EFFECTS OF TRAVEL FREQUENCY ON THE PERSISTENCE OF MOSQUITO-BORNE DISEASES

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Abstract. Travel frequency of people varies widely with occupation, age, gender, ethnicity, income, climate and other factors. Meanwhile, the distribution of the numbers of times people in different regions or with different travel behaviors bitten by mosquitoes may be nonuniform. To reflect these two heterogeneities, we develop a multipatch model to study the impact of travel frequency and human biting rate on the spatial spread of mosquito-borne diseases. The human population in each patch is divided into four classes: susceptible unfrequent, infectious unfrequent, susceptible frequent, and infectious frequent. The basic reproduction number \( R_0 \) is defined. It is shown that the disease-free equilibrium is globally asymptotically stable if \( R_0 \leq 1 \), and there is a unique endemic equilibrium that is globally asymptotically stable if \( R_0 > 1 \). A more detailed study is conducted on the single patch model. We use analytical and numerical methods to demonstrate that the model without considering the difference of humans in travel frequency mostly underestimates the risk of infection. Numerical simulations suggest that the greater the difference in travel frequency, the larger the underestimate of the transmission potential. In addition, the basic reproduction number \( R_0 \) may decreasingly, or increasingly, or nonmonotonically vary when more people travel frequently.

1. Introduction. Mosquito-borne diseases (MBDs) like malaria, West Nile fever, dengue fever, Zika, yellow fever, and chikungunya pose a big threat to public health worldwide [22]. An estimated 219 million new malaria cases occurred and 435,000 people died worldwide in 2017 [50]. Zika virus infected more than one million people and resulted in thousand of infants with birth defects during 2015-2016 Zika virus epidemic [30]. The 2015-2016 yellow fever outbreak in Angola and Democratic Republic of the Congo caused 7,334 suspected cases and 498 deaths [49]. West Nile virus swiftly spread across the United States, causing thousands of infections and hundreds of deaths annually after the first reported human case in New York in 1999 [29]. Dengue fever is endemic in more than 100 countries consisting of about half of the world’s population, as many as 400 million people are infected each
year [23]. These diseases also have a serious impact on the economy of endemic areas. For example, the per capita income growth rate of the countries with severe malaria from 1965 to 1990 was 0.4% per year, while the average growth rate of other countries was 2.3% [13].

Mathematical modeling plays a crucial role in the prevention and control of mosquito-borne diseases [38]. The initial model is the so-called Ross–Macdonald model for malaria transmission which was proposed by Ross [40] in 1911 and later extended by Macdonald in the 1950s [33]. The model captures the essential feature of malaria transmission process but ignores many important factors in ecology and epidemiology, leading to a large number of generalized malaria models [34]. For example, we can consider acquired immunity [3], age structure [4], spatial heterogeneity [19], incubation periods of the parasites [41], drug resistance [28], climate change [36], biological control [32], and mass drug administration [15]. Among these, we focus on the models that incorporate the movement of humans and/or mosquitoes. Human travel and migration facilitate the geographic spread of malaria and threaten the control and elimination of the disease [35, 44]. Dye and Hasibeder [12] and Hasibeder and Dye [25] proposed and analyzed a malaria model where a mosquito from one vector patch can commute to and bite in any of the host patches. They showed that non-homogeneous mixing between vectors and hosts results in a larger basic reproduction number. Auger et al. [5] developed a malaria patch model where only humans migrate among patches and some patches could be vector free. Cosner et al. [9] considered two modeling approaches that mimic migration and visitation, respectively. They numerically found that a vector-borne disease can become persist in a two-patch environment when human movement occurs even though the disease dies out in both isolated patches. Recently, Gao et al. [21] showed that the finding of Dye and Hasibeder [12] and Hasibeder and Dye [25] remains true for a malaria model with human and/or mosquito migration. For more studies on this topic, the readers may refer to two review articles by Cosner [8] and Gao and Ruan [20].

Travel survey and transportation data indicate that factors like age, occupation, gender, ethnicity, region, family size, and socioeconomic status lead to a nonuniform distribution of travel frequency among individuals [31]. In the United States, the high income households take more trips and travel more miles per household than low-medium income households, and people aged 35-54 take the most trips among all age groups [46]. In UK, a recent study observed that young women may travel more than young men [45]. People in Finland make an average of 7.5 trips per person per year, which is more than any other countries around the world [51]. The age factor has a positive but nonlinear effect on travel likelihood [1]. An intercept survey found that the proportions of respondents with less than two and over 20 round-trip flights per year within Norway are 57% and 2.2%, respectively [10]. A small proportion of people who make disproportionately more journeys than others may accelerate the global spread of epidemics [26]. Meanwhile, the number of bites a person received per unit time is affected by many factors such as blood type [2], health status [39], quality of bed nets [43], age or host size [37], and skin color [24].

To the best of our knowledge, there is no modeling study on the influence of heterogeneity in travel frequency on the spatial spread of mosquito-borne diseases. In the next section, based on the Ross–Macdonald model, we formulate a multipatch mosquito-borne disease model where the human population in each patch is divided into four classes with respect to disease status and travel behavior. In Section 3, the
global threshold dynamics of the model are established in terms of the reproduction number and a more detailed analysis for the single patch case is performed. In addition, we compare the new model and the simple multipatch Ross–Macdonald model through their basic reproduction numbers. In Section 4, extensive numerical simulations are given to further investigate the influence of the trend that more people become frequent travelers and the application of the current modeling study. A brief discussion on the findings and future work is given at the end.

2. Model formulation. Taking both human and mosquito movements into consideration, Cosner et al. [9] proposed and studied a multipatch Ross–Macdonald model as follows

\[
\begin{align*}
\frac{dH_i}{dt} &= \sum_{j=1}^{p} c_{ij} H_j, \ 1 \leq i \leq p, \\
\frac{dV_i}{dt} &= \sum_{j=1}^{p} d_{ij} V_j, \ 1 \leq i \leq p, \\
\frac{dh_i}{dt} &= a_i b_i \frac{v_i}{H_i} (H_i - h_i) - \gamma_i h_i + \sum_{j=1}^{p} c_{ij} h_j, \ 1 \leq i \leq p, \\
\frac{dv_i}{dt} &= a_i c_i \frac{h_i}{H_i} (V_i - v_i) - \mu_i v_i + \sum_{j=1}^{p} d_{ij} v_j, \ 1 \leq i \leq p,
\end{align*}
\]

with nonnegative initial conditions

\[(H_1(0), \ldots, H_p(0), V_1(0), \ldots, V_p(0), h_1(0), \ldots, h_p(0), v_1(0), \ldots, v_p(0))\]

satisfying

\[\sum_{i=1}^{p} H_i(0) = H > 0 \text{ and } \sum_{i=1}^{p} V_i(0) = V > 0.\]

Here \(p\) is the total number of patches. The state variables \(H_i\) and \(V_i\) represent the total population sizes for humans and mosquitoes in patch \(i\), respectively; \(h_i\) and \(v_i\) denote the numbers of infected humans and infected mosquitoes in patch \(i\), respectively. The parameters \(b_i\) and \(c_i\) are the transmission probabilities from infected mosquitoes to susceptible humans and from infected humans to susceptible mosquitoes in patch \(i\), respectively; \(\gamma_i\) and \(\mu_i\) are the recovery rate of humans and mortality rate of mosquitoes in patch \(i\), respectively; \(c_{ij}\) and \(d_{ij}\) are the migration rates of humans and mosquitoes from patches \(j\) to \(i\) for \(1 \leq i, j \leq p\) and \(i \neq j\), respectively; \(-c_{ii} = \sum_{j \neq i} c_{ji}\) and \(-d_{ii} = \sum_{j \neq i} d_{ji}\) are the emigration rates of humans and mosquitoes in patch \(i\) for \(1 \leq i \leq p\), respectively. The migration matrices of humans and mosquitoes, \(C = (c_{ij})_{p \times p}\) and \(D = (d_{ij})_{p \times p}\), are assumed to be irreducible. Since most mosquitoes fly no more than three miles from where they hatch [48], we can ignore the movement of mosquitoes when a medium-large geographic scale is considered.

Using the next generation matrix approach [11, 47], the basic reproduction number is defined as

\[\hat{R}_0 = \sqrt{\rho(A^{-1}B^{-1})},\]

where \(A = \text{diag}\{a_1 b_1, \ldots, a_p b_p\}\), \(B = \text{diag}\{a_1 c_1 V^*_1/H^*_1, \ldots, a_p c_p V^*_p/H^*_p\}\), \(C = \text{diag}\{\gamma_1, \ldots, \gamma_p\} - C\), \(D = \text{diag}\{\mu_1, \ldots, \mu_p\} - D\). In addition, \((H^*_1, \ldots, H^*_p)\) and
\((V_1^*, \ldots, V_p^*)\) are respectively the unique positive solutions to the systems of linear equations

\[
\sum_{j=1}^{p} c_{ij} H_j = 0, \quad 1 \leq i \leq p - 1, \quad \text{and} \quad \sum_{i=1}^{p} H_i = H,
\]

and

\[
\sum_{j=1}^{p} d_{ij} V_j = 0, \quad 1 \leq i \leq p - 1, \quad \text{and} \quad \sum_{i=1}^{p} V_i = V.
\]

It is shown that the disease dies out in all patches if \(\hat{R}_0 \leq 1\), but persists at a unique endemic equilibrium if \(\hat{R}_0 > 1\) [9].

