# BIRDS MOVEMENT IMPACT ON THE TRANSMISSION OF WEST NILE VIRUS BETWEEN PATCHES\*

Juping Zhang<sup>1,2</sup>, Zhen Jin<sup>1,2,†</sup> and Huaiping Zhu<sup>1,3</sup>

Abstract Spatial heterogeneity plays an important role in the distribution and persistence of many infectious disease. In the paper, a multi-patch model for the spread of West Nile virus among n discrete geographic regions is presented that incorporates a mobility process. In the mobility process, we assume that the birds can move among regions, but not the mosquitoes based on scalespace. We show that the movement of birds between patches is sufficient to maintain disease persistence in patches. We compute the basic reproduction number  $R_0$ . We prove that if  $R_0 < 1$ , then the disease-free equilibrium of the model is globally asymptotically stable. When  $R_0 > 1$ , we prove that there exists a unique endemic equilibrium, which is globally asymptotically stable on the biological domain. Finally, numerical simulations demonstrate that the disease becomes endemic in both patches when birds move back and forth between two regions.

**Keywords** West Nile virus, patch model, birds movement, basic reproduction number, stability.

**MSC(2010)** 34A34, 34D23.

### 1. Introduction

West Nile virus(WNV) was first isolated and identified as a distinct pathogen from the blood of a woman in the West Nile region of Uganda in 1937 [19]. In the African tropics, Middle East and temperate Eurasia, WNV has been found to be a rather common pathogen [9]. However, WNV was first recorded in the New World during August 1999 in New York City [20]. Until the outbreak in New York, the occurrence of WNV had never previously been documented in the Western Hemisphere. In the subsequent five years the epidemic has spread spatially to most of the west coast of North America. Migratory birds have long been suspected as the principal

<sup>&</sup>lt;sup>†</sup>the corresponding author. Email address:jinzhn@263.net(Z. Jin)

<sup>&</sup>lt;sup>1</sup>Complex Systems Research Center, Shanxi University, Taiyuan City, Shanxi Province, 030006, China

<sup>&</sup>lt;sup>2</sup>Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention, Shanxi University, Taiyuan City, Shanxi Province, 030006, China

<sup>&</sup>lt;sup>3</sup>LAMPS and Department of Mathematics and Statistics, York University, Toronto, Ontario, M3J 1P3, Canada

<sup>\*</sup>The authors were supported by supported by the National Natural Sciences Foundation of China (11301491, 11331009, 11501339), Research Project Supported by Shanxi Scholarship Council of China(2014-020), the One Hundred Talents Project of Shanxi Province.

introductory hosts of WNV into new regions. Migratory birds have been linked with transporting related viruses in the Western Hemisphere [2,13]. Wonham et al. [25] have suggested that the WNV model should be extended biologically to consider bird migration. Rappole et al. [16] speculated that migratory birds might serve as the principal introductory hosts for the virus in the New World. Rappole et. al. [17] have provided some factors supporting the hypothesis that the migrant bird is an introductory host for the spread of WNV. Owen et al. [15] have demonstrated that migrating passerine birds are potential dispersal vehicles for WNV. These studies show that the importance of birds migration on the distribution and maintenance of WNV can hardly be underestimated.

Spatial structures play an important role in describing the spreading of communicable diseases, not only because the environment is heterogeneous but also because individuals move around in space. In modeling spatial effects on the spread of a disease, one is the diffusion model, which denote migration of individuals between continuous adjacent zones. These model commonly use partial differential equation(PDE). The other is dispersal model, which denote migration of individuals between discrete geographical regions [26]. The discrete geographical regions can be families, villages, cities, towns, states, countries or other appropriate community divisions. These model commonly use the metapopulation concept. One can refer to the survey articles of [23] and [14].

