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Transmission Dynamics and Control Mechanisms of Vector-Borne Diseases with Active and Passive Movements Between Urban and Satellite Cities

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Abstract
A metapopulation model which explicitly integrates vector-borne and sexual transmission of an epidemic disease with passive and active movements between an urban city and a satellite city is formulated and analysed. The basic reproduction number of the disease is explicitly determined as a combination of sexual and vector-borne transmission parameters. The sensitivity analysis reveals that the disease is primarily transmitted via the vector-borne mode, rather than via sexual transmission, and that sexual transmission by itself may not initiate or sustain an outbreak. Also, increasing the population movements from one city to the other leads to an increase in the basic reproduction number of the later city but a decrease in the basic reproduction number of the former city. The influence of other significant parameters is also investigated via the analysis of suitable partial rank correlation coefficients. After gauging the effects of mobility, we explore the potential effects of optimal control strategies relying upon several distinct restrictions on population movement.

Keywords Vector-borne disease · Passive mobility · Metapopulation model · Sexual transmission · Control mechanism

1 Introduction

Technological advancements in contemporary transportation have enabled people to travel more often and on longer routes. Epidemic outbreaks occurring in local communities can now easily spread to the world at large, potentially threatening public health at a global scale. Therefore, adopting and enacting effective, coordinated epidemic control strategies through international and inter-community cooperation is of

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vital importance in order to mitigate the threat of global epidemics (MacPherson et al. 2007).

The detection and containment of several recent epidemics (Ebola in West Africa and Zika in the Americas) lead to significant logistic challenges (Pompon 2017; Shen et al. 2015). Part of these challenges has been addressed by the coordination of the World Health Organization (WHO) and the resources of the Global Outbreak Alert and Response Network (GOARN) (WHO 2016a; Majumder et al. 2016). Research institutes have also been collaborating with the involved health administrations to set up and enact effective control strategies and policies, for better and more comprehensive epidemic control.

In this context, appropriate modelling of geographical communities and population movements becomes mandatory. It should be noted that accounting for the effects of population movements on the spread of an infectious disease can be accomplished in several ways, illustrating the fact that human mobility encompasses a wide variety of social phenomena taking place on vastly different time scales. An approach towards modelling population heterogeneity and movements relies on the use of metapopulation models, which consider distinct spatial locations called patches, corresponding to countries, cities or local communities harbouring distinctive traits, connected by pathways for human migration and mobility (Arino and van den Driessche 2003; Hethcote 1978; Sattenspiel and Dietz 1995).

The cooperation and complementarity between urban and satellite cities create successful urban-satellite relationships. Large urban cities are often linked with several satellite cities, with only a few local jobs depending on the urban city for employment, shopping and essential services such as specialized medical care. More often than not, people from the urban cities also move to satellite cities for recreational activities, especially on weekends and national holidays.

In this paper, the movements of people between the urban city and the satellite city are classified as active and passive. The active movements are the casual movements of people acting on their own free will, while passive movements are the movements of some severely infected individuals who require intensive treatment of their disease. The satellite clinics move these severely infected individuals from the satellite city to the urban city through ambulance services. The individuals who move passively to the urban city are typically to be confined to a hospital to receive intensive medical care, while individuals who move actively can decide whether or not to move to the urban city because solutions to their problems can be found in either city.

The theory of metapopulations was first introduced in 1969 (Levins 1969) in the field of ecology. The succeeding years have witnessed an increased focus on the development of metapopulation models aiming at epidemic containment. Disease containment strategies such as travel restrictions (Colizza et al. 2007; Meloni 2011) and vaccination have been widely employed in metapopulation models (Hufnagel et al. 2004; Lima et al. 2015). Further attempts at realism have accounted for the effects of behavioural changes of individuals and timely dissemination of information upon disease containment (Meloni 2011).

However, only a few of these models have considered a metapopulation model that incorporates a vector-borne disease which also spreads via sexual transmission. Zika is one of the few diseases, if not the only one, that is transmitted through both

sexual and vector avenues (Duffy 2009; Gao 2016; WHO 2016b). ZIKV, the Zika virus, is a Flavivirus with functional similarities to DENV, the dengue virus, and is predominantly transmitted to humans by an infected female mosquito of the Aedes genus. ZIKV has other possible routes of transmission including mother to child, sexual and blood transfusions (Duffy 2009). ZIKV has been detected in serum, saliva, urine and semen. In some cases, ZIKV may not be detected in the blood, but is still present in urine and semen for 27 and 62 days after the onset of febrile illness, respectively (Musso 2015a). The World Health Organization (WHO) declared the Zika epidemic as a Public Health Emergency of International Concern (PHEIC) (WHO 2016b).

A study formulated two control models for investigating the impact of infecting Aedes aegypti mosquitoes with Wolbachia strains on the transmission of ZIKV in Brazil and concluded that the release of the male Wolbachia-infected mosquitoes is the best strategy for controlling the spread of Zika virus, possibly even eradicating wild mosquitoes eventually (Aliota et al. 2016; Wang et al. 2017). Further studies suggested that risky human behaviours involving multiple sexual partners, particularly among male populations, substantially increase the population of infected individuals, contributing significantly to the disease burden in the community (Agusto et al. 2017). Therefore, prevention and control efforts against ZIKV should target both the mosquito-borne and sexual transmission routes (Gao 2016). A comparison analysis of ZIKV outbreaks in French Polynesia, Colombia and the state of Bahia in Brazil concluded that there are variations within the attack rates in the three different locations, but there exists an association between the amount of precipitations and ZIKV outbreaks (He et al. 2017). However, in the USA, the Food and Drug Administration issued guidelines restricting the use of genetically modified animals to control vector-borne diseases. Consequently, it is crucial to explore alternative control scenarios by assessing the potential impact of mobility restrictions on the magnitude and time-line of an epidemic.

Mathematical modelling has become a vital tool for formulating and testing prevention and control measures for infectious diseases (Zhang et al. 2017). A recent study Arino and Portet (2015) used an SIR metapopulation model for investigating the spread of infectious disease between a larger urban centre and several smaller communities, using a mix of standard incidence (for the urban centre) and mass action incidence (for the satellite cities). In this regard, the disease spread could be caused by either human to human contact or through sexual intercourse with an infected person. It is therefore instructive to investigate the role of passive and active human movements on the spread of vector-borne and sexually transmitted infectious diseases. For the sake of simplicity, we formulate a metapopulation model which incorporates both vector and sexual transmissions together with active and passive movements of people between an urban city and a single satellite city and then investigate its effect on the spatial and temporal spread of the disease. The stability of a conceptually related model devised to describe the spread of cholera between two communities connected by migration has been discussed in Njagarah and Nyabadza (2014).

In this paper, we formulate a metapopulation model for the propagation of a vector-borne disease which is also sexually transmitted, keeping track of two patches linked together by means of active and passive mobilities. We then investigate the following problems:

- What is the main driving force for disease propagation in our concrete metapopulation model?
- To what extent can active and passive mobilities lead to the spread of the disease in both cities?
- Will restriction mechanisms acting upon active and passive movements between the two cities lead to the elimination of the disease?

2 Model Formulation

To formulate our model, we divide the human population into two communities (urban and satellite cities). Each community is further divided into five compartments according to the disease status of the individuals. In each community, the individuals are classified in the following way: susceptible individuals (S), exposed but not yet infected individuals (E), infected individuals harbouring mild symptoms (I_a), infected individuals with severe symptoms (I_b) and recovered individuals (R). Within each community, the subpopulations are assumed to be homogeneous. To distinguish between similar populations in both communities, of humans or vectors, we associate with the variables and parameters related to the satellite city the subscript 1, while the corresponding parameters and variables for the urban city have no subscript.

Our metapopulation model accounts for two movement patterns, active and passive. First, all populations except for the severely infected can freely move from one community to the other, being assumed that no new infections occur during travel. Typically, they show mild symptoms or no symptoms at all. Once reaching their destination, they conform to the disease dynamics of the respective community.

Secondly, we assume that the infected humans harbouring severe symptoms (I_b and I_{b1}) are to be confined to a hospital, as they would be seeking strict medical attention to recover from the disease. Therefore, the movement of infected individuals with severe symptoms across communities is assumed to take place only from the smaller community to the larger one (as a passive movement (q)), since only the urban city is assumed to possess the appropriate facilities to treat them.

New susceptible individuals are recruited in the urban and satellite communities at rates b and b_1, respectively. Conversely, the susceptible individuals are depleted through contact with infected vectors at a rate θ, as well as through sexual intercourse with the exposed population at rate κ and with the mildly infected population at rate τ, respectively, in the urban community, and at rates κ_1 and τ_1 with the exposed and mildly infected populations, respectively, in the satellite community.

The exposed individuals transfer to the respective compartments of infected individuals with mild symptoms in both communities (I_a and I_{a1}) at rates γ and γ_1, respectively, and the infected individuals with mild symptoms have their health condition further deteriorate into becoming infected individuals with severe symptoms at rates of δ and δ_1 for both communities, respectively. The infected individuals with severe symptoms (I_b and I_{b1}) undergo medical treatment and recover at rates α and α_1, respectively. The recovered individuals acquire permanent immunity to the disease, in both communities. Individuals in each compartment suffer natural mortality at rates of d and d_1 for the first and second communities, respectively.

Exposed and infected individuals transfer the pathogens (viruses) to the susceptible vector populations at rates $\beta_v \lambda_v \kappa_v$ for the urban city and $\beta_{v1} \lambda_{v1} \tau_{v1}$ for the satellite city, respectively. The susceptible vectors are recruited at the respective rates b_v and b_{v1} for both cities and move to the exposed compartment after coming into contact with exposed and infected human populations. The exposed vectors either die by natural death at rates d_v and d_{v1} for both cities or transfer to the infected vector population at rates ψ_v and ψ_{v1}, respectively. The vector populations are assumed to be present in both communities, and hence, their transmission dynamics is considered to be the same in both communities.

To gauge the effectiveness and impact of mobility restrictions, we employ an optimal control approach using the Pontryagin's maximum principle to determine the necessary optimality conditions. We incorporate three time-dependent controls ($u_1(t)$, $u_2(t)$, $u_3(t)$) into the model (3) to determine the optimal strategy to control the disease.

- $u_1(t)$: The efforts of the satellite city to restrict the movement of human populations (S_1, E_1, I_{a1}, R_1) to reduce the movement of individuals that may be infectious into the urban city.
- $u_2(t)$: The efforts of the urban city to restrict the movement of human populations (S, E, I_a, R) to reduce the movement of individuals that may be infectious into the satellite city.
- $u_3(t)$: The efforts of the satellite city to improve the treatment of the severely infected individuals (I_{b1}) to reduce the movement of the severely infected individuals into the urban city.

We assume that the satellite city will be able to upgrade the medical facilities during a disease outbreak to improve the treatments of the inhabitants.

The control variables, $u_1(t)$, $u_2(t)$ and $u_3(t)$, are bounded and Lebesgue integrable functions. Our control problem involves a situation in which the number of mildly infectious individuals, severely infected individuals, the cost of applying control mechanisms $u_1(t)$, $u_2(t)$ and the cost of the improvement of treatment $u_3(t)$ are minimized subject to the system (12). The given objective function is defined as

$$J(u_1, u_2, u_3) = \int_0^T \left[c_1 I_a + c_2 I_b + c_3 I_{a1} + c_4 I_{b1} + c_5 u_1^2 + c_6 u_2^2 + c_7 u_3^2 \right] \mathrm{d}t, \quad (1)$$

where I_a, I_{a1}, I_b and I_{b1} are the total infected human populations, T is the final time and the coefficients c_1, c_2, c_3, c_4 are the unit costs of control mechanisms on the mildly and severely infected individuals, and c_5, c_6 and c_7 are the unit costs of the other control mechanisms. Our aim is to minimize the total cost of controlling infected humans with mild and severe symptoms and at the same time save money on applying control mechanisms $u_1(t), u_2(t), u_3(t)$. Thus, we search for an optimal control (u_1^*, u_2^*, u_3^*) such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{u_1,\, u_2,\, u_3} \{ J(u_1, u_2, u_3) | u_1, u_2, u_3 \in \Omega \}, \quad (2)$$

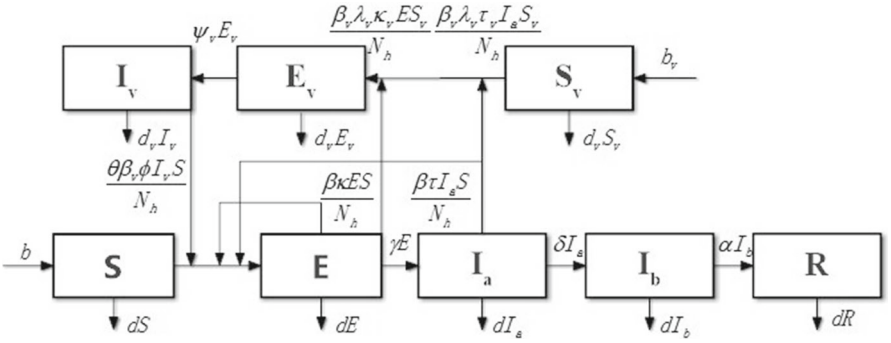

Fig. 1 The flow chart of disease progression in an isolated community

in which the control set is

$$\Omega = \{(u_1, u_2, u_3) | u_i : [0, T] \rightarrow [0, 1] \text{ Lebesgue measurable}, \quad i = 1, 2, 3\}.$$

The flow charts of disease progression are shown in Figs. 1 and 2, respectively. Figure 1 shows the transmission of the disease in an isolated community, while Fig. 2 shows the transmission of the disease in two communities linked together by means of transportation, with active and passive movements occurring between the urban and the satellite city. Here, $N_h = S + E + I_a + R$.

3 Model Analysis

For the sake of simplicity, we consider in this paper only the case of a single satellite city linked to a single urban city. The corresponding equations of the model are given below.