Recently, Gao [14] proposed an SIS patch model for directly transmitted diseases with the consideration of travel frequency. Using a similar idea, we generalize the multipatch Ross–Macdonald model (1) by further splitting the humans in each patch into four classes: susceptible unfrequent, infectious unfrequent, susceptible frequent, and infectious frequent. Let \(H^u_i, H^f_i\) and \(V_i\) be the total numbers of unfrequent travelers, frequent travelers and mosquitoes in patch \(i\), respectively; \(h^u_i, h^f_i\) and \(v_i\) be the numbers of infected unfrequent travelers, infected frequent travelers and infected mosquitoes in patch \(i\), respectively. Let \(P^u_i\) and \(P^f_i\) be the human biting rates (HBR, i.e., the number of bites per human per unit time) of unfrequent and frequent travelers in patch \(i\), respectively. The fact that the total number of bites made by mosquitoes equals the total number of bites received by humans gives

\[
a_i V_i = P^u_i H^u_i + P^f_i H^f_i = P^u_i (H^u_i + \sigma_i H^f_i),
\]

where \(\sigma_i = P^f_i / P^u_i\) represents the relative HBR of frequent travelers to unfrequent travelers. Hence, the forces of infection for susceptible unfrequent and susceptible frequent travelers in patch \(i\) are respectively given by

\[
P^u_i \frac{v_i}{V_i} h^u_i = \frac{a_i h^u_i v_i}{H^u_i + \sigma_i H^f_i} \quad \text{and} \quad P^f_i \frac{v_i}{V_i} h^f_i = \frac{\sigma_i a_i h^f_i v_i}{H^u_i + \sigma_i H^f_i},
\]

and the force of infection for susceptible mosquitoes in patch \(i\) is

\[
a_i \frac{P^u_i H^u_i}{a_i V_i} h^u_i + a_i \frac{P^f_i H^f_i}{a_i V_i} h^f_i = \frac{a_i c^u_i h^u_i}{H^u_i + \sigma_i H^f_i} + \frac{\sigma_i a_i c^f_i h^f_i}{H^u_i + \sigma_i H^f_i}.
\]

In addition, we make the following assumptions

1. People migrating from one patch to another do not immediately change their travel frequency;
2. The exchange rates of travel frequency are independent of disease status;
3. People do not immediately change their travel frequency after getting sick or recovering;
4. Unfrequent and frequent travelers may be subject to different numbers of mosquito bites.
Based on the above assumptions and the flowchart in Figure 1, we propose the following two-group multipatch mosquito-borne disease model \((1 \leq i \leq p)\)

\[
\frac{dH^u_i}{dt} = -\phi^u_i H^u_i + \phi^f_i H^f_i + \sum_{j=1}^{p} c^u_{ij} H^u_j, \tag{2a}
\]

\[
\frac{dH^f_i}{dt} = \phi^u_i H^u_i - \phi^f_i H^f_i + \sum_{j=1}^{p} c^f_{ij} H^f_j, \tag{2b}
\]

\[
\frac{dV_i}{dt} = \frac{\sum_{j=1}^{p} d_{ij} V_j}{p}, \tag{2c}
\]

\[
\frac{dh^u_i}{dt} = \frac{a_i b^u_i v_i}{H^u_i + \sigma_i H^f_i} (H^u_i - h^u_i) - \gamma^u_i h^u_i - \phi^u_i h^u_i + \phi^f_i h^f_i + \sum_{j=1}^{p} c^u_{ij} h^u_j, \tag{2d}
\]

\[
\frac{dh^f_i}{dt} = \frac{\sigma_i a_i h^f_i v_i}{H^u_i + \sigma_i H^f_i} (H^f_i - h^f_i) - \gamma^f_i h^f_i + \phi^u_i h^u_i - \phi^f_i h^f_i + \sum_{j=1}^{p} c^f_{ij} h^f_j, \tag{2e}
\]

\[
\frac{dv_i}{dt} = \frac{a_i c^f_i h^u_i}{H^u_i + \sigma_i H^f_i} (V_i - v_i) + \frac{\sigma_i a_i c^f_i h^f_i}{H^u_i + \sigma_i H^f_i} (V_i - v_i) - \mu_i v_i + \sum_{j=1}^{p} d_{ij} v_j, \tag{2f}
\]

with nonnegative initial conditions satisfying

\[
\sum_{i=1}^{p} (H^u_i(0) + H^f_i(0)) = H > 0 \quad \text{and} \quad \sum_{i=1}^{p} V_i(0) = V > 0.
\]

All parameters of model (2) are summarized in Table 1, where the ranges are mainly based on malaria and adopted from the references \([7, 14, 17, 41]\). In particular, the emigration rates of unfrequent travelers, frequent travelers, and mosquitoes
in patch \(i\) satisfy respectively
\[-c_{ii}^u = \sum_{j \neq i} c_{ji}^u, \quad -c_{ii}^f = \sum_{j \neq i} c_{ji}^f, \quad \text{and} \quad -d_{ii} = \sum_{j \neq i} d_{ji}, \quad 1 \leq i \leq p.\]

Unless otherwise indicated, the sum of migration matrices for unfrequent and frequent travelers, \(C^u + C^f = (c_{ij}^u)_{p \times p} + (c_{ij}^f)_{p \times p} = (c_{ij}^u + c_{ij}^f)_{p \times p}\), and the migration matrix for mosquitoes, \(D = (d_{ij})_{p \times p}\), are assumed to be irreducible.

**Table 1.** Descriptions and ranges of parameters (time unit is day).

| Description | Range |
|-------------|-------|
| \(a_i\) | mosquito biting rate | 0.1–1 |
| \(b_i^u\) | transmission probability from an infectious mosquito to a susceptible unfrequent traveler per bite | 0.01–0.8 |
| \(b_i^f\) | transmission probability from an infectious mosquito to a susceptible frequent traveler per bite | 0.01–0.8 |
| \(c_i^u\) | transmission probability from an infectious unfrequent traveler to a susceptible mosquito per bite | 0.072–0.64 |
| \(c_i^f\) | transmission probability from an infectious frequent traveler to a susceptible mosquito per bite | 0.072–0.64 |
| \(\gamma_i^u\) | recovery rate of infectious unfrequent humans | 0.005–0.05 |
| \(\gamma_i^f\) | recovery rate of infectious frequent humans | 0.005–0.05 |
| \(\mu_i\) | mosquito mortality rate | 0.05–0.2 |
| \(\sigma_i\) | relative HBR of frequent travelers to unfrequent travelers | 0.2–5 |
| \(c_{ij}^f\) | travel rate of frequent travelers from patches \(j\) to \(i\) | 0.03–0.1 |
| \(\tau_{ij}\) | relative travel rate of unfrequent travelers to frequent travelers | 0–0.4 |
| \(c_{ij}^u\) | travel rate of unfrequent travelers from patches \(j\) to \(i\) | \(c_{ij}^u = \tau_{ij}c_{ij}^f\) |
| \(d_{ij}\) | travel rate of mosquitoes from patch \(j\) to patch \(i\) | 0.001–0.03 |
| \(\phi_i^f\) | change rate from frequent travelers to unfrequent travelers | \(2.7 \times 10^{-4}\) |
| \(\tau_i\) | relative change rate of unfrequent travelers to frequent travelers | \(9 \times 10^{-4}\) |
| \(\phi_i^u\) | change rate from unfrequent travelers to frequent travelers | 0.1–0.5 |
| \(V/H\) | ratio of mosquitoes to humans | 1–10 |

**3. Mathematical results.** In this section, we first show the existence and uniqueness of the disease-free equilibrium and define the basic reproduction number for model (2). Then the global threshold dynamics of the model system are established and the single patch case is analyzed in detail. Finally, the basic reproduction numbers of the traditional model (1) and the new model (2) are compared to explore the impact of travel difference on the transmission of mosquito-borne diseases.

**3.1. Basic reproduction number.** The irreducibility of \(C^u + C^f\) implies that the coefficient matrix of (2a)–(2b), i.e.,
\[
A = \begin{pmatrix} A_{11} & -A_{12} \\ -A_{21} & A_{22} \end{pmatrix},
\]
is irreducible, where
\[ A_{11} = \text{diag}\{\phi_u^i, \ldots, \phi_u^p\} - C^u, \quad A_{12} = \text{diag}\{\phi_f^i, \ldots, \phi_f^p\}, \]
\[ A_{21} = \text{diag}\{\phi_u^i, \ldots, \phi_u^p\}, \quad A_{22} = \text{diag}\{\phi_f^i, \ldots, \phi_f^p\} - C^f. \]

By Lemma 1 in Cosner et al. [9], the human and mosquito movement models (2a)–(2b) and (2c) have unique positive equilibria, \((H^{u*}, H^f) = (H_1^{u*}, \ldots, H_p^{u*}, H_1^{f*}, \ldots, H_p^{f*})\) and \(V^* = (V_1^*, \ldots, V_p^*)\), respectively, which satisfy

\[-\phi_u^i H_i^{u*} + \phi_f^i H_i^{f*} + \sum_{j=1}^p c_{ij}^u H_j^{u*} = 0, \quad 1 \leq i \leq p,\]
\[\phi_u^i H_i^{u*} - \phi_f^i H_i^{f*} + \sum_{j=1}^p c_{ij}^f H_j^{f*} = 0, \quad 1 \leq i \leq p - 1,\]
\[\sum_{i=1}^p (H_i^{u*} + H_i^{f*}) = H,\]

and
\[\sum_{j=1}^p d_{ij} V_j = 0, \quad 1 \leq i \leq p - 1, \quad \text{and} \quad \sum_{i=1}^p V_i = V,\]
respectively. Thus model (2) admits a unique disease-free equilibrium, denoted by \(E_0 = (H^{u*}, H^f, V^*, 0, 0, 0).\)

