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On the Effect of Host Migration in Host-Vector Transmission

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Abstract

Host-vector transmission is challenging to model, due to human and environmental factors influence complexity. The effect of human mobility on the dispersion of vector-borne diseases is studied here. The effect of human mobility among two-patches will be investigated through a host-vector model with a standard incidence rate and constant mobility rate of humans between both patches. If all hosts could migrate between patches, this model gives us a disease-free equilibrium and a co-endemic equilibrium. The basic reproduction number, R0 carried out as a threshold classifying the dynamics of the models when all hosts could migrate. We also consider a scenario if only unidirectional of infected host occurs which shows up a trans-critical bifurcation. The infected migration rate becomes an important parameter to change the co-endemic behavior to be a disease-free condition. Another scenario implement was a migration of healthy host only which shows that the basic reproduction ratio, R_0 not the only threshold for co-endemic equilibria existence. The analytical results also show that increasing healthy host migration from patch i to j may be a helpful control strategy for disease management in patch i. The host migration can also turn out to be one of the driving forces to the disease dispersal for some specific conditions.

Keywords: Host-vector; host migration; basic reproduction number

1. Introduction

The vector-borne disease which includes long-established scourges, i.e. malaria, dengue, and chikungunya, has emerged as an international public health problem (WHO, 2009). The maintenance and resurgence of vector-borne diseases are related to ecological changes that favor increased vector densities or vector-host interactions, among other factors. There have been profound increases in the magnitude of vector-borne disease problems as a result of urbanization, deforestation, globalization, economic development, among other factors (Gratz, 1999). Experts recognize mobility as one of the most important drivers of global change and predict that rapid increases in urban populations throughout the world will have major implications for human health in general and vector-borne diseases specifically (Sutherst, 2004).

Reasons for the resurgence of vector-borne in the tropics and subtropics are complex and include population growth factors like urbanization with substandard living conditions, inadequate public health, sanitation infrastructure, lack of vector control, international travels, and virus evolution (Gubler, 1998; Annelies & Gubler, 2008). Of all these factors, urbanization has probably had the most impact on the amplification of vector-borne disease within a country, and travel has had the most impact on the spread of infection from country to country and continent to continent. Epidemics of vector-borne disease, their seasonality, and oscillations over time are reflected by the epidemiology of vector-borne in travelers (Annelies & Gubler, 2008).

Travel and transport have also contributed to the spread of vector-borne diseases. There are many reasons to believe that the spatial movement of humans may be important for the epidemiology of vector-borne diseases (Ostroff, 2012; Chen & Wilson, 2008; Gratz, 1999; Gushulak & MacPherson, 2004; Stoddard, et al., 2009; Annelies & Gubler, 2008). As an example, Martens (2000) found out that one of the factors contributing to the re-emergence of malaria is human migration. Some empirical studies also by Domarle (2006) and Ronald (2006) supporting the idea that travel outside urban areas is an important factor in maintaining malaria in urban areas where transmission is low. Mosquitoes borne diseases like dengue and malaria do not have an avian intermediary. Humans are the only hosts that amplifying these viruses within the body and effectively moving these viruses from place to place (Cavrini, et al., 2009; Annelies & Gubler, 2008; Rezza, et al., 2007).

Okubo and Levin (2001) are using both continuous reaction-diffusion systems and discrete patchy models to study spatial heterogeneity. The reaction-diffusion system is suitable for random spatial dispersal, while patchy

models are used to describe directed movement among patches. Modeling the spread of infectious diseases in spatially heterogeneous host populations by directed movement is to captured migration or among countries and regions or travel among cities. Arino (2006) formulated *n*-city SIS epidemic models to investigate the propagation of disease in a population of individuals who travel between *n* cities. The mobility component is represented as a directed graph with cities as vertex and arcs determined by outgoing travel. Wang (2004) studied an *n*-patch SIS model with bilinear incidence and the same dispersal rate of susceptible and infectious individuals on each patch. With the same assumption, Jin (2005) showed that the *n* patch SIS model can be reduced to a monotone system and used the theory of monotone dynamical systems to prove the uniqueness and global stability of endemic equilibrium. Li (2009) utilizes the graph-theoretical approach to construct the global Lyapunov function for the SIR epidemic model with a bilinear incidence in a patchy environment, while Salmani (2006) used standard incidence. Differ with Li (2009), Ma (2008) proposed a similar n-patch model without global stability analysis. The proposed model by Li (2009) is a generalization of two patch SIS model of Wang (2004). A two patch SIRS model in Brauer (2008) also becomes a special case of model in Li (2009) if we assume that the disease has permanent immunity.

Until now, only a few studies discuss the effect of mobility on the spread of host-vector disease in general and specifically their diseases. Pongsumpun (2004) describes the transmission of dengue fever (DF) in an endemic region and focus on the number of travelers who become infected. Cosner (2009) develop spatial models of vector-borne disease dynamics on a network of patches to examine how the movement of humans in heterogeneous environments affects transmission. They show that the movement of humans between patches can to maintain disease persistence in patches with zero transmission. Cai (2010) analyzed a vector-host epidemic model with direct transmission by SIS and SIRS model.

We proposed the transmission model of vector-borne diseases in the presence of human mobility as an important factor of disease spread. Since vector flight limited capability, this model assumes in the absence of vector mobility. Different from Iddi (2011), which proposed a mathematical model that just involved the inflow rate of infected immigrants entering one host population, we introduced a complex model with host migration between two patches. Further, some strategies also applied to illustrate the effect of host migration on disease spreading. Numerical simulation will be given to illustrate the analytic results for the two patches model.

2. Mathematical Model

A two-patch host-vector transmission model is introducing to explore host migration roles on vector-borne disease transmission. In this section, we will particularly discuss a two-patch model following the SIR-SI model with a standard incidence rate. The compartment diagram for the two-patch model illustrated in *Figure 1*. This model included two-patches that we called by Patch 1 and Patch 2. The index i in the next discussion will refer to 1 and 2 as the identity name of the patch.

The total host population of patch i, N_{hi} is divided into three compartments: the susceptible hosts S_{hi}, the infected hosts I_{hi}, and the recovered hosts R_{hi}. Total vector population in patch i, N_{vi} also divided into the susceptible vector, S_{vi} and infected vector, I_{vi}. The new-born birth rate of a host in patch i occurs at constant rate Λ_{hi}, while the recruitment rate of a host in patch i denoted by Λ_{vi}. All newborns are assumed to become a susceptible sub-population. The natural death rate of humans and vector in patch i denoted by μ_{hi} and μ_{vi}, respectively, and there is no disease-induced death.

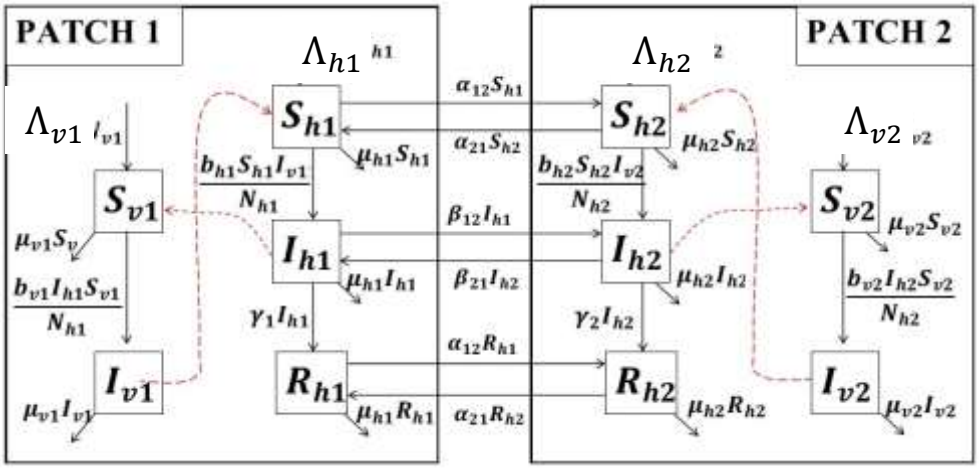


Figure 1. Transmission Diagram Host-Vector in Two Patch

A susceptible host in patch i can get infected through contacts at the per capita rate b_{hi} per infected vectors in patch i. Moreover, a susceptible vector in patch i can get infected through contacts at the per capita rate b_{vi} per infected host from patch i. Then we apply the standard incidence rate at which humans and vectors get infected, $b_{hi}S_hI_v/N_{hi}$ and $b_{vi}S_vI_h/N_{hi}$, respectively. It is different from Mishra (2018) that used mass incidence rate which not reliable since the total host population might change over time due to host migration. The infective host in patch i recover at a constant rate γ_i and will become immune to reinfection within a certain period.

The effect of host mobility in host-vector transmission dynamics shows using different migration rate of healthy host and infected ones. Healthy host, i.e. S_{hi} and R_{hi} can cross the border between patches with migration rate $\alpha_{ij}, i \neq j$. Infected host, I_{hi} also can transfer to another patch with migration rate $\beta_{ij}I_{hi}, i \neq j$ per unit time. Disease infection during transport is negligible. Moreover, the vector population does not relocate between patch due to their short flight distance ability. The *Aedes aegypti* mosquitoes usually fly an average of 400 meters (WHO, 2009). This short flight distance makes human rapidly move the virus within and between communities and places, rather than mosquitoes.