Vector-borne diseases in recent years has been seriously endanger human health. Migration patterns of the hosts, birds and humans, is one of the important reasons that cause the worldwide spread of the vector-borne diseases. Some articles have considered the effect of host or vector migration among multiple patches on the dynamics of vector-borne diseases. Dye and Hasibeder [6], Hasibeder and Dye [8] investigated the persistence of a mosquito-borne disease(malaria) in a system where mosquitoes and hosts are grouped in patches containing any number of individuals. Rodriguez and Torres-Sorando [18] studied models with hosts distributed in subpopulations as a consequence of spatial partitioning. The results indicate the importance of knowing the spatial distribution and mobility patterns to understand the dynamics of infectious diseases. Torres-Sorando and Rodriguez [22] described the dynamics of malaria in time in a heterogeneous environment, including migration between patches with no return, and visitation in which the individuals return to their patch of origin after visiting other patches. Smith et al. [21] consider the malaria model that mosquitoes are assumed to move but humans are not. Liu et al. [12] studied the impact of directional dispersal of birds on the spatial spreading of WNV. Auger et. al. [1] formulate a Ross-MacDonald model on n patches to describe the transmission dynamics of malaria. Cosner et. al. [4] consider the impact of both short term host movement and long-term host migration on the dynamics of vector-borne diseases. Gao and Ruan [7] proposed a multi-patch model to study the effects of population dispersal on the spatial spread of malaria between patches.

In the paper, we use spatial model in heterogenous environments affect the transmission of WNV. Based on the model [4], a type of movement is where birds are commuting between locations (or changing their activities) on a regularly scheduled basis, so that there is a well defined fraction of time that any given individual spends in any given location or state of activity. Our description identifies birds as resident in a given patch and assumes that they remain in that patch, but may visit other patches often for spending some time in staying in the patch. We formulate the spatial WNV model with birds movement between patches.

#### 2. Formulation of the model

Let  $M_{jS}(t), M_{jI}(t)$  denote the numbers of susceptible and infective in the mosquitoes population, and  $B_{jS}(t), B_{jI}(t)$ , and  $B_{jR}(t)$  denote the numbers of susceptible, infective, and recovered in the birds population in the *j*-th patch,  $j = 1, 2, \dots, n$ . Due to the vector's short life, a mosquito never recovers from the infection, and we do not consider the recovered class in this population. When the *n* patches are connected, we assume that only birds can move among the patches since mosquitoes move only small distances during their lifetime. We define transmission rates by averaging the rates across patches weighted by the fractions of their time that birds spend in each patch. We denote  $p_{jk}$ , note that  $\sum_{k=1}^{n} p_{jk} = 1$ . The flow chart shown in Fig.1.



Figure 1. Flow chart for the transmission of WNV between patches via visit of birds.

The following system of differential equations captures WNV spread among n patches

$$\begin{aligned} \frac{dM_{jS}}{dt} &= b_m M_{jS} + (1-q) b_m M_{jI} - d_m M_{jS} - b\beta_1 \sum_{k=1}^n \frac{p_{kj} B_{kI} M_{jS}}{N_{kB}}, \\ \frac{dM_{jI}}{dt} &= q b_m M_{jI} + b\beta_1 \sum_{k=1}^n \frac{p_{kj} B_{kI} M_{jS}}{N_{kB}} - d_m M_{jI}, \\ \frac{dB_{jS}}{dt} &= b_b N_{jB} - d_b B_{jS} - b\beta_2 \sum_{k=1}^n \frac{p_{jk} M_{kI} B_{jS}}{N_{jB}}, \qquad j = 1, 2, \cdots, n, \end{aligned}$$
(2.1)  
$$\begin{aligned} \frac{dB_{jI}}{dt} &= b\beta_2 \sum_{k=1}^n \frac{p_{jk} M_{kI} B_{jS}}{N_{jB}} - (d_b + \gamma_b + \alpha_b) B_{jI}, \\ \frac{dB_{jR}}{dt} &= \gamma_b B_{jI} - d_b B_{jR}, \end{aligned}$$

where  $N_{jM} = M_{jS} + M_{jI}$ , and  $N_{jB} = B_{jS} + B_{jI} + B_{jR}$  are the total number of mosquitoes and birds in the *j*-th patch,  $j = 1, 2, \dots, n$ . The interpretations

of parameters are described in Table 1. We also give some parameters value for simulation and summarize them in Table 1. The parameters in this model are all positive constants.