Using the recipe of van den Driessche and Watmough [47], the new infection and transition matrices are given by

\[ F = \begin{pmatrix} 0 & 0 & F_{13} \\ 0 & 0 & F_{23} \\ F_{31} & F_{32} & 0 \end{pmatrix} = \begin{pmatrix} 0 & F_h \\ F_v & 0 \end{pmatrix}, \]

and
\[ V = \begin{pmatrix} V_{11} & -V_{12} & 0 \\ -V_{21} & V_{22} & 0 \\ 0 & 0 & V_{33} \end{pmatrix} = \begin{pmatrix} V_h & 0 \\ 0 & V_v \end{pmatrix}, \]

respectively, where

\[ F_{13} = \left( \delta_{ij} \frac{a_i b_i H_i^{u*}}{H_i^{u*} + \sigma_i H_i^{f*}} \right)_{p \times p}, \quad F_{23} = \left( \delta_{ij} \frac{\sigma_i a_i c_i V_i^{*}}{H_i^{u*} + \sigma_i H_i^{f*}} \right)_{p \times p}, \]
\[ F_{31} = \left( \delta_{ij} \frac{a_i c_i H_i^{u*}}{H_i^{u*} + \sigma_i H_i^{f*}} \right)_{p \times p}, \quad F_{32} = \left( \delta_{ij} \frac{\sigma_i a_i c_i V_i^{*}}{H_i^{u*} + \sigma_i H_i^{f*}} \right)_{p \times p}, \]
\[ V_{11} = \left( \delta_{ij} (\gamma_i^{u*} + \phi_i^{u*}) \right)_{p \times p} - C^u, \quad V_{12} = \left( \delta_{ij} \phi_i^{f*} \right)_{p \times p}, \]
\[ V_{21} = \left( \delta_{ij} \phi_i^{u*} \right)_{p \times p}, \quad V_{22} = \left( \delta_{ij} (\gamma_i^{f*} + \phi_i^{f*}) \right)_{p \times p} - C^f, \]
\[ V_{33} = \left( \delta_{ij} \mu_i \right)_{p \times p} - D. \]

Here \(\delta_{ij}\) denotes the Kronecker delta (i.e., 1 when \(i = j\) and 0 otherwise). The next generation matrix [11] for model (2) is

\[ FV^{-1} = \begin{pmatrix} 0 & F_h V_v^{-1} \\ F_v V_h^{-1} & 0 \end{pmatrix}. \]
For model (2) using the theory of monotone dynamical systems [42]. We next study the global behavior of system (2) using the theory of monotone dynamical systems [42].

3.2. Global dynamics. It is easy to see that the feasible region

\[ \Gamma = \left\{ (H^u, H^f, V, h^u, h^f, v) \in \mathbb{R}^{6p} : \sum_{i=1}^{p} (H^u_i + H^f_i) = H > 0, \right. \]
\[ \left. \sum_{i=1}^{p} V_i = V > 0, h^u_i \leq H^u_i, h^f_i \leq H^f_i, v_i \leq V_i, i = 1, \ldots, p \right\} \]

is positively invariant with respect to system (2). We next study the global behavior of system (2) using the theory of monotone dynamical systems [42].

**Theorem 3.1.** For model (2), the disease-free equilibrium \( E_0 \) is globally asymptotically stable in \( \Gamma \) if \( R_0 \leq 1 \), and there is a unique endemic equilibrium, denoted by \( E^* = (H^{u*}, H^{f*}, V^*, h^{u*}, h^{f*}, v^*) \), which is globally asymptotically stable in \( \Gamma \) minus the set of all disease-free states if \( R_0 > 1 \).

**Proof.** For subsystem (2a)–(2c), the equilibrium \( (H^{u*}, H^{f*}, V^*) \) is globally asymptotically stable in \( \mathbb{R}^{3p}_+ \). Thus system (2) is topologically equivalent to the limiting system

\[
\frac{dh^u_i}{dt} = \frac{a_i b^{u}_i v_i}{H^{u*}_i + \sigma_i H^{f*}_i} (H^{u}_i - h^{u}_i) - \gamma^{u}_i h^{u}_i - \phi^{u}_i h^{u}_i + \phi^{f}_i h^{f}_i + \sum_{j=1}^{p} c^{u}_{ij} h^{u}_j, \\
\frac{dh^f_i}{dt} = \frac{\sigma_i a_i b^{f}_i v_i}{H^{u*}_i + \sigma_i H^{f*}_i} (H^{f}_i - h^{f}_i) - \gamma^{f}_i h^{f}_i + \phi^{u}_i h^{u}_i - \phi^{f}_i h^{f}_i + \sum_{j=1}^{p} c^{f}_{ij} h^{f}_j, \\
\frac{dv_i}{dt} = \frac{a_i c^{f}_i h^{f}_i + \sigma_i a_i c^{f}_i h^{f}_i}{H^{u*}_i + \sigma_i H^{f*}_i} (V^*_i - v_i) - \mu_i v_i + \sum_{j=1}^{p} d_{ij} v_j. 
\]

Let \( f = (f_1, \ldots, f_{3p}) \) be the vector field defined by (3), \( \psi_t \) the corresponding semiflow. System (3) is cooperative and irreducible on the region \( \mathbb{D} = \{(h^u, h^f, v) \in \mathbb{R}^{3p}_+ : h^u_i \leq H^{u*}_i, h^f_i \leq H^{f*}_i, v_i \leq V^*_i, i = 1, \ldots, p \} \).

For any \( \alpha \in (0,1) \) and any \( x = (h^u_1, \ldots, h^u_p, h^f_1, \ldots, h^f_p, v_1, \ldots, v_p) \in \mathbb{D} \) with \( x \gg 0 \), we have

\[
f_i(\alpha x) - \alpha f_i(x) = \begin{cases} 
    \alpha(1 - \alpha) a_i b^{u}_i v_i h^{u}_i > 0, & i = 1, \ldots, p, \\
    \alpha(1 - \alpha) a_i b^{f}_i v_i h^{f}_i > 0, & i = p + 1, \ldots, 2p, \\
    \alpha(1 - \alpha) a_i c^{f}_i h^{f}_i (c^{u}_i h^{u}_i + \sigma_i c^{f}_i h^{f}_i) > 0, & i = 2p + 1, \ldots, 3p.
\end{cases}
\]

The above means that \( f \) is strongly sublinear on \( \mathbb{D} \). Note that

\[
f_i(x) \geq 0, \text{ if } x \in \mathbb{D} \text{ and } x_i = 0, i = 1, \ldots, 3p.
\]
If \( x \in \mathbb{D} \) and \( x_i = H_i^{u*} \), \( i = 1, \ldots, p \), then
\[
f_i(x) = -\gamma_i H_i^{u*} - \phi_i^u H_i^{u*} + \phi_i^f h_i^f + \sum_{j \neq i} c_{ij}^u h_j^u - \sum_{j \neq i} c_{ij}^u H_j^{u*}
\]
\[
= -\gamma_i H_i^{u*} + \phi_i^f h_i^f + \sum_{j \neq i} c_{ij}^u h_j^u - \left( \phi_i^u H_i^{u*} + \sum_{j \neq i} c_{ij}^u H_j^{u*} \right)
\]
\[
= -\gamma_i H_i^{u*} - \phi_i^f (H_i^{f*} - h_i^f) - \sum_{j \neq i} c_{ij}^u (H_j^{u*} - h_j^u) \leq 0.
\]

Similarly, \( f_i(x) \leq 0 \) if \( x \in \mathbb{D} \) and \( x_i = H_i^{f*} \), \( i = p + 1, \ldots, 2p \); \( f_i(x) \leq 0 \) if \( x \in \mathbb{D} \) and \( x_i = V_i^{u*} \), \( i = 2p + 1, \ldots, 3p \). These facts imply that the set \( \mathbb{D} \) is positively invariant with respect to system (3).

The proof of Theorem 2 in van den Driessche and Watmough [47] indicates that \( R_0 - 1 \) has the same sign as \( s(Df(0)) = s(F - V) \), where \( Df(0) \) denotes the Jacobian matrix of system (3) at \( x = 0 \). By Corollary 3.2 in Zhao and Jing [53], the origin is globally asymptotically stable with respect to \( \mathbb{D} \) if \( R_0 \leq 1 \), and the system (3) has a unique positive equilibrium \( x^* = (h^{u*}, h^{f*}, v^*) \in \mathbb{D} \) which is globally asymptotically stable with respect to \( \mathbb{D} \setminus \{0\} \) otherwise. The strong monotonicity of \( \psi_i \) in \( \mathbb{D} \) implies that \( 0 < (h^{u*}, h^{f*}, v^*) < (H^{u*}, H^{f*}, V^*) \). Therefore, applying the theory of asymptotically autonomous systems or chain transitive sets [6, 52], if \( R_0 \leq 1 \), then the disease-free equilibrium \( E_0 \) of system (2) is globally asymptotically stable in \( \Gamma \); if \( R_0 > 1 \), then system (2) admits a unique endemic equilibrium \( E^* = (H^{u*}, H^{f*}, V^*, h^{u*}, h^{f*}, v^*) \) that is globally asymptotically stable.