Based on this, we get the dynamical system as shown in system (1) below.

$$
\begin{aligned}
\frac{dS_{h1}}{dt} &= \Lambda_{h1} - \mu_{h1}S_{h1} - \alpha_{12}S_{h1} + \alpha_{21}S_{h2} - \frac{b_{h1}I_{v1}S_{h1}}{N_{h1}} \\
\frac{dI_{h1}}{dt} &= \mu_{h1}I_{h1} - \beta_{12}I_{h1} + \beta_{21}I_{h2} + \frac{b_{h1}I_{v1}S_{h1}}{N_{h1}} - \gamma_1 I_{h1} \\
\frac{dR_{h1}}{dt} &= \gamma_1 I_{h1} - \alpha_{12}R_{h1} - \mu_{h1}R_{h1} + \alpha_{21}R_{h2} \\
\frac{dS_{v1}}{dt} &= \Lambda_{v1} - \frac{b_{v1}S_{v1}I_{h1}}{N_{h1}} - \mu_{v1}S_{v1} \\
\frac{dI_{v1}}{dt} &= \frac{b_{v1}S_{v1}I_{h1}}{N_{h1}} - \mu_{v1}I_{v1} \\
\frac{dS_{h2}}{dt} &= \Lambda_{h2} - \mu_{h2}S_{h2} - \alpha_{21}S_{h2} + \alpha_{12}S_{h1} - \frac{b_{h2}I_{v2}S_{h2}}{N_{h2}} \\
\frac{dI_{h2}}{dt} &= \frac{b_{h2}I_{v2}S_{h2}}{N_{h2}} - \gamma_2 I_{h2} - \mu_{h2}I_{h2} - \beta_{21}I_{h2} + \beta_{12}I_{h1} \\
\frac{dR_{h2}}{dt} &= \gamma_2 I_{h2} + \alpha_{12}R_{h1} - \alpha_{21}R_{h2} - \mu_{h2}R_{h2} \\
\frac{dS_{v2}}{dt} &= \Lambda_{v2} - \frac{b_{v2}S_{v2}I_{h2}}{N_{h2}} - \mu_{v2}S_{v2} \\
\frac{dI_{v2}}{dt} &= \frac{b_{v2}S_{v2}I_{h2}}{N_{h2}} - \mu_{v2}I_{v2}
\end{aligned}
\tag{1}
$$

The non-negative initial conditions are given by

$$S_{hi}(0), S_{vi}(0) > 0, I_{hi}(0), I_{vi}(0), R_{hi}(0) \geq 0, \text{and } I_{h1}(0) + I_{h2}(0) > 0. \tag{2}$$

Noted that $N_{hi} = S_{hi}(t) + I_{hi}(t) + R_{hi}(t)$ and $N_{vi} = S_{vi}(t) + I_{vi}(t)$ for $i = 1,2$ as the total population of humans and mosquitos in patch i respectively.

When both patches are isolated, i.e. $\alpha_{ij} = \beta_{ij} = 0, i \neq j$, the long behavior of the host population in patch i tends to recruitment rate of human in patch i during host lifetime, A_{hi}/μ_{hi}. Let $\mu_h^* = min\{\mu_{h1}, \mu_{h2}\}$. If migration between patches occurs, i.e. $\alpha_{ij} \neq 0, \beta_{ij} \neq 0$, for $i \neq j$, the total host population dynamics are given by

$$\frac{d}{dt}\sum_{i=1}^{2} N_{hi} = \Lambda_{h1} + \Lambda_{h2} - \mu_{h1}N_{h1} - \mu_{h2}N_{h2} \leq \Lambda_{h1} + \Lambda_{h2} - \mu_h^* \sum_{i=1}^{2} N_{hi} \tag{3}$$

The initial conditions (2) make sure that $\sum_{i=1}^{2} N_{hi}(0) \geq 0$. Thus, the total populations of host $\sum_{i=1}^{2} N_{hi}$ remain positive and bounded for all finite time $t > 0$.

3. Dynamical Analysis

Direct calculation shows that system (1) has a disease-free equilibrium point given by

$$E_0 = \left(N_{h1}^*, 0, 0, \frac{\Lambda_{v1}}{\mu_v}, 0, N_{h2}^*, 0, 0, \frac{\Lambda_{v2}}{\mu_v}, 0\right),$$

where $N_{h1}^* = \frac{\Lambda_{h1}\alpha_{21} + \Lambda_{h1}\mu_{h2} + \Lambda_{h2}\alpha_{21}}{\alpha_{12}\mu_{h2} + \alpha_{21}\mu_{h1} + \mu_{h1}\mu_{h2}}$ and $N_{h2}^* = \frac{\Lambda_{h1}\alpha_{12} + \Lambda_{h2}\mu_{h1} + \Lambda_{h2}\alpha_{12}}{\alpha_{12}\mu_{h2} + \alpha_{21}\mu_{h1} + \mu_{h1}\mu_{h2}}$.

Let construct transmission matrix $\mathbf{F}$ and transition matrix $\mathbf{V}$ as follows

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & b_{h1} & 0 \\ 0 & 0 & 0 & b_{h2} \\ \frac{b_{v1}\Lambda_{v1}}{N_{h1}^*\mu_{v1}} & 0 & 0 & 0 \\ 0 & \frac{b_{v2}\Lambda_{v2}}{N_{h2}^*\mu_{v1}} & 0 & 0 \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} \mu_{h1} + \gamma_1 + \beta_{12} & -\beta_{21} & 0 & 0 \\ -\beta_{12} & \mu_{h2} + \gamma_2 + \beta_{21} & 0 & 0 \\ 0 & 0 & \mu_{v1} & 0 \\ 0 & 0 & 0 & \mu_{v2} \end{pmatrix}.$$

Defined $R_0^{(i)} = \frac{b_h b_v \Lambda_{vi}}{\mu_v^2 N_{hi}^*(\mu_h + \gamma)}$ and $\eta_i = \frac{\beta_{ji} + \mu_h + \gamma}{\beta_{ij} + \beta_{ji} + \mu_h + \gamma}, j \neq i$. Using the Next Generation Method by (Driessche & Watmough, 2002), we obtain the quantity R_0 as follows:

$$R_0 = \rho(\mathbf{F}\mathbf{V}^{-1}) = \frac{1}{2}\left(R_0^{(1)}\eta_1 + R_0^{(2)}\eta_2 + \sqrt{\left(R_0^{(1)}\eta_1 - R_0^{(2)}\eta_2\right)^2 + 4\frac{R_0^{(1)}R_0^{(2)}\beta_{12}\beta_{21}}{(\beta_{12} + \beta_{21} + \mu_h + \gamma)^2}}\right) \tag{4}$$

The threshold R_0 called the basic reproduction number which represents an average number of secondary cases produced by a single infective individual which is introduced into an entirely susceptible population. For classical epidemics models, it is common that R_0 is a threshold in a sense that if $R_0 < 1$, on average each infected individual infects fewer than one individual, and the disease dies out. If $R_0 > 1$, on average each infected individual infects more than on individuals, so the diseases are expected to spread. When there is no migration between two patches, i.e. $\alpha_{ij} = 0$ and $\beta_{ij} = 0$, the R_0 for host-vector in (Driessche & Watmough, 2002) is resolved as R_0 of each patch of our results.

Theorem 1. If $R_0 \leq 1$ the disease-free equilibrium E_0 is globally asymptotically stable in

$$\Omega = \left\{(\mathrm{S_{h1}}, \mathrm{I_{h1}}, \mathrm{R_{h1}}, \mathrm{S_{v1}}, \mathrm{I_{v1}}, \mathrm{S_{h2}}, \mathrm{I_{h2}}, \mathrm{R_{h2}}, \mathrm{S_{v2}}, \mathrm{I_{v2}}) \middle| \sum_{i=1}^{2} N_{hi} \leq \frac{\Lambda_{h1} + \Lambda_{h2}}{\mu_h^*} \text{ and } N_{vi} \leq \frac{\Lambda_{vi}}{\mu_v}, i = 1,2\right\}.$$

Proof. Let $\mathbf{F}$ and $\mathbf{V}$ as define before. Since all off-diagonal entries of $\mathbf{V}$ are non-positive and the sum of the entries in each column of $\mathbf{V}$ positive, and thus $\mathbf{V}$ is a non-singular matrix. $\mathbf{V^{-1}}$ is also irreducible. By Perron-Frobenius Theorem (Horn & Johnson, 2013), nonnegative irreducible matrix $\mathbf{V^{-1}F}$ has a positive left eigenvector $\mathbf{w} = (w_1, w_2, w_3, w_4)$ corresponding to eigenvalue $\rho(\mathbf{V^{-1}F})$. Since $\mathbf{F}$ is block matrix with diagonal sub-matrices, then $\rho(\mathbf{V}^{-1}\mathbf{F}) = \rho(\mathbf{F}\mathbf{V}^{-1}) = R_0$. Consequently, we have $\mathbf{w}\mathbf{V^{-1}F} = R_0\mathbf{w}$ and thus

$$\frac{1}{R_0}\mathbf{w} = \mathbf{w}\mathbf{F^{-1}}\mathbf{V} \tag{5}$$

Let $\mathbf{c} = \mathbf{w}\mathbf{F^{-1}} = \left(\frac{w_3}{b_{h1}}, \frac{w_4}{b_{h2}}, \frac{w_1\bar{S}_{h1}}{b_{v1}\bar{S}_{v1}}, \frac{w_2\bar{S}_{h2}}{b_{v2}\bar{S}_{v2}}\right)$ and $\mathbf{I^h} = (I_{h1}, I_{h2}, I_{v1}, I_{v2})^T$. Set $L = \sum_{i=1}^{n} c_i I_i^h$. Differentiating L along system (1) and using identity (5), we obtain

$$\frac{dL}{dt} = \mathbf{c}\left(\frac{\mathrm{d}}{\mathrm{dt}}\mathbf{I^h}\right) = \mathbf{c}\begin{pmatrix} -(\mu_{h1}+\gamma_1+\beta_{12}) & \beta_{21} & \frac{b_{h1}S_{h1}}{N_{h1}^*} & 0 \\ \beta_{12} & -(\mu_{h2}+\gamma_2+\beta_{21}) & 0 & \frac{b_{h2}S_{h2}}{N_{h2}^*} \\ \frac{b_{v1}S_{v1}}{N_{h1}^*} & 0 & -\mu_{v1} & 0 \\ 0 & \frac{b_{v2}S_{v2}}{N_{h2}^*} & 0 & -\mu_{v2} \end{pmatrix}\begin{pmatrix} I_{h1} \\ I_{h2} \\ I_{v1} \\ I_{v2} \end{pmatrix}$$

$$\frac{dL}{dt} \leq \mathbf{c}(\mathbf{F}-\mathbf{V})\mathbf{I^h} = \mathbf{w}\mathbf{F^{-1}}(\mathbf{F}-\mathbf{V})\mathbf{I^h} = (\mathbf{w}-\mathbf{w}\mathbf{F^{-1}}\mathbf{V})\mathbf{I^h}$$

