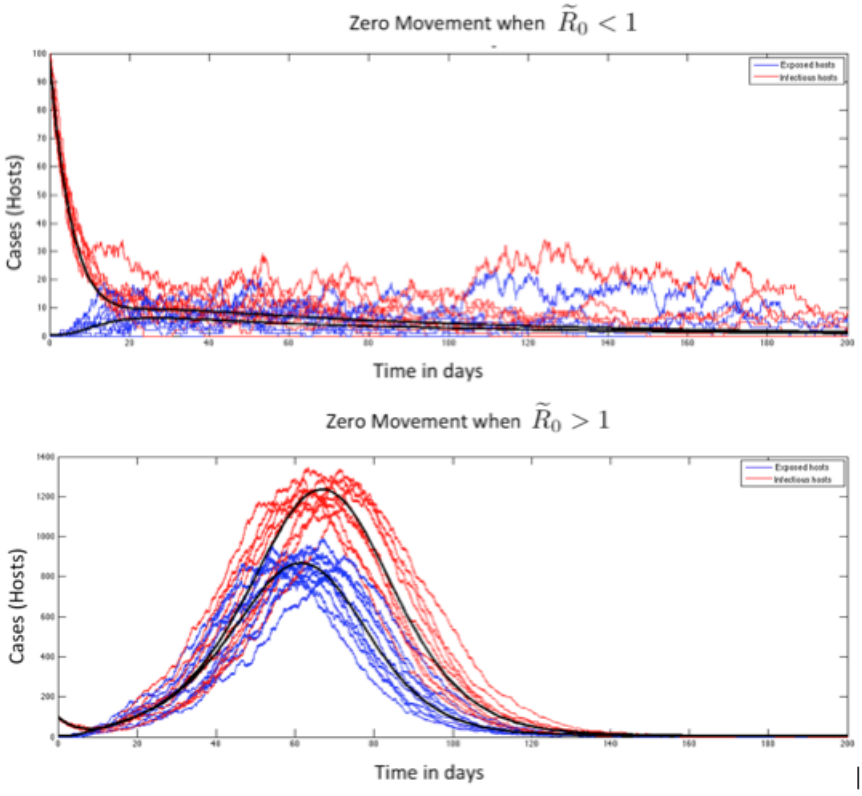

Figure 15: Infectious and exposed hosts, vectors where $\widetilde{R}_0 < 1$ and $\widetilde{R}_0 > 1$

In this section, simulations are done using a stochastic Markov process. The deterministic model is matched closely by the stochastic models, which suggests that the parameter values are accurate means. Stochastic models were used only for the zero movement case. Infectious and exposed classes for hosts and vectors were examined for cases where $R_0 < 1$ and where $R_0 > 1$. Stochastic models for the more complicated cases are not included as they are extremely computationally expensive and inefficient.

7 Cumulative Plots

Finally, we assess the cumulative amounts of infectious hosts for several scenarios. Initially, we consider the cases in which $R_0 < 1$ for five simulations. The first simulation concerns a single population of 10,000 hosts with no movement. The other four simulations concern

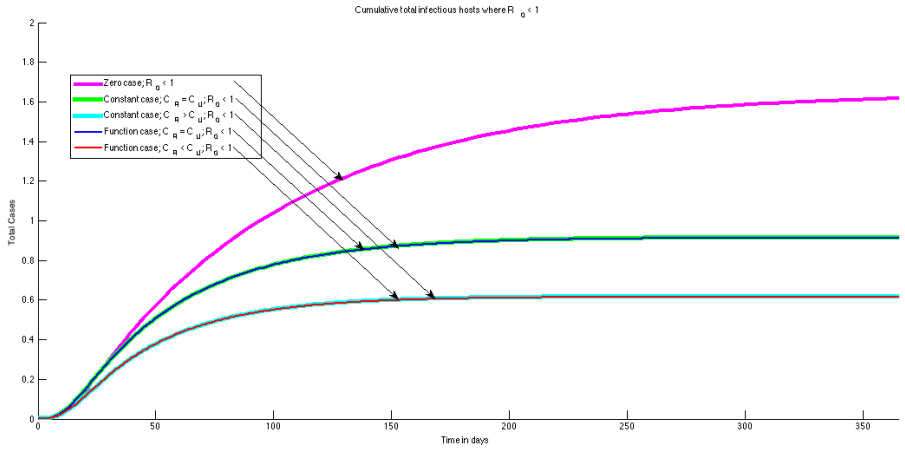

Figure 16: Cumulative infectious cases for multiple scenarios where the DFE is locally asymptotically stable.

two host populations between which movement occurs. The two populations are a rural population of 2,000 hosts and an urban population of 8,000 hosts. The second and third simulations consider cases of constant movement. In the second simulation, movement in both directions is equal, that is, $C_R = C_U$, while in the third simulation, $C_R > C_U$. Similarly, the last two simulations describe a case in which movement follows the functions described above, where movement is equal or preferential in the fourth and fifth simulations, respectively. When $R_0 < 1$, all cases show stability at the disease free equilibrium, so the disease dies out and the cumulative cases of infection reach a maximum and plateau there. All scenarios reach their respective plateaus at approximately the same time. The total cases of infection are greatest for the zero movement case and least for the cases of preferential movement, but in all cases, the total infectious cases are between 0.4 and 1.6 cases.