3.3. Single patch model. When only one patch is considered, model (2) is reduced to
\[
\begin{align*}
\frac{dh^u}{dt} &= \frac{ab_{uv}}{H^u + \sigma H^f} (H^u - h^u) - \gamma^u h^u - \phi^u h^u + \phi^f h^f, \\
\frac{dh^f}{dt} &= \frac{\sigma ab_{uv}}{H^u + \sigma H^f} (H^f - h^f) - \gamma^f h^f + \phi^u h^u - \phi^f h^f, \\
\frac{dv}{dt} &= \frac{ac_{uv} h^u}{H^u + \sigma H^f} (V - v) + \frac{\sigma ac_{uv}}{H^u + \sigma H^f} (V - v) - \mu v,
\end{align*}
\]
where \( H = H^u + H^f > 0 \) and \( V > 0 \) are the total population sizes of humans and mosquitoes, respectively; and the numbers of unfrequent and frequent travelers are respectively
\[ H^u = \frac{\phi^f}{\phi^u + \phi^f} H \quad \text{and} \quad H^f = \frac{\phi^u}{\phi^u + \phi^f}. \]

Clearly, model (4) has a unique disease-free equilibrium \( E_1 = (0, 0, 0) \). Using the next generation matrix method [47], we can write the incidence and transition matrices as
\[
F_1 = \begin{pmatrix}
0 & 0 & F_{1}^{uv} \\
0 & 0 & F_{1}^{uv} \\
F_{1}^{uv} & F_{1}^{vf} & 0
\end{pmatrix}
\quad \text{and} \quad
V_1 = \begin{pmatrix}
\gamma^u + \phi^u & -\phi^f & 0 \\
-\phi^u & \gamma^f + \phi^f & 0 \\
0 & 0 & \mu
\end{pmatrix},
\]
where
\[
F_1^{uv} = \frac{ab^uH^u}{H^u + \sigma H^f}, \quad F_1^{fu} = \frac{\sigma ab^fH^f}{H^u + \sigma H^f}, \quad F_1^{vu} = \frac{ac^uV}{H^u + \sigma H^f}, \quad F_1^{vf} = \frac{\sigma ac^fV}{H^u + \sigma H^f}.
\]
The basic reproduction number for model (4) is defined as the spectral radius of the next generation matrix \(F_1V_1^{-1}\), i.e.,
\[
\mathcal{R}_1 = \rho(F_1V_1^{-1}) = \sqrt{\langle \mathcal{R}_1^{uv} \rangle^2 + \langle \mathcal{R}_1^{uf} \rangle^2 + \langle \mathcal{R}_1^{fu} \rangle^2 + \langle \mathcal{R}_1^{vf} \rangle^2},
\]
where
\[
\langle \mathcal{R}_1^{uv} \rangle^2 = \frac{F_1^{uv}F_1^{vu}(\gamma^f + \phi^f)}{\mu \Delta}, \quad \langle \mathcal{R}_1^{uf} \rangle^2 = \frac{F_1^{uf}F_1^{fu}}{\mu \Delta},
\]
\[
\langle \mathcal{R}_1^{fu} \rangle^2 = \frac{F_1^{fu}F_1^{fu}q}{\mu \Delta}, \quad \langle \mathcal{R}_1^{vf} \rangle^2 = \frac{F_1^{vf}F_1^{vf}(\gamma^u + \phi^u)}{\mu \Delta},
\]
and \(\Delta = (\gamma^u + \phi^u)(\gamma^f + \phi^f) - \phi^u\phi^f > 0\).

Similar to the multipatch model (2), following the theory of monotone dynamical systems, the disease dynamics of system (4) are governed by the associated basic reproduction number.

**Corollary 1.** For the single patch model (4), if \(\mathcal{R}_1 \leq 1\), then the disease-free equilibrium \(E_1 = (0, 0, 0)\) is globally asymptotically stable in \(\Gamma_1 = \{(h^u, h^f, v) \in \mathbb{R}_+^3 : h^u \leq H^u, h^f \leq H^f, v \leq V\}\); if \(\mathcal{R}_1 > 1\), then there is a unique endemic equilibrium \(E^*_1 = (h^{u*}, h^{f*}, v^*)\) which is globally asymptotically stable in \(\Gamma_1 \setminus \{E_1\}\).

In case there is no epidemiological difference between unfrequent and frequent travelers, i.e., \(b^u = b^f = b, c^u = c^f = c, \gamma^u = \gamma^f = \gamma\), then system (4) can be rewritten as
\[
\begin{aligned}
\frac{dh^u}{dt} &= \frac{abv}{H^u + \sigma H^f}(H^u - h^u) - \gamma h^u - \phi^u h^u + \phi^f h^f, \\
\frac{dh^f}{dt} &= \frac{\sigma abv}{H^u + \sigma H^f}(H^f - h^f) - \gamma h^f + \phi^u h^u - \phi^f h^f, \\
\frac{dv}{dt} &= \frac{ach^u}{H^u + \sigma H^f}(V - v) + \frac{\sigma ach^f}{H^u + \sigma H^f}(V - v) - \mu v.
\end{aligned}
\]

Furthermore, if the HBRs of unfrequent and frequent travelers are the same, i.e., \(\sigma = 1\), then model (5) can be reduced to the classical Ross–Macdonald model
\[
\begin{aligned}
\frac{dh}{dt} &= \frac{abv}{H}(H - h) - \gamma h, \\
\frac{dv}{dt} &= \frac{ach}{H}(V - v) - \mu v,
\end{aligned}
\]
where \(h = h^u + h^f\) represents the total number of infected humans.

**Remark 1.** In comparison to model (5) with biting difference, the Ross–Macdonald model (6) underestimates the risk of infection. In fact, the basic reproduction numbers of models (5) and (6) are respectively
\[
\mathcal{R}_1 = \sqrt{\frac{a^2bcV(\phi^u + \phi^f)(\phi^f + \sigma \phi^u)^2 + \gamma(\phi^f + \sigma \phi^u)^2}{\mu \gamma H}(\phi^f + \phi^u + \gamma)(\phi^f + \sigma \phi^u)^2}} \quad \text{and} \quad \mathcal{R}_c = \sqrt{\frac{a^2bcV}{\mu \gamma H}}.
\]

Direct calculation yields
\[
\langle \mathcal{R}_1 \rangle^2 - \langle \mathcal{R}_c \rangle^2 = \frac{a^2bcV(1 - \sigma)^2 \phi^u \phi^f}{\mu H(\phi^f + \phi^u + \gamma)(\phi^f + \sigma \phi^u)^2} \geq 0,
\]
with equality if and only if $\sigma = 1$.

Next we solve the endemic equilibrium of system (4). It follows from the first and second equilibrium equations associated with system (4) that

\[
\begin{align*}
    h^u &= \frac{\lambda_{vf}^* + \gamma^f + \phi^f}{\lambda_{vu}^* H^u + \phi^f \lambda_{vf}^* H^f}
    \left(\lambda_{vf}^* + \gamma^f + \phi^f\right) + \gamma^u + \phi^u - \phi^f \phi^u, \\
    h^f &= \frac{\phi^u \lambda_{vu}^* H^u + \left(\lambda_{vu}^* + \gamma^u + \phi^u\right) \lambda_{vf}^* H^f}{\lambda_{vf}^* + \gamma^f + \phi^f\left(\lambda_{vu}^* + \gamma^u + \phi^u\right) - \phi^f \phi^u},
\end{align*}
\]

where

\[
\lambda_{vu}^* = \frac{ab^u v^*}{H^u + \sigma H^f} \quad \text{and} \quad \lambda_{vf}^* = \frac{ab^f v^*}{H^u + \sigma H^f}.
\]

By substituting $h^u$ and $h^f$ into the third equilibrium equation of system (4), we obtain an equation of $v^*$ as follows

\[
d_2(v^*)^2 + d_1 v^* + d_0 = 0,
\]

where

\[
\begin{align*}
d_2 &= \sigma a^2 b^u b^f (H^u (a c^u + \mu) + \sigma H^f (a c^f + \mu)) > 0, \\
d_1 &= a(-\sigma a^2 V b^u b^f (H^u c^u + \sigma H^f c^f) + \mu (H^u + \sigma H^f)^2 (b^u (\gamma^f + \phi^f) \\
    &+ \sigma b^f (\gamma^u + \phi^u)) + a (H^u + \sigma H^f) (H^u b^u (c^u (\gamma^f + \phi^f) + \sigma c^f \phi^u) \\
    &+ \sigma H^f b^f (c^u \phi^f + \sigma c^f (\gamma^u + \phi^u)))], \\
d_0 &= (H^u + \sigma H^f) (\mu (H^u + \sigma H^f)^2 (\gamma^u \phi^f + \gamma^f (\gamma^u + \phi^u)) \\
    &- a^2 V (H^u b^u (c^u (\gamma^f + \phi^f) + \sigma c^f \phi^u) + \sigma H^f b^f (c^u \phi^f + \sigma c^f (\gamma^u + \phi^u))) \]
\]

The above quadratic equation has a unique positive solution

\[
v^* = \frac{-d_1 + \sqrt{d_1^2 - 4d_0 d_2}}{2d_2},
\]

whenever $d_0 < 0$, or equivalently, $R_1 > 1$. The result agrees with Corollary 1.

Remark 2. For model (2) without the movement of unfrequent travelers, the fraction of frequent travelers in patch $i$ tends to

\[
\frac{H^u_i}{H^u_i + H^u_i} = \frac{\phi^u_i}{\phi^u_i + \phi^f_i}.
\]

If the per capita net change rate of migrants for the unfrequent travelers is small enough in relative to the frequency exchange rates, then the fraction of frequent travelers in patch $i$ converges to

\[
\begin{align*}
    H^f_i &= \frac{\phi^u_i H^u_i - \sum_{j=1}^p c^u_{ij} H^u_j}{\phi^u_i + \phi^f_i} H^u_i - \sum_{j=1}^p c^u_{ij} H^u_j \\
    &\approx \frac{\phi^u_i}{\phi^u_i + \phi^f_i}.
\end{align*}
\]

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So the proportion of frequent travelers of patch \( i \) mainly depends on the frequency exchange rates \( \phi^u_i \) and \( \phi^f_i \). Furthermore, we find that both the size and proportion of frequent travelers of the \( i \)th patch increase as \( \phi^u_i \) increases or \( \phi^f_i \) decreases.

**Theorem 3.2.** Consider a simple single population patch model

\[
\frac{dN_i}{dt} = \sum_{j=1}^{p} c_{ij}N_j, \quad 1 \leq i \leq p, \quad \text{and} \quad \sum_{i=1}^{p} N_i(0) = N > 0,
\]

where \( C = (c_{ij})_{p \times p} \) is an essentially nonnegative and irreducible matrix with zero column sums. Then the system has a unique positive equilibrium \( \mathbf{N}^* = (N_i^*, \ldots, N_p^*) \) which is globally asymptotically stable. Furthermore, we have

\[
\frac{\partial N_i^*}{\partial c_{ij}} > 0 \quad \text{and} \quad \frac{\partial N_j^*}{\partial c_{ij}} < 0,
\]

for \( 1 \leq i, j \leq p \) and \( i \neq j \).