Substitute identity (5) in above equation obtained that $\frac{dL}{dt} \leq \mathbf{w}\left(1-\frac{1}{R_0}\right)\mathbf{I^h} \leq 0$, if $R_0 \leq 1$. Therefore, L is a Lyapunov function for system (1). Since all element $\mathbf{c}$ are positive, $\frac{dL}{dt} = 0$ when $I_{hi} = 0$ and $I_{vi} = 0$ for $i = 1,2$. This condition implied that the only invariant subset of the set

$$\{(\mathrm{S_{hi}}, \mathrm{I_{hi}}, \mathrm{R_{hi}}, \mathrm{S_{vi}}, \mathrm{I_{vi}}, \mathrm{i} = 1,2) \in \Omega |\ \mathrm{I_{hi}} = 0, \mathrm{I_{vi}} = 0, i = 1,2\}$$

is the singleton E_0. Therefore, E_0 is globally asymptotically stable in Ω. ■

From Eq.(4), if $R_0^{(1)}R_0^{(2)} \geq \left(1+\frac{\mu_h+\gamma+\beta_{12}}{\beta_{21}}\right)\left(1+\frac{\mu_h+\gamma+\beta_{12}}{\beta_{21}}\right)$ then it easily deduce that $R_0 > 1$ holds for any $\left(R_0^{(1)}, R_0^{(2)}\right) \in \mathbb{R}_+^2$. Let either $\beta_{12} = 0$ or $\beta_{21} = 0$, then $R_0 = \bar{R}_0 = \max\left(R_0^{(1)}\eta_1, R_0^{(2)}\eta_2\right)$. If $\beta_{12} > 0$ and $\beta_{21} > 0$ then $R_0 > \bar{R}_0$. This means that the infected host migration increasing value of R_0. In the next discussion, we used a same value of $b_h = b_{h1} = b_{h2}$, $b_v = b_{v1} = b_{v2}$, $\mu_h = \mu_{h1} = \mu_{h2}$, and $\mu_v = \mu_{v1} = \mu_{v2}$ since the value is not significant different between two patch.

Recalling that $N_{hi} = S_{hi} + I_{hi} + R_{hi}$ and $N_{vi} = S_{vi} + I_{vi}$, we achieve the following limit system of (1) as follows

$$\begin{aligned}
\frac{dS_{h1}}{dt} &= \Lambda_{h1} - \frac{b_h I_{v1} S_{h1}}{N_{h1}^*} - (\mu_h + \alpha_{12})S_{h1} + \alpha_{21}S_{h2} \\
\frac{dI_{h1}}{dt} &= \frac{b_h I_{v1} S_{h1}}{N_{h1}^*} - (\mu_h + \beta_{12} + \gamma)I_{h1} + \beta_{21}I_{h2} \\
\frac{dI_{v1}}{dt} &= \frac{b_v(\Lambda_{v1}/\mu_v - I_{v1})I_{h1}}{N_{h1}^*} - \mu_v I_{v1} \\
\frac{dS_{h2}}{dt} &= \Lambda_{h2} - \frac{b_h I_{v2} S_{h2}}{N_{h2}^*} - (\mu_h + \alpha_{21})S_{h2} + \alpha_{12}S_{h1} \\
\frac{dI_{h2}}{dt} &= \frac{b_h I_{v2} S_{h2}}{N_{h2}^*} - (\mu_h + \beta_{21} + \gamma)I_{h2} + \beta_{12}I_{h1} \\
\frac{dI_{v2}}{dt} &= \frac{b_v(\Lambda_{v2}/\mu_v - I_{v2})I_{h2}}{N_{h2}^*} - \mu_v I_{v2}
\end{aligned} \tag{6}$$

The dynamical behaviors of S_{hi}, I_{hi}, and $I_{vi}, i = 1,2$ in (1) is asymptotically same as in (6) by the theory of asymptotically autonomous systems (Chavez & Thieme, 1995). The disease-free equilibrium point of system (1) still being a disease-free equilibrium point of system (6) with reduced dimension, i.e. $E_0 = (N_{h1}^*, 0, 0, N_{h2}^*, 0, 0)$. Therefore, in what following, we study system (6) to see the infective population instead.

3.1 Existence of equilibria

System (6) has a disease-free equilibrium, E_0 and a co-endemic equilibrium of the form $E^* = (S_{h1}^*, I_{h1}^*, I_{v1}^*, S_{h2}^*, I_{h2}^*, I_{v2}^*)$, i.e. $I_{h1}^* \neq 0$ and $I_{h2}^* \neq 0$ corresponding to disease persistent in both patches. The co-endemic population in the form of I_{h2}^* is given by

$$S_{h1}^* = \frac{-c_2(I_{h2}^*)^2 + c_1 I_{h2}^* + c_0}{N_{h2}^*\mu_h\xi_1(\alpha_{12}+\alpha_{21}+\mu_h)};\ I_{v2}^* = \frac{I_{h2}^* b_v \Lambda_{v2}/\mu_v}{I_{h2}^* b_v + N_{h2}^*\mu_v};\ I_{v1}^* = \frac{I_{h1}^* b_v \Lambda_{v1}/\mu_v}{I_{h1}^* b_v + N_{h1}^*\mu_v} \tag{7}$$

$$S_{h2}^* = \frac{(\gamma+\mu_h)(\gamma+\beta_{12}+\beta_{21}+\mu_h)(I_{h2}^* b_v + N_{h2}^* \mu_v)}{(\alpha_{12}+\alpha_{21}+\mu_h)\mu_h \xi_1}\left(-I_{h2}^*\alpha_{12} + \frac{\mu_h \beta_{12} N_{h2}^* (\alpha_{12}+\alpha_{21}+\mu_h)}{(\gamma+\mu_h)(\gamma+\beta_{12}+\beta_{21}+\mu_h)}\right)$$; and

$$I_{h1}^* = \frac{I_{h2}^*}{\xi_1}\left(\frac{\xi_2 \mu_v R_0^{(2)}(\mu_h+\gamma)}{\alpha_{12}+\alpha_{21}+\mu_h} + b_v(\gamma+\beta_{21}+\mu_h)\right)\left(I_{h2}^* - \frac{N_{h2}^*\mu_v(\alpha_{12}+\alpha_{21}+\mu_h)\left(R_0^{(2)}(\mu_h+\gamma) - (\beta_{21}+\mu_h+\gamma)\right)}{\xi_2(\mu_h+\gamma)\mu_v R_0^{(2)} + b_v(\alpha_{12}+\alpha_{21}+\mu_h)(\beta_{21}+\mu_h+\gamma)}\right)$$

where $c_1 \in \mathbb{R}$,

$$\xi_1 = N_{h2}^*\beta_{12}\mu_v + \left(b_v + \frac{R_0^{(2)}\mu_v(\mu_h+\gamma)}{\alpha_{12}+\alpha_{21}+\mu_h}\right)\left(\beta_{12} - \frac{\alpha_{12}R_0^{(2)}(\mu_h+\gamma)^2\mu_v}{\mu_h\left(b_v(\alpha_{12}+\alpha_{21}+\mu_h) + R_0^{(2)}(\mu_h+\gamma)\mu_v\right)}\right) I_{h2}^*,$$

$$\xi_2 = \frac{\gamma\alpha_{12}}{\mu_h} + \gamma + \alpha_{12} + \beta_{21} + \mu_h$$

$$c_2 = b_v(\gamma+\mu_h)(\gamma+\beta_{12}+\beta_{21}+\mu_h)(b_v\Lambda_{v2}/\mu_v + (\alpha_{21}+\mu_h)N_{h2}^*)$$

$$c_0 = (N_{h2}^*)^2\beta_{12}\mu_h\mu_v N_{h1}^*(\alpha_{12}+\alpha_{21}+\mu_h).$$

The I_{h2}^* is a root of a polynomial third order below

$$\delta(I_{h2}) = a_3(I_{h2})^3 + a_2(I_{h2})^2 + a_1 I_{h2} + a_0, \quad \text{where } a_3 > 0, a_2, a_1 \in \mathbb{R} \tag{8}$$

and $a_0 = N_{h1}^*(N_{h2}^*)^2\beta_{12}\mu_h^2\mu_v(\mu_h+\alpha_{12}+\alpha_{21})^2\left(1 - \left(R_0^{(1)}\eta_1 + R_0^{(2)}\eta_2\right) + \frac{R_0^{(1)}R_0^{(2)}(\gamma+\mu_h)}{\gamma+\mu_h+\beta_{12}+\beta_{21}}\right)$.

Theorem 2. *If* $R_0 > 1$ *then Equation* (8) *has minimal one positive root,* I_{h2}^*.