	<b>Table 1.</b> Parameters of the model $(2.1)$ .		
Parameter	Interpretation	Value	Resource
$b_m$	The birth rates of mosquitos(per day)	Parameter	
$d_m$	The natural death rates of mosquitos(per day)	Parameter	
$b_b$	The birth rates of birds(per day)	Parameter	
$d_b$	The natural death rates of birds(per day)	Parameter	
$\alpha_b$	The WNV-induced death rates of birds(per day)	Parameter	
q	The vertical transmission rate of infectious mosquitoes	0.007	[3]
b	Biting rate of mosquito on bird	0.50	[3]
$\beta_1$	Trans. probability from bird to mosquito	0.26	[3]
$\beta_2$	Trans. probability from mosquito to bird	1.00	[3]
$\gamma_b$	Recovery rate of infected bird (per day)	0.36	[3]
$p_{jk}$	The fraction of time a bird resident in patch $j$		
	spends visiting patch $k$	Parameter	
$N_{jM}$	The total number of mosquitos in Patch $j$	Parameter	
$N_{jB}$	The total number of birds in Patch $j$	Parameter	

For sake of simplicity, we assume that the parameters  $b_m$ ,  $b_b$ , q, b,  $d_m$ ,  $d_b$ ,  $\alpha_b$ ,  $\gamma_b$ ,  $\beta_1$  and  $\beta_2$  are the same for *n* patches. However, the analysis presented here can be extended when these parameters differ from patch to patch. Based on the model (2.1), we assume that bird and mosquito populations are fixed but there is turnover in the bird and mosquito population because of adult mortality. Therefore, we constraint parameters  $b_m = d_m$ ,  $b_b = d_b$ ,  $\alpha_b = 0$ .

In order to reduce the number of parameters and simplify the analysis of the system (2.1), we normalize the bird and mosquito population by letting

$$M_{js} = \frac{M_{jS}}{N_{jM}}, M_{ji} = \frac{M_{jI}}{N_{jM}}, B_{js} = \frac{B_{jS}}{N_{jB}}, B_{ji} = \frac{B_{jI}}{N_{jB}},$$
$$B_{jr} = \frac{B_{jR}}{N_{iB}}, a_{kj} = \frac{N_{kM}}{N_{iB}}, \quad j, k = 1, \cdots, n.$$

Since  $B_{jr} = 1 - B_{js} - B_{ji}$  and  $M_{js} = 1 - M_{ji}$ , we can omit the equations for  $M_{js}$ and  $B_{ir}$ . Then the system (2.1) is equivalent to the following system

$$\frac{dM_{ji}}{dt} = b\beta_1 \sum_{k=1}^n p_{kj} B_{ki} (1 - M_{ji}) - (1 - q) d_m M_{ji},$$
  
$$\frac{dB_{js}}{dt} = d_b - d_b B_{js} - b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki} B_{js}, \qquad j = 1, 2, \cdots, n, \qquad (2.2)$$
  
$$\frac{dB_{ji}}{dt} = b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki} B_{js} - (d_b + \gamma_b) B_{ji}.$$

For model (2.2), all trajectories stay inside the region  $\Omega = \{0 \leq B_{js}, B_{ji}, B_{js} + B_{ji} \leq 0\}$ 

 $1, 0 \leq M_{ji} \leq 1, j = 1, \dots, n$ . One can observe that if  $p_{jj} = 1$ , then  $p_{jk} = 0$   $(j \neq k)$ , and the model (2.1) becomes a single-patch model of WNV.