**Proof.** The existence, uniqueness and global asymptotic stability of the positive equilibrium \( \mathbf{N}^* = (N_1^*, \ldots, N_p^*) \) are guaranteed by applying Lemma 1 in Cosner et al. [9]. For the monotonicity of population size of patch \( i \) on the travel rate from/to patch \( i \), without loss of generality, it suffices to consider

\[
\frac{\partial N_1^*}{\partial c_{12}} \quad \text{and} \quad \frac{\partial N_i^*}{\partial c_{12}}.
\]

Since \( \mathbf{N}^* \) is the unique solution of the system of linear equations

\[
\sum_{j=1}^{p} c_{ij}N_j = 0, \quad 1 \leq i \leq p - 1, \quad \text{and} \quad \sum_{i=1}^{p} N_i = N > 0,
\]

we have

\[
0 = \sum_{j=1}^{p} c_{ij}N_j^* = \sum_{j=1}^{p-1} c_{ij}N_j^* + c_{ip}N_p^* = \sum_{j=1}^{p-1} c_{ij}N_j^* + c_{ip} \left( N - \sum_{j=1}^{p-1} N_j^* \right)
\]

\[
= \sum_{j=1}^{p-1} (c_{ij} - c_{ip})N_j^* + c_{ip}N, \quad i = 1, \ldots, p - 1.
\]  

Denote the coefficient matrix of the system of linear equations \( (7) \) with respect to \( N_1^*, \ldots, N_{p-1}^* \) by \( \tilde{C} = (c_{ij} - c_{ip})_{(p-1) \times (p-1)} \). The migration matrix \( C \) is essentially nonnegative and irreducible with zero column sums, so the spectral bound of \( C \) is \( s(C) = 0 \) and the spectrum of \( C \) can be written as

\[
\sigma(C) = \{0, \lambda_1, \ldots, \lambda_{p-1}\},
\]

where \( \lambda_i < 0 \) for \( 1 \leq i \leq p - 1 \). Direct calculation gives
Note that \( \lambda \) is the (\( ji \)) minor of \( \tilde{C} \). So, 
\[
\lambda = \lambda \big| \lambda I_{p-1} - \tilde{C} \big|
\]
which implies that the spectrum of \( \tilde{C} \) is \( \sigma(\tilde{C}) = \{ \lambda_1, \ldots, \lambda_{p-1} \} \). Thus, \( |\tilde{C}| = \lambda_1 \times \lambda_2 \times \cdots \times \lambda_{p-1} \) and hence \( \text{sgn}(|\tilde{C}|) = (-1)^{p-1} \).

By the implicit function theorem, \( \partial N_1^* / \partial c_{12} \) exists for \( i = 1, \ldots, p - 1 \). Differentiating both sides of (7) with respect to \( c_{12} \) gives
\[
\sum_{j=1}^{p-1} (c_{ij} - c_{ip}) \frac{\partial N_1^*}{\partial c_{12}} + \sum_{j=1}^{p-1} \frac{\partial (c_{ij} - c_{ip})}{\partial c_{12}} N_j^* + \frac{\partial c_{ip}}{\partial c_{12}} N = 0, \; 1 \leq i \leq p - 1. \tag{8}
\]

For \( p = 2 \), a straightforward calculation gives
\[
N^* = \begin{pmatrix} N_1^* \\ N_2^* \end{pmatrix} = \begin{pmatrix} c_{12} \\ c_{12} + c_{21} \end{pmatrix} N = \begin{pmatrix} c_{21} \\ c_{12} + c_{21} \end{pmatrix} N,
\]
so
\[
\frac{\partial N_1^*}{\partial c_{12}} = \frac{c_{21}}{(c_{12} + c_{21})^2} N > 0 \quad \text{and} \quad \frac{\partial N_2^*}{\partial c_{12}} = -\frac{\partial N_1^*}{\partial c_{12}} < 0.
\]

For \( p \geq 3 \), equations (8) can be rewritten as
\[
\tilde{C} \left( \begin{array}{c}
\frac{\partial N_1^*}{\partial c_{12}} \\
\frac{\partial N_2^*}{\partial c_{12}} \\
\vdots \\
\frac{\partial N_{p-1}^*}{\partial c_{12}}
\end{array} \right)^T = (-N_2^*, N_2^*, 0, \ldots, 0)^T.
\]
Note that \( \tilde{C}^{-1} = \tilde{C}^*/|\tilde{C}| \), where \( \tilde{C}^* = ((-1)^{i+j} c_{ji}^*)_{(p-1) \times (p-1)} \) is the adjoint matrix of \( \tilde{C} \) and \( c_{ji}^* \) is the (\( ji \)) minor of \( \tilde{C} \). So,
\[
\left( \begin{array}{c}
\frac{\partial N_1^*}{\partial c_{12}} \\
\frac{\partial N_2^*}{\partial c_{12}}
\end{array} \right)^T = \frac{1}{|\tilde{C}|} \begin{pmatrix}
-c_{11}^* & -c_{21}^* \\
-c_{12}^* & -c_{22}^*
\end{pmatrix} \left( \begin{array}{c}
-N_2^* \\
N_2^*
\end{array} \right) = \frac{N_2^*}{|\tilde{C}|} \begin{pmatrix}
-c_{11}^* & -c_{21}^* \\
N_2^* & c_{12}^* + c_{22}^*
\end{pmatrix}.
\]
Now let us determine the sign of $c_{11}^* + c_{21}^*$. Direct calculation yields

$$c_{11}^* + c_{21}^* = \begin{bmatrix} 1 & c_{12} - c_{1p} & \cdots & c_{1p-1} - c_{1p} \\ -1 & c_{22} - c_{2p} & \cdots & c_{2p-1} - c_{2p} \\ 0 & c_{32} - c_{3p} & \cdots & c_{3p-1} - c_{3p} \\ \vdots & \vdots & \ddots & \vdots \\ 0 & c_{p-12} - c_{p-1p} & \cdots & c_{p-1p-1} - c_{p-1p} \\ 0 & -c_{p2} + c_{pp} & \cdots & -c_{pp-1} + c_{pp} \\ 0 & c_{22} - c_{2p} & \cdots & c_{2p-1} - c_{2p} \\ 0 & c_{32} - c_{3p} & \cdots & c_{3p-1} - c_{3p} \\ \vdots & \vdots & \ddots & \vdots \\ 0 & c_{p-12} - c_{p-1p} & \cdots & c_{p-1p-1} - c_{p-1p} \end{bmatrix}$$

$$= (-1)(-1)^{p-1-2} \begin{bmatrix} c_{32} - c_{3p} & c_{33} - c_{3p} & \cdots & c_{3p-1} - c_{3p} \\ \vdots & \vdots & \ddots & \vdots \\ c_{p-12} - c_{p-1p} & c_{p-13} - c_{p-1p} & \cdots & c_{p-1p-1} - c_{p-1p} \\ c_{p2} - c_{pp} & c_{p3} - c_{pp} & \cdots & c_{pp-1} - c_{pp} \end{bmatrix}$$

$$= (-1)^p(-1)^{1+p-1} \begin{bmatrix} 1 & 1 & \cdots & 1 & 1 \\ c_{32} & c_{33} & \cdots & c_{3p-1} & c_{3p} \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ c_{p-12} & c_{p-13} & \cdots & c_{p-1p-1} & c_{p-1p} \\ c_{p2} & c_{p3} & \cdots & c_{pp-1} & c_{pp} \end{bmatrix} = |W|,$$

where

$$W = \begin{bmatrix} 1 & 1 & \cdots & 1 & 1 \\ c_{32} & c_{33} & \cdots & c_{3p-1} & c_{3p} \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ c_{p-12} & c_{p-13} & \cdots & c_{p-1p-1} & c_{p-1p} \\ c_{p2} & c_{p3} & \cdots & c_{pp-1} & c_{pp} \end{bmatrix} = \begin{bmatrix} W_{11} & W_{12} \\ W_{21} & W_{22} \end{bmatrix}$$

is an essentially nonnegative matrix of order $p - 1$ with submatrices $W_{11} = 1, W_{12} = (1, \ldots, 1)_{1 \times (p-2)}, W_{21} = (c_{32}, \ldots, c_{p2})^T, \text{ and } W_{22} = (c_{i+2,j+2})_{(p-2) \times (p-2)}$.

**Claim.** $\text{sgn}(|W|) = (-1)^p$. Using the Schur complement, we have

$$|W| = |W_{22}| \cdot |W_{11} - W_{12}W_{22}^{-1}W_{21}|$$

provided that $W_{22}$ is nonsingular. Note that $W_{22}$ is the negative of a nonsingular $M$-matrix. In fact, if $W_{22}$ is irreducible, then it is an irreducibly diagonally dominant matrix and hence it is nonsingular by Corollary 6.2.27 in Horn and Johnson [27]; if $W_{22}$ is reducible, then up to a permutation it is similar to a block upper triangular matrix where each diagonal block is either a single negative entry or an irreducibly diagonally dominant submatrix (see the proof of Lemma 3.1 in Gao and Dong [16]).