Proof. Formula R_0 in Eq. (4) also can be written as follows

$$R_0 = \frac{1}{2}\left(R_0^{(1)}\eta_1 + R_0^{(2)}\eta_2 + \sqrt{\left(R_0^{(1)}\eta_1 + R_0^{(2)}\eta_2\right)^2 - 4\frac{R_0^{(1)}R_0^{(2)}(\mu_h+\gamma)}{(\beta_{12}+\beta_{21}+\mu_h+\gamma)}}\right).$$

The other forms of $R_0 > 1$ based on the above formula is

$$-\frac{R_0^{(1)}R_0^{(2)}(\mu_h+\gamma)}{(\beta_{12}+\beta_{21}+\mu_h+\gamma)} > 1 - \left(R_0^{(1)}\eta_1 + R_0^{(2)}\eta_2\right).$$

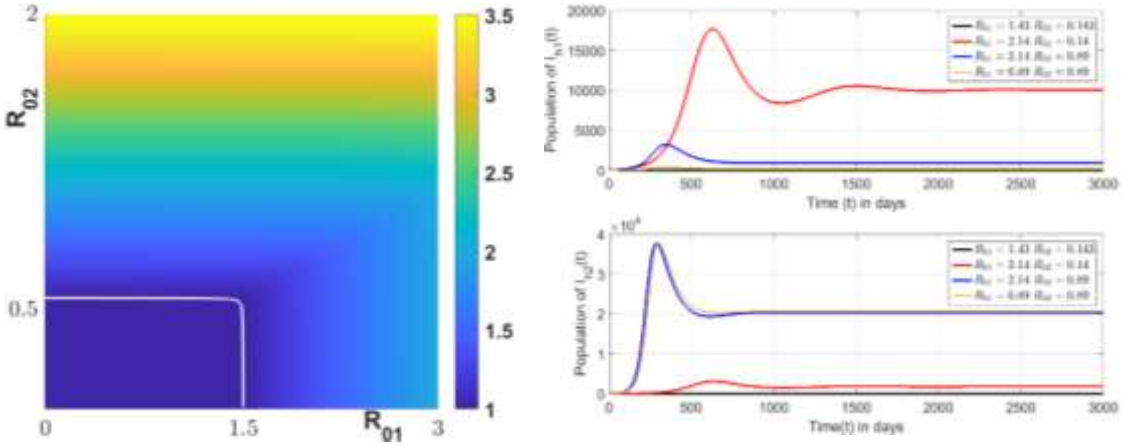
Condition $R_0 > 1$ equivalent with $a_0 < 0$. This means that $R_0 > 1$ becomes the sufficient condition for the existence of a minimal positive root, I_{h2}^* of the polynomial (8). ■

Let $f(R_{01}) = \frac{N_{h2}^*\left(-\Lambda_{h1}/\mu_h(\gamma+\beta_{21}+\mu_h)R_{01} + N_{h1}^*(\gamma+\beta_{12}+\beta_{21}+\mu_h)\right)}{\Lambda_{h2}/\mu_h\left(-\Lambda_{h1}/\mu_h(\mu_h+\gamma)R_{01} + N_{h1}^*(\mu_h+\gamma+\beta_{12})\right)}$, for $R_{01} \in \left[0, \frac{N_{h1}^*}{N_{h1}}\frac{\gamma+\beta_{12}+\beta_{21}+\mu_h}{\mu_h+\gamma+\beta_{21}}\right]$. The level set of R_0 using parameters as shown in Table 1 can be seen in *Figure 2*(a). Along with the white curve, R_0 equal one, or $f(R_{01})$. This curve becomes a boundary for diseases dies out or disease persistence in both patches. *Figure 2* (b) shows the behavior of I_{h1} and I_{h2} using the same set migration parameter and confirms the dynamical results that R_0 becomes the only threshold for determining the long term behavior of the system (6).

Using the set mobility parameter as above, the region in $R_{01} \in [0,1]$ and $R_{02} \in [0.52,1]$ will make the disease persist in both patches even though if no migration occurs disease dies out in both patches. On the contrary, in region $R_{01} \in [1,1.5]$ and $R_{02} \in [0,1]$ migration make the disease vanish in both patched even though if no migration occurs the disease persists in Patch 2. This means that host migration can be a control strategy for disease eradication but also can be a driven force for disease spread out. The migration parameter must be chosen very wisely for this goal.

Table 1. Parameters Values

Notation	Parameter Description	Value	Ref
b_v	Transmission probability from host to vector	0.05	(Esteva & Vargas, 1998)
b_h	transmission probability from vector to host	0.5 x 0.75	
γ	the recovery rate of an infected host	1/7	
μ_v	The natural death rate of vector	1/14	
μ_h	The natural death rate of host	1/(65x365)	
Λ_{h1}	The recruitment rate of a host in Patch 1	$10^6/(65x365)$	by simulation
Λ_{h2}	The recruitment rate of a host in Patch 2	$10^6/(65x365)$	
Λ_{v1}	The recruitment rate of a host in Patch 2	$2/14 \times 10^6$	
Λ_{v2}	The recruitment rate of a host in Patch 2	$8/14 \times 10^5$ $8/14 \times 10^4$	



(a) Level set of R_0 (b) The solution of I_{h1} and I_{h2}

Figure 2. Simulation using the set of migration parameter as $\{\alpha_{12} = 0.01, \alpha_{21} = 0.035, \beta_{21} = 0.02, \beta_{12} = 0.001\}$

3.2 Unidirectional Infected Host Migration

We want to show the importance of the infected host transfer to an isolated patch. Assumed endemic happen in Patch 1 which is a central city. Since we want to reduce the infected host population in Patch 1, the government will transfer the infected host to Patch 2 which assumed to be an isolated area. Infected people will be quarantined in Patch 2 so they cannot enter Patch 1 due to a perfect border screening of infected people from isolated Patch 2. For this scenario, the migration parameter β_{21} becomes equal to zeros and using Eq.(4), the basic reproduction ratio R_0 becomes $R_0 = max\left\{R_0^{(1)}\eta_1, R_0^{(2)}\right\}$. Condition $R_0^{(1)}\eta_1 < 1$ and $R_0^{(2)} < 1$ equivalent with $R_0 < 1$. This means E_0 is globally asymptotically stable if and only if $R_0^{(1)}\eta_1 < 1$ and $R_0^{(2)} < 1$. Setting $\beta_{21} = 0$ implied that $a_0 < 0$ if and only if

$$\left(R_0^{(2)} - 1\right)\left(R_0^{(1)}\eta_1 - 1\right) < 0.$$

Let $\bar{I}_{h2}^{**} = \frac{\mu_h(\mu_h+\alpha_{12}+\alpha_{21})(N_{h2}^*)^2\mu_v\left(R_0^{(2)}-1\right)}{b_v(\Lambda_{v2}b_h(\alpha_{12}+\mu_h)/\mu_v+\mu_h(\mu_h+\alpha_{12}+\alpha_{21})N_{h2}^*)}$. Substitute $\beta_{21} = 0$ to $\delta(I_{h2})$ of Eq. (8) can be factorized as

$$\delta(I_{h2}) = (I_{h2} - \bar{I}_{h2}^{**})(b_v^2(\gamma + \mu_h)k_2 I_{h2} + k_1 I_{h2} + k_0),$$

where $k_2 > 0$, $k_1 \in \mathbb{R}$, and $k_0 = (N_{h2}^*)^2\beta_{12}\mu_h\mu_v^2(N_{h1}^*)^2(\mu_h + \alpha_{12} + \alpha_{21})\left(1 - R_0^{(1)}\eta_1\right)$. Thus, if $R_0^{(2)} > 1$ we have a minimal a positive root of $\delta(I_{h2})$ denoted by $\bar{I}_{h2}^{**}$ with corresponding state as follow

$\bar{S}_{h1}^{**} = \frac{\mu_h(\mu_h+\alpha_{12}+\alpha_{21})N_{h1}^*b_v+\alpha_{21}\mu_v(\gamma+\mu_h)N_{h2}^*}{b_v(\mu_h+\alpha_{12}+\alpha_{21})(\mu_hN_{h2}^*(\mu_h+\alpha_{12}+\alpha_{21})+\Lambda_{v2}b_h(\alpha_{12}+\mu_h)/\mu_v)}$; $\bar{I}_{v2}^{**} = \frac{\bar{I}_{h2}^{**}\Lambda_{v2}b_v/\mu_v}{\bar{I}_{h2}^{**}b_v+N_{h2}^*\mu_v}$, $\bar{I}_{v1}^{**} = 0$,

$\bar{I}_{h1}^{**} = 0$, and $\bar{S}_{h2}^{**} = \frac{N_{h2}^*(\mu_h(\mu_h+\alpha_{12}+\alpha_{21})b_v+\mu_v(\mu_h+\alpha_{12})(\gamma+\mu_h))}{b_v(\mu_hN_{h2}^*(\mu_h+\alpha_{12}+\alpha_{21})+\Lambda_{v2}b_h(\alpha_{12}+\mu_h)/\mu_v)}$.

We called this condition as a boundary endemic equilibria $E_1 = (\bar{S}_{h1}^{**}, 0, 0, \bar{S}_{h2}^{**}, \bar{I}_{h2}^{**}, I_{v2}^*)$.