# 3. The disease-free equilibrium and basic reproduction number

In the following, we first find  $R_0$  by the approach in van den Driessche and Watmough [24]. The model as follows

$$\frac{dx_j}{dt} = f_j(x) = \mathscr{F}_j(x) - \mathscr{V}_j(x),$$

where  $x = (x_1, \ldots, x_n)$ ,  $\mathscr{F}_j$  is the rate at which new infections occur in compartment j and  $-\mathscr{V}_j$  is the rate of movement of individuals into or out of that compartment by other means. The rate  $\mathscr{V}_j$  is broken down further as  $\mathscr{V}_j = \mathscr{V}_j^+ - \mathscr{V}_j^-$ , where  $\mathscr{V}_j^+, \mathscr{V}_j^-$  are rates of individuals entering and leaving compartment j, respectively. If f(x) satisfies the conditions (A1)-(A5) [24], then Theorem 2 in [24] tells us that the disease-free equilibrium is locally asymptotically stable if  $R_0 < 1$  but unstable if  $R_0 > 1$ , where  $R_0 = \rho(FV^{-1})$ , the spectral radius of the matrix  $FV^{-1}$ , F and V are  $[\frac{\partial \mathscr{F}_j}{\partial x_k}(x^0)]$  and  $[\frac{\partial \mathscr{V}_j}{\partial x_k}(x^0)]$  with  $1 \leq j, k \leq n$ , respectively,  $x^0$  is the disease-free equilibrium(DFE).

In our case, the system contain 2n infected populations, namely  $M_{ji}$  and  $B_{ji}$ ,  $j = 1, 2, \cdots, n$ . We have  $x = (M_{1i}, \ldots, M_{ni}, B_{1i}, \cdots, B_{ni})$ . Then  $\mathscr{F}_j(x) = b\beta_1 \sum_{k=1}^n p_{kj} B_{ki}$  (1– $M_{ji}$ ),  $j = 1, \cdots, n$ ,  $\mathscr{F}_j(x) = b\beta_2 \sum_{k=1}^n p_{(j-n)k} a_{k(j-n)} M_{ki} B_{(j-n)s}$ ,  $j = n+1, \cdots, 2n$ , and  $\mathscr{V}_j^+ = (1-q)d_m M_{ji}$ ,  $j = 1, \cdots, n$ ,  $\mathscr{V}_j^+ = (d_b + \gamma_b)B_{(j-n)i}$ ,  $j = n+1, \cdots, 2n$ ,  $\mathscr{V}_j^- = 0$  for  $j = 1, \ldots, 2n$ . We can see that the hypotheses (A1)-(A5) [24] can be readily verified. Note that, in our models (2.2) the disease-free equilibrium is  $E^0(0, 1, 0, \cdots, 0, 1, 0)$ . Linearizing of  $\mathscr{F}$  and  $\mathscr{V}$  about the trivial equilibrium, we have that

$$F = \begin{pmatrix} 0 & \mathcal{A} \\ \mathcal{B} & 0 \end{pmatrix}, V = \begin{pmatrix} \mathcal{C} & 0 \\ 0 & \mathcal{D} \end{pmatrix}$$

where  $\mathcal{A} = (b\beta_1 p_{kj}), \mathcal{B} = (b\beta_2 p_{jk} a_{kj}), \mathcal{C} = \text{diag}((1-p)d_m)$  and  $\mathcal{D} = \text{diag}(d_b + \gamma_b)$ . By calculating, it follows that

$$FV^{-1} = \begin{pmatrix} 0 & \mathcal{AD}^{-1} \\ \mathcal{BC}^{-1} & 0 \end{pmatrix}.$$

Further, we have

$$(FV^{-1})^2 = \begin{pmatrix} \mathcal{A}\mathcal{D}^{-1}\mathcal{B}\mathcal{C}^{-1} & 0\\ 0 & \mathcal{B}\mathcal{C}^{-1}\mathcal{A}\mathcal{D}^{-1} \end{pmatrix},$$

so that  $R_0^2 = \rho(\mathcal{BC}^{-1}\mathcal{AD}^{-1})$ , where  $\rho(\mathcal{BC}^{-1}\mathcal{AD}^{-1})$  represents the spectral radius of the matrix  $\mathcal{BC}^{-1}\mathcal{AD}^{-1}$ .