Next, we consider the same scenarios for the case in which $R_0 > 1$. When $R_0 > 1$, all cases show stability at the endemic equilibrium, so the disease persists as new susceptible individuals enter the population and become infectious. In all cases, the disease eventually affects effectively every member of the population, so the cumulative infectious cases approach and plateau at 10,000 cases (the total host population for all simulations). The scenarios reach this plateau at slightly different times. The case of a single patch with zero movement is the first to reach the plateau, while the cases which include preferential movement are the last to reach the plateau. This is reasonable, as an outbreak spread more quickly in a single population of 10,000 than an outbreak which began in one population and took time to travel to another population via host movement.

Analysis of the total infectious cases suggests while movement has some effect on the severity (when $R_0 < 1$) and speed (when $R_0 > 1$) of a Dengue outbreak, it causes minimal practical change. The greatest effect of host movement is its ability to spread Dengue to

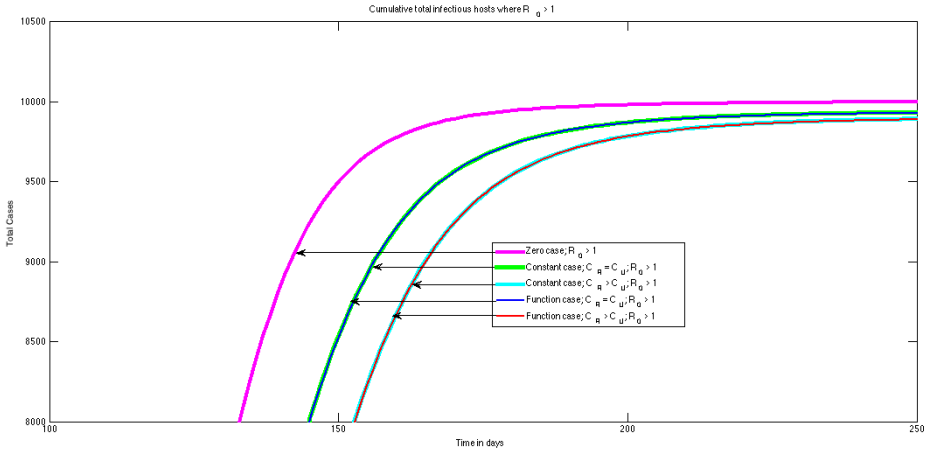

Figure 17: Cumulative infectious cases for multiple scenarios where the endemic equilibrium is stable.

new areas, rather than its ability to change other qualitative traits of an outbreak.

8 Discussion

The purpose of this paper is to model the spread of Dengue fever, taking into consideration movement between rural and urban areas. It is assumed that urban and rural vectors do not leave their respective areas and will only bite mobile hosts. The total populations of vectors, N_{VR} and N_{VU}, are constant, as is the total population of hosts, N_H. The host subpopulations, N_{HR} and N_{HU}, are not constant when movement occurs. It is assumed that exposure to DENV does not impede or change movement in the host, but some proportion of the infectious class is immobile and therefore also unable to be bitten by and infect susceptible vectors. The model considers only one serotype, to which all recovered hosts are permanently immune. Finally, the model considers the populations of hosts and vectors to be homogeneously mixed, so that any mobile host has an equal probability of being bitten by any vector in that area.

In the deterministic model, the simulations show that when $R_0 < 1$, DENV reaches the disease free equilibrium for both the host and vector. The infectious and exposed classes slightly increase, but infectious individuals leave the class via recovery or natural death before infecting an average of one host or vector. Also, the deterministic models demonstrate the existence of an endemic equilibrium. At the endemic equilibrium, the disease is present in the population but does not cause disturbance. Many subtropical and temperate regions undergo seasonal endemics; however, with an increased tendency for hosts to travel, this can quickly lead to an epidemic in multiple areas. The numerical simulations suggest that over a very long time period, the outbreaks of Dengue become

more infrequent and decrease in magnitude. Damping oscillations occur as the populations approach the endemic equilibrium, when $R_0 > 1$.