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \beta \frac{\kappa E + \tau I_a}{N_h} S - \frac{\theta \beta_v \phi I_v}{N_h} S - dS + m_2 S_1 - mS, \\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta \frac{\kappa E + \tau I_a}{N_h} S + \frac{\theta \beta_v \phi I_v}{N_h} S - dE - \gamma E + m_2 E_1 - mE, \\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \delta I_a + m_2 I_{a1} - mI_a, \\
\frac{\mathrm{d}I_b}{\mathrm{d}t} &= \delta I_a - \alpha I_b - dI_b + q I_{b1}, \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I_b - dR + m_2 R_1 - mR, \\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= b_v - \beta_v \lambda_v \frac{\kappa_v E + \tau_v I_a}{N_h} S_v - d_v S_v, \\
\frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v \lambda_v \frac{\kappa_v E + \tau_v I_a}{N_h} S_v - d_v E_v - \psi_v E_v,
\end{aligned}$$

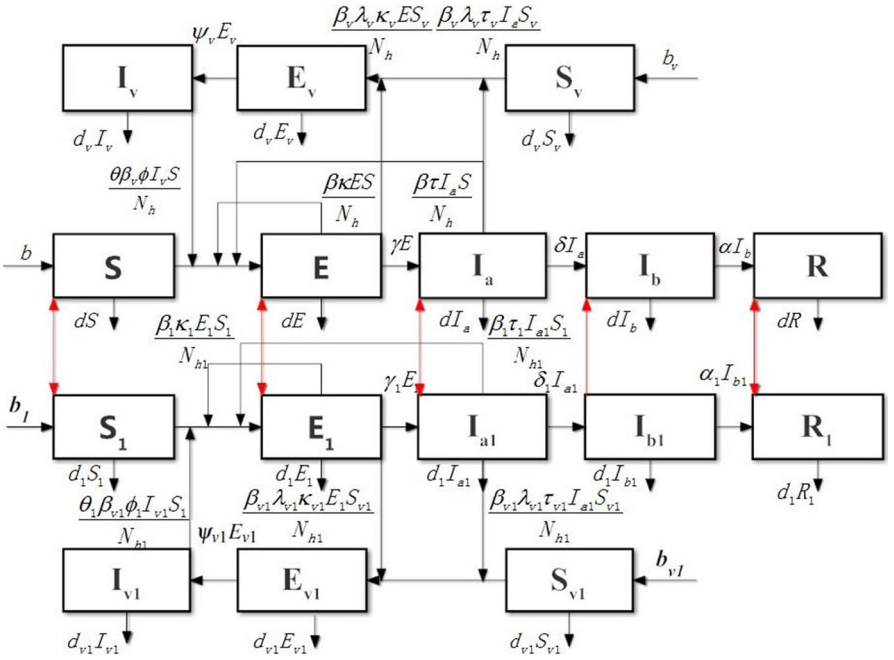

Fig. 2 (Colour figure online) The flow chart of disease progression in an urban city linked to a satellite city. The red arrows represent the migration of the human population

$$
\begin{aligned}
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_v E_v - d_v I_v,\\
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= b_1 - \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 - \frac{\theta_1\beta_{v1}\phi_1 I_{v1} S_1}{N_{h1}} - d_1 S_1 - m_2 S_1 + mS,\\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 + \frac{\theta_1\beta_{v1}\phi_1 I_{v1} S_1}{N_{h1}} - d_1 E_1 - \gamma_1 E_1 - m_2 E_1 + mE,\\
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1 E_1 - d_1 I_{a1} - \delta_1 I_{a1} - m_2 I_{a1} + mI_a,\\
\frac{\mathrm{d}I_{b1}}{\mathrm{d}t} &= \delta_1 I_{a1} - \alpha_1 I_{b1} - d_1 I_{b1} - q I_{b1},\\
\frac{\mathrm{d}R_1}{\mathrm{d}t} &= \alpha_1 I_{b1} - d_1 R_1 - m_2 R_1 + mR,\\
\frac{\mathrm{d}S_{v1}}{\mathrm{d}t} &= b_{v1} - \beta_{v1}\lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}} S_{v1} - d_{v1} S_{v1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1}\lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}} S_{v1} - d_{v1} E_{v1} - \psi_{v1} E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1} E_{v1} - d_{v1} I_{v1}.
\end{aligned}
\tag{3}
$$

3.1 Positive Invariance

The total human population and the total vector population are given below. We assume that the severely infected individuals do not infect anyone because they are hospitalized. That is, the severely infected and recovered ones have no infectivity to humans or mosquitoes and are effectively removed from the population. The system (3) is epidemiologically feasible and mathematically well-posed in $\mathbb{D} = \mathbb{D}_h \times \mathbb{D}_v \subset \mathbb{R}_+^{10} \times \mathbb{R}_+^6$, in which $\mathbb{D}_h$ is the domain for the human subpopulations and $\mathbb{D}_v$ is the domain for vector subpopulations.

3.2 The Disease-Free Equilibrium

At the disease-free equilibrium, there is no infection in either the human or the vector population. First of all, let us assume that there is no movement between the satellite city and the urban city. The system (3) without mobility then has a disease-free equilibrium E^0, given by

$$E^0 = \left(\frac{b}{d}, 0, 0, 0, 0, \frac{b_v}{d_v}, 0, 0, \frac{b_1}{d_1}, 0, 0, 0, 0, \frac{b_{v1}}{d_{v1}}, 0, 0\right) \in \mathbb{R}_+^{16}. \tag{4}$$

Secondly, to obtain the disease-free equilibrium E_m^0 for the system (3) with mobility, we need to solve the equilibrium subsystem associated to the susceptible populations, in the form

$$\begin{aligned} b + m_2 S_1 - (d + m)S &= 0, \\ b_1 + mS - (d_1 + m_2)S_1 &= 0. \end{aligned} \tag{5}$$

It then follows that

$$E_m^0 = \left(\frac{b(d_1 + m_2) + b_1 m_2}{dd_1 + dm_2 + md_1}, 0, 0, 0, 0, \frac{b_v}{d_v}, 0, 0, \frac{b_1(d + m) + mb}{dd_1 + dm_2 + md_1}, 0, 0, 0, 0, \frac{b_{v1}}{d_{v1}}, 0, 0\right).$$

3.3 The Basic Reproduction Number for an Isolated Community

The basic reproduction number is obtained using the next-generation method presented in van den Driessche and Watmough (2002). It follows that the basic reproduction number of an isolated community, described by the first eight equations of the system (3) with $m = m_2 = 0$, is given by

$$R_0 = \frac{R_{hh} + \sqrt{R_{hh}^2 + 4R_{hv}^2}}{2},$$

in which R_{hh} is the basic reproduction number for sexual transmission and R_{hv} is the basic reproduction number for vector-borne transmission, given by

$$R_{hh} = \frac{\beta(\tau\gamma + \kappa(d + \delta))}{(d + \gamma)(d + \delta)},$$

$$R_{hv} = \sqrt{\frac{\theta\beta_v^2\phi\psi_v\lambda_v b_v d\left(\tau_v(d_v+\psi_v)+\kappa_v(d+\delta)\right)}{bd_v^2(d+\gamma)(d+\delta)(d_v+\psi_v)}}.$$

If the infection occurs in an isolated community, then the corresponding condition for its persistence in terms of the basic reproduction number of the disease is given by $R_0 > 1$, which holds only if $R_{hh} + R_{hv}^2 > 1$.

3.4 The Basic Reproduction Number for the Connected System

If the infection occurs in a community which is connected to another one via population mobility, the movements of individuals should be reflected in the reproduction number of the disease. The reproduction numbers of the connected communities are then given by

$$R_{01M} = \frac{R_{hh1M} + \sqrt{R_{hh1M}^2 + 4R_{hv1M}^2}}{2}$$
$$R_{02M} = \frac{R_{hh2M} + \sqrt{R_{hh2M}^2 + 4R_{hv2M}^2}}{2},$$

in which

$$R_{hh1M} = \frac{\beta(\tau\gamma + \kappa(d+\delta+m))}{(d+\gamma+m)(d+\delta+m)},$$
$$R_{hv1M} = \sqrt{\frac{\theta\beta_v^2\phi\psi_v\lambda_v b_v(dd_1+dm_2+md_1)(\tau_v(d_v+\psi_v)+\kappa_v(d+\delta+m))}{d_v^2(b(d_1+m_2)+b_1m_2)(d+\gamma+m)(d+\delta+m)(d_v+\psi_v)}},$$
$$R_{hh2M} = \frac{\beta_1(\tau_1\gamma_1 + \kappa_1(d_1+\delta_1+m_2))}{(d_1+\gamma_1+m_2)(d_1+\delta_1+m_2)},$$
$$R_{hv2M} = \sqrt{\frac{\theta_1\beta_{v1}^2\phi_1\psi_{v1}\lambda_{v1}b_{v1}(dd_1+dm_2+md_1)(\tau_{v1}(d_{v1}+\psi_{v1})+\kappa_{v1}(d_1+\delta_1+m_2))}{d_{v1}^2(b_1(d+m)+mb)(d_1+\gamma_1+m_2)(d_1+\delta_1+m_2)(d_{v1}+\psi_{v1})}}.$$

An estimation for the 'global' basic reproduction number R_0 (somewhat less computationally intensive than the exact value, as it does not involve finding the inverse of a higher-dimensional matrix) can then be given as the maximum of the reproduction numbers associated with each community,

$$R_0^m = \max\{R_{01M}, R_{02M}\}.$$

The exact value of R_0 can be obtained via the next-generation method. It is seen that

$$F = \begin{bmatrix} \beta\kappa & \beta\tau & 0 & 0 & \theta\beta_v\phi & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_v\lambda_v\kappa_v b_v d}{bd_v} & \frac{\beta_v\lambda_v\tau_v b_v d}{bd_v} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_1\kappa_1 & \beta_1\tau_1 & 0 & 0 & 0 & \theta_1\beta_{1v}\phi_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_{vI}\lambda_{vI}\kappa_{vI}b_{vI}d_1}{b_1 d_{vI}} & \frac{\beta_{vI}\lambda_{vI}\tau_{vI}b_{vI}d_1}{b_1 d_{vI}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V^{-1} = \begin{bmatrix} v_1 & 0 & 0 & 0 & 0 & v_2 & 0 & 0 & 0 & 0 \\ v_3 & v_4 & 0 & 0 & 0 & v_5 & v_6 & 0 & 0 & 0 \\ v_7 & v_8 & (\alpha+d)^{-1} & 0 & 0 & v_9 & v_{10} & v_{11} & 0 & 0 \\ 0 & 0 & 0 & (d_v+\psi_v)^{-1} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_v}{(d_v+\psi_v)d_v} & {d_v}^{-1} & 0 & 0 & 0 & 0 & 0 \\ v_{12} & 0 & 0 & 0 & 0 & v_{13} & 0 & 0 & 0 & 0 \\ v_{14} & v_{15} & 0 & 0 & 0 & v_{16} & v_{17} & 0 & 0 & 0 \\ v_{18} & v_{19} & 0 & 0 & 0 & v_{20} & v_{21} & (d_1+\alpha_1+q)^{-1} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & (d_{vI}+\psi_{vI})^{-1} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\psi_{vI}}{(d_{vI}+\psi_{vI})d_{vI}} & {d_{vI}}^{-1} \end{bmatrix},$$

where

$$\begin{aligned}
b_1 &= (dd_1 + dm_2 + d\gamma_1 + d_1\gamma + d_1 m + \gamma m_2 + \gamma\gamma_1 + m\gamma_1) \\
b_2 &= (dd_1 + dm_2 + d\delta_1 + d_1\delta + d_1 m + \delta m_2 + \delta\delta_1 + m\delta_1) \\
a_3 &= {d_1}^2\gamma + 2d_1\gamma m_2 + d_1\gamma\delta_1 + d_1\gamma\gamma_1 + \gamma {m_2}^2 + \gamma m_2\delta_1 + \gamma m_2\gamma_1 \\
&\quad + \gamma\delta_1\gamma_1 + mm_2\gamma_1 \\
a_5 &= d\gamma_1 + d_1\gamma + \gamma m_2 + \gamma\delta_1 + \gamma\gamma_1 + m\gamma_1 \\
a_7 &= ((d_1+\alpha_1+q)(\gamma_1+d_1+m_2)(\delta_1+d_1+m_2)\gamma + \gamma_1(m_2 d_1 + (\alpha_1+q)m_2 \\
&\quad + \delta_1 q)m)\delta + m\delta_1 q((\gamma_1+d_1+m_2)\gamma + \gamma_1(d+m)) \\
b_7 &= (\alpha+d)((\gamma_1+d_1+m_2)\gamma + (d+m)d_1 + dm_2 + \gamma_1(d+m)) \\
&\quad \times (d_1+\alpha_1+q)\cdot((\delta_1+d_1+m_2)\delta + (d+m)d_1 + dm_2 + \delta_1(d+m)) \\
a_9 &= \Big(\gamma(d_1+\alpha_1+q){m_2}^2 + (d_1+\alpha_1+q)((d+m+\gamma)\gamma_1 + \gamma(\delta_1+d_1))m_2 \\
&\quad + \delta_1\gamma_1 q(d+m+\gamma))\delta + \delta_1 q(\gamma mm_2 + \gamma_1(d+m)(d+m+\gamma)) \\
b_9 &= (\alpha+d)((d+\gamma)m_2 + (\gamma_1+d_1)(d+m+\gamma))(d_1+\alpha_1+q) \\
&\quad \cdot((\delta_1+d_1+m_2)\delta + dm_2 + (\delta_1+d_1)(d+m)) \\
a_{16} &= d^2\gamma_1 + d\delta\gamma_1 + d\gamma\gamma_1 + 2dm\gamma_1 + \delta\gamma\gamma_1 + \delta m\gamma_1 + \gamma mm_2 \\
&\quad + \gamma m\gamma_1 + m^2\gamma_1 \\
v_1 &= \frac{d_1+\gamma_1+m_2}{b_1} \\
v_2 &= \frac{m_2}{b_1}
\end{aligned}$$

$$
\begin{aligned}
v_3 &= \frac{a_3}{b_1 b_2} \\
v_4 &= \frac{d_1 + \delta_1 + m_2}{b_2} \\
v_5 &= \frac{a_5 m_2}{b_1 b_2} \\
v_6 &= \frac{m_2}{b_2} \\
v_7 &= \frac{a_7}{b_7} \\
v_8 &= \frac{a_3}{(\alpha + d)\, b_2\, (d_1 + \alpha_1 + q)} \\
v_9 &= \frac{a_9}{b_9} \\
v_{10} &= \frac{d q \delta_1 + d_1\, \delta\, m_2 + \delta\, m_2\, q + \delta\, m_2\, \alpha_1 + \delta\, q \delta_1 + m q \delta_1}{(\alpha + d)\, b_2\, (d_1 + \alpha_1 + q)} \\
v_{11} &= \frac{q}{(d_1 + \alpha_1 + q)\, (\alpha + d)} \\
v_{12} &= \frac{m}{b_1} \\
v_{13} &= \frac{d + \gamma + m}{b_1} \\
v_{14} &= \frac{(d \gamma_1 + d_1\, \gamma + \delta\, \gamma_1 + \gamma\, m_2 + \gamma\, \gamma_1 + m \gamma_1)\, m}{b_2 b_1} \\
v_{15} &= \frac{m}{b_2} \\
v_{16} &= \frac{a_{16}}{b_2 b_1} \\
v_{17} &= \frac{d + \delta + m}{b_2} \\
v_{18} &= \frac{\delta_1\ (d \gamma_1 + d_1\, \gamma + \delta\, \gamma_1 + \gamma\, m_2 + \gamma\, \gamma_1 + m \gamma_1)\, m}{b_2 b_1\, (d_1 + \alpha_1 + q)} \\
v_{19} &= \frac{m \delta_1}{b_2\, (d_1 + \alpha_1 + q)} \\
v_{20} &= \frac{\delta_1\, a_{16}}{b_2 b_1\, (d_1 + \alpha_1 + q)} \\
v_{21} &= \frac{\delta_1\ (d + \delta + m)}{b_2\, (d_1 + \alpha_1 + q)}
\end{aligned}
$$

$$FV^{-1} = \begin{bmatrix} w_1 & w_2 & 0 & \frac{\theta\,\beta_v\,\phi\,\psi_v}{(d_v+\psi_v)d_v} & \frac{\theta\,\beta_v\,\phi}{d_v} & w_3 & w_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ w_5 & w_6 & 0 & 0 & 0 & w_7 & w_8 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ w_9 & 0 & 0 & \frac{\beta_1\,\kappa_1\,\psi_v}{(d_v+\psi_v)d_v} & \frac{\beta_1\,\kappa_1}{d_v} & w_{10} & 0 & 0 & w_{11} & \frac{\theta_1\,\beta_{1v}\,\phi_1}{d_{vI}} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ w_{12} & w_{13} & 0 & 0 & 0 & w_{14} & w_{15} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

where

$$\begin{aligned}
w_1 &= \frac{\beta\,\kappa\ (d_1+\gamma_1+m_2)}{b_1} + \frac{\beta\,\tau\,a_3}{b_2 b_1} \\
w_2 &= \frac{\beta\,\tau\ (d_1+\delta_1+m_2)}{dd_1 + dm_2 + d\delta_1 + d_1\,\delta + d_1\,m + \delta\,m_2 + \delta\,\delta_1 + m\delta_1} \\
w_3 &= \frac{\beta\,\kappa\,m_2}{b_1} + \frac{\beta\,\tau\,a_5 m_2}{b_2 b_1} \\
w_4 &= \frac{\beta\,\tau\,m_2}{dd_1 + dm_2 + d\delta_1 + d_1\,\delta + d_1\,m + \delta\,m_2 + \delta\,\delta_1 + m\delta_1} \\
w_5 &= \frac{\beta_v\,\lambda_v\,\kappa_v\,b_v\,d\,(d_1+\gamma_1+m_2)}{bd_v\,b_1} + \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,da_3}{bd_v\,b_2 b_1} \\
w_6 &= \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,d\,(d_1+\delta_1+m_2)}{bd_v\,b_2} \\
w_7 &= \frac{\beta_v\,\lambda_v\,\kappa_v\,b_v\,dm_2}{bd_v\,b_1} + \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,da_5 m_2}{bd_v\,b_2 b_1} \\
w_8 &= \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,dm_2}{bd_v\,b_2} \\
w_9 &= \frac{\beta_1\,\tau_1\,m}{b_1} \\
w_{10} &= \frac{\beta_1\,\tau_1\ (d+\gamma+m)}{b_1} \\
w_{11} &= \frac{\theta_1\,\beta_{1v}\,\phi_1\,\psi_{vI}}{(d_{vI}+\psi_{vI})\,d_{vI}} \\
w_{12} &= \frac{\beta_{vI}\,\lambda_{vI}\,\kappa_{vI}\,b_{vI}\,d_1\,m}{b_1\,d_{vI}\,b_1} \\
&\quad + \frac{\beta_{vI}\,\lambda_{vI}\,\tau_{vI}\,b_{vI}\,d_1\ (d\gamma_1 + d_1\,\gamma + \delta\,\gamma_1 + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)\,m}{b_1\,d_{vI}\,b_1 b_2} \\
w_{13} &= \frac{\beta_{vI}\,\lambda_{vI}\,\tau_{vI}\,b_{vI}\,d_1\,m}{b_1\,d_{vI}\,b_2}
\end{aligned}$$