**Theorem 3.1.** If  $R_0 < 1$ , then the disease-free equilibrium  $E^0$  of system (2.2) is locally asymptotically stable, if  $R_0 > 1$ , then  $E^0$  is unstable.

In following, we prove the global stability of  $E^0$ .

**Theorem 3.2.** If  $R_0 < 1$ , then the disease-free equilibrium  $E^0$  of system (2.2) is globally asymptotically stable.

**Proof.** Let  $(M_{1i}, B_{1s}, B_{1i}, \dots, M_{ni}, B_{ns}, B_{ni})$  be a non-negative solution of system (2.2). To complete the proof, it is sufficient to show that this non-negative solution tends to the disease-free equilibrium  $E^0$  as  $t \to +\infty$ .

The first and third equations of the system (2.2) with  $M_{js}, B_{js} \leq 1$  gives the inequality

$$\frac{dM_{ji}}{dt} \le b\beta_1 \sum_{k=1}^n p_{kj} B_{ki} - (1-q) d_m M_{ji}, 
\frac{dB_{ji}}{dt} \le b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki} - (d_b + \gamma_b) B_{ji}, \qquad j = 1, 2, \cdots, n.$$
(3.1)

Define an auxiliary linear system by (3.1), namely

$$\frac{dM_{ji}}{dt} = b\beta_1 \sum_{k=1}^n p_{kj} B_{ki} - (1-q) d_m M_{ji},$$

$$\frac{dB_{ji}}{dt} = b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki} - (d_b + \gamma_b) B_{ji}, \qquad j = 1, 2, \cdots, n.$$
(3.2)

The right side of (3.2) has coefficient matrix F - V. For  $R_0 = \rho(FV^{-1}) < 1$ , each eigenvalue of F - V lies in the left half plane. Thus each non-negative solution of (3.2) satisfies  $\lim_{t\to\infty} M_{ji}(t) = 0$  and  $\lim_{t\to\infty} B_{ji}(t) = 0, j = 1, \cdots, n$ . Since (3.2) is a linear system, the DFE of (3.2) is globally asymptotically stable. By the comparison principle, it is easy to see that each non-solution of the first and third equation of the system (2.2) satisfies  $\lim_{t\to\infty} M_{ji}(t) = 0$  and  $\lim_{t\to\infty} B_{ji}(t) = 0, j = 1, \cdots, n$ . From the second equation of the system (2.2), since  $\lim_{t\to\infty} M_{ji}(t) = 0$ , then

$$\frac{dB_{js}}{dt} = d_b - d_b B_{js}, \qquad j = 1, 2, \cdots, n.$$
 (3.3)

Thus  $\lim_{t\to\infty} B_{js}(t) = 1, j = 1, \dots, n$ , completing the proof that the DFE is globally asymptotically stable.

# 4. The existence and stability of the endemic equilibrium

To obtain all positive equilibrium points, we let  $\chi_j = B_{js} + B_{ji}$ , and  $M_{ji} = 1 - M_{js}$ , then the system (2.2) can be rewritten as

$$\frac{dM_{js}}{dt} = (1-q)d_m - (1-q)d_m M_{js} - b\beta_1 \sum_{k=1}^n p_{kj}\chi_k M_{js} + b\beta_1 \sum_{k=1}^n p_{kj}B_{ks}M_{js},$$

$$\frac{dB_{js}}{dt} = d_b - d_b B_{js} - b\beta_2 \sum_{k=1}^n p_{jk}a_{kj}B_{js} + b\beta_2 \sum_{k=1}^n p_{jk}a_{kj}M_{ks}B_{js},$$

$$\frac{d\chi_j}{dt} = d_b - (d_b + \gamma_b)\chi_j + \gamma_b B_{js},$$

$$j = 1, 2, \cdots, n.$$
(4.1)