Again by Corollary 6.2.27 in Horn and Johnson [27], $W_{22}$ is nonsingular. Since $-W_{22}$ is a nonsingular $M$-matrix, $(-W_{22})^{-1}$ is nonnegative and all the eigenvalues of $-W_{22}$ have positive real parts. Thus, $W_{11} - W_{12}W_{22}^{-1}W_{21} \geq 1$ and hence $\text{sgn}(|W|) = \text{sgn}(|W_{22}|) = (-1)^{p-2}$. The claim is proved and therefore

$$\text{sgn}(\partial N_1^*/\partial c_{12}) = (-1) \text{sgn}(c_{11}^* + c_{21}^*) \text{sgn}(|\tilde{c}|) = (-1)^{1+p+p-1} = 1.$$
Namely, $\partial N_1^*/\partial c_{12} > 0$. Similarly, we can prove that the sign of

$$c_{12}^* + c_{22}^* = \begin{vmatrix}
1 & 1 & \cdots & 1 & 1 \\
c_{31} & c_{33} & \cdots & c_{3p-1} & c_{3p} \\
\vdots & \vdots & \ddots & \vdots & \vdots \\
c_{p-11} & c_{p-13} & \cdots & c_{p-1p-1} & c_{p-1p} \\
c_p & c_p & \cdots & c_{pp-1} & c_{pp}
\end{vmatrix}$$

is $\text{sgn}(c_{12}^* + c_{22}^*) = (-1)^p$ and hence $\partial N_2^*/\partial c_{12} < 0$. 

3.4. Model comparison. Similar to the work of Gao [14], we examine the differences between the traditional model (1) and the new model (2) in describing the transmission potential through comparing their basic reproduction numbers. The parameter values of the traditional model are determined by these of the new model (2). Equilibria of the human migration models associated with the new and traditional models are $(H^{u*}, H^{f*})$ and $H^*$, respectively. So the numbers of residents in patch $i$ and travelers from patch $i$ to patch $j$ satisfy

$$H_i^* = H_i^{u*} + H_i^{f*}, \quad 1 \leq i \leq p,$$

$$c_{ji}H_i^* = c_{ji}H_i^{u*} + c_{ji}H_i^{f*}, \quad 1 \leq i, j \leq p,$$

which imply that the migration rate of humans from the $i$th patch to the $j$th patch for the traditional model (1) is

$$c_{ji} = \frac{c_{ji}H_i^{u*} + c_{ji}H_i^{f*}}{H_i^{u*} + H_i^{f*}}. \quad (9)$$

Assume that there are no epidemiological and biting differences between unfrequent and frequent travelers, that is, $b_i^u = b_i^f = b_i$, $c_i^u = c_i^f = c_i$, $\gamma_i^u = \gamma_i^f = \gamma_i$, and $\sigma_i = 1$ for $1 \leq i \leq p$. In what follows, we show that the traditional model underestimates the initial disease transmission when only frequent travelers migrate in a two-patch environment.

**Theorem 3.3.** Consider a two-patch submodel of (2), assume that $b_i^u = b_i^f = b_i$, $c_i^u = c_i^f = c_i$, $\gamma_i^u = \gamma_i^f = \gamma_i$, $\sigma_i = 1$, $c_{12}^u = c_{21}^u = 0$, $d_{12} = d_{21} = 0$, and $V_i > 0$, $i = 1, 2$. Then the basic reproduction number of the new model (2) is greater than or equal to that of the corresponding traditional model (1) with equality if and only if the basic reproduction numbers of the two patches in isolation are the same.

**Proof.** We first solve the disease-free equilibrium of the new model (2), then determine the disease-free equilibrium and the human movement rates of the traditional model (1), and lastly calculate and compare the basic reproduction numbers of the two models.

Since $c_{12}^u = c_{21}^u = 0$, it follows from the new model (2) that

$$\begin{align*}
- \phi_1^u H_1^{u*} + \phi_1^f H_1^{f*} &= 0, \\
- \phi_2^u H_2^{u*} + \phi_2^f H_2^{f*} &= 0, \\
- c_{21}^f H_1^{f*} + c_{12}^f H_2^{f*} &= 0, \\
H_1^{u*} + H_2^{u*} + H_1^{f*} + H_2^{f*} &= H,
\end{align*}$$

where $H$ is the total human population. Theorem 3.3 follows.
which has a unique positive solution
\[
H_1^{u*} = \frac{c_{12}^f \phi_2^u}{\Psi} H, \quad H_2^{u*} = \frac{c_{21}^f \phi_1^u}{\Psi} H, \\
H_1^{f*} = \frac{c_{12}^f \phi_2^u (\phi_1^u + \phi_1^f)}{\Psi} H, \quad H_2^{f*} = \frac{c_{21}^f \phi_1^u (\phi_2^u + \phi_2^f)}{\Psi} H
\]

with \( \Psi = c_{12}^f \phi_2^u (\phi_1^u + \phi_1^f) + c_{21}^f \phi_1^u (\phi_2^u + \phi_2^f) \). So the numbers of humans in patches 1 and 2 at the disease-free equilibrium of the traditional model (1) are
\[
H_1^* = \frac{c_{12}^f \phi_2^u (\phi_1^u + \phi_1^f)}{\Psi} H \quad \text{and} \quad H_2^* = \frac{c_{21}^f \phi_1^u (\phi_2^u + \phi_2^f)}{\Psi} H,
\]
respectively. Following (9) and the first two equations of (10), the human migration rates for the traditional model are
\[
c_{12} = \frac{\phi_2^u}{\phi_1^u + \phi_2^f} c_{12}^f \quad \text{and} \quad c_{21} = \frac{\phi_1^u}{\phi_1^u + \phi_2^f} c_{21}^f.
\]

Using the next generation matrix method [11, 47], the new infection and transition matrices of the new model (2) and the traditional model (1) are respectively
\[
F_2 = \begin{pmatrix}
0 & 0 & 0 & 0 & \frac{a_{1b_1} H_1^{u*}}{H_1^*} & 0 \\
0 & 0 & 0 & 0 & 0 & \frac{a_{2b_2} H_2^{u*}}{H_2^*} \\
0 & 0 & 0 & 0 & \frac{a_{1b_1} H_1^{f*}}{H_1^*} & 0 \\
0 & 0 & 0 & 0 & 0 & \frac{a_{2b_2} H_2^{f*}}{H_2^*} \\
\frac{a_{1c_1} V_1}{H_1^*} & 0 & \frac{a_{1c_1} V_1}{H_1^*} & 0 & 0 & 0 \\
0 & \frac{a_{2c_2} V_2}{H_2^*} & 0 & \frac{a_{2c_2} V_2}{H_2^*} & 0 & 0
\end{pmatrix} := \left( \begin{array}{c}
0 \\
0 \\
\bar{F}_h
\end{array} \right),
\]
\[
V_2 = \begin{pmatrix}
\gamma_1 + \phi_1^u & 0 & -\phi_1^f & 0 & 0 & 0 \\
0 & \gamma_2 + \phi_2^u & 0 & -\phi_2^f & 0 & 0 \\
-\phi_1^u & 0 & \gamma_1 + \phi_1^f + c_{21}^f & -c_{21}^f & 0 & 0 \\
0 & -\phi_2^u & -c_{21}^f & \gamma_2 + \phi_2^f + c_{21}^f & \mu_1 & 0 \\
0 & 0 & 0 & 0 & \mu_2 & 0
\end{pmatrix} := \left( \begin{array}{c}
\bar{V}_h \\
0 \\
\bar{V}_v
\end{array} \right),
\]
and
\[
F_3 = \begin{pmatrix}
0 & 0 & a_{1b_1} & 0 \\
0 & 0 & 0 & a_{2b_2} \\
\frac{a_{1c_1} V_1}{H_1^*} & 0 & 0 & 0 \\
0 & \frac{a_{2c_2} V_2}{H_2^*} & 0 & 0
\end{pmatrix} := \left( \begin{array}{c}
0 \\
0 \\
\bar{F}_h
\end{array} \right),
\]
\[
V_3 = \begin{pmatrix}
\gamma_1 + \phi_1^u & \frac{c_{21}^f}{\phi_1^u + \phi_1^f} & -\phi_2^f & 0 & 0 \\
-\phi_1^u & \gamma_2 + \phi_2^u + \phi_2^f & \frac{c_{21}^f}{\phi_2^u + \phi_2^f} & \mu_1 & 0 \\
0 & 0 & 0 & \mu_2 & 0
\end{pmatrix} := \left( \begin{array}{c}
\bar{V}_h \\
0 \\
\bar{V}_v
\end{array} \right).
Thus the basic reproduction numbers of the new model (2) and the traditional model (1) are respectively

\[ R_2 = \rho(F_h V_h^{-1}) = \sqrt{\rho(F_h V_h^{-1} F_h V_h^{-1})} = \sqrt{\rho(F_h V_h^{-1} F_h V_h^{-1})}, \]
\[ R_3 = \rho(F_3 V_3^{-1}) = \sqrt{\rho(F_h V_h^{-1} F_h V_h^{-1})} = \sqrt{\rho(F_h V_h^{-1} F_h V_h^{-1})}. \]