If $k_0 < 0$ then we have another a positive root $I_{h2}^* = \frac{-k_1+\sqrt{k_1^2-4b_v^2(\gamma+\mu_h)k_2k_0}}{2b_v^2(\gamma+\mu_h)k_2}$. Condition $k_0 < 0$ equivalent with $R_0^{(1)}\eta_1 > 1$, and this means that if $R_0^{(1)}\eta_1 > 1$ then a positive $I_{h2}^* = \frac{-k_1+\sqrt{k_1^2-4b_v^2(\gamma+\mu_h)k_2k_0}}{2b_v^2(\gamma+\mu_h)k_2}$ which correspond with an interior endemic equilibrium $E^* = (S_{h1}^*, I_{h1}^*, I_{v1}^*, S_{h2}^*, I_{h2}^*, I_{v2}^*)$ exist.

The stability analysis of E_1 and E^* is quite complicated. Thus, the below theorem applied in a specific scenario when healthy people are isolated in their patch, i.e. $\alpha_{12} = 0$ and $\alpha_{21} = 0$. The stability of E^* will be shown by the numerical results.

Theorem 3. A boundary endemic equilibria $E_1 = (\bar{S}_{h1}^{**}, 0, 0, \bar{S}_{h2}^{**}, \bar{I}_{h2}^{**}, I_{v2}^*)$ is locally asymptotically stable if and only if $R_0^{(2)} > 1$ and $R_0^{(1)}\eta_1 < 1$.

Proof: It is clear if $R_0^{(2)} > 1$ then $\bar{I}_{h2}^{**}$ has a positive value. The local stability of E_1 can be examined by the linearizing system (6) around E_1. The characteristic polynomial of Jacobian from the system (6) when all mobility is zeros, except $\beta_{12} \neq 0$ and evaluated in E_1 can be written as follows.

$$p(\lambda) = (\lambda + \mu_h)(K_3\lambda^3 + K_2\lambda^2 + K_1\lambda + K_0)\,\delta(\lambda) \quad (9)$$

where $\delta(\lambda) = \left(\lambda^2 + (\mu_h + \mu_v + \gamma + \beta_{12})\lambda + \mu_v(\gamma + \mu_h + \beta_{12})\left(1 - R_0^{(1)}\eta_1\right)\right)$,

$K_3 = N_{h2}(\gamma\mu_v + b_v\mu_h + \mu_h\mu_v)(\Lambda_{h2} + \Lambda_{v2}b_h/\mu_v)$,

$K_2 = \mu_h\left(R_0^{(2)}\mu_v(\gamma + \mu_h)(\Lambda_{h2}/\mu_h(\gamma + 3\mu_h + 2\mu_v) + \Lambda_{v2}b_h/\mu_v) + \Lambda_{h2}(\gamma + 2\mu_h)b_v\right) +$
$\Lambda_{h2}\mu_h\mu_v b_v R_0^{(2)} + \mu_v(\gamma + \mu_h)b_h(\gamma + \mu_h + \mu_v)\Lambda_{v2}/\mu_v + \Lambda_{h2}\mu_v(\gamma + \mu_h)^2$,

$K_1 = b_v(\Lambda_{h2} + \Lambda_{v2}b_h/\mu_v)\left(R_0^{(2)}\Lambda_{h2}\mu_v + b_h(\gamma + \mu_h + \mu_v)\Lambda_{v2}/\mu_v + \Lambda_{h2}(\gamma + \mu_h)\right) +$
$(\Lambda_{h2}/\mu_h)^2\mu_v(\gamma + \mu_h)(\gamma\mu_v + b_v\mu_h + \mu_h\mu_v)\left(R_0^{(2)} - 1\right)$, and

$K_0 = (\gamma + \mu_h)(\gamma\mu_v + b_v\mu_h + \mu_h\mu_v)(\Lambda_{h2} + \Lambda_{v2}b_h/\mu_v)\Lambda_{h2}\mu_v\left(R_0^{(2)} - 1\right)$

All eigenvalues of the last polynomial third order have the negative real part if they satisfy Routh-Hurwitz Criteria, such that $K_i > 0$ for i=0,1,2,3 with $K_2K_1 > K_3K_0$. For $R_0^{(2)} > 1$, we obtain $K_i > 0$ for all i. Thus all roots of Eq. *(9)*(9) have negative real parts if and only if $R_0^{(2)} > 1$ and $R_0^{(1)}\eta_1 < 1$, which shows that E_1 is locally asymptotically stable in this condition. ■

Remark 4. For $R_0^{(2)} < 1$ then E_1 does not exist, since $\bar{I}_{h1} < 0$. Further, for $R_0^{(2)} > 1$ and $R_0^{(1)}\eta_1 > 1$ then $\delta(0) < 0$ and $\lim_{\lambda\to\infty}\delta(\lambda) = \infty$ when $\lambda \in \mathbb{R}$. Then, there exists $\lambda^* > 0$ such that $\delta(\lambda^*) = 0$ which proves instability of E_1 even though this boundary equilibrium exists.

Stability and existence diagram for this scenario in $\left(R_0^{(1)}\eta_1, R_0^{(2)}\right)$-plane can be seen in *Figure 3* which indicated a trans-critical bifurcation of the equilibrium point. For $R_0^{(2)} < 1$, the dynamical behavior of (6) can change twice when $R_0^{(1)}\eta_1 < 1$ varies from zeros to infinity; the condition goes to E_0 if $R_0^{(1)}\eta_1 < 1$, but if $R_0^{(1)}\eta_1 > 1$ then the solution of the system (6) goes to E^*. Besides, when $R_0^{(2)} > 1$, the dynamical behavior of (6) also change twice, $R_0^{(1)}\eta_1 < 1$ varies from zeros to infinity; the condition goes to E_1 if $R_0^{(1)}\eta_1 < 1$, but if $R_0^{(1)}\eta_1 > 1$ then the behavior of the system (6) go to E^*.

Since we want to see the effect of infected host transfer, we look β_{12} as the bifurcation parameter. To find trans-critical bifurcation, we set k_0 to be zero and solve the critical value of β_{12}, denoted by $\hat{\beta}_{12}$ is given by $\hat{\beta}_{12} = (R_{01} - 1)(\gamma + \mu_h)$ where R_{01} is the basic reproduction ratio of patch 1 before any migration or transfer occurs between two-patch, i.e. $R_{01} = \frac{\Lambda_{v1}b_h b_v\mu_h}{\Lambda_{h1}(\gamma+\mu_h)\mu_v^2}$. Since β_{12} is the migration rate of the infected host, then $0 < \beta_{12} < 1$. This implied that a trans-critical bifurcation occurs when $1 < R_{01} < 1 + \frac{1}{\gamma+\mu_h}$.

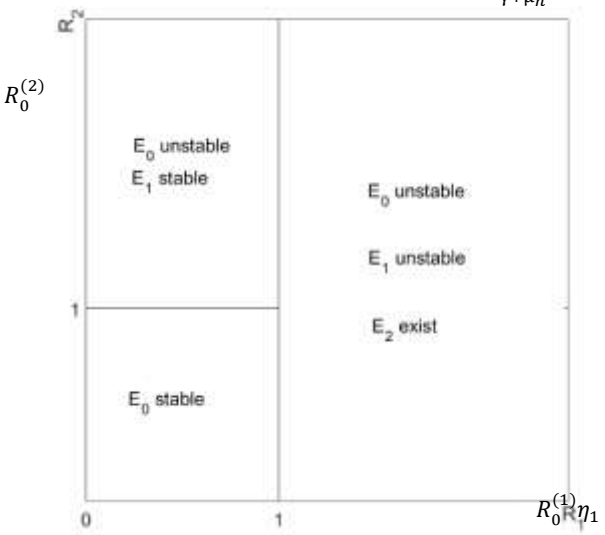
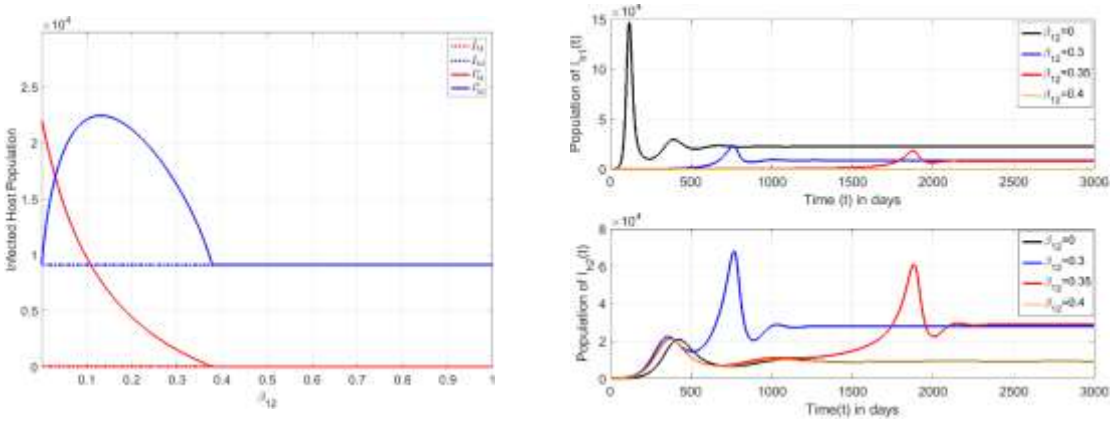


Figure 3. Stability and existence diagram when only $\beta_{12} \neq 0$

This strategy can be used if the government want to eradicate or reduce the number of infected hosts in Patch 1 by transferring infected host from Patch 1 to Patch 2 as an isolated patch. There is two probability of Patch 2 before transferring occurs, a free infection patch or a less endemic patch than Patch 1. Figure 4 illustrated the dynamical behavior if the isolated patch less endemic than the small one. Figure 5 illustrated the dynamical behavior if the isolated patch is free from disease.