Table 1 Descriptions of notations for variables

Description	Variable
Total susceptible human population	$S^T = (S, S_1)$
Total exposed human population	$E^T = (E, E_1)$
Total mildly infected human population	$I_a^T = (I_a, I_{a1})$
Total severely infected human population	$I_b^T = (I_b, I_{b1})$
Total recovered human population	$R^T = (R, R_1)$
Total susceptible vector population	$S_v^T = (S_v, S_{v1})$
Total exposed vector population	$E_v^T = (E_v, E_{v1})$
Total infected vector population	$I_v^T = (I_v, I_{v1})$
Total vector population	$N_v = (S_v^T, E_v^T, I_v^T)$
Non-susceptible human population	$J = (E^T, I_a^T, I_b^T, R^T)$
Non-susceptible vector population	$J_v = (E_v^T, I_v^T)$
Generic epidemiological population	$\mathbb{C} = (S^T, J, S_v^T, J_v)$
Total human population size	$\overline{N} = \sum_{i=1}^{10} N_i = \|\|(S^T, J)\|\|$
Total vector population size	$\overline{N}_v = \|\|N_v\|\|$

$$w_{14} = \frac{\beta_{v1}\,\lambda_{v1}\,\kappa_{v1}\,b_{v1}\,d_1\,(d+\gamma+m)}{b_1\,d_{v1}\,b_1} + \frac{\beta_{v1}\,\lambda_{v1}\,\tau_{v1}\,b_{v1}\,d_1\,a_{16}}{b_1\,d_{v1}\,b_1 b_2}$$
$$w_{15} = \frac{\beta_{v1}\,\lambda_{v1}\,\tau_{v1}\,b_{v1}\,d_1\,(d+\delta+m)}{b_1\,d_{v1}\,b_2}$$

The global basic reproduction number (R_0) is then given as the dominant eigenvalue of the matrix FV^{-1} given above. An outcome is the fact that R_0 does not depend upon the passive movement rate q, which is perhaps because the severely infected individuals are supposed to be hospitalized and isolated from the susceptible individuals. This changes if a single category of infectives is considered for each location (see "Appendix B" for details).

3.5 Uniform Strong Disease Persistence and Existence of Endemic Equilibria

Under the assumption of the constant recruitment, it is easy to see that the host is strongly uniformly persistent. To simplify our results, we make the notations summarized in Table 1. Since the recruitment rates b and b_1 are positive constants, $S^T(t) > (0, 0)$ for all $t > 0$, and there exist two positive constants δ_1^* and δ_2^* such that

$$\liminf_{t\to\infty} S^T(t) \geq (\delta_1^*, \delta_2^*)$$

for all non-negative solutions in the system (3). In fact, by the first subsystem in the system (3)

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \Big(d + \frac{\beta(\kappa E + \tau I_a)}{N_h} + \frac{\theta\beta_v\phi I_v}{N_h}\Big)S + m_2 S_1 - mS,\\ &> b - \big(d + \beta(\kappa+\tau) + \theta\beta_v\phi\big)S - mS.\end{aligned}$$

Consequently, there exists $\delta_1^* \in (0, +\infty)$, independent of the solution, such that

$$\liminf_{t\to\infty} S(t) \geq \frac{b}{d + \beta(\kappa+\tau) + \theta\beta_v\phi + m} =: \delta_1^*.$$

Similarly, there exists a $\delta_2^* \in (0, +\infty)$ such that

$$\liminf_{t\to\infty} S_1(t) \geq \delta_2^*.$$

Hence, if $R_0 > 1$, the disease is then uniformly strongly persistent as well. Since $S^T(t) >> (0,0)$ for $t > 0$, the subsequent persistence results do not need the solutions of system (3) to satisfy $S^T(0) >> (0,0)$. Also, if $R_0 > 1$, and all recruitment rates b and b_1 are positive constants, then there exists some $\epsilon > 0$ such that

$$\liminf_{t\to\infty} \ C_i(t) \geq \epsilon, \ i = 1, 2, \ C \in \mathbb{C} \text{ and } \mathrm{C} = (\mathrm{C}_1, \mathrm{C}_2)$$

for all non-negative solutions of system (3) with

$$\big(E^T(0), I_a^T(0), I_b^T(0), E_v^T(0), I_v^T(0)\big) > 0.$$

Let

$$\begin{aligned}\mathbb{X} = \{&(S^T, E^T, I_a^T, I_b^T, R^T, S_v^T, E_v^T, I_v^T) \in (0,\infty)^{16} | (S^T, S_v^T) \in (0,+\infty)^4 \text{ and}\\ &(E^T, I_a^T, I_b^T, R^T, E_v^T, I_v^T) \in \mathbb{R}_+^{12}\}.\end{aligned}$$

By Theorem A.32 of Thieme (2003), the solution takes its values in $\mathbb{X}$ for $t > 0$. Define $\rho : \mathbb{X} \to \mathbb{R}_+$ by

$$\rho(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = I,$$

for fixed $I \in \{I_a, I_{a1}, I_b, I_{b1}, I_v, I_{v1}\}$, and $\widetilde{\rho} : \mathbb{X} \to \mathbb{R}_+$ by

$$\widetilde{\rho}(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = \frac{I_a + I_b}{N_h} + \frac{I_{a1} + I_{b1}}{N_{h1}} + \frac{I_v + I_{v1}}{\overline{N_v}}.$$

In the language of Sect. A.5 of Thieme (2003), the semiflow Φ induced by the solutions of system (3) is uniformly weakly ρ-persistent by Theorem 4.3 in Dhirasakdanon et al. (2007). The compactness condition in Sect. A.5 of Thieme (2003) follows from the results above. Notice that every total orbit $\omega : \mathbb{R} \to X$ of Φ is associated with a solution of system (3) that is defined for all times and takes value in $\mathbb{X}$. By the irreducibility

of the matrix $\begin{pmatrix} 0 & m \\ m_2 & 0 \end{pmatrix}$, $\widetilde{\rho}(\omega(0)) > 0$ whenever $\rho(\omega(t)) > 0$ for all $t \in \mathbb{R}$. The claim for $C \in \{I_a^T, I_b^T, I_v^T\}$ now follows from Theorem A.34 in Thieme (2003). For $C \in \{E^T, R^T, E_v^T\}$, modify $\widetilde{\rho}(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = C_i$. For $C = S^T$, the statement has already been shown in the content above. Similarly, for $C = S_v^T$, the statement should be easily shown. The existence of an (endemic) equilibrium of system (3) in $(0, \infty)^{16}$ follows from Theorem 1.3.7. in Dhirasakdanon et al. (2007).

4 Sensitivity Analysis

A sensitivity analysis is carried out to find the dependence of the basic reproduction number upon the parameters of the model. This analysis is essential, enabling us to identify the critical parameters to be acted upon if the disease is to be controlled and eliminated. The sensitivity and uncertainty analyses are performed by using the Latin hypercube sampling (LHS) scheme, which is a Monte–Carlo stratified sampling method leading to an unbiased estimate of the model output for a given set of input parameter values (Chitnis et al. 2008; Blower and Dowlatabadi 1994). The parameter space is sampled without replacement, assuming statistical independence between the parameters. The selected sample is then used to determine unbiased estimates of output values for the reproduction numbers R_{01M} and R_{02M} of the model, respectively. R_{01M} is the average number of infections that a single individual in the urban city causes over the duration of the infectious period accounting for movements of humans between both cities, and R_{02M} is the average number of infections that a single individual in the satellite city causes over the duration of the infectious period accounting for movements of humans between both cities.

The partial rank correlation coefficients determined for the reproduction numbers are graphically presented in tornado plots, as shown in Figs. 3 and 4. A positive (negative) correlation coefficient corresponds to an increase (decrease) of the reproduction numbers as a result of an increase in the corresponding parameter. The parameters ψ_v, θ, ϕ and λ_v have the lowest partial rank correlation coefficients (PRCCs) with respect to R_{01M}, while τ_{v1}, d_1 and ψ_{v1} have the lowest PRCCs with respect to R_{02M}. However, their influences are visible. The human to human transmission parameters τ, κ, τ_1 and κ_1 for both cities, respectively, have positive correlations indicating that an increase in sexual transmission will lead to an increase in the basic reproduction number.

The respective correlation values of κ and τ are too small to cause an outbreak of the disease, but their combined contribution is large enough to prolong an outbreak in the urban city. In this regard, any measures that increase vector mortality or reduce their multiplication decrease not only the burden of infection but also the risk of contracting it. Also, the rate θ at which susceptible individuals become infected and the contact rate β increase the basic reproduction number when increased. Further, increasing the movement of infected individuals m and m_2 from one patch to the other leads to an increase of the threshold for the latter and a decrease of the threshold for the former, which increases the risk of local disease outbreak.

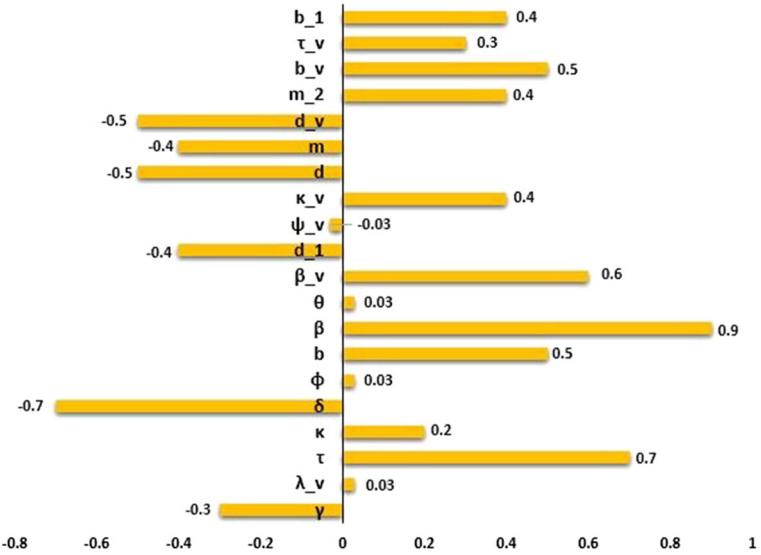

Fig. 3 Tornado plot showing partial rank correlation coefficients (PRCCs) of the reproduction number (R_{01M}) with respect to the parameter values

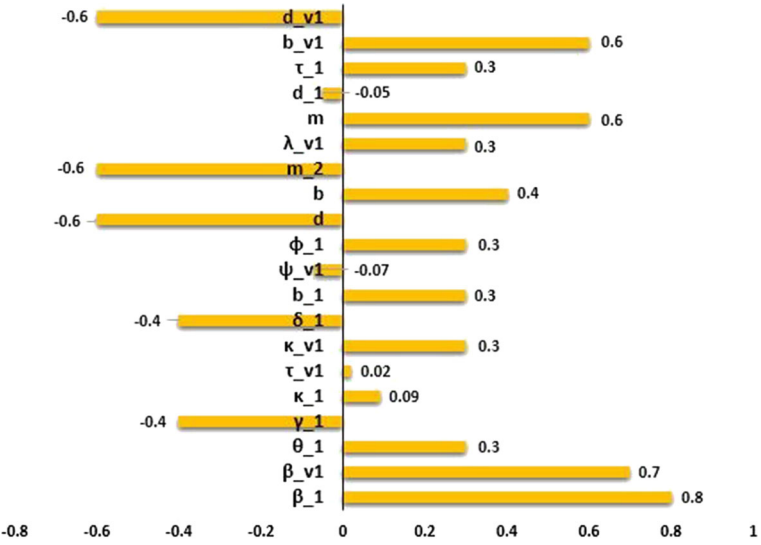

Fig. 4 Tornado plot showing partial rank correlation coefficients (PRCCs) of the reproduction number (R_{02M}) with respect to the parameter values

5 Optimal Control Strategies

Vector control techniques may be an effective way of reducing epidemic transmission, as shown by our sensitivity analysis. However, we try to stay away from the controversial issue of using genetically modified vectors to control the vector population.

After the emergence of the H1N1 influenza in 2009, travel-related control strategies during the early stage of the epidemic outbreak were used to slow down its spread. Consequently, in this work we incorporate three time-dependent control variables into the model (3) to determine the optimal strategy for controlling the disease. The model (3) then becomes

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \beta\frac{\kappa E + \tau I_a}{N_h}S - \frac{\theta\beta_v\phi I_v}{N_h}S - dS + (1-u_1(t))m_2S_1 - (1-u_2(t))mS,\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta\frac{\kappa E + \tau I_a}{N_h}S + \frac{\theta\beta_v\phi I_v}{N_h}S - dE - \gamma E + (1-u_1(t))m_2E_1\\
&\quad - (1-u_2(t))mE,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \delta I_a + (1-u_1(t))m_2I_{a1} - (1-u_2(t))mI_a,\\
\frac{\mathrm{d}I_b}{\mathrm{d}t} &= \delta I_a - \alpha I_b - dI_b + (1-u_3(t))qI_{b1},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I_b - dR + (1-u_1(t))m_2R_1 - (1-u_2(t))mR,\\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= b_v - \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vS_v,\\
\frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vE_v - \psi_vE_v,\\
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_vE_v - d_vI_v,\\
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= b_1 - \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 - \frac{\theta_1\beta_{v1}\phi_1I_{v1}}{N_{h1}}S_1 - d_1S_1 - (1-u_1(t))m_2S_1\\
&\quad + (1-u_2(t))mS,\\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 + \frac{\theta_1\beta_{v1}\phi_1I_{v1}}{N_{h1}}S_1 - d_1E_1 - \gamma_1E_1 - (1-u_1(t))m_2E_1\\
&\quad + (1-u_2(t))mE,\\
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1E_1 - d_1I_{a1} - \delta_1I_{a1} - (1-u_1(t))m_2I_{a1} + (1-u_2(t))mI_a,\\
\frac{\mathrm{d}I_{b1}}{\mathrm{d}t} &= \delta_1I_{a1} - \alpha_1I_{b1} - d_1I_{b1} - (1-u_2(t))qI_{b1},\\
\frac{\mathrm{d}R_1}{\mathrm{d}t} &= \alpha_1I_{b1} - d_1R_1 - (1-u_1(t))m_2R_1 + (1-u_2(t))mR,\\
\frac{\mathrm{d}S_{v1}}{\mathrm{d}t} &= b_{v1} - \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}S_{v1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}E_{v1} - \psi_{v1}E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1}E_{v1} - d_{v1}I_{v1}.
\end{aligned}
\tag{6}
$$

Pontryagin's maximum principle (Pontryagin et al. 1962) states a necessary condition that must hold on an optimal trajectory. This principle converts the equations (12) and (13) into a problem of minimizing pointwise a Hamiltonian H with respect to u_1, u_2 and u_3.