In addition to the deterministic model, we also constructed a stochastic model in order to confirm the accuracy of our parameters by adding randomness to the model. The deterministic model is used to make predictions based on average parameters and the schematics of the system. This type of model allows us to understand the basic dynamics of transmission where the state variables follow a flow chart and depend on previous states. On the other hand, the stochastic model incorporates a memoryless tool that contributes to the model's realistic outputs. The transmission rates of Dengue between hosts and vectors are dependent on random occurrences, which is reflected in the stochastic model. Finally, it allows us to observe many different qualitative outcomes as well as the probabilities of each outcome. Plotting the stochastic and deterministic models together shows that the deterministic model is the approximate average of the stochastic plots, which supports the accuracy of our model.

In conclusion, we notice that the transmission rates β and ϕ are directly proportional to R_0, such that an increase in the transmission rates will result in an increase in R_0. We modeled the dynamics of Dengue transmission with host movement between rural and urban areas and observed the effects on transmission between the hosts and the stationary vectors. We conclude that while movement between rural and urban areas does not have a notable effect on the speed or severity of a Dengue outbreak in one area, host movement is solely responsible for the spread of Dengue to new areas and populations. Analysis of the system both with and without movement did not reveal a major change in transmission dynamics due to the addition of movement.

For future studies, we will model movement with time-dependent rates. In the future, we also would consider the potential effects of a vaccination, for which we would need to take into account all separate serotypes of DENV. If multiple serotypes are included in a future model, we would also consider instances of DSS as a result of secondary infection, as well as disease-related death.

9 Acknowledgments

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Special thanks to our faculty advisors Dr. Fabio Sanchez and Dr. Omayra Ortega. We would like to express our appreciation for the wisdom and continuous encouragement from our graduate mentors Komi Messan and I Made Eka Dwipayana, as well as Dr. Abba Gumel and Dr. Susan Holechek. We also thank Dr. Ricardo Sá enz for his insight and suggestions.

A.10 Appendix

In this appendix we give some mathematical results that are not shown in the previous sections.

A.10.1 Basic Reproductive Number

A.10.1.1 Case without movement

To find the reproductive number $\widetilde{R}_0$, we follow the Next Generation Operator Matrix Method [28]. $\mathcal{F}$ represents the rate of the new infections caused by transition to theinfected group. $\mathcal{V}$ represents the rates of transfer of individuals into or out of the infected classes by other means. This is,

$$\mathcal{F} = \begin{pmatrix} \beta S_H \frac{I_V}{N_V} \\ \phi S_V p \frac{I_H}{N_H} \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} \alpha E_H + \mu_H E_H \\ \theta E_V \ + \mu_V E_V \\ -\alpha E_H + \delta E_V + \mu_H E_V \\ \theta E_V + \mu_V I_V \end{pmatrix}.$$

The partial derivatives of each matrix are taken with respect to the variables representing the new exposed and infectious classes. Both matrices will be evaluated at the DFE, resulting in

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta \frac{S_H}{N_V} \\ 0 & 0 & \phi p \frac{S_V}{N_V} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \alpha + \mu_H & 0 & 0 & 0 \\ 0 & \theta + \mu_V & 0 & 0 \\ -\alpha & 0\delta + \mu_H & 0 & 0 \\ 0 & -\theta & 0 & \mu_V \end{pmatrix}.$$

We determine the spectral radius of FV^{-1}. Then, the basic reproductive number is

$$\widetilde{R}_0 = \rho(FV^{-1}) = \sqrt{\frac{\beta}{\delta + \mu_H} \cdot \frac{\phi p}{\mu_V} \cdot \frac{\theta}{\mu_V + \theta} \cdot \frac{\alpha}{\alpha + \mu_H}}$$

A.10.1.2 Constant movement case: $f_U(\boldsymbol{I}, \boldsymbol{N}) = C_U$ and $f_R(\boldsymbol{I}, \boldsymbol{N}) = C_R$.

We use the next generation operator matrix method to calculate the basic reproductive number and analyze local stability of the DFE. The vector X consists of infected classes and Y of all other classes are given by:

$$X = \begin{pmatrix} E_{HR} \\ E_{HU} \\ E_{VR} \\ E_{VU} \\ I_{HR} \\ I_{HU} \\ I_{VR} \\ I_{VU} \end{pmatrix} \quad \text{and} \quad Y = \begin{pmatrix} S_{HR} \\ S_{HU} \\ S_{VR} \\ S_{VU} \\ R_{HR} \\ R_{HU} \end{pmatrix},$$

where $\frac{dx}{dt} = \mathcal{F}(X,Y) + \mathcal{V}(X,Y)$. We calculate $\mathcal{F}$, which consists of the newly infectious rates, and $\mathcal{V}$, which consists of rates of transfer in and out of the infectious classes by other means. Following this method, we have:

$$\mathcal{F} = \begin{pmatrix} \beta_R S_{HR} \frac{I_{VR}}{N_{VR}} + (C_R E_{HR} + C_U E_{HU}) \left(\frac{I_{HU}}{N_{HU}}\right)^2 \\ \beta_U S_{HU} \frac{I_{VU}}{N_{VU}} (C_R E_{HR} + C_U E_{HU}) \left(\frac{I_{HR}}{N_{HR}}\right)^2 \\ \phi_R p S_{VR} \frac{I_{HR}}{N_{HR}} \\ \phi_U p S_{VU} \frac{I_{HU}}{N_{HU}} \\ C_R p I_{HR} \left(\frac{I_{HU}}{N_{HU}}\right)^2 + C_U p I_{HU} \left(1 + \left(\frac{I_{HU}}{N_{HU}}\right)^2\right) \\ C_U p I_{HU} \left(\frac{I_{HR}}{N_{HR}}\right)^2 + C_R p I_{HR} \left(1 + \left(\frac{I_{HR}}{N_{HR}}\right)^2\right) \\ 0 \\ 0 \end{pmatrix}$$

and

$$\mathcal{V} = \begin{pmatrix} (C_R E_{HR} + C_U E_{HU}) \left(\frac{I_{HR}}{N_{HR}}\right)^2 + (\alpha + C_R + \mu_H) E_{HR} - C_U E_{HU} \\ (C_R E_{HR} + C_U E_{HU}) \left(\frac{I_{HU}}{N_{HU}}\right)^2 + (\alpha + C_U + \mu_H) E_{HU} - C_R E_{HR} \\ (\theta_R + \mu_{VR}) E_{VR} \\ (\theta_U + \mu_{VU}) E_{VU} \\ C_U p I_{HU} \left(\frac{I_{HR}}{N_{HR}}\right)^2 + C_R p I_{HR} \left(1 + \left(\frac{I_{HR}}{N_{HR}}\right)^2\right) - \alpha E_{HR} + (\delta + \mu_H) I_{HR} \\ C_R p I_{HR} \left(\frac{I_{HU}}{N_{HU}}\right)^2 + C_U p I_{HU} \left(1 + \left(\frac{I_{HU}}{N_{HU}}\right)^2\right) - \alpha E_{HU} + (\delta + \mu_H) I_{HU} \\ -\theta_R E_{VR} + \mu_{VR} I_{VR} \\ -\theta_U E_{VU} + \mu_{VU} I_{VU} \end{pmatrix}.$$

Then, we take the partial derivatives with respect to the infectious classes, X, and evaluate at the DFE.

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & \beta_R \frac{N_{HR}}{N_{VR}} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_U \frac{N_{HU}}{N_{VU}} \\ 0 & 0 & 0 & 0 & \phi_R p \frac{N_{VR}}{N_{HR}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi_U p \frac{N_{VU}}{N_{HU}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} C_R + \alpha + \mu_H & -C_U & 0 & 0 & 0 & 0 & 0 & 0 \\ -C_R & C_U + \alpha + \mu_H & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_R + \mu_{VR} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta_U + \mu_{VU} & 0 & 0 & 0 & 0 \\ -\alpha & 0 & 0 & 0 & \delta + \mu_H & 0 & 0 & 0 \\ 0 & -\alpha & 0 & 0 & 0 & \delta + \mu_H & 0 & 0 \\ 0 & 0 & -\theta_R & 0 & 0 & 0 & \mu_{VR} & 0 \\ 0 & 0 & 0 & -\theta_U & 0 & 0 & 0 & \mu_{VU} \end{pmatrix}$$

.

The next generation matrix is FV^{-1} where $\rho(FV^{-1})$ is the basic reproduction number, R_0. The R_0 is given by:

$$R_0 = \frac{1}{2}\left(\widetilde{R}_{0_R}^2 + \widetilde{R}_{0_U}^2\right) + \sqrt{\frac{1}{4}\left(\frac{C_U+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}_{0_R}^2 - \frac{C_R+\alpha+\mu_H}{C_R+C_U+\alpha+\mu_H}\widetilde{R}_{0_U}^2\right)^2 + \left(C_R\widetilde{R}_{0_R}^2\right)\left(C_U\widetilde{R}_{0_U}^2\right)}$$

where

$$\widetilde{R}_{0_R}^2 = \frac{\beta_R}{\mu_H+\delta} \cdot \frac{\phi_R p}{\mu_{VR}} \cdot \frac{\theta_R}{\mu_{VR}+\theta_R} \cdot \frac{\alpha}{\alpha+\mu_H}$$

$$\widetilde{R}_{0_U}^2 = \frac{\beta_U}{\mu_H+\delta} \cdot \frac{\phi_U p}{\mu_{VU}} \cdot \frac{\theta_U}{\mu_{VU}+\theta_U} \cdot \frac{\alpha}{\alpha+\mu_H}$$

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