If we now set

$$\mathbf{z} = (\underbrace{M_{1s}, \cdots, M_{ns}}_{n}, \underbrace{B_{1s}, \cdots, B_{ns}}_{n}, \underbrace{\chi_{1}, \cdots, \chi_{n}}_{n})^{T},$$

$$\mathbf{e} = (\underbrace{-(1-q)d_{m}, \cdots, -(1-q)d_{m}}_{n}, \underbrace{-(d_{b}+b\beta_{2}\sum_{k=1}^{n}p_{1k}a_{k1}), \cdots, -(d_{b}+b\beta_{2}\sum_{k=1}^{n}p_{nk}a_{kn})}_{n},$$

$$\underbrace{-(d_{b}+\gamma_{b}), \cdots, -(d_{b}+\gamma_{b})}_{n})^{T},$$

$$\mathbf{c} = (\underbrace{(1-q)d_{m}, \cdots, (1-q)d_{m}}_{n}, \underbrace{d_{b}, \cdots, d_{b}}_{n}, \underbrace{d_{b}, \cdots, d_{b}}_{n})^{T},$$

$$A = \begin{pmatrix} 0 & A_{12} & A_{13} \\ A_{21} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, A_{12} = (b\beta_{1}p_{kj}), A_{13} = (-b\beta_{1}p_{kj}), A_{21} = (b\beta_{2}p_{jk}a_{kj}).$$

$$B = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & B_{32} & 0 \end{pmatrix}, B_{32} = \operatorname{diag}(\gamma_{b}).$$

The system (4.1) becomes

$$\frac{d\mathbf{z}}{dt} = \operatorname{diag}(\mathbf{z})(\mathbf{e} + A\mathbf{z}) + (\mathbf{c} + B\mathbf{z}).$$
(4.2)

Lemma 4.1 (Corollary 2.4. [5]). If the vector c in (4.2) is strictly positive, then the system (4.2) admits a strictly positive equilibrium  $z^* \in \Omega_+$ , where  $\Omega_+ = \Omega - \{0\}$ ,  $\Omega$  is positively invariant.

From Lemma 4.1, we obtain that system (4.1) have a strictly positive equilibrium  $(M_{1s}^*, \cdots, M_{ns}^*, B_{1s}^*, \cdots, B_{ns}^*, \chi_1^*, \cdots, \chi_n^*).$  **Namely**, system (2.2) have the endemic equilibrium  $E^*(M_{1i}^*, \cdots, M_{ni}^*, B_{1s}^*, \cdots, M_{ni}^*, M_{$ 

 $B_{ns}^*, B_{1i}^*, \cdots, B_{ni}^*) \in \Omega_+$ , where  $M_{ji}^*, B_{js}^*, B_{ji}^* > 0$  satisfy the equilibrium equations

$$(1-q)d_m M_{ji}^* = b\beta_1 \sum_{k=1}^n p_{kj} B_{ki}^* (1-M_{ji}^*), \qquad (4.3)$$

$$d_b = d_b B_{js}^* - b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki}^* B_{js}^*, \qquad (4.4)$$

$$(d_b + \gamma_b)B_{ji}^* = b\beta_2 \sum_{k=1}^n p_{jk} a_{kj} M_{ki}^* B_{js}^*.$$
(4.5)

In the remaining of this section, we shall show that system (2.2) has only one endemic equilibrium, which is globally asymptotically stable. We first introduce a theoretical result which plays an important role in our discussion.

Lemma 4.2 (Theorem 3.1 and Corollary 3.3 [10]). Assume that the following assumptions are satisfied.