Figure 4(a) is associated with the trans-critical bifurcation diagram using a set of parameters as shown in Table 1 with $N_{v1} = 8 \times 10^5$. This figure shows the solution of system (6) goes to co-endemic equilibria E^* if $\beta_{12} < \hat{\beta}_{12} = 0.378$, however, the dynamic behavior of the system (6) goes to E_1 if $\beta_{12} > \hat{\beta}_{12}$. The number of infected hosts in each patch, i.e. I_{h1} and I_{h2} using different values of β_{12} shown in Figure 4(b) confirm the analytical results. A trans-critical bifurcation and infected host number in Figure 4 shows the effect of transferring infected hosts from Patch 1 to Patch 2.

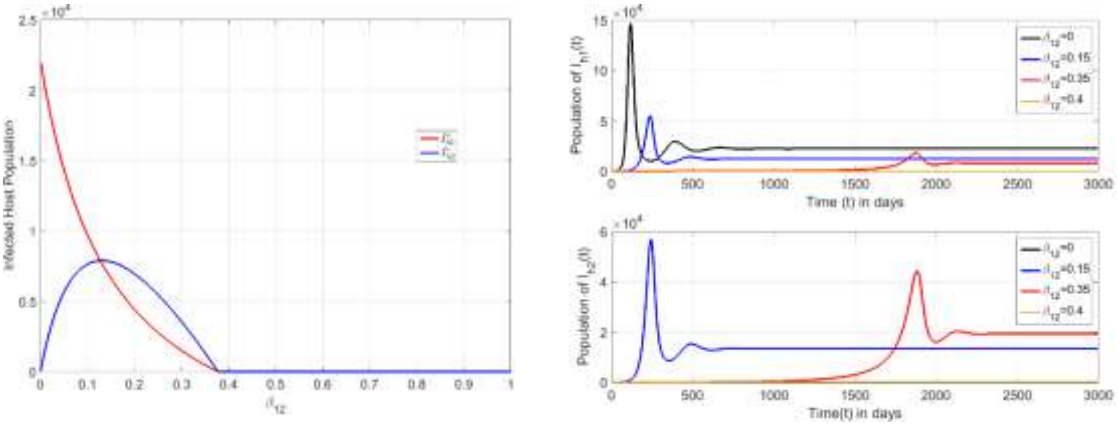
At the beginning of the disease spreading, both patches are endemic but Patch 1 is more endemic than Patch 2, since $R_{01} = 3.5$ and $R_{02} = 1.4$. The increasing number of infected hosts who will be transferred from Patch 1 to Patch 2 less than 37.8% can reduce the number of infected hosts in Patch 1 and an increasing number of I_{h2}. This is reasonable due to the infected host transferring from Patch 1 to Patch 2. The increasing number of infected hosts transferring as many as 37.8% cause Patch 1 free from the disease and the disease persist in Patch 2. However, transferring infected host more than 37.8% does not give any effect, since the Patch 1 free from the disease and Patch 2 become endemic.



(a) Value I_{h1}^* and I_{h2}^* as a function of β_{12} (b) The solution of $I_{h1}(t)$ and $I_{h2}(t)$

Figure 4. Simulation of only $\beta_{12} \neq 0$, $R_{01} = 3.5$, and $R_{02} = 1.4$

Figure 5 shows a scenario when Patch 1 is the only endemic patch before transferring happens. Patch 2 is a free disease area. For several reasons, the government of Patch 1 wants to isolate the infected people in Patch 2 by transferring them to Patch 2. If they transfer less than 38% infected host, the condition gets worse since the disease will be persistent in Patch 2. However, if they transfer more than 38% infected host then the disease will be eradicated from both patches.



(a) Value I_{h1}^* and I_{h2}^* as a function of β_{12} (b) The solution of $I_{h1}(t)$ and $I_{h2}(t)$

Figure 5. Simulation of only $\beta_{12} \neq 0$, $R_{01} = 3.5$, and $R_{02} = 0.7$

3.3 Healthy Host Migration

Assume that all infected populations cannot migrate to another patch since medical screening at the border. Introducing some new notation to make the calculation simpler, i.e.

$Q_1 = \frac{b_v\mu_h(\mu_h+\alpha_{12}+\alpha_{21})}{\mu_v(\gamma+\mu_h)}, R_1 = \frac{R_0^{(1)}\left(Q_1+R_0^{(2)}\frac{\Lambda_{h1}}{N_{h1}^*}+\alpha_{21}\frac{N_{h2}^*}{N_{h1}^*}\right)}{R_0^{(2)}(\alpha_{12}+\mu_h)+Q_1}, R_2 = \frac{R_0^{(2)}\left(Q_1+R_0^{(1)}\frac{\Lambda_{h2}}{N_{h2}^*}+\alpha_{12}\frac{N_{h1}^*}{N_{h2}^*}\right)}{R_0^{(1)}(\alpha_{21}+\mu_h)+Q_1}$, and $\varrho = \frac{(\gamma+\mu_h)N_{h1}^*}{b_v}\left(R_0^{(1)}R_0^{(2)}\mu_v(\gamma+\mu_h)+b_v\left(R_0^{(1)}(\alpha_{21}+\mu_h)+R_0^{(2)}(\alpha_{12}+\mu_h)\right)+b_vQ_1\right)$.

Let $I_{h2}^* = \frac{N_{h1}^*N_{h2}^*\mu_v(\gamma+\mu_h)\left(R_0^{(1)}(\alpha_{21}+\mu_h)+Q_1\right)(R_2-1)}{b_v\varrho}$.

The only healthy host migration indicates that $\beta_{12} = 0$ and $\beta_{21} = 0$, then polynomial (8), $\delta(I_{h2})$, can be rewritten as $\delta(I_{h2}) = b_v(\gamma+\mu_h)I_{h2}(I_{h2}-\bar{I}_{h2}^{**})(I_{h2}-I_{h2}^*)$. This means that $\delta(I_{h2})$ has three roots, $\bar{I}_{h2}^* = 0$, $\bar{I}_{h2}^{**}$, and I_{h2}^*. Condition $I_{h2} = \bar{I}_{h2}^* = 0$ correspond with a boundary equilibrium $E_1 = (\bar{S}_{h1}^*, \bar{I}_{h1}^*, \bar{I}_{v1}^*, \bar{S}_{h2}^*, 0, 0)$ where

$\bar{S}_{h1}^* = \frac{N_{h1}^*Q_1+N_{h2}^*(\alpha_{21}+\mu_h)}{R_0^{(1)}(\alpha_{21}+\mu_h)+Q_1}, \bar{I}_{h1}^* = \frac{N_{h1}^*\mu_h(\mu_h+\alpha_{12}+\alpha_{21})\left(R_0^{(1)}-1\right)}{\left((\alpha_{21}+\mu_h)R_0^{(1)}+Q_1\right)(\gamma+\mu_h)}, \bar{I}_{v1}^* = \frac{\bar{I}_{h1}^*\Lambda_{v1}b_v/\mu_v}{\bar{I}_{h1}^*b_v+N_{h1}^*\mu_v}$, and $\bar{S}_{h2}^* = \frac{\Lambda_{h2}R_0^{(1)}+N_{h2}^*Q_1+N_{h1}^*\alpha_{12}}{(\alpha_{21}+\mu_h)R_0^{(1)}+Q_1}$.

Further, condition $I_{h2} = \bar{I}_{h2}^{**}$ correspond with $E_2 = (\bar{S}_{h1}^{**}, 0, 0, \bar{S}_{h2}^{**}, \bar{I}_{h2}^{**}, \bar{I}_{v2}^{**})$ where

$$\bar{S}_{h1}^{**} = \frac{N_{h1}^*Q_1+\alpha_{21}N_{h2}^*}{(\mu_h+\alpha_{12}+\alpha_{21})N_{h2}^*\left(Q_1+R_0^{(2)}(\alpha_{12}+\mu_h)\right)}, \bar{S}_{h2}^{**} = \frac{Q_1+\alpha_{12}+\mu_h}{Q_1+R_0^{(2)}(\alpha_{12}+\mu_h)}, \bar{I}_{v2}^{**} = \frac{\bar{I}_{h2}^{**}\Lambda_{v2}b_v/\mu_v}{\bar{I}_{h2}^{**}b_v+N_{h2}^*\mu_v}.$$

The last possibility condition $I_{h2} = I_{h2}^*$ correspond with a co-endemic equilibrium $E^* = (S_{h1}^*, I_{h1}^*, I_{v1}^*, S_{h2}^*, I_{h2}^*, I_{v2}^*)$, where

$S_{h1}^* = \frac{N_{h1}^*Q_1+\alpha_{21}N_{h2}^*}{(\mu_h+\alpha_{12}+\alpha_{21})N_{h2}^*\left(Q_1+R_0^{(2)}(\alpha_{12}+\mu_h)\right)}, S_{h2}^* = \frac{Q_1+\alpha_{12}+\mu_h}{Q_1+R_0^{(2)}(\alpha_{12}+\mu_h)}, I_{h1}^* = \frac{N_{h1}^*\mu_v\left(R_0^{(2)}(\alpha_{12}+\mu_h)+Q_1\right)(R_1-1)}{(\alpha_{21}+\mu_h)R_0^{(1)}b_v+(\alpha_{12}+\mu_h)R_0^{(2)}b_v+R_0^{(1)}R_0^{(2)}\mu_v(\gamma+\mu_h)+Q_1b_v}, I_{v1}^* = \frac{I_{h1}^*\Lambda_{v1}b_v/\mu_v}{I_{h1}^*b_v+N_{h1}^*\mu_v}, I_{v2}^* = \frac{I_{h2}^*\Lambda_{v2}b_v/\mu_v}{I_{h2}^*b_v+N_{h2}^*\mu_v}$.

This co-endemic equilibrium exists if $R_1 > 1$ and $R_2 > 1$.