Denote \( P_1 = \gamma_1 + \phi_1^P, P_2 = \gamma_2 + \phi_2^P, P_3 = \phi_3^P + \phi_3^P, \) and \( P_4 = \gamma_2 + \phi_2^P. \) The characteristic equations corresponding to matrices \( F_h V_h^{-1} F_h V_h^{-1} \) and \( F_h V_h^{-1} F_h V_h^{-1} \) are respectively

\[ A_2 \lambda^2 - B_2 \lambda + C_2 = 0 \quad \text{and} \quad A_3 \lambda^2 - B_3 \lambda + C_3 = 0, \]

where

\[ A_2 = H^2 c_{12}^f c_{12}^f c_{12}^u c_{12}^u \mu_1 \mu_2 P_3^2 P_4^2 (c_{12}^f \gamma_1 (\gamma_2 + \phi_2^P) P_1 + \gamma_2 (c_{21}^f (\gamma_1 + \phi_1^P) + \gamma_1 P_1) P_2), \]
\[ B_2 = H (c_{12}^f c_{12}^u P_3 + c_{21}^f \phi_1^P P_4) \]
\[ \quad \times (V_1 a_1^2 b_1 c_1 c_{12}^f \mu_2 P_1^2 (c_{12}^f (\gamma_2 + \phi_2^P) P_1 P_3 + \gamma_2 (c_{21}^f \phi_1^P + P_1 P_1) P_2) \]
\[ + V_2 a_2^2 b_2 c_2 c_{12}^f \mu_1 P_1^2 (c_{12}^f (\gamma_1 + \phi_1^P) P_2 P_3 + \gamma_1 (c_{12}^f \phi_2^P + P_2 P_4) P_1)), \]
\[ C_2 = V_1 V_2 a_1^2 a_2^2 b_1 b_2 c_1 c_2 (c_{12}^f \phi_2^P P_3 + c_{21}^f \phi_1^P P_4)^2 (c_{12}^f \phi_2^P P_1 P_3 + (c_{21}^f \phi_1^P + P_1 P_3) P_2 P_4), \]

and

\[ A_3 = H^2 c_{12}^f c_{12}^f c_{12}^u \mu_1 \mu_2 (c_{12}^f c_{12}^f \phi_2^P P_4 + \gamma_1 (c_{12}^f \phi_2^P + \gamma_2 P_4) P_3), \]
\[ B_3 = H (c_{12}^f c_{12}^u P_3 + c_{21}^f \phi_1^P P_4) (V_1 a_1^2 b_1 c_1 c_{12}^f \mu_2 \phi_1^P (c_{12}^f \phi_2^P + \gamma_2 P_4) \]
\[ + V_2 a_2^2 b_2 c_2 c_{12}^f \mu_1 P_1^2 (c_{12}^f \phi_1^P + \gamma_1 P_3)), \]
\[ C_3 = V_1 V_2 a_1^2 a_2^2 b_1 b_2 c_1 c_2 (c_{12}^f \phi_2^P P_3 + c_{21}^f \phi_1^P P_4)^2. \]

Direct calculation yields

\[ (R_2)^2 = \frac{B_2 + \sqrt{B_2^2 - 4A_2C_2}}{2A_2} \quad \text{and} \quad (R_3)^2 = \frac{B_3 + \sqrt{B_3^2 - 4A_3C_3}}{2A_3}. \]

To compare the two basic reproduction numbers, we subtract them

\[ (R_2)^2 - (R_3)^2 = \frac{A_3 B_2 - A_2 B_3 + A_3 \sqrt{B_2^2 - 4A_2C_2} - A_2 \sqrt{B_3^2 - 4A_3C_3}}{2A_3}. \]

Note that

\[ A_3 B_2 - A_2 B_3 \]
\[ = H^3 c_{12}^f c_{12}^f c_{12}^u \mu_1 \mu_2 (c_{12}^f \phi_2^P P_3 + c_{21}^f \phi_1^P P_3) (c_{12}^f \phi_2^P P_1 P_3 + c_{21}^f \phi_1^P P_1 P_3) \]
\[ \times (V_1 a_1^2 b_1 c_1 (c_{21}^f)^2 \gamma_2^2 \mu_2 (\phi_1^P)^2 P_1^2 + V_2 a_2^2 b_2 c_2 (c_{12}^f)^2 \gamma_1^2 \mu_1 (\phi_2^P)^2 P_2^2) > 0 \]

and

\[ (A_3 B_2 - A_2 B_3)(B_3 C_2 - B_2 C_3) - (A_3 C_2 - A_2 C_3)^2 \]
\[ = H^4 V_1 V_2 a_1^2 a_2^2 b_1 b_2 c_1 c_2 (c_{12}^f)^2 (c_{21}^f)^2 \mu_1 \mu_2 (\phi_1^P)^2 (\phi_2^P)^2 (c_{12}^f \phi_2^P P_3 + c_{21}^f \phi_1^P P_4)^4 \]
\[ \times (a_1^2 b_1 c_1 V_1 c_{12}^f \mu_2 \gamma_2 \phi_2^P P_4 - a_2^2 b_2 c_2 V_2 c_{12}^f \mu_1 \gamma_1 \phi_2^P P_3)^2 \]
\[ \times (c_{12}^f \phi_2^P P_1 P_3 + c_{21}^f \phi_1^P P_2 P_4)^2 \geq 0. \]
Hence, similar to the proof of Theorem 3.9 in Gao [14], we have
\[(A_3B_2 - A_2B_3)(B_3C_2 - B_2C_3) \geq (A_3C_2 - A_2C_3)^2\]
\[\iff (B_3^2 - 4A_2C_2)(B_2^2 - 4A_3C_3) \geq (B_2B_3 - 2A_2C_3 - 2A_3C_2)^2\]
\[\Rightarrow \sqrt{B_3^2 - 4A_2C_2} \sqrt{B_2^2 - 4A_3C_3} \geq B_2B_3 - 2A_2C_3 - 2A_3C_2\]
\[\iff (A_3B_2 - A_2B_3)^2 \geq (A_2\sqrt{B_3^2 - 4A_3C_3} - A_3\sqrt{B_2^2 - 4A_2C_2})\]
\[\Rightarrow A_3B_2 - A_2B_3 \geq A_2\sqrt{B_3^2 - 4A_3C_3} - A_3\sqrt{B_2^2 - 4A_2C_2}\]
\[\iff R_2 \geq R_3,\]
with equality if and only if
\[R_0^{(1)} := \frac{a_1^2 b_1 c_1 V_1}{\gamma_1 H_1^{*}} = R_0^{(2)} := \frac{a_2^2 b_2 c_2 V_2}{\gamma_2 H_2^{*}},\]
i.e., the basic reproduction numbers of the two patches under isolation are exactly the same.

Within biologically reasonable parameter ranges, we will numerically show that the underestimate phenomenon is still very common when both unfrequent and frequent travelers move. However, traditional model can evidently overestimate the risk of infection when both human and mosquito move.

4. **Numerical results.** Human movement has undergone tremendous changes with the progress of globalization and urbanization [46]. In particular, more people travel more often than ever before, resulting in a significant increase in the proportion of frequent travelers in the whole population. In this section, some numerical simulations are carried out for the two-patch submodel to study the effects of differences in human travel behavior and human biting rate on the disease transmission.

**Example 4.1. Effect of travel frequency on** $R_0$. For model (2), the parameters are taken as follows:

- $a_1 = 0.10, a_2 = 0.15, \sigma_1 = \sigma_2 = 1,$
- $b_1^u = b_1^f = 0.2, b_2^u = b_2^f = 0.3, c_1^u = c_1^f = 0.3, c_2^u = c_2^f = 0.3,$
- $\gamma_1^u = \gamma_1^f = 0.012, \gamma_2^u = \gamma_2^f = 0.02, \mu_1 = 0.14, \mu_2 = 0.2,$
- $H = 10000, V_1 = 8000, V_2 = 11500,$
- $\phi_1^u = 2 \times 10^{-4}, \phi_2^u = 3 \times 10^{-4}, \phi_1^f = 5 \times 10^{-4}, \phi_2^f = 8 \times 10^{-4},$
- $c_{12}^u = 0.09, c_{21}^u = 0.06, c_{12}^f = 0.01, c_{21}^f = 0.01, d_{12} = d_{21} = 0.$

Thus, the respective basic reproduction numbers of patches 1 and 2 in isolation are $R_0^{(1)} = 0.7347$ and $R_0^{(2)} = 1.1122$, i.e., in the absence of population dispersal, the disease dies out in patch 1 but persists in patch 2. When the two patches are connected by human movement, the basic reproduction number of the new model is $R_0 = 1.0048$, i.e., the disease becomes endemic in both patches. Therefore, human movement can lead to disease outbreak in areas with low risk of infection.
Under the above parameter setting, the parameter values of the associated traditional model (1) are

\[ a_1 = 0.10, a_2 = 0.15, b_1 = 0.2, b_2 = 0.3, c_1 = 0.3, c_2 = 0.3, \]
\[ \gamma_1 = 0.012, \gamma_2 = 0.02, \mu_1 = 0.14, \mu_2 = 0.2, \]
\[ H = 10000, V_1 = 8000, V_2 = 11500, \]
\[ c_{12} = 0.02955, c_{21} = 0.02627, d_{12} = d_{21} = 0. \]

Thus, the basic reproduction number of the traditional model is \( \hat{R}_0 = 0.9935 \), i.e., the disease goes extinct. This scenario suggests that the traditional model underestimates disease persistence. Furthermore, using the same parameter values except that

\[ c_{12}^u = \tau_{12} c_{12}^f \quad \text{and} \quad c_{21}^u = \tau_{21} c_{21}^f, \quad \tau_{12}, \tau_{21} \in [0,1], \]

the difference of the basic reproduction numbers of the new model and the traditional model, \( R_0 - \hat{R}_0 \), with respect to \( \tau_{12} \) and \( \tau_{21} \), is shown in Figure 2. It can be seen that for this parameter set the traditional model always underestimates the transmission potential. Particularly, the amplitude of underestimate is large when the travel rate of unfrequent travelers is sufficiently small in relative to that of frequent travelers in low risk patch. More generally, we assume that both patches are homogeneous, that is, there is no epidemiological difference between unfrequent and frequent travelers in each patch. In other words, \( b_i^u = b_i^f, c_i^u = c_i^f, \) and \( \gamma_i^u = \gamma_i^f, \) \( i = 1, 2. \) With parameter ranges in Table 1, we use Latin hypercube sampling (LHS) method to randomly generate \( 10^5 \) parameter sets and calculate \( R_0 - \hat{R}_0 \) for each set. The following results are obtained under different assumptions:
(1) No mosquito movement ($d_{12} = d_{21} = 0$) and no biting difference ($\sigma_1 = \sigma_2 = 1$). The difference $R_0 - \hat{R}_0$ is constantly positive, i.e., the traditional model always underestimates the risk of infection.

(2) No mosquito movement ($d_{12} = d_{21} = 0$) but with biting difference $\sigma_i \in [0, 2.5]$. Only one scenario has negative $R_0 - \hat{R}_0$ with the difference being around $10^{-6}$. So the traditional model almost always underestimates the infection risk.