The Hamiltonian is given by

$$
\begin{aligned}
H = {} & c_1 I_a + c_2 I_b + c_3 I_{a1} + c_4 I_{b1} + c_5 u_1^2 + c_6 u_2^2 + c_7 u_3^2, \\
& + \lambda_S \left\{ b - \beta \frac{\kappa E + \tau I_a}{N_h} S - \frac{\theta \beta_v \phi I_v}{N_h} S - dS + (1 - u_1(t)) m_2 S_1 \right. \\
& \left. - (1 - u_2(t)) m S \right\}, \\
& + \lambda_E \left\{ \beta \frac{\kappa E + \tau I_a}{N_h} S + \frac{\theta \beta_v \phi I_v}{N_h} S - dE - \gamma E + (1 - u_1(t)) m_2 E_1 \right. \\
& \left. - (1 - u_2(t)) m E \right\}, \\
& + \lambda_{I_a} \{ \gamma E - d I_a - \delta I_a + (1 - u_1(t)) m_2 I_{a1} - (1 - u_2(t)) m I_a \}, \\
& + \lambda_{I_b} \{ \delta I_a - \alpha I_b - d I_b + (1 - u_3(t)) q I_{b1} \}, \\
& + \lambda_R \left\{ \alpha I_b - dR + (1 - u_1(t)) m_2 R_1 - (1 - u_2(t)) m R \right\}, \\
& + \lambda_{S_v} \left\{ b_v - \beta_v \lambda_v \frac{\kappa_v E + \tau_v I_a}{N_h} S_v - d_v S_v \right\}, \\
& + \lambda_{E_v} \left\{ \beta_v \lambda_v \frac{\kappa_v E + \tau I_a}{N_h} S_v - d_v E_v - \psi_v E_v \right\}, \\
& + \lambda_{I_v} \{ \psi_v E_v - d_v I_v \}, \\
& + \lambda_{S_1} \left\{ b_1 - \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 - \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} S_1 - d_1 S_1 \right. \\
& \left. - (1 - u_1(t)) m_2 S_1 + (1 - u_2(t)) m S \right\}, \\
& + \lambda_{E_1} \left\{ \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 + \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} S_1 - d_1 E_1 - \gamma_1 E_1 \right. \\
& \left. - (1 - u_1(t)) m_2 E_1 + (1 - u_2(t)) m E \right\}, \\
& + \lambda_{I_{a1}} \{ \gamma_1 E_1 - d_1 I_{a1} - \delta_1 I_{a1} - (1 - u_1(t)) m_2 I_{a1} + (1 - u_2(t)) m I_a \}, \\
& + \lambda_{I_{b1}} \{ \delta_1 I_{a1} - \alpha_1 I_{b1} - d_1 I_{b1} - (1 - u_3(t)) q I_{b1} \}, \\
& + \lambda_{R_1} \{ \alpha_1 I_{b1} - d_1 R_1 - (1 - u_1(t)) m_2 R_1 + (1 - u_2(t)) m R \}, \\
& + \lambda_{S_{v1}} \left\{ b_{v1} - \beta_{v1} \lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}} S_{v1} - d_{v1} S_{v1} \right\}, \\
& + \lambda_{E_{v1}} \left\{ \beta_{v1} \lambda_{v1} \frac{\kappa_v E_1 + \tau_v I_{a1}}{N_{h1}} S_{v1} - d_{v1} E_{v1} - \psi_{v1} E_{v1} \right\}, \\
& + \lambda_{I_{v1}} \{ \psi_{v1} E_{v1} - d_{v1} I_{v1} \}, \qquad (7)
\end{aligned}
$$

where $\lambda_S, \lambda_E, \lambda_{I_a}, \lambda_{I_b}, \lambda_R, \lambda_{S_v}, \lambda_{E_v}, \lambda_{I_v}, \lambda_{S_1}, \lambda_{E_1}, \lambda_{I_{a1}}, \lambda_{I_{b1}}, \lambda_{R_1}, \lambda_{S_{v1}}, \lambda_{E_{v1}}$ and $\lambda_{I_{v1}}$ are the adjoint variables or co-state variables. The system of equations is found by taking the appropriate partial derivatives of the Hamiltonian (15) with respect to the associated state variables. Furthermore, the transversality conditions are

$$\begin{aligned}\lambda_S(T) &= \lambda_E(T) = \lambda_{I_a}(T) = \lambda_{I_b}(T) = \lambda_R(T) = \lambda_{S_v}(T) = \lambda_{E_v}(T) = \lambda_{I_v}(T)\\ &= \lambda_{S_1}(T) = \lambda_{E_1}(T) = \lambda_{I_{a1}}(T) = \lambda_{I_{b1}}(T) = \lambda_{R_1}(T) = \lambda_{S_{v1}}(T) = \lambda_{E_{v1}}(T)\\ &= \lambda_{I_{v1}}(T) = 0.\end{aligned}$$

Finally, since in our optimal control problem there are no terminal values for the state variables, we give transversality conditions at the final time T by $\lambda_i(T) = 0$, $i = 1,\ 2,\ 3$.

On the interior of the control set, where $0 < u_i < 1$, for $i = 1,\ 2,\ 3$, we have

$$\begin{aligned}\frac{\partial H}{\partial u_1} &= 2c_5u_1 - m_2S_1\lambda_S - m_2E_1\lambda_E - m_2I_{a1}\lambda_{I_a} - m_2R_1\lambda_R + m_2S_1\lambda_{S_1}\\ &\quad + m_2E_1\lambda_{E_1} + m_2I_{a1}\lambda_{I_{a1}} + m_2R_1\lambda_{R_1} = 0,\\ \frac{\partial H}{\partial u_2} &= 2c_6u_2 + mS\lambda_S + mE\lambda_E + mI_a\lambda_{I_a} + mR\lambda_R - mS\lambda_{S_1} - mE\lambda_{E_1}\\ &\quad - mI_a\lambda_{I_{a1}} - mR\lambda_{R_1} = 0,\\ \frac{\partial H}{\partial u_3} &= 2c_7u_3 - qI_{b1}\lambda_{I_b} + qI_{b1}\lambda_{I_{b1}} = 0.\end{aligned}$$

By the standard control arguments involving the bounds on the control variables, we obtain

$$\begin{aligned}u_1^* &= \min\left\{1, \max\left(0, u_1^{**}\right)\right\},\\ u_2^* &= \min\left\{1, \max\left(0, u_2^{**}\right)\right\},\\ u_3^* &= \min\left\{1, \max\left(0, u_3^{**}\right)\right\},\end{aligned}$$

where

$$\begin{aligned}u_1^{**} &= \frac{1}{2c_5}\Big[m_2S_1\lambda_S + m_2E_1\lambda_E + m_2I_{a1}\lambda_{I_a} + m_2R_1\lambda_R - m_2S_1\lambda_{S_1} - m_2E_1\lambda_{E_1}\\ &\quad - m_2I_{a1}\lambda_{I_{a1}} - m_2R_1\lambda_{R_1}\Big],\\ u_2^{**} &= \frac{1}{2c_6}\Big[-mS\lambda_S - mE\lambda_E - mI_a\lambda_{I_a} - mR\lambda_R + mS\lambda_{S_1} + mE\lambda_{E_1}\\ &\quad + mI_a\lambda_{I_{a1}} + mR\lambda_{R_1}\Big],\end{aligned}$$

Table 2 Average daily number of individuals travelling in each direction between Winnipeg and the satellite community Steinbach. Data from 2011 Canadian census (Manitoba)

City	*Population*	Distance (km)	Average daily travellers
Winnipeg	663, 617	66	7505
Steinbach	13, 524	66	7505

$$u_3^{**} = \frac{1}{2c_7}\left[qI_{b1}\lambda_{I_b} - qI_{b1}\lambda_{I_{b1}}\right].$$

6 Estimation of Parameters

To estimate the movement rates, we use the approach employed in Arino and Portet (2015). Let us consider the urban city S and its population N_S. Assume that the rate at which individuals leave city S to move to the satellite city S_1 is m_{ss_1}. Thus, *ceteris paribus*, $N_s^{'} = -m_{ss_1}N_s$, which implies that $N_s(t) = N_s(0)e^{-m_{ss_1}t}$. Therefore, after a day, $N_s(1) = N_s(0)e^{-m_{ss_1}}$, that is,

$$m_{ss_1} = -\ln\left(\frac{N_s(1)}{N_s(0)}\right).$$

Now, $N_s(1) = N_s(0) - P_{ss_1}$, where P_{ss_1} is the number of individuals going from S to S_1 each day. It follows that

$$m_{ss_1} = -\ln\left(1 - \frac{P_{ss_1}}{N_S(0)}\right).$$

This is computed for the pair of cities given in Table 2, using data from the 2011 Canadian census (Arino and Portet 2015).

All parameter descriptions are summarized in Tables 3, 4, 5, 6 and 7, together with their explored ranges. Certain parameters are chosen to have the same values in both cities (patches) because of their geographical proximity. The disease-related parameters are taken to match those of Zika.

7 Numerical Simulations

In this section, we explore from a numerical point of view the potential effects of optimal control strategies with restriction of movements on the dynamics of disease transmission. The optimal control profiles are determined by solving the optimality system consisting of 32 ordinary differential equations, representing the state and adjoint equations.

We deal with the corresponding two-point boundary value problem with boundary conditions at $t = 0$ and $t = T$ by using a fourth-order Runge–Kutta method

Table 3 Description of each variable used for the model

Description	Symbol
Size of the susceptible human population in the urban city	$S(t)$
Size of the exposed human population in the urban city	$E(t)$
Size of the infected human population with mild symptoms in the urban city	$I_a(t)$
Size of the infected human population with severe symptoms in the urban city	$I_b(t)$
Size of the recovered human population in the urban city	$R(t)$
Size of the susceptible human population in the satellite city	$S_1(t)$
Size of the exposed human population in the satellite city	$E_1(t)$
Size of the infected human population with mild symptoms in the satellite city	$I_{a1}(t)$
Size of the infected human population with severe symptoms in the satellite city	$I_{b1}(t)$
Size of the recovered human population in the satellite city	$R_1(t)$
Size of the susceptible mosquito population in the urban city	$S_v(t)$
Size of the exposed mosquito population in the urban city	$E_v(t)$
Size of the infected mosquito population in the urban city	$I_v(t)$
Size of the susceptible mosquito population in the satellite city	$S_{v1}(t)$
Size of the exposed mosquito population in the satellite city	$E_{v1}(t)$
Size of the infected mosquito population in the satellite city	$I_{v1}(t)$

implemented in Matlab. Combining two control measures at a time or, in an instance, combining all three control measures, we then compare the corresponding numerical results. We choose the values for most variables and parameters based on available information from a literature survey as shown in Tables 4, 5, 6 and 7, all other values being either estimated or assumed. The initial conditions used for simulations are given in Table 8. Also, to illustrate the effect of each optimal scenario on the spread of the disease in the population, we use the following cost factors: $c_1 = 85$, $c_2 = 80$, $c_3 = 70$, $c_4 = 75$, $c_5 = 85$, $c_6 = 80$ and $c_7 = 75$. The approach of cost factors is necessary and common in optimal control problems (Okosun et al. 2013; Cai et al. 2017; Zhang et al. 2017; Rodrigues et al. 2014; Ştefănescu and Dimitriu 2012).

Next, we provide a quantitative discussion of the optimal control strategy by investigating different mobility restrictions.

Table 4 Description of variables and parameters used for the urban city (I)

Para-meter	Description	Value	Range explored	Source
β	Contact rate between exposed or mildly infected humans and susceptible humans (day^{-1})	0.05	0.04–0.06	Gao (2016)
κ	Probability of transmission from exposed humans to susceptible humans (dimensionless)	0.3	0–1	Gao (2016)
τ	Probability of transmission from mildly infected humans to susceptible humans (dimensionless)	0.3	0–1	Gao (2016)
γ	Human progression rate from exposed to mildly infected (day^{-1})	0.6	0–1	Gao (2016)
δ	Human progression rate from mildly infected to severely infected (day^{-1})	0.2	0.2–0.24	Bearcroft (1956)
α	Human progression rate from severely infected to recovered (day^{-1})	0.053	0.04–0.07	Musso (2015b)
θ	Probability of transmission from an infectious mosquito to a susceptible human (dimensionless)	0.4	0.1–0.75	Andraud et al. (2012)

Table 5 Description of variables and parameters used for the urban city (II)

Para-meter	Description	Value	Range explored	Source
β_v	Vector biting rate (day^{-1})	0.5	0.3–1	Andraud et al. (2012)
ϕ	Vector contact rate with humans (day^{-1})	0.502	0.34 - 0.52	Blayneh et al. (2009)
λ_v	Probability of transmission from an exposed or mildly infectious human to a susceptible mosquito (dimensionless)	0.5	0.3–0.75	Chikaki and Ishikawa (2009)
κ_v	Vector contact rate with exposed human (day^{-1})	0.5	0.3–0.75	Chikaki and Ishikawa (2009)
τ_v	Vector contact rate with infected human (day^{-1})	0.5	0.3–0.75	Chikaki and Ishikawa (2009)
b	Natural birth rate of human population (day^{-1})	5	1–10	Chitnis et al. (2008)
d	Natural death rate of human population (day^{-1})	0.00004		Nishiura et al. (2016)
b_v	Natural birth rate of the vector population (day^{-1})	5000	400–5000	Andraud et al. (2012)
d_v	Natural death rate of the vector population (day^{-1})	0.02	0.013–0.134	Chitnis et al. (2006)
m	Movement from urban city to satellite city (day^{-1})	0.5	0.25–0.73	Arino and Portet (2015)
ψ_v	Vector progression rate from exposed to infected (day^{-1})	0.091	0.029–0.33	Chitnis et al. (2008)

Table 6 Description of variables and parameters used for the satellite city (I)

Para-meter	Description	Value	Range explored	Source
β_1	Contact rate between exposed or mildly infected humans and susceptible humans (day^{-1})	0.054	0.04–0.06	Gao (2016)
κ_1	Probability of transmission from exposed humans to susceptible humans (dimensionless)	0.3	0–1	Gao (2016)
τ_1	Probability of transmission from mildly infected humans to susceptible humans (dimensionless)	0.3	0–1	Gao (2016)
γ_1	Human progression rate from exposed to mildly infected (day^{-1})	0.6	0–1	Gao (2016)
δ_1	Human progression rate from mildly infected to severely infected (day^{-1})	0.2	0.2–0.21	Bearcroft (1956)
α_1	Human progression rate from severely infected to recovered (day^{-1})	0.05	0.04–0.07	Musso (2015b)
θ_1	Probability of transmission from an infectious mosquito to a susceptible human (dimensionless)	0.4	0.1–0.75	Andraud et al. (2012)

7.1 Control Strategies

- Strategy A: the combination of $u_1(t)$ and $u_2(t)$.
- Strategy B: the combination of $u_1(t)$ and $u_3(t)$.
- Strategy C: the combination of $u_2(t)$ and $u_3(t)$.
- Strategy D: the combination of $u_1(t)$, $u_2(t)$ and $u_3(t)$.

Contour plots of the basic reproduction number in terms of certain significant parameters are shown in Fig. 5. From Panels a, b, it is seen that the movement parameters (m and m_2) act somewhat differently on the dynamics of the disease propagation in the urban city and the satellite city, respectively, as evidenced through their influence upon the value of the respective reproduction numbers. In the meantime, it is observed from Panels c, d that the sexual transmission parameters (κ, τ, κ_1, and τ_1) have similar effects on the dynamics of the disease propagation in the urban city and the satellite city, respectively, as viewed through the same lens of influence upon the value of respective reproduction numbers.

The estimation of the basic reproduction number $R_0^m = \max\{R_{01M}, R_{02M}\}$ is seen to have the value $R_0^m = 2.1126$, where the component of R_0 characterizing sexual transmission is $R_{hh} = 0.1954$ and the component characterizing vector transmission is $R_{hv} = 2.0125$. This implies that vector transmission plays a critical role in the spread of the disease, while sexual transmission is mostly responsible for increasing the prevalence of the disease. Panels e, f show the effects of R_{hh} and R_{hv} on the basic reproduction number R_0.