Theorem 5. An equilibrium E_1 is locally asymptotically stable if $R_0^{(1)} > 1$ and $R_2 < 1$.

Proof. The local stability of E_1 can be examined by linearizing the system (6) around E_1. A characteristic equation of the Jacobian evaluated in E_1 can be written as

$$\left(\lambda^2+(\mu_v+\gamma+\mu_h)\lambda+\mu_v(\mu_h+\gamma)(1-R_2)\right)(a_4\lambda^4+a_3\lambda^4+a_2\lambda^4+a_1\lambda+a_0) = 0 \qquad (10)$$

with $a_i > 0$ for $i = 1,2,3,4$. All eigenvalues have the negative real part if $R_0^{(2)} < 1$ and the last fourth-order polynomial satisfy the Routh-Hurwitz criteria (Allen, 2007), such that $a_i > 0$ for $i = 1,2,3,4$ with $a_3a_2a_1 > a_1^2+a_3^2a_0$. If $R_0^{(1)} < 1$, we obtain $a_i > 0$ for i = 1,2,3,4. Thus, all the eigenvalues of the polynomial (10) have

negative real parts if and only if $R_2 < 1$ which shows the locally asymptotically stable behavior of E_1.

■

Theorem 6. An equilibrium E_2 is locally asymptotically stable if $R_0^{(2)} > 1$ and $R_1 < 1$.

Proof. The characteristic equation of Jacobian from the system (6) which evaluated in E_2 can be written as follows

$$\left(\lambda^2 + (\gamma + \mu_h + \mu_v)\lambda + \mu_v(\mu_h + \gamma)(1 - R_1)\right)(\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0) = 0$$

where $A_3, A_2, A_1 \in \mathbb{R}^+$ and $A_0 = N_{h2}^{*2}\mu_v\mu_h(\alpha_{12} + \alpha_{21} + \mu_h)(\mu_h + \gamma)\left(R_0^{(2)} - 1\right)$. These four eigenvalues have the negative real part if they satisfy the Routh-Hurwitz Criteria (Allen, 2007), such that $A_i > 0$ for $i = 1,2,3,4$ with $A_3A_2A_1 > A_1^2 + A_3^2A_0$. For $R_0^{(2)} > 1$, we obtain $A_i > 0$ for $i = 1,2,3,4$. Thus, all eigenvalues have negative real parts if and only if $R_1 > 1$ which implied that E_2 is locally asymptotically stable.

■

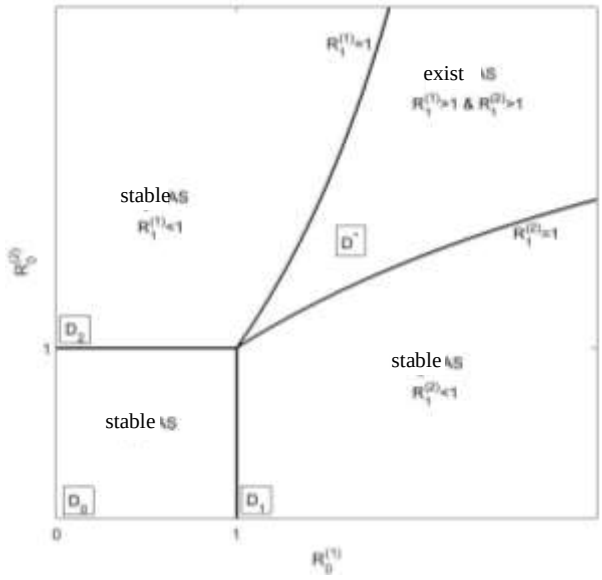


Figure 6. Stability diagram in $\left(R_0^{(1)}, R_0^{(2)}\right)$-plane

It is easy to see that $R_0^{(1)}$ and $R_0^{(2)}$ are a threshold for disease-free steady-state stability and threshold for both boundary endemic equilibrium existence, E_1 and E_2. The basic reproduction number of (6) when all infected host isolated is $R_0 = max\left\{R_0^{(1)}, R_0^{(2)}\right\}$. Observing Theorem 5-6 implied that R_0 cannot determine the dynamics of the system (6) completely. R_0 can only determine if the disease dies out in total population including two patches. According to Theorem 5-6, another threshold R_1 and R_2 are also necessary to obtain the existence of co-endemic equilibrium E^*.

From Figure 6, it can be seen that this boundary separates the parameter plane into four distinct regions, D_0, D_1, D_2, and D^*. In $D_0 = \left\{\left(R_0^{(1)}, R_0^{(2)}\right) | R_0^{(1)} \le 1, R_0^{(2)} \le 1\right\}$, E_0 is locally asymptotically stable; E_1 is locally asymptotically stable in $D_1 = \left\{\left(R_0^{(1)}, R_0^{(2)}\right) | R_0^{(1)} > 1, R_2 \le 1\right\}$; E_2 is locally asymptotically stable in $D_2 = \left\{\left(R_0^{(1)}, R_0^{(2)}\right) | R_1 \le 1, R_0^{(2)} > 1\right\}$; and E^* is locally asymptotically stable in the region $D^* = \left\{\left(R_0^{(1)}, R_0^{(2)}\right) | R_1 > 1, R_2 > 1\right\}$.

Since $R_1 = 1$ is equivalent to $R_0^{(2)} = \frac{(N_{h1}^*Q_1 + N_{h2}^*\alpha_{21})R_0^{(1)} - N_{h1}^*Q_1}{N_{h1}^*(\alpha_{12}+\mu_h) - R_0^{(1)}\Lambda_{h1}}$. Note that $R_0^{(1)} = \frac{N_{h1}^*(\alpha_{12}+\mu_h)}{\Lambda_{h1}} > 1$ is the asymptotic line of the curve $R_1 = 1$. Similarly, $R_2 = 1$ has the asymptotic line $R_0^{(2)} = \frac{N_{h2}^*(\alpha_{21}+\mu_h)}{\Lambda_{h2}} > 1$. Thus, it is not difficult to see the following facts from Figure 6: when $1 < R_0^{(1)} < \frac{N_{h1}^*(\alpha_{12}+\mu_h)}{\Lambda_{h1}}$, the dynamical behavior of (6) can change twice when $R_0^{(2)}$ varies from zero to infinity. E_1 is stable for $0 < R_0^{(2)} \le \frac{N_{h2}^*\left(Q_1 + R_0^{(1)}(\alpha_{21}+\mu_h)\right)}{R_0^{(1)}\Lambda_{h2} + N_{h1}^*\alpha_{12} + N_{h2}^*Q_1}$; E^* is

stable for $\frac{N_{h2}^{*}\left(Q_1+R_0^{(1)}(\alpha_{21}+\mu_h)\right)}{R_0^{(1)}\Lambda_{h2}+N_{h1}^{*}\alpha_{12}+N_{h2}^{*}Q_1} < R_0^{(2)} < \frac{(N_{h1}^{*}Q_1+N_{h2}^{*}\alpha_{21})R_0^{(1)}-N_{h1}^{*}Q_1}{-R_0^{(1)}\Lambda_{h1}+N_{h1}^{*}(\alpha_{12}+\mu_h)}$; E_2 is stable for $R_0^{(2)} \geq \frac{\left(N_{h1}^{*}Q_1+N_{h2}^{*}\alpha_{21}\right)R_0^{(1)}-N_{h1}^{*}Q_1}{-R_0^{(1)}\Lambda_{h1}+N_{h1}^{*}(\alpha_{12}+\mu_h)}$.

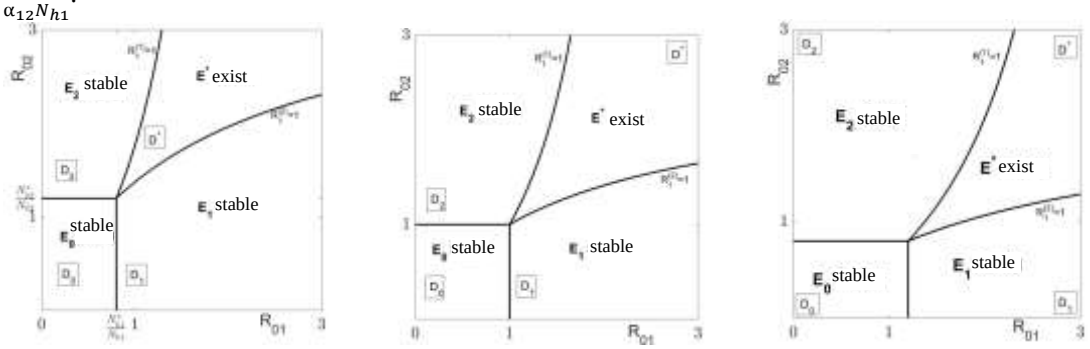
Further, if $R_0^{(1)} < 1$ or $R_0^{(1)} > \frac{N_{h1}^{*}(\alpha_{12}+\mu_h)}{\Lambda_{h1}}$, the dynamical behavior of (6) can change only once when $R_0^{(2)}$ vary from zeros to infinity. Similarly, when $1 < R_0^{(2)} < \frac{N_{h2}^{*}(\alpha_{12}+\mu_h)}{\Lambda_{h2}}$, the dynamical behavior of (6) can all change twice when $R_0^{(1)}$ varies from zeros to infinity; when $R_0^{(2)} < 1$ or $R_0^{(2)} > \frac{N_{h2}^{*}(\alpha_{12}+\mu_h)}{\Lambda_{h2}}$, it can change only once.