(3) Mosquitoes migrate between patches ($d_{ij} > 0$) and human biting rate is relevant to travel frequency ($\sigma_i \in [0, 2.5]$). There are 181 scenarios whose $R_0 - \hat{R}_0$ are negative with the maximum difference being less than 0.24.

In summary, ignoring the difference in travel frequency tends to underestimate the transmission potential when a homogeneous patchy environment is concerned.

**Example 4.2. Effect of more frequent travelers.** Consider a homogeneous two-patch environment without mosquito movement ($d_{12} = d_{21} = 0$) and biting difference ($\sigma_1 = \sigma_2 = 1$). Let $\phi_i^u = \tau_i \phi_i^f$ for $i = 1, 2$. It follows from Theorem 3.2 that an increase in $\tau_i$ will increase the size and fraction of frequent travelers in patch $i$. Choosing four parameter sets listed in Table 2 with ranges from Table 1, their associated contour plots of $R_0$ with respect to $\tau_1$ and $\tau_2$ are plotted in Figure 3. It shows that $R_0$ can simultaneously decrease or increase, or inconsistently monotonically or non-monotonically change in $\tau_1$ and $\tau_2$. Similar phenomena can be observed when unfrequent travelers receive more or less bites. Therefore, the dependence of the basic reproduction number of the new model (2) on the frequency exchange rates are very complicated even under strong restrictions.

**Example 4.3. Effect of travel frequency on disease prevalence.** We again assume that the two patches are homogeneous and there is no mosquito movement between the two patches. Moreover, a patch is called a high-risk or low-risk patch if its patch reproduction number is larger or smaller than that of the other patch. When $R_0 > 1$, there exists a unique endemic equilibrium $E^*$. We calculate the respective disease prevalences of the unfrequent and frequent travelers at $E^*$ in each patch, i.e., $h_i^u / H_i^u$ and $h_i^f / H_i^f$ for $i = 1, 2$, and compare them to see which group is at higher infection risk within patch and the highest infection risk across patches. Adopting the parameter ranges in Table 1, we again use the LHS method to generate $10^5$ random parameter sets and get the following results:

(1) No biting difference ($\sigma_1 = \sigma_2 = 1$). We get 99,601 parameter sets with basic reproduction numbers greater than one. Among these qualified parameter sets, 91,900 (92.27%) scenarios have unfrequent travelers in high-risk patch and frequent travelers in low-risk patch being high-risk groups. In addition, 91,941 parameter sets have the unfrequent travelers in high-risk patch being at the highest risk of infection and 92,813 parameter sets have the unfrequent travelers in low-risk patch being the lowest risk group. However, we obtain 99,661 parameter sets whose corresponding basic reproduction numbers are over one. For the qualified parameter sets, 69,126 (69.36%) scenarios have unfrequent travelers in both patches being high-risk groups, and 29,972 (30.07%) scenarios have unfrequent travelers in high-risk patch and frequent travelers in low-risk patch being high-risk groups. In addition, 92,260 parameter sets have unfrequent traveler in high-risk patch being the highest risk group, and 30,282 and 59,357 parameter sets have unfrequent and frequent travelers in low-risk patch being the lowest risk group, respectively.
Table 2. Parameter settings for Figure 3.

| Symbol | Figure 3a | Figure 3b | Figure 3c | Figure 3d |
|--------|-----------|-----------|-----------|-----------|
| $a_1$  | 0.438     | 0.380     | 0.535     | 0.161     |
| $a_2$  | 0.402     | 0.100     | 0.147     | 0.051     |
| $b_1$  | 0.548     | 0.686     | 0.373     | 0.656     |
| $b_2$  | 0.642     | 0.159     | 0.178     | 0.716     |
| $c_1$  | 0.398     | 0.324     | 0.608     | 0.586     |
| $c_2$  | 0.636     | 0.085     | 0.618     | 0.403     |
| $\gamma_1$ | 0.049    | 0.044     | 0.044     | 0.009     |
| $\gamma_2$ | 0.026    | 0.029     | 0.014     | 0.020     |
| $\mu_1$ | 0.135     | 0.066     | 0.094     | 0.053     |
| $\mu_2$ | 0.165     | 0.194     | 0.174     | 0.103     |
| $\sigma_1$ | 1         | 1         | 1         | 1         |
| $\sigma_2$ | 1         | 1         | 1         | 1         |
| $c_{12}^u$ | 0.021    | 0.006     | 0.0128    | 0.0028    |
| $c_{21}^u$ | 0.018    | 0.002     | 0.0182    | 0.0005    |
| $c_{12}^f$ | 0.094    | 0.060     | 0.0534    | 0.0550    |
| $c_{21}^f$ | 0.092    | 0.076     | 0.0943    | 0.0497    |
| $d_{12}$ | 0         | 0         | 0         | 0         |
| $d_{21}$ | 0         | 0         | 0         | 0         |
| $\phi_1^u$ | $7.73 \times 10^{-5}$ | $2.80 \times 10^{-4}$ | $1.40 \times 10^{-4}$ | $1.50 \times 10^{-4}$ |
| $\phi_2^u$ | $5.11 \times 10^{-5}$ | $3.46 \times 10^{-4}$ | $2.10 \times 10^{-4}$ | $1.50 \times 10^{-4}$ |
| $\phi_1^f$ | $7.16 \times 10^{-4}$ | $7.58 \times 10^{-4}$ | $5.35 \times 10^{-4}$ | $4.82 \times 10^{-4}$ |
| $\phi_2^f$ | $3.24 \times 10^{-4}$ | $7.62 \times 10^{-4}$ | $4.67 \times 10^{-4}$ | $5.55 \times 10^{-4}$ |
| $V_1$ | 5859     | 23633     | 6065      | 13838     |
| $V_2$ | 18790    | 29698     | 17129     | 19918     |
| $H$   | 10000    | 10000     | 10000     | 10000     |

(3) Frequent travelers receive more bites ($\sigma_i \in [1, 5]$). There are 99,725 parameter sets whose basic reproduction numbers are more than one. Among them, 96,555 (96.82%) scenarios have frequent travelers in both patches being high-risk groups. In addition, 89,679 parameter sets have frequent travelers in high-risk patch being at the highest infection risk and 90,186 parameter sets have unfrequent travelers in low-risk patch being at the lowest infection risk.

From the above simulations, we can see that the disease prevalence in humans is not only affected by travel frequency, but also by human biting rate.

5. Discussion. Statistical data show that, affected by age, gender, income, ethnicity, climate and other factors, there are significant differences in travel frequency among people. Meanwhile, the number of mosquito bites per person is affected by many factors like blood type, age, quality of bed nets and location. In this paper, based on the classical Ross–Macdonald model, we developed a two-group multipatch model to study the impact of heterogeneity in travel frequency and human biting rate on the transmission of mosquito-borne diseases. The disease-free equilibrium is calculated and the basic reproduction number $R_0$ is defined. It is proved that the disease-free equilibrium is globally asymptotically stable when $R_0 \leq 1$ and there exists a unique endemic equilibrium which is globally asymptotically stable when $R_0 > 1$. For the single patch case, the basic reproduction number and the unique
Figure 3. The contour plot of $R_0$ versus the relative frequency change rates $\tau_1$ and $\tau_2$. (a) simultaneously decreases, (b) simultaneously increases, (c) increases in $\tau_1$ but decreases $\tau_2$, (d) non-monotonically varies with $\tau_1$ and decreases in $\tau_2$.

endemic equilibrium are explicitly solved, and the same global dynamics result is given. The relationship between frequency change rates and the size of frequent travelers is established. When there are no epidemiological and biting differences between unfrequent and frequent travelers and only frequent travelers migrate between two patches, we rigorously prove that the traditional model which does not distinguish travel frequency underestimates the transmission potential in comparison to the new model. Further, through numerical approach, it is concluded that the traditional model still underestimates the risk of infection as long as mosquitoes do not move. However, traditional model may occasionally slightly overestimates the infection risk when both human and mosquito move. In addition, the amplitude of the difference of the basic reproduction numbers of the new model and the traditional model depends on the relative travel rates of unfrequent travelers to frequent travelers. An increase in the proportion of frequent travelers may decreasingly, increasingly or non-monotonously change the basic reproduction number $R_0$. The human disease prevalence is affected by patch reproduction number, and heterogeneities in travel frequency and human biting rate.
The current study indicates that the model with the consideration of travel frequency and biting difference can better describe the spatial spread of mosquito-borne diseases. From the aspect of application, the subdivision of the human population by travel frequency enables us to identify high-risk groups and to optimize the control strategy when limited medical resources are available. However, the present model is relatively simple and ignores some important epidemiological and ecological factors. For example, the symptoms of some mosquito-borne diseases like malaria could seriously reduce the mobility of infected people and cause up to 2 million deaths annually worldwide [19]. The extrinsic incubation period in mosquitoes is crucial since most infected mosquitoes are likely to die before they can transmit the infectious agents to humans [41]. The gonotrophic cycle (from blood feeding to oviposition) for malaria parasite in adult Anopheles mosquitoes can take over a week. Temporary immunity to diseases can be acquired after repeat infections [7]. The birth rate and survival rate of mosquitoes are influenced by climatic factors like temperature, rainfall and humidity such that the mosquito abundance is seasonally varying [36]. Thus, we may study the joint effect of seasonality, travel frequency and biting difference on the geographic spread of mosquito-borne diseases by generalizing the periodic multipatch Ross–Macdonald model proposed by Gao et al. [18]. Intervention strategies such as case management, vector control, vaccine, and prophylactic medicine need to be included for disease control. Human population may be divided into more subgroups according to the frequency of travel. The influence of increasing percentage of frequent travelers on the spread of vector-borne diseases need a qualitative study. We leave these interesting but more challenging problems for future consideration.

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