Table 7 Description of variables and parameters used for the satellite city (II)

Para-meter	Description	Value	Range explored	Source
β_{v1}	Vector biting rate (day^{-1})	0.56	0.3–1	Andraud et al. (2012)
ϕ_1	Vector contact rate with humans (day^{-1})	0.512	0.34–0.52	Blayneh et al. (2009)
λ_{v1}	Probability of transmission from an exposed or mildly infectious human to a susceptible mosquito (dimensionless)	0.51	0.3–0.75	Chikaki and Ishikawa (2009)
κ_{v1}	Vector contact rate with exposed human (day^{-1})	0.54	0.3–0.75	Chikaki and Ishikawa (2009)
τ_{v1}	Vector contact rate with infected human (day^{-1})	0.6	0.3–0.75	Chikaki and Ishikawa (2009)
b_1	Natural birth rate of human population (day^{-1})	3	1–10	Chitnis et al. (2008)
d_1	Natural death rate of human population (day^{-1})	0.00004		Nishiura et al. (2016)
b_{v1}	Natural birth rate of the vector population (day^{-1})	5000	400–5000	Andraud et al. (2012)
d_{v1}	Natural death rate of the vector population (day^{-1})	0.02	0.013–0.134	Chitnis et al. (2006)
m_2	Movement from satellite city to urban city (day^{-1})	0.80954	0.25–1.235	Arino and Portet (2015)
q	Passive movement of severely infected humans from satellite city to urban city (day^{-1})	0.2	0–1	Assumed
ψ_{v1}	Vector progression rate from exposed to infected (day^{-1})	0.091	0.029–0.33	Chitnis et al. (2008)

Table 8 Initial conditions used for simulations

Description	Symbol	$Initial values$
Size of the susceptible human population in the urban city	$S(t)$	125, 000
	$S(t)$	125, 000
Size of the exposed human population in the urban city	$E(t)$	5000
Size of the infected human population with mild symptoms in the urban city	$I_a(t)$	2000
Size of the infected human population with severe symptoms in the urban city	$I_b(t)$	2000
Size of the recovered human population in the urban city	$R(t)$	1000
Size of the susceptible human population in the satellite city	$S_1(t)$	75, 000
Size of the exposed human population in the satellite city	$E_1(t)$	4000
Size of the infected human population with mild symptoms in the satellite city	$I_{a1}(t)$	2000
Size of the infected human population with severe symptoms in the satellite city	$I_{b1}(t)$	2000
Size of the recovered human population in the satellite city	$R_1(t)$	800
Size of the susceptible mosquito population in the urban city	$S_v(t)$	250, 000
Size of the exposed mosquito population in the urban city	$E_v(t)$	50, 000
Size of the infected mosquito population in the urban city	$I_v(t)$	20, 000
Size of the susceptible mosquito population in the satellite city	$S_{v1}(t)$	250, 000
Size of the exposed mosquito population in the satellite city	$E_{v1}(t)$	40, 000
Size of the infected mosquito population in the satellite city	$I_{v1}(t)$	20, 500

From Fig. 6a, it is observed that an increase in the passive movements of infectives leads to an increase in the global basic reproduction number of the simplified model (shown in "Appendix B"). Figure 6b indicates that an increase in the passive movements alone within the specified values will lead to an increase of approximately 10% in the global basic reproduction number.

From the optimal control simulation results (Figs. 7, 8 and 9), it is observed that the combination of the control measures $u_1(t)$ and $u_3(t)$ decreases the size of the infected human population in the urban city compared to the satellite city. This strategy suggests

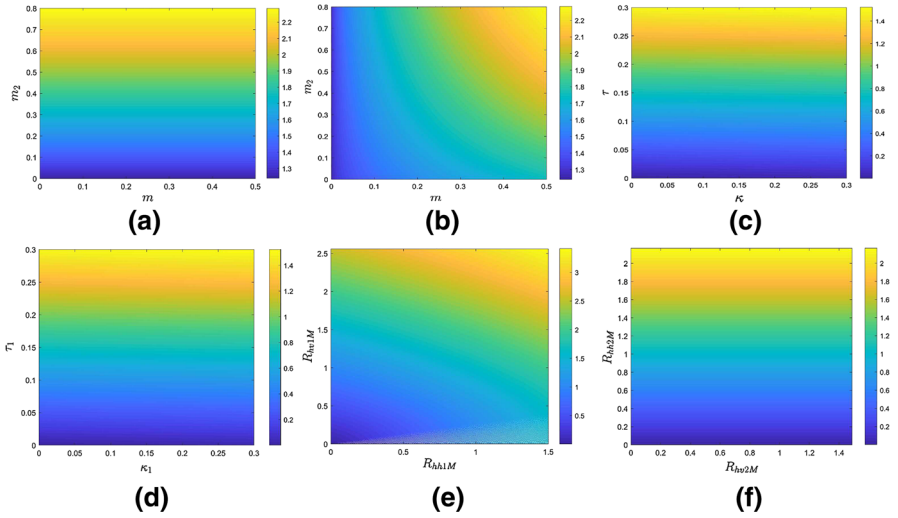

Fig. 5 (Colour figure online) Contour plots for the basic reproduction number in terms of significant parameters. Panel **a** shows the contour plot of R_{01M} in terms of m_2 and m. Panel **b** shows the contour plot of R_{02M} in terms of m_2 and m. Panel **c** shows the contour plot of R_{01M} in terms of κ and τ. Panel **d** shows the contour plot of R_{02M} in terms of κ_1 and τ_1. Panel **e** shows the contour plot of R_{01M} in terms of R_{hv1M} and R_{hh1M}. Panel **f** shows the contour plot of R_{02M} in terms of R_{hv2M} and R_{hh2M}.

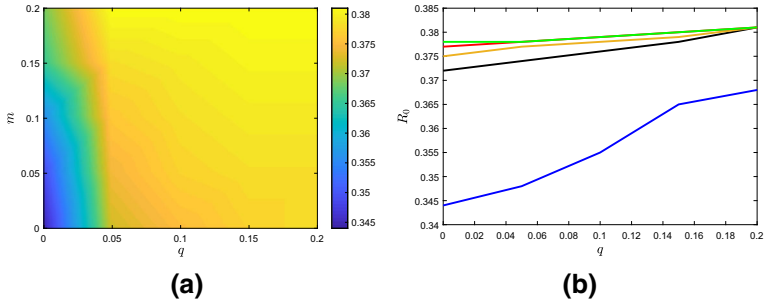

Fig. 6 (Colour figure online) Simulations showing the effects of passive and active mobility on the global basic reproduction number of the simplified model (R_0^s)

that the restriction of movements of individuals from the satellite city into the urban city has a significant effect on the control of the disease in the urban city. Similarly, the combination of the control measures $u_1(t)$ or $u_3(t)$ with $u_2(t)$ visibly decreases the number of the infected human population in the satellite city. This suggests that the restriction of movements of individuals from the urban city into the satellite city has a significant effect on the control of the disease in the satellite city. Total restriction of movements into both cities (the combination of all three control measures $u_1(t)$, $u_2(t)$ and $u_3(t)$) significantly decreases the number of the infected population when compared to the strategies combining only two control measures.

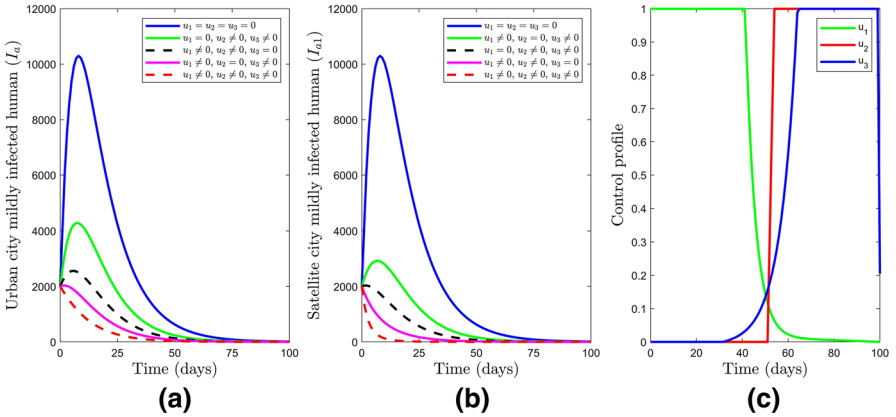

Fig. 7 (Colour figure online) **a**, **b** A comparison between the effects of all four control strategies A, B, C and D on the sizes of the mildly infected human populations in the urban and the satellite cities, respectively. The outcome of the control strategy D is a significant decrease in the disease incidence in both cities. The reduction of the disease incidence is even more prominent in the satellite city when compared to the urban city. This result shows that if both cities adhere to the effective implementation of the strategy D, then the disease incidence will be greatly reduced. The profile of the control is shown in (**c**)

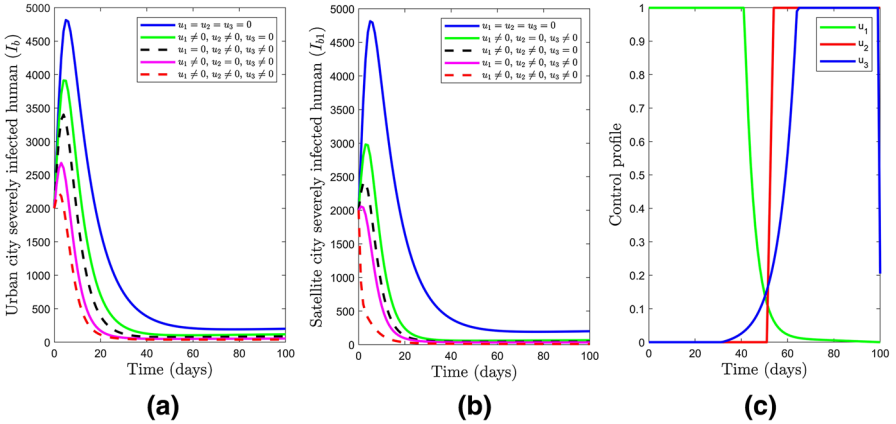

Fig. 8 (Colour figure online) **a**, **b** A comparison between the effects of all four control strategies A, B, C and D on the sizes of the severely infected human populations in the urban and the satellite cities, respectively. The outcome of the control strategy D is a significant decrease in the disease incidence in both cities. The disease incidence is significantly reduced in the satellite city compared to the urban city. This result shows that if both cities adhere to the effective implementation of the strategy D, then the disease incidence will be greatly reduced. The profile of the control is shown in (**c**)

8 Conclusion

A metapopulation model is formulated in order to investigate the role of active and passive mobilities on the spread of an epidemic between an urban city and a satellite city. Mathematically relevant features such as the biologically significant invariant region and the basic reproduction number of the disease for isolated communities and

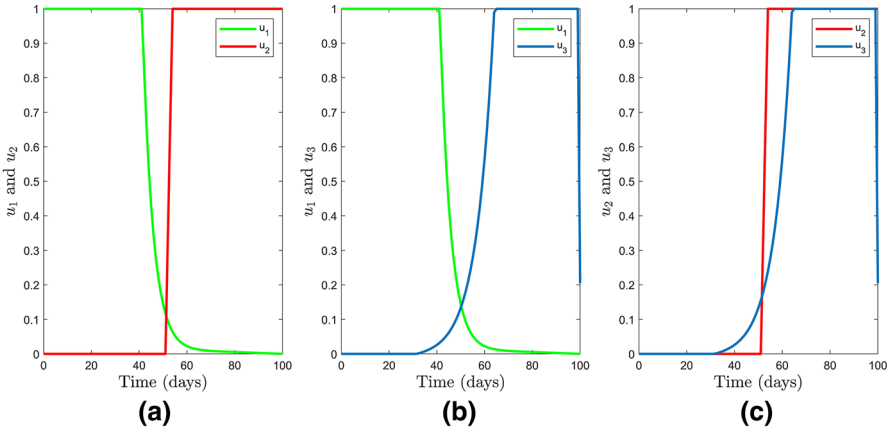

Fig. 9 (Colour figure online) Simulations of the combinations of the control measures u_1, u_2 and u_3

communities connected by migration, respectively, are introduced, and control strategies are investigated. The basic reproduction number of the disease R_0 is explicitly determined as a combination of sexual and vector-borne transmission parameters.

From our sensitivity analysis, we observe that the basic reproduction number of the satellite city (R_{02M}) has a positive correlation with the rate of movement from the urban city to the satellite city (m), which indicates that an increase in the movement rate m will lead to an increase in the basic reproduction number of the satellite city. Similarly, the basic reproduction number of the urban city (R_{01M}) has a positive correlation with the rate of movement from the satellite city to the urban city (m_2), which indicates that increasing the rate of movement from the satellite city to the urban city will increase the basic reproduction number of the urban city. As an increase in the basic reproduction number leads to a corresponding increase in the disease prevalence, this will in turn lead to an increase in the disease prevalence.

It is also observed that the basic reproduction number is least sensitive to the sexual transmission parameter κ, with sensitivity index equal to 0.2, which suggests that sexual transmission by itself may not initiate or sustain an outbreak and can only increase the risk of infection and the size of the epidemic, leading to a complicated scenario for the controlling of the disease. What is then realised from the sensitivity analysis is that in our metapopulation model of a vector-borne disease which is also sexually transmitted, the disease spreads mainly through the vector-borne mode of transmission rather than through sexual transmission, which is attributed to the facts that vectors abound and the vector to human contact rate is larger than both the human to human contact rate and the rate of sexual activity.

In order to analyse the impact of mobility in disease propagation, we further include three time-dependent control measures on the movement of individuals from the urban city to the satellite city and vice versa. It is observed from the simulation results that the optimal control strategies result in a significant decrease in the infected human population in both cities (to be noted that this also depends on the weights chosen for optimal control). When strategies A, C and D are employed, the decrease in the

infected population of the satellite city is more prominent, while when strategy B is employed, the decrease in the infected population in the urban city is more prominent. The best control strategy is strategy D, the combination of all three control measures. Another outcome of the simulations, consistent with the findings of related studies, Arino and van den Driessche (2003); Arino and Portet (2015) the disease prevalence can be controlled by targeting the urban city with major control measures.

The model formulated and analysed in this paper is related to the ones presented in Arino and van den Driessche (2003), Arino and Portet (2015). However, while Arino and van den Driessche (2003) and Arino and Portet (2015) discuss SIR metapopulation models (host–host transmission only, that is, albeit from an arbitrary number of groups/cities), we further consider the effects of vector–host transmission. The basic reproduction number of our model is explicitly computed for a given scenario (without movement), an explicit estimation and a numerical estimation being provided for another scenario (with movement), while in Arino and van den Driessche (2003), Arino and Portet (2015) the basic reproduction number can only be numerically estimated. Also, we incorporate control strategies in our model and investigated the effects of the optimal restrictive measures upon the dynamics of our model, while neither Arino and van den Driessche (2003) nor Arino and Portet (2015) is concerned with control problems. Both our model and the those of Arino and van den Driessche (2003), Arino and Portet (2015) agree in the finding that the movement rates may have a significant impact upon the basic reproduction number of a single city (local basic reproduction number), but their impact upon the global basic reproduction number is smaller. (Particularly, they are not the main driving force of the infection for our model.)

For our model, which considers both host–host transmission and vector–host transmission, we have observed that the main driving force of the disease spread is the vector population. The fact that active movements have comparatively little influence upon the global basic reproduction number of the model indicates that although travel restrictions restriction from the urban city to the satellite city may reduce the prevalence of the disease in the satellite city, significant control measures targeting the densely populated cities will be required in order to eradicate the disease in the entire region.

Like many other mechanistic attempts at distilling the essence of biological processes, our approach has certain limitations. The model presented in this paper assumes homogeneous mixing, which is stringent to achieve in vectors (mosquitoes) and in sexual transmission of a disease. In practice, susceptibility to Zika virus varies, because of differences in behavioural, social and environmental factors. Despite these setbacks, this model presents a unique attempt to link the dynamics of a metapopulation model with both vector and sexual transmission to the use of certain control measures, mathematically and numerically. The model can also be extended by incorporating additional interventions such as behaviour change and media campaigns.

Further, we assumed for the sake of simplicity that no new infections occur during travelling, an assumption which is shared with most usual metapopulation models. There are, however, documented instances of influenza transmission during train transportation (Le 2010; Furuya 2007). Disease propagation is also known to occur on-board aircrafts, although the occurrence risk is regarded as being significantly lower due to

the nature of the modern ventilation systems and being concentrated close to infected passengers with symptoms (Baker 2010) (A/H1N1), (Kenyon 1996) (tuberculosis). See also Mangili and Gendreau (2005) for a generic assessment of transmission risks during commercial air travel. In this regard, models accounting for infections occurring during transportation have been considered in Arino et al. (2016), Knipl (2016) and we see this avenue as another way of extending our model.