Note $N_{hi} = \frac{\Lambda_{hi}}{\mu_h}, (i = 1,2)$ represents the size of the population in patch i at the equilibrium in the absence of disease and mobility, while the basic reproduction number in patch i is given by R_{0i}. The direct calculation shows that

$$R_{0i} - R_0^{(i)} = \frac{b_h b_v N_{vi}\left(N_{hj}\alpha_{ji} - N_{hi}\alpha_{ij}\right)}{(\mu_h + \alpha_{12} + \alpha_{21})N_{hi}\mu_v(\mu_h + \gamma)N_{hi}^{*}}, i, j = 1,2, i \neq j$$

This means that $R_{0i} > R_0^{(i)}$ if and only if $N_{hj}\alpha_{ji} > N_{hj}\alpha_{ij}$. It implies that under the condition that mobility individuals go out from patch i to j is greater than mobility individuals go to patch i from j, the basic reproductions number in patch i in the absence of dispersal is less than in the presence of dispersal. So, increasing the mobility of healthy people (susceptible and recovered) from patch j to i may be a helpful control strategy for the dengue disease eradicated in patch i.

Next, we will figure out the stability region of equilibria in (R_{01}, R_{02})-plane as shown in Figure 7. Curve $R_1 = 1$ and $R_2 = 1$ will be intersect in $(R_{01}, R_{02}) = \left(\frac{N_{h1}^{*}}{N_{h1}}, \frac{N_{h2}^{*}}{N_{h2}}\right)$. This gives us a different illustration that depends on ratio $\frac{\alpha_{21}N_{h2}}{\alpha_{12}N_{h1}}$.



(a) $\alpha_{21}N_{h2} < \alpha_{12}N_{h1}$ (b) $\alpha_{21}N_{h2} = \alpha_{12}N_{h1}$ (c) $\alpha_{21}N_{h2} > \alpha_{12}N_{h1}$

Figure 7. Stability Diagram in (R_{01}, R_{02}) −plane

Let R_{01} and R_{02} before mobility occurs are given. We want to see the effect of healthy host mobility only in the dengue spread out. The only threshold for interior endemic equilibrium was R_1 and R_2. Using a similar set value of the parameter, and plot $R_1 = 1$ and $R_2 = 1$ in $(\alpha_{12}, \alpha_{21})$-parameter plane gives us stability region as shown in Figure 7.

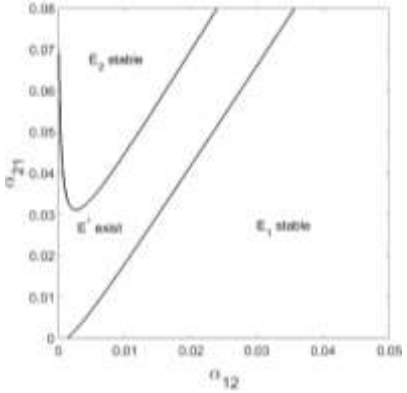


Figure 8. Stability and existence diagram in $(\alpha_{12}, \alpha_{21})$- plane, setting $R_{01} = 3.5$ and $R_{02} = 1.4$

Figure 8 shows that changes in mobility parameter α_{12} and α_{21} contributed to the stability of endemic equilibrium. If we fix one parameter, called α_{21} equal to 0.035, the equilibrium can change third times along the

α_{12} axis, from E^* become E_2 to E^* then E_1 as shown in Figure 9. Otherwise, set a fixed α_{12} equal to 0.025, the equilibrium can change third times along the α_{21} axis, from E_1 becomes E^* then E_2.

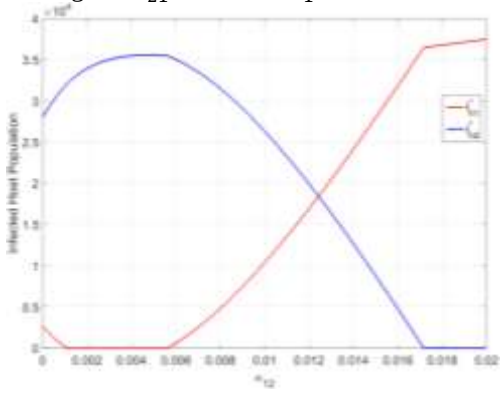


Figure 9. Trans-critical Bifurcation diagram of I_{h1}^* and I_{h2}^* setting $\alpha_{21} = 0.035$

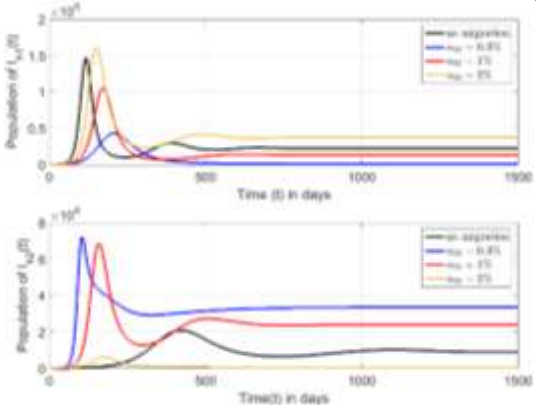


Figure 10. The solution of I_{h1} and I_{h2} with a fixed value of $\alpha_{21} = 0.035$

Figure 10 shows up different dynamic behavior of solution with a different value of α_{12}. Both patches isolated figure out by the black line, which both patches are endemic with Patch 1 more endemic than Patch 2 since $R_{01} = 3.5$, and $R_{02} = 1.4$. Setting $\alpha_{12} = 0.003$ and $\alpha_{21} = 0.035$, healthy host migration makes Patch 1 free from infection but disease persists in Patch 2. Increasing healthy host migration from Patch 1 to Patch 2 as much as $\alpha_{12} = 0.01$ increasing infected host of Patch 1 and decreasing infected host number in Patch 2, which makes both patches endemic. However, increasing α_{12} as much as 0.02 will make disease dies out in Patch 2 corresponding with the persistence of infection in Patch 1. This means that the control of people's mobility can be a control strategy for disease eradication in patchy environments. From this scenario also, we show that the mobility healthy human can both intensify and reduce the spread of the disease in patches.

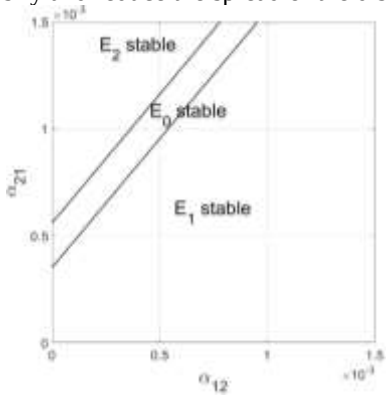


Figure 11 Stability and existence diagram in $(\alpha_{12}, \alpha_{21})$- plane, setting $R_{01} = 1.07$ and $R_{02} = 0.89$

In the second example, we consider a case where at the beginning Patch 1 endemic but Patch 2 not. Figure 11 shows that if only the healthy host migrated from Patch 1 to Patch 2, i.e. α_{21} equal with zeros, then there is no chance to make the disease vanish from both patches. Further, let α_{21} as small as 0.001 and $\alpha_{12} \in (0.000366, 0.000539)$ will result in the disease dies out in both patches. However, α_{12} less than 0.000366 makes a reverse condition, which Patch 1 free from infection but Patch 2 is not. This result is strengthened by the

dynamical behavior of the infected population as shown in Figure 12.

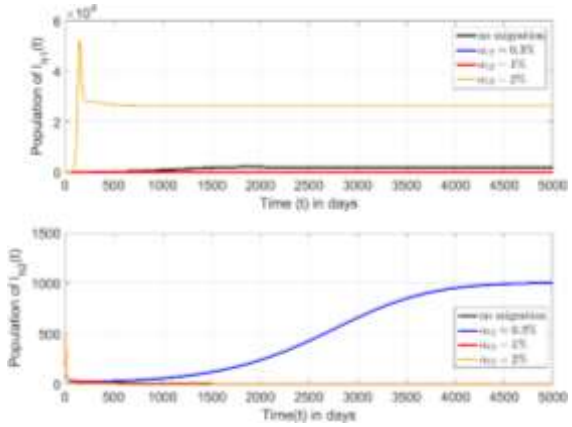


Figure 12. The solution of I_{h1} and I_{h2} using a fixed value of $\alpha_{21} = 0.001$

4. Conclusion

We have analyzed the two-patch model for host-vector transmission. In this model, we study the effect of host migration using a different rate for a healthy host and infected host. The disease transmission is assumed could happen when the host stays in a patch since infection during the transportation is negligible. The vector population cannot migrate to other patches due to the limited flying ability. When migration between patch occurs, we found that the R_0 is the threshold condition for the global stability of the disease-free state, $\tilde{R}_0 = \sqrt{R_0}$ is the basic reproductive number of the disease. If the one-directional host migration is not considered, either $\beta_{12} = 0$ or $\beta_{21} = 0$, then the basic reproduction number becomes $\widehat{R_0} = \max\left\{R_0^{(1)}\eta_1, R_0^{(2)}\eta_2\right\} < R_0$. It implied that the host migration would increase the basic reproduction number. If we only migrated infected host from Patch 1 to Patch 2, the infection level within Patch 1 will decrease and become a disease-free state as β_{12} increased. Moreover, the infection level within Patch 2 as increasing of β_{12} will be increasing at the beginning, going to its maximum level and then decrease to its endemic level as shown in Figure 4 and Figure 5.

The healthy people migration only shown leads to the disease spread both patches if R_1 and R_2 are greater than 1. The basic reproduction number of this model is $R_0 = \left\{R_0^{(1)}, R_0^{(2)}\right\}$, but from Theorem 5 and 6, R_0 cannot determine their dynamics completely since the threshold R_1 and R_2 are also necessary. From Figure 8 and Figure 11, we can see that the disease vanishes in a patch if the healthy host migration is suitable.

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