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Compliance with Ethical Standards

Conflicts of interest The authors declare no conflicts of interest.

Data Accessibility Estimation of parameters have been stated in the body of the paper and included in the reference section.

9 Appendix A

Model equation

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \beta\frac{\kappa E + \tau I_a}{N_h}S - \frac{\theta\beta_v\phi I_v}{N_h}S - dS + m_2S_1 - mS,\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta\frac{\kappa E + \tau I_a}{N_h}S + \frac{\theta\beta_v\phi I_v}{N_h}S - dE - \gamma E + m_2E_1 - mE,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \delta I_a + m_2I_{a1} - mI_a,\\
\frac{\mathrm{d}I_b}{\mathrm{d}t} &= \delta I_a - \alpha I_b - dI_b + qI_{b1},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I_b - dR + m_2R_1 - mR,\\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= b_v - \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vS_v,\\
\frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vE_v - \psi_vE_v,\\
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_vE_v - d_vI_v,\\
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= b_1 - \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 - \frac{\theta_1\beta_{v1}\phi_1I_{v1}S_1}{N_{h1}} - d_1S_1 - m_2S_1 + mS,\\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 + \frac{\theta_1\beta_{v1}\phi_1I_{v1}S_1}{N_{h1}} - d_1E_1 - \gamma_1E_1 - m_2E_1 + mE,
\end{aligned}
$$

$$
\begin{aligned}
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1 E_1 - d_1 I_{a1} - \delta_1 I_{a1} - m_2 I_{a1} + m I_a,\\
\frac{\mathrm{d}I_{b1}}{\mathrm{d}t} &= \delta_1 I_{a1} - \alpha_1 I_{b1} - d_1 I_{b1} - q I_{b1},\\
\frac{\mathrm{d}R_1}{\mathrm{d}t} &= \alpha_1 I_{b1} - d_1 R_1 - m_2 R_1 + m R,\\
\frac{\mathrm{d}S_{v1}}{\mathrm{d}t} &= b_{v1} - \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}} S_{v1} - d_{v1} S_{v1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}} S_{v1} - d_{v1} E_{v1} - \psi_{v1} E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1} E_{v1} - d_{v1} I_{v1}.
\end{aligned} \tag{8}
$$

Positive Invariance

By Theorem A.4 in Thieme (2003), there exists a unique solution with values in $\mathbb{R}^{16}_+$ that is defined on some interval $[0, a)$ with $a \in (0, \infty)$. If $a < \infty$, then $\limsup_{t\to a-} \overline{N}(t) = \infty$. We add all host equations in the system (3) and then derive the following inequality

$$\frac{\mathrm{d}\overline{N}}{\mathrm{d}t} \le b^* - d^*\overline{N},$$

in which $b^* = b + b_1$ and $d^* = \min\{d, d_1\}$. Assume that there exists a positive number c such that

$$b^* \le \frac{d^*}{2}\overline{N} \text{ whenever } ||(S^T, J)|| = \overline{N} \ge c.$$

By Diekmann and Heesterbeek (2000), $\frac{d\overline{N}(t)}{dt} \le -\frac{d^*}{2}\overline{N}(t)$ whenever $t \in [0, a)$ and $\overline{N}(t) \ge c$. This implies $\overline{N}(t) \le \max\{c, \overline{N}(0)\}$ for all $t \in [0, a)$. So $a = \infty$, and $\limsup_{t\to\infty} \overline{N}(t) \le c$. Similarly, summing the total vector equations together, we have

$$\frac{\mathrm{d}\overline{N}_v}{\mathrm{d}t} \le b_v^* - d_v^*\overline{N}_v,$$

in which $b_v^* = b_v + b_{v1}$ and $d_v^* = \min\{d_v, d_{v1}\}$. If there exists a positive number c_v such that

$$b_v^* \le \frac{d_v^*}{2}\overline{N}_v \quad \text{whenever} \quad ||(S_v^T, J_v)|| = \overline{N}_v \ge c_v,$$

then the total vector population $\overline{N}_v(t) \le \max\{c_v, \overline{N}_v(0)\}$ for all $t \in [0, a)$. So $a = \infty$, and $\limsup_{t\to\infty} \overline{N_v}(t) \le c_v$. We then conclude that system (3) is epidemiologically feasible and mathematically well-posed in $\mathbb{D} = \mathbb{D}_h \cup \mathbb{D}_v \subset \mathbb{R}^{10}_+ \times \mathbb{R}^6_+$, in which $\mathbb{D}_h$

is the domain of the total human population and $\mathbb{D}_v$ is the domain for the total vector population.

The Disease-Free Equilibrium

At the disease-free equilibrium, there is no infection in either the human or the vector population. First, let us assume that there is no movement between the satellite city and the urban city. The system (3) without mobility then has a disease-free equilibrium E^0, given by

$$E^0 = \left(\frac{b}{d}, 0, 0, 0, 0, \frac{b_v}{d_v}, 0, 0, \frac{b_1}{d_1}, 0, 0, 0, 0, \frac{b_{v1}}{d_{v1}}, 0, 0\right) \in \mathbb{R}_+^{16}. \tag{9}$$

To obtain the disease-free equilibrium E_m^0 for the system (3) with mobility, we note first that we are led to solving the equilibrium subsystem associated to the susceptible populations, in the form

$$\begin{aligned} b + m_2 S_1 - (d + m)S &= 0, \\ b_1 + mS - (d_1 + m_2)S_1 &= 0. \end{aligned} \tag{10}$$

This subsystem has the solutions

$$S^{**} = \frac{b(d_1 + m_2) + b_1 m_2}{dd_1 + dm_2 + md_1}, \qquad S_1^{**} = \frac{b_1(d + m) + mb}{dd_1 + dm_2 + md_1}.$$

It then follows that

$$E_m^0 = \left(\frac{b(d_1 + m_2) + b_1 m_2}{dd_1 + dm_2 + md_1}, 0, 0, 0, 0, \frac{b_v}{d_v}, 0, 0, \frac{b_1(d + m) + mb}{dd_1 + dm_2 + md_1}, 0, 0, 0, 0, \frac{b_{v1}}{d_{v1}}, 0, 0\right).$$

Computation of the Basic Reproduction Number R_0

To use the next-generation method, we note that the equations which model the dynamics of the infected compartments E, I_a, I_b, E_v and I_v of the urban city and E_1, I_{a1}, I_{b1}, E_{v1} and I_{v1} of the satellite city in (3) are

$$\begin{aligned} \frac{\mathrm{d}E}{\mathrm{d}t} &= \beta \frac{\kappa E + \tau I_a}{N_h} S + \frac{\theta \beta_v \phi I_v}{N_h} S - dE - \gamma E + m_2 E_1 - mE, \\ \frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \delta I_a + m_2 I_{a1} - mI_a, \\ \frac{\mathrm{d}I_b}{\mathrm{d}t} &= \delta I_a - \alpha I_b - dI_b + qI_{b1}, \\ \frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v \lambda_v \frac{\kappa_v E + \tau_v I_a}{N_h} S_v - d_v E_v - \psi_v E_v, \\ \frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_v E_v - d_v I_v, \end{aligned}$$

$$
\begin{aligned}
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 + \frac{\theta_1 \beta_{v1} \phi_1 I_{v1} S_1}{N_{h1}} - d_1 E_1 - \gamma_1 E_1 - m_2 E_1 + mE,\\
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1 E_1 - d_1 I_{a1} - \delta_1 I_{a1} - m_2 I_{a1} + mI_a,\\
\frac{\mathrm{d}I_{b1}}{\mathrm{d}t} &= \delta_1 I_{a1} - \alpha_1 I_{b1} - d_1 I_{b1} - q I_{b1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1} \lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}} S_{v1} - d_{v1} E_{v1} - \psi_{v1} E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1} E_{v1} - d_{v1} I_{v1}.
\end{aligned}
\tag{11}
$$

To fix our ideas, let us focus on the urban city, that is, on the first five equations of (11). With the notations of van den Driessche and Watmough (2002), $\mathcal{F}$ and $\mathcal{V}$ are given by

$$
\mathcal{F} = \begin{bmatrix} \beta \dfrac{\kappa E + \tau I_a}{N_h} S + \dfrac{\theta \beta_v \phi I_v}{N_h} S \\ 0 \\ 0 \\ \beta_v \lambda_v \dfrac{\kappa_v E + \tau_v I_a}{N_h} S_v \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} dE + \gamma E - m_2 E_1 + mE \\ -\gamma E + dI_a + \delta I_a - m_2 I_{a1} + mI_a \\ -\delta I_a + \alpha I_b + dI_b - q I_{b1} \\ d_v E_v + \psi_v E_v \\ -\psi_v E_v + d_v I_v \end{bmatrix}.
$$

Assuming that $m = m_2 = 0$, the associated Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium are given by

$$
F = \begin{bmatrix} \beta\kappa & \beta\tau & 0 & 0 & \theta\beta_v\phi \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \dfrac{\beta_v \lambda_v \kappa_v b_v d}{b d_v} & \dfrac{\beta_v \lambda_v \tau_v b_v d}{b d_v} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},
$$

$$
V = \begin{bmatrix} (d+\gamma) & 0 & 0 & 0 & 0 \\ -\gamma & (d+\delta) & 0 & 0 & 0 \\ 0 & -\delta & (\alpha+d) & 0 & 0 \\ 0 & 0 & 0 & (d_v+\psi_v) & 0 \\ 0 & 0 & 0 & -\psi_v & d_v \end{bmatrix}.
$$

The basic reproduction number R_0 equals the spectral radius (dominant eigenvalue) of the matrix FV^{-1}, given by:

$$
R_0 = \frac{R_{hh} + \sqrt{R_{hh}^2 + 4R_{hv}^2}}{2},
$$

where

$$R_{hh} = \frac{\beta(\tau\gamma + \kappa(d+\delta))}{(d+\gamma)(d+\delta)},$$

$$R_{hv} = \sqrt{\frac{\theta\beta_v^2\phi\psi_v\lambda_v b_v d\left(\tau_v(d_v+\psi_v)+\kappa_v(d+\delta)\right)}{bd_v^2(d+\gamma)(d+\delta)(d_v+\psi_v)}}.$$

The Basic Reproduction Number for the System with Mobility

If the infection exists in a single community which is connected to another community through population mobility, the phenomenon related to the movements of individuals should be reflected in the disease threshold. When the communities are connected by migration, the community-specific reproduction numbers are given by

$$R_{01M} = \frac{R_{hh1M} + \sqrt{R_{hh1M}^2 + 4R_{hv1M}^2}}{2} \text{ and } R_{02M} = \frac{R_{hh2M} + \sqrt{R_{hh2M}^2 + 4R_{hv2M}^2}}{2}.$$

To compute R_{01M} and R_{02M}, we substitute the disease-free equilibrium points with movement for

$$S^* = \frac{b(d_1+m_2)+b_1m_2}{dd_1+dm_2+md_1}, \quad S_1^* = \frac{b_1(d+m)+mb}{dd_1+dm_2+md_1}, \quad S_v^* = \frac{b_v}{d_v}, \quad S_{v1}^* = \frac{b_{v1}}{d_{v1}}$$

into the Jacobian matrix for F before computing for the eigenvalues of the matrix FV^{-1}. We obtain the following results

$$R_{hh1M} = \frac{\beta(\tau\gamma + \kappa(d+\delta+m))}{(d+\gamma+m)(d+\delta+m)},$$

$$R_{hv1M} = \sqrt{\frac{\theta\beta_v^2\phi\psi_v\lambda_v b_v(dd_1+dm_2+md_1)(\tau_v(d_v+\psi_v)+\kappa_v(d+\delta+m))}{d_v^2(b(d_1+m_2)+b_1m_2)(d+\gamma+m)(d+\delta+m)(d_v+\psi_v)}},$$

$$R_{hh2M} = \frac{\beta_1(\tau_1\gamma_1 + \kappa_1(d_1+\delta_1+m_2))}{(d_1+\gamma_1)(d_1+\delta_1+m_2)},$$

$$R_{hv2M} = \sqrt{\frac{\theta_1\beta_{v1}^2\phi_1\psi_{v1}\lambda_{v1}b_{v1}(dd_1+dm_2+md_1)(\tau_{v1}(d_{v1}+\psi_{v1})+\kappa_{v1}(d_1+\delta_1+m_2))}{d_{v1}^2(b_1(d+m)+mb)(d_1+\gamma_1+m_2)(d_1+\delta_1+m_2)(d_{v1}+\psi_{v1})}}.$$

An estimation of the basic reproduction number, hereby denoted as R_0^m, can then be given as the maximum of the community-specific reproduction numbers

$$R_0^m = \max\{R_{01M}, R_{02M}\}.$$

For a single isolated community, the corresponding persistence condition is $R_0 > 1$, which holds only if $R_{hh} + R_{hv}^2 > 1$. That is,

$$
\begin{aligned}
& R_0 > 1 \\
\Longrightarrow\ & \frac{R_{hh} + \sqrt{R_{hh}^2 + 4R_{hv}^2}}{2} > 1, \\
\Longrightarrow\ & R_{hh} + \sqrt{R_{hh}^2 + 4R_{hv}^2} > 2, \\
\Longrightarrow\ & \sqrt{R_{hh}^2 + 4R_{hv}^2} > 2 - R_{hh}, \\
\Longrightarrow\ & \left(\sqrt{R_{hh}^2 + 4R_{hv}^2}\right)^2 > (2 - R_{hh})^2, \\
\Longrightarrow\ & R_{hh}^2 + 4R_{hv}^2 > 4 - 4R_{hh} + R_{hh}^2, \\
\Longrightarrow\ & 4R_{hh} + 4R_{hv}^2 > 4, \\
\Longrightarrow\ & R_{hh} + R_{hv}^2 > 1.
\end{aligned}
$$

Uniform Strong Disease Persistence and Existence of Endemic Equilibria

Under the assumption of the constant recruitment, it is easy to see that the host is strongly uniformly persistent. Since the recruitment rates b and b_1 are positive constants, $S^T(t) > (0,0)$ for all $t > 0$, and there exist two positive constants δ_1^* and δ_2^* such that

$$\lim_{t\to\infty} \inf S^T(t) \geq (\delta_1^*, \delta_2^*)$$

for all non-negative solutions in model system (3). In fact, by the first subsystem in the system (3)

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \left(d + \frac{\beta(\kappa E + \tau I_a)}{N_h} + \frac{\theta\beta_v\phi I_v}{N_h}\right)S + m_2 S_1 - mS, \\
&> b - \big(d + \beta(\kappa + \tau) + \theta\beta_v\phi\big)S - mS.
\end{aligned}
$$

Then, there exists a $\delta_1^* \in (0, +\infty)$, independent of the solution, such that

$$\lim_{t\to\infty} \inf S(t) \geq \frac{b}{d + \beta(\kappa + \tau) + \theta\beta_v\phi + m} =: \delta_1^*.$$

Similarly, there exists a $\delta_2^* \in (0, +\infty)$ such that

$$\lim_{t\to\infty} \inf S_1(t) \geq \delta_2^*.$$

Since $S^T(t) >> (0,0)$ for $t > 0$, the subsequent persistence results do not need the solutions of system (3) to satisfy $S^T(0) >> (0,0)$. Also, if $R_{0M} > 1$, and all recruitment rates b and b_1 are positive constants, then there exists some $\epsilon > 0$ such that

$$\liminf_{t\to\infty} \; C_i(t) \geq \epsilon, \; i = 1, 2, \; C \in \mathbb{C} \text{ and } \mathrm{C} = (\mathrm{C}_1, \mathrm{C}_2)$$

for all non-negative solutions of system (3) with

$$\left(E^T(0), I_a^T(0), I_b^T(0), E_v^T(0), I_v^T(0)\right) > 0.$$

Let

$$\mathbb{X} = \Big\{(S^T, E^T, I_a^T, I_b^T, R^T, S_v^T, E_v^T, I_v^T) \in (0,\infty)^{16} | (S^T, S_v^T) \in (0, +\infty)^4 \text{ and } (E^T, I_a^T, I_b^T, R^T, E_v^T, I_v^T) \in \mathbb{R}_+^{12}\Big\}.$$

By Theorem A.32 of Thieme (2003), the solution takes its values in $\mathbb{X}$ for $t > 0$. Define $\rho : \mathbb{X} \to \mathbb{R}_+$ by

$$\rho(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = I,$$

for fixed $I \in \{I_a, I_{a1}, I_b, I_{b1}, I_v, I_{v1}\}$, and $\widetilde{\rho} : \mathbb{X} \to \mathbb{R}_+$ by

$$\widetilde{\rho}(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = \frac{I_a + I_b}{N_h} + \frac{I_{a1} + I_{b1}}{N_{h1}} + \frac{I_v + I_{v1}}{\overline{N_v}}.$$

In the language Sect. A.5 of Thieme (2003), the semiflow Φ induced by the solutions of system (3) is uniformly weakly ρ-persistent by Theorem 4.3 in Dhirasakdanon et al. (2007). The compactness condition in Sect. A.5 of Thieme (2003) follows from the known results above. Notice that every total orbit $\omega : \mathbb{R} \to X$ of Φ is associated with a solution of system (3) that is defined for all times and takes value in $\mathbb{X}$. By the irreducibility of the matrix $\begin{pmatrix} 0 & m \\ m_2 & 0 \end{pmatrix}$, $\widetilde{\rho}(\omega(0)) > 0$ whenever $\rho(\omega(t)) > 0$ for all $t \in \mathbb{R}$. The claim for $C \in \{I_a^T, I_b^T, I_v^T\}$ now follows from Theorem A.34 in Thieme (2003). For $C \in \{E^T, R^T, E_v^T\}$, modify $\widetilde{\rho}(S^T, E^T, I_a^T, I_b^T, R^T, N_v) = C_i$. For $C = S^T$, the statement has already been shown in the content above. Similarly, for $C = S_v^T$, the statement should be easily shown. The existence of an (endemic) equilibrium of system (3) in $(0,\infty)^{16}$ follows from Theorem 1.3.7. in Dhirasakdanon et al. (2007).

Optimal Control Strategies

We use Pontryagin's maximum principle to determine the necessary conditions for optimal control of the epidemic disease. We incorporate three time-dependent control variables into the model (3) to determine the optimal strategy for controlling the disease. The model (3) then becomes

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \beta\frac{\kappa E + \tau I_a}{N_h}S - \frac{\theta\beta_v\phi I_v}{N_h}S - dS + (1-u_1(t))m_2S_1 - (1-u_2(t))mS,\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta\frac{\kappa E + \tau I_a}{N_h}S + \frac{\theta\beta_v\phi I_v}{N_h}S - dE - \gamma E + (1-u_1(t))m_2E_1\\
&\quad - (1-u_2(t))mE,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \delta I_a + (1-u_1(t))m_2I_{a1} - (1-u_2(t))mI_a,\\
\frac{\mathrm{d}I_b}{\mathrm{d}t} &= \delta I_a - \alpha I_b - dI_b + (1-u_3(t))qI_{b1},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I_b - dR + (1-u_1(t))m_2R_1 - (1-u_2(t))mR,\\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= b_v - \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vS_v,\\
\frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vE_v - \psi_vE_v,\\
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_vE_v - d_vI_v,\\
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= b_1 - \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 - \frac{\theta_1\beta_{v1}\phi_1I_{v1}}{N_{h1}}S_1 - d_1S_1 - (1-u_1(t))m_2S_1\\
&\quad + (1-u_2(t))mS,\\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 + \frac{\theta_1\beta_{v1}\phi_1I_{v1}}{N_{h1}}S_1 - d_1E_1 - \gamma_1E_1 - (1-u_1(t))m_2E_1\\
&\quad + (1-u_2(t))mE,\\
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1E_1 - d_1I_{a1} - \delta_1I_{a1} - (1-u_1(t))m_2I_{a1} + (1-u_2(t))mI_a,\\
\frac{\mathrm{d}I_{b1}}{\mathrm{d}t} &= \delta_1I_{a1} - \alpha_1I_{b1} - d_1I_{b1} - (1-u_2(t))qI_{b1},\\
\frac{\mathrm{d}R_1}{dt} &= \alpha_1I_{b1} - d_1R_1 - (1-u_1(t))m_2R_1 + (1-u_2(t))mR,\\
\frac{\mathrm{d}S_{v1}}{\mathrm{d}t} &= b_{v1} - \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}S_{v1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}E_{v1} - \psi_{v1}E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1}E_{v1} - d_{v1}I_{v1}.
\end{aligned}
\tag{12}
$$

The control variables, $u_1(t)$, $u_2(t)$ and $u_3(t)$, are bounded, Lebesgue integrable functions. Our control problem involves a situation in which the number of mildly infectious individuals, severe infected individuals and the cost of applying screening control $u_1(t)$, $u_2(t)$ and $u_3(t)$ are minimized subject to the system (12). The objective function is defined as

$$J(u_1, u_2, u_3) = \int_0^T \left[c_1 I_a + c_2 I_b + c_3 I_{a1} + c_4 I_{b1} + c_5 u_1^2 + c_6 u_2^2 + c_7 u_3^2\right] \mathrm{d}t, \tag{13}$$

where I_a, I_{a1}, I_b and I_{b1} are the total infected human populations, T is the final time and the coefficients $c_1, c_2, c_3, c_4, c_5, c_6, c_7$ are positive weights. Our aim is to minimize the total number of infected humans and minimize the costs of control mechanisms $u_1(t)$, $u_2(t)$ and $u_3(t)$ at the same time. Thus, we search for an optimal control $(u_1^*,\ u_2^*,\ u_3^*)$ such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{u_1,\ u_2,\ u_3} \{J(u_1, u_2, u_3) | u_1, u_2, u_3 \in \Omega\}, \tag{14}$$

where the control set

$$\Omega = \{(u_1,\ u_2,\ u_3) | u_i :\ [0, T\ ] \to\ [0, 1\ ] \text{ Lebesgue measurable},\quad i = 1,\ 2,\ 3\}.$$

The existence of an optimal control is a result of the convexity of the integrand of J with respect to u_1, u_2 and u_3, a priori boundedness of the state variables, and the Lipschitz property of the state system with regard to the state variables. The Pontryagin's maximum principle (Pontryagin et al. 1962) converts the equation (12) and the equation (13) into a problem of minimizing a Hamiltonian H with respect to u_1, u_2 and u_3.

$$\begin{aligned}
H &= c_1 I_a + c_2 I_b + c_3 I_{a1} + c_4 I_{b1} + c_5 u_1^2 + c_6 u_2^2 + c_7 u_3^2, \\
&+ \lambda_S \left\{ b - \beta \frac{\kappa E + \tau I_a}{N_h} S - \frac{\theta \beta_v \phi I_v}{N_h} S - dS + (1 - u_1(t)) m_2 S_1 - (1 - u_2(t)) m S \right\}, \\
&+ \lambda_E \left\{ \beta \frac{\kappa E + \tau I_a}{N_h} S + \frac{\theta \beta_v \phi I_v}{N_h} S - dE - \gamma E + (1 - u_1(t)) m_2 E_1 - (1 - u_2(t)) m E \right\}, \\
&+ \lambda_{I_a} \{\gamma E - d I_a - \delta I_a + (1 - u_1(t)) m_2 I_{a1} - (1 - u_2(t)) m I_a\}, \\
&+ \lambda_{I_b} \{\delta I_a - \alpha I_b - d I_b + (1 - u_3(t)) q I_{b1}\}, \\
&+ \lambda_R \{\alpha I_b - dR + (1 - u_1(t)) m_2 R_1 - (1 - u_2(t)) m R\}, \\
&+ \lambda_{S_v} \left\{ b_v - \beta_v \lambda_v \frac{\kappa_v E + \tau_v I_a}{N_h} S_v - d_v S_v \right\}, \\
&+ \lambda_{E_v} \left\{ \beta_v \lambda_v \frac{\kappa_v E + \tau I_a}{N_h} S_v - d_v E_v - \psi_v E_v \right\}, \\
&+ \lambda_{I_v} \{\psi_v E_v - d_v I_v\}, \\
&+ \lambda_{S_1} \left\{ b_1 - \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 - \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} S_1 - d_1 S_1 \right. \\
&\left. -(1 - u_1(t)) m_2 S_1 + (1 - u_2(t)) m S \right\}, \\
&+ \lambda_{E_1} \left\{ \beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} S_1 + \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} S_1 - d_1 E_1 - \gamma_1 E_1 \right. \\
&\left. -(1 - u_1(t)) m_2 E_1 + (1 - u_2(t)) m E) \right\}, \\
&+ \lambda_{I_{a1}} \{\gamma_1 E_1 - d_1 I_{a1} - \delta_1 I_{a1} - (1 - u_1(t)) m_2 I_{a1} + (1 - u_2(t)) m I_a\},
\end{aligned}$$

$$
\begin{aligned}
&+\lambda_{I_{b1}}\left\{\delta_1 I_{a1}-\alpha_1 I_{b1}-d_1 I_{b1}-(1-u_3(t))q I_{b1}\right\},\\
&+\lambda_{R_1}\left\{\alpha_1 I_{b1}-d_1 R_1-(1-u_1(t))m_2 R_1+(1-u_2(t))mR\right\},\\
&+\lambda_{S_{v1}}\left\{b_{v1}-\beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1+\tau_{v1}I_{a1}}{N_{h1}}S_{v1}-d_{v1}S_{v1}\right\},\\
&+\lambda_{E_{v1}}\left\{\beta_{v1}\lambda_{v1}\frac{\kappa_v E_1+\tau_v I_{a1}}{N_{h1}}S_{v1}-d_{v1}E_{v1}-\psi_{v1}E_{v1}\right\},\\
&+\lambda_{I_{v1}}\left\{\psi_{v1}E_{v1}-d_{v1}I_{v1}\right\},
\end{aligned}
\tag{15}
$$

where $\lambda_S, \lambda_E, \lambda_{I_a}, \lambda_{I_b}, \lambda_R, \lambda_{S_v}, \lambda_{E_v}, \lambda_{I_v}, \lambda_{S_1}, \lambda_{E_1}, \lambda_{I_{a1}}, \lambda_{I_{b1}}, \lambda_{R_1}, \lambda_{S_{v1}}, \lambda_{E_{v1}}$ and $\lambda_{I_{v1}}$ are the adjoint variables or co-state variables. The system of equations is found by taking the appropriate partial derivatives of the Hamiltonian (15) with respect to the associated state variables.

$$
\begin{aligned}
-\frac{\mathrm{d}\lambda_S}{\mathrm{d}t} &= \left[\beta\frac{\kappa E+\tau I_a}{N_h}\left(1-\frac{S}{N_h}\right)+\frac{\theta\beta_v\phi I_v}{N_h}\left(1-\frac{S}{N_h}\right)+d+(1-u_2(t))m\right]\lambda_S\\
&\quad-\left[\beta\frac{\kappa E+\tau I_a}{N_h}\left(1-\frac{S}{N_h}\right)+\frac{\theta\beta_v\phi I_v}{N_h}\left(1-\frac{S}{N_h}\right)\right]\lambda_E,\\
&\quad+\left[\beta_v\lambda_v\frac{\kappa_v E+\tau_v I_a}{N_h^2}S_v\right](\lambda_{S_v}-\lambda_{E_v})-(1-u_2(t))m\lambda_{S_1},\\
-\frac{\mathrm{d}\lambda_E}{\mathrm{d}t} &= \left[\beta\frac{\kappa}{N_h}\left(1-\frac{E}{N_h}\right)\right]\lambda_S-\left[\beta\frac{\kappa}{N_h}\left(1-\frac{E}{N_h}\right)+d+\gamma+(1-u_2(t))m\right]\lambda_E\\
&\quad-\gamma\lambda_{I_a}+\left[\beta_v\lambda_v\frac{\kappa_v}{N_h}\left(1-\frac{E}{N_h}\right)\right]\lambda_{S_v}-\left[\beta_v\lambda_v\frac{\kappa_v}{N_h}\left(1-\frac{E}{N_h}\right)\right]\lambda_{E_v}\\
&\quad-(1-u_2(t))m\lambda_{E_1},\\
-\frac{\mathrm{d}\lambda_{I_a}}{\mathrm{d}t} &= -c_1+\left[\beta\frac{\tau}{N_h}\left(1-\frac{I_a}{N_h}\right)\right]\lambda_S-\left[\beta\frac{\tau}{N_h}\left(1-\frac{I_a}{N_h}\right)\right]\lambda_E\\
&\quad+\left[(d+\delta)+(1-u_2(t))m\right]\lambda_{I_a}-\delta\lambda_{I_b}+\left[\beta_v\lambda_v\frac{\tau_v}{N_h}\left(1-\frac{I_a}{N_h}\right)\right]\lambda_{S_v}\\
&\quad-\left[\beta_v\lambda_v\frac{\tau_v}{N_h}\left(1-\frac{I_a}{N_h}\right)\right]\lambda_{E_v}-(1-u_2(t))m\lambda_{I_{a1}},\\
-\frac{\mathrm{d}\lambda_{I_b}}{\mathrm{d}t} &= -c_2+(\alpha+d)\lambda_{I_b}-\alpha\lambda_R,\\
-\frac{\mathrm{d}\lambda_R}{\mathrm{d}t} &= (d+(1-u_2(t))m)\lambda_R-(1-u_2(t))m\lambda_{R_1},\\
-\frac{\mathrm{d}\lambda_{S_v}}{\mathrm{d}t} &= \left[\beta_v\lambda_v\frac{\kappa_v E+\tau_v I_a}{N_h}+d_v\right]\lambda_{S_v}-\left[\beta_v\lambda_v\frac{\kappa_v E+\tau_v I_a}{N_h}\right]\lambda_{E_v},\\
-\frac{\mathrm{d}\lambda_{E_v}}{\mathrm{d}t} &= \left[d_v+\psi_v\right]\lambda_{E_v}-\psi_v\lambda_{I_v},\\
-\frac{\mathrm{d}\lambda_{I_v}}{\mathrm{d}t} &= (d_v)\lambda_{I_v}+\left[\frac{\theta\beta_v\phi}{N_h}S\right]\lambda_S-\left[\frac{\theta\beta_v\phi}{N_h}S\right]\lambda_E,
\end{aligned}
$$

$$
\begin{aligned}
\frac{\mathrm{d}\lambda_{S_1}}{\mathrm{d}t} &= \left[\beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} \left(1 - \frac{S_1}{N_{h1}}\right) + \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} \left(1 - \frac{S_1}{N_{h1}}\right) \right. \\
&\quad \left. + d_1 + (1 - u_1(t)) m_2 \right] \lambda_{S_1} \\
&\quad - \left[\beta_1 \frac{\kappa_1 E_1 + \tau_1 I_{a1}}{N_{h1}} \left(1 - \frac{S_1}{N_{h1}}\right) + \frac{\theta_1 \beta_{v1} \phi_1 I_{v1}}{N_{h1}} \left(1 - \frac{S_1}{N_{h1}}\right)\right] \lambda_{E_1} \\
&\quad + \left[\beta_{v1} \lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}^2} S_{v1}\right] (\lambda_{S_{v1}} - \lambda_{E_{v1}}) - (1 - u_1(t)) m_2 \lambda_S, \\
-\frac{\mathrm{d}\lambda_{E_1}}{\mathrm{d}t} &= \left[\beta_1 \frac{\kappa_1}{N_{h1}} \left(1 - \frac{E_1}{N_{h1}}\right)\right] \lambda_{S_1} - \left[\beta_1 \frac{\kappa_1}{N_{h1}} \left(1 - \frac{E_1}{N_{h1}}\right) \right. \\
&\quad \left. + d_1 + \gamma_1 + (1 - u_1(t)) m_2 \right] \lambda_{E_1} \\
&\quad - \gamma_1 \lambda_{I_{a1}} + \left[\beta_{v1} \lambda_{v1} \frac{\kappa_{v1}}{N_{h1}} \left(1 - \frac{E_1}{N_{h1}}\right)\right] (\lambda_{S_{v1}} - \lambda_{E_{v1}}) - (1 - u_1(t)) m_2 \lambda_E, \\
-\frac{\mathrm{d}\lambda_{I_{a1}}}{\mathrm{d}t} &= -c_3 + \left[\beta_1 \frac{\tau_1}{N_{h1}} \left(1 - \frac{I_{a1}}{N_{h1}}\right)\right] \lambda_{S_1} - \left[\beta_1 \frac{\tau_1}{N_{h1}} (1 - \frac{I_{a1}}{N_{h1}})\right] \lambda_{E_1} \\
&\quad + \left[d_1 + \delta_1 + (1 - u_1(t)) m_2\right] \lambda_{I_{a1}} - \delta_1 \lambda_{I_{b1}} \\
&\quad + \left[\beta_{v1} \lambda_{v1} \frac{\tau_{v1}}{N_{h1}} \left(1 - \frac{I_{a1}}{N_{h1}}\right)\right] (\lambda_{S_{v1}} - \lambda_{E_{v1}}) - \left[(1 - u_1(t)) m_2\right] \lambda_{I_a}, \\
-\frac{\mathrm{d}\lambda_{I_{b1}}}{\mathrm{d}t} &= -c_4 + \left[\alpha_1 + d_1 + (1 - u_3(t) q\right] \lambda_{I_{b1}} - \left[(1 - u_3(t)) q\right] \lambda_{I_b} - \alpha_1 \lambda_{R_1}, \\
-\frac{\mathrm{d}\lambda_{R_1}}{\mathrm{d}t} &= (d_1 + (1 - u_1(t)) m_2) \lambda_{R_1} - \left[(1 - u_1(t)) m_2\right] \lambda_R, \\
-\frac{\mathrm{d}\lambda_{S_{v1}}}{\mathrm{d}t} &= \left[\beta_{v1} \lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}} + d_{v1}\right] \lambda_{S_{v1}} - \left[\beta_{v1} \lambda_{v1} \frac{\kappa_{v1} E_1 + \tau_{v1} I_{a1}}{N_{h1}}\right] \lambda_{E_{v1}}, \\
-\frac{\mathrm{d}\lambda_{E_{v1}}}{\mathrm{d}t} &= \left[d_{v1} + \psi_{v1}\right] \lambda_{E_{v1}} - \psi_{v1} \lambda_{I_{v1}}, \\
-\frac{\mathrm{d}\lambda_{I_{v1}}}{\mathrm{d}t} &= (d_{v1}) \lambda_{I_{v1}} + \left[\frac{\theta_1 \beta_{v1} \phi_{v1} S_1}{N_{h1}}\right] \lambda_{S_1} - \left[\frac{\theta_1 \beta_{v1} \phi_{v1} S_1}{N_{h1}}\right] \lambda_{E_1}.
\end{aligned}
$$

Furthermore, the transversality conditions are

$$
\begin{aligned}
&\lambda_S(T) = \lambda_E(T) = \lambda_{I_a}(T) = \lambda_{I_b}(T) = \lambda_R(T) = \lambda_{S_v}(T) = \lambda_{E_v}(T) = \lambda_{I_v}(T) \\
&\quad = \lambda_{S_1}(T) = \lambda_{E_1}(T) = \lambda_{I_{a1}}(T) = \lambda_{I_{b1}}(T) = \lambda_{R_1}(T) = \lambda_{S_{v1}}(T) = \lambda_{E_{v1}}(T) \\
&\quad = \lambda_{I_{v1}}(T) = 0.
\end{aligned}
$$

Finally, since in our optimal control problem, there are no terminal value for the state variable, we give transversality conditions at the final time T by

$$
\lambda_i(T) = 0, i = 1, 2, 3.
$$

On the interior of the control set, where $0 < u_i < 1$, for $i = 1,\ 2,\ 3$, we have

$$\begin{aligned}
\frac{\partial H}{\partial u_1} &= 2c_5u_1 - m_2S_1\lambda_S - m_2E_1\lambda_E - m_2I_{a1}\lambda_{I_a} - m_2R_1\lambda_R + m_2S_1\lambda_{S_1} \\
&\quad + m_2E_1\lambda_{E_1} + m_2I_{a1}\lambda_{I_{a1}} + m_2R_1\lambda_{R_1} = 0, \\
\frac{\partial H}{\partial u_2} &= 2c_6u_2 + mS\lambda_S + mE\lambda_E + mI_a\lambda_{I_a} + mR\lambda_R - mS\lambda_{S_1} - mE\lambda_{E_1} \\
&\quad - mI_a\lambda_{I_{a1}} - mR\lambda_{R_1} = 0, \\
\frac{\partial H}{\partial u_3} &= 2c_7u_3 - qI_{b1}\lambda_{I_b} + qI_{b1}\lambda_{I_{b1}} = 0.
\end{aligned}$$

We obtain

$$\begin{aligned}
u_1^{**} &= \frac{1}{2c_5}\Big[m_2S_1\lambda_S + m_2E_1\lambda_E + m_2I_{a1}\lambda_{I_a} + m_2R_1\lambda_R - m_2S_1\lambda_{S_1} - m_2E_1\lambda_{E_1} \\
&\quad - m_2I_{a1}\lambda_{I_{a1}} - m_2R_1\lambda_{R_1}\Big], \\
u_2^{**} &= \frac{1}{2c_6}\Big[-mS\lambda_S - mE\lambda_E - mI_a\lambda_{I_a} - mR\lambda_R + mS\lambda_{S_1} + mE\lambda_{E_1} \\
&\quad + mI_a\lambda_{I_{a1}} + mR\lambda_{R_1}\Big], \\
u_3^{**} &= \frac{1}{2c_7}\Big[qI_{b1}\lambda_{I_b} - qI_{b1}\lambda_{I_{b1}}\Big].
\end{aligned}$$

By the standard control arguments involving the bounds on the control variables, we conclude that

$$u_1^* = \begin{cases} 0 & \text{if } u_1^{**} \le 0 \\ u_1^{**} & \text{if } 0 < u_1^{**} < 1, \\ 1 & \text{if } u_1^{**} \ge 1 \end{cases}$$

$$u_2^* = \begin{cases} 0 & \text{if } u_2^{**} \le 0 \\ u_2^{**} & \text{if } 0 < u_2^{**} < 1, \\ 1 & \text{if } u_2^{**} \ge 1 \end{cases}$$

$$u_3^* = \begin{cases} 0 & \text{if } u_3^{**} \le 0 \\ u_3^{**} & \text{if } 0 < u_3^{**} < 1 \\ 1 & \text{if } u_3^{**} \ge 1 \end{cases},$$

that is,

$$\begin{aligned}
u_1^* &= \min\left\{1, \max\left(0, u_1^{**}\right)\right\}, \\
u_2^* &= \min\left\{1, \max\left(0, u_2^{**}\right)\right\}, \\
u_3^* &= \min\left\{1, \max\left(0, u_3^{**}\right)\right\}.
\end{aligned}$$

10 Appendix B

Consider a simplified model with a single category of infectives for each location and no active movements for infectives, in the form.

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - \beta\frac{\kappa E + \tau I_a}{N_h}S - \frac{\theta\beta_v\phi I_v}{N_h}S - dS + m_2S_1 - mS,\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta\frac{\kappa E + \tau I_a}{N_h}S + \frac{\theta\beta_v\phi I_v}{N_h}S - dE - \gamma E + m_2E_1 - mE,\\
\frac{\mathrm{d}I_a}{\mathrm{d}t} &= \gamma E - dI_a - \alpha I_a + qI_{a1},\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \alpha I_a - dR + m_2R_1 - mR,\\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= b_v - \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vS_v,\\
\frac{\mathrm{d}E_v}{\mathrm{d}t} &= \beta_v\lambda_v\frac{\kappa_v E + \tau_v I_a}{N_h}S_v - d_vE_v - \psi_vE_v,\\
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= \psi_vE_v - d_vI_v,\\
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= b_1 - \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 - \frac{\theta_1\beta_{v1}\phi_1I_{v1}S_1}{N_{h1}} - d_1S_1 - m_2S_1 + mS,\\
\frac{\mathrm{d}E_1}{\mathrm{d}t} &= \beta_1\frac{\kappa_1E_1 + \tau_1I_{a1}}{N_{h1}}S_1 + \frac{\theta_1\beta_{v1}\phi_1I_{v1}S_1}{N_{h1}} - d_1E_1 - \gamma_1E_1 - m_2E_1 + mE,\\
\frac{\mathrm{d}I_{a1}}{\mathrm{d}t} &= \gamma_1E_1 - d_1I_{a1} - \alpha_1I_{a1} - qI_{a1},\\
\frac{\mathrm{d}R_1}{\mathrm{d}t} &= \alpha_1I_{a1} - d_1R_1 - m_2R_1 + mR,\\
\frac{\mathrm{d}S_{v1}}{\mathrm{d}t} &= b_{v1} - \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}S_{v1},\\
\frac{\mathrm{d}E_{v1}}{\mathrm{d}t} &= \beta_{v1}\lambda_{v1}\frac{\kappa_{v1}E_1 + \tau_{v1}I_{a1}}{N_{h1}}S_{v1} - d_{v1}E_{v1} - \psi_{v1}E_{v1},\\
\frac{\mathrm{d}I_{v1}}{\mathrm{d}t} &= \psi_{v1}E_{v1} - d_{v1}I_{v1}.
\end{aligned}
\tag{16}
$$

Using the next-generation method, we obtain F and V as follows:

$$
F = \begin{bmatrix}
\beta\kappa & \beta\tau & 0 & \theta\beta_v\phi & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
\frac{\beta_v\lambda_v\kappa_vb_vd}{bd_v} & \frac{\beta_v\lambda_v\tau_vb_vd}{bd_v} & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \beta_1\kappa_1 & \beta_1\tau_1 & 0 & \theta_1\beta_{v1}\phi_1\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \frac{\beta_{v1}\lambda_{v1}\kappa_{v1}b_{v1}d_1}{b_1d_{v1}} & \frac{\beta_{v1}\lambda_{v1}\tau_{v1}b_{v1}d_1}{b_1d_{v1}} & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix},
$$

$$V = \begin{bmatrix} (d+\gamma+m) & 0 & 0 & 0 & -m_2 & 0 & 0 & 0 \\ -\gamma & (d+\alpha) & 0 & 0 & 0 & -q & 0 & 0 \\ 0 & 0 & (d_v+\psi_v) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\psi_v & d_v & 0 & 0 & 0 & 0 \\ -m & 0 & 0 & 0 & (d_1+\gamma_1+m_2) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma & (d_1+\alpha_1+q) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & (d_{v1}+\psi_{v1}) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\psi_{v1} & d_{v1} \end{bmatrix}.$$

The matrix FV^{-1} is given by

$$FV^{-1} = \begin{bmatrix} a & b & 0 & c & d & e & f & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ g & h & 0 & 0 & k & l & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ m & 0 & 0 & 0 & n & p & q & r \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ s & 0 & 0 & 0 & t & u & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

where

$$a = \frac{\beta\,\kappa\ (d_1+\gamma_1+m_2)}{dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1} + \frac{\beta\,\tau\ ((d_1+m_2+q+\alpha_1+\gamma_1)d_1\,\gamma\ +\gamma\,m_2\,q+\gamma\,m_2\,\alpha_1+\gamma\,q\gamma_1+\gamma\,\alpha_1\,\gamma_1+mq\gamma_1)}{(\alpha+d)\,(dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)\,(d_1+\alpha_1+q)},$$

$$b = \frac{\beta\,\tau}{\alpha+d},$$

$$c = \frac{\theta\,\beta_v\,\phi\,\psi_v}{(d_v+\psi_v)\,d_v},$$

$$d = \frac{\theta\,\beta_v\,\phi}{d_v},$$

$$e = \frac{\beta\,\kappa\,m_2}{dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1} + \frac{\beta\,\tau\ (dq\gamma_1+d_1\,\gamma\,m_2+\gamma\,m_2\,q+\gamma\,m_2\,\alpha_1+\gamma\,q\gamma_1+mq\gamma_1)}{(\alpha+d)\,(dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)\,(d_1+\alpha_1+q)},$$

$$f = \frac{\beta\,\tau\,q}{(d_1+\alpha_1+q)\,(\alpha+d)},$$

$$g = \frac{\beta_v\,\lambda_v\,\kappa_v\,b_v\,d\,(d_1+\gamma_1+m_2)}{bd_v\ (dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)} + \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,d\,((d_1+m_2+q+\alpha_1+\gamma_1)d_1\,\gamma\ +\gamma\,m_2\,q+\gamma\,m_2\,\alpha_1+\gamma\,q\gamma_1+\gamma\,\alpha_1\,\gamma_1+mq\gamma_1)}{bd_v\ (\alpha+d)\,(dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)\,(d_1+\alpha_1+q)},$$

$$h = \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,d}{bd_v\ (\alpha+d)},$$

$$k = \frac{\beta_v\,\lambda_v\,\kappa_v\,b_v\,dm_2}{bd_v\ (dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)} + \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,d\,(dq\gamma_1+d_1\,\gamma\,m_2+\gamma\,m_2\,q+\gamma\,m_2\,\alpha_1+\gamma\,q\gamma_1+mq\gamma_1)}{bd_v\ (\alpha+d)\,(dd_1+dm_2+d\gamma_1+d_1\,\gamma+d_1\,m+\gamma\,m_2+\gamma\,\gamma_1+m\gamma_1)\,(d_1+\alpha_1+q)},$$

$$l = \frac{\beta_v\,\lambda_v\,\tau_v\,b_v\,dq}{bd_v\ (d_1+\alpha_1+q)\,(\alpha+d)},$$

$$
\begin{aligned}
m &= \frac{\beta_1\,\kappa_1\,m}{dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1} \\
&\quad + \frac{\beta_1\,\tau_1\,\gamma_1\,m}{(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)\,(d_1 + \alpha_1 + q)}, \\
n &= \frac{\beta_1\,\kappa_1\,(d + \gamma + m)}{dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1} \\
&\quad + \frac{\beta_1\,\tau_1\,\gamma_1\,(d + \gamma + m)}{(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)\,(d_1 + \alpha_1 + q)}, \\
p &= \frac{\beta_1\,\tau_1}{d_1 + \alpha_1 + q}, \\
q &= \frac{\theta_1\,\beta_{1v}\,\phi_1\,\psi_{v1}}{(d_{v1} + \psi_{v1})\,d_{v1}}, \\
r &= \frac{\theta_1\,\beta_{1v}\,\phi_1}{d_{v1}}, \\
s &= \frac{\beta_{v1}\,\lambda_{v1}\,\kappa_{v1}\,b_{v1}\,d_1\,m}{b_1\,d_{v1}\,(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)} \\
&\quad + \frac{\beta_{v1}\,\lambda_{v1}\,\tau_{v1}\,b_{v1}\,d_1\,\gamma_1\,m}{b_1\,d_{v1}\,(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)\,(d_1 + \alpha_1 + q)}, \\
t &= \frac{\beta_{v1}\,\lambda_{v1}\,\kappa_{v1}\,b_{v1}\,d_1\,(d + \gamma + m)}{b_1\,d_{v1}\,(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)} \\
&\quad + \frac{\beta_{v1}\,\lambda_{v1}\,\tau_{v1}\,b_{v1}\,d_1\,\gamma_1\,(d + \gamma + m)}{b_1\,d_{v1}\,(dd_1 + dm_2 + d\gamma_1 + d_1\,\gamma + d_1\,m + \gamma\,m_2 + \gamma\,\gamma_1 + m\gamma_1)\,(d_1 + \alpha_1 + q)}, \\
u &= \frac{\beta_{v1}\,\lambda_{v1}\,\tau_{v1}\,b_{v1}\,d_1}{b_1\,d_{v1}\,(d_1 + \alpha_1 + q)}.
\end{aligned}
$$

The basic reproduction number R_0^s (we use the superscript s to denote the fact that we refer to the simplified model) is the spectral radius (dominant eigenvalue) of the matrix FV^{-1}. We substitute different values of the passive (q) and active (m and m_2) movement rates to obtain the associated values of R_0^s. The effects of the passive movement on the global basic reproduction number R_0^s are shown in Fig. 6.

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