(i) There exist functions  $U_j(t, u_j)$ ,  $F_{jk}(t, u_j, u_k)$ , and constants  $b_{jk} \ge 0$  such that

$$\dot{U}_j \le \sum_{k=1}^n b_{jk} F_{jk}(t, u_j, u_k), \quad t > 0, \quad u_j \in D_j \subset R^{m_j}, \quad j = 1, \cdots, n.$$

(ii) Along each directed cycle  $\mathbb{C}$  the weighted digraph  $(\mathcal{G}, \mathbb{A}), \mathbb{A} = (\partial_{jk}),$ 

$$F_{jk}(t, u_j, u_k) \le G_j(t, u_j) - G_k(t, u_k),$$

where  $G_i(t, u_i)$  is arbitrary functions.

(iii) Constants  $r_i$  are given in (2.2) of [10].

Then the function  $U(t, u) = \sum_{j=1}^{n} r_j U_j$  satisfies  $U(t, u) \le 0$  for t > 0 and, namely, t is a Lyapupov function for system  $u'_i = f_i(t, u_i) + \sum_{j=1}^{n} a_{ij}(t, u_j, u_j)$ 

U is a Lyapunov function for system  $u'_j = f_j(t, u_j) + \sum_{k=1}^n g_{jk}(t, u_j, u_k).$ 

Let  $\mathcal{G}$  be a digraph with *n* vertices, in which each vertex represents a group. An arc (j,k) exists if and only if  $b\beta_1 p_{kj} > 0$  or  $b\beta_2 p_{jk} a_{kj} > 0$ , namely, if the disease can be transmitted from group *j* to group *k*. System (2.2) can thus be regarded as a coupled system on  $\mathcal{G}$ . We note that  $\mathcal{G}$  is strongly connected if and only if transmission matrix  $(b\beta_1 p_{kj})$  or  $(b\beta_2 p_{jk} a_{kj})$  is irreducible.

**Theorem 4.1.** Assume that the matrix  $(b\beta_1 p_{kj})$  or  $(b\beta_2 p_{jk} a_{kj})$  is irreducible, if  $R_0 > 1$ , and the following conditions

$$(M_{ki} - M_{ki}^*)(M_{ki}B_{ki}^* - M_{ki}^*B_{ki}) \le 0,$$

$$(B_{ki}(1 - M_{ji}) - B_{ki}^*(1 - M_{ji}^*))(M_{ki}^*B_{ki}(1 - M_{ji}) - M_{ki}B_{ki}^*(1 - M_{ji}^*)) \le 0,$$

$$(4.6)$$

 $(j,k,i = 1,2,\cdots,n)$  are true, then there exists a unique endemic equilibrium  $E^*$  for system (2.2), and  $E^*$  is globally asymptotically stable in  $\Omega_+$ .

**Proof.** Consider a Lyapunov funcation for a single-patch model

$$U_j(M_{ji}, B_{js}, B_{ji}) = (M_{ji} - M_{ji}^* \ln M_{ji}) + (B_{js} - B_{js}^* \ln B_{js}) + (B_{ji} - B_{ji}^* \ln B_{ji}).$$

We verify that  $U_j$  satisfies the assumption of Lemma 4.2. Using equilibrium equations (4.3)-(4.5), we obtain

$$\begin{split} \dot{U}_{j} &= \left(1 - \frac{M_{ji}^{*}}{M_{ji}}\right) \left(b\beta_{1} \sum_{k=1}^{n} p_{kj} B_{ki} (1 - M_{ji})\right) + \left(1 - \frac{B_{js}^{*}}{B_{js}}\right) \left(d_{b} - d_{b} B_{js} - b\beta_{2} \sum_{k=1}^{n} p_{jk} a_{kj} M_{ki} B_{js}\right) \\ &+ \left(1 - \frac{B_{ji}^{*}}{B_{ji}}\right) \left(b\beta_{2} \sum_{k=1}^{n} p_{jk} a_{kj} M_{ki} B_{js} - (d_{b} + \gamma_{b}) B_{ji}\right) \\ &= -d_{b} B_{js}^{*} \left(\frac{B_{js}^{*}}{B_{js}} + \frac{B_{js}}{B_{js}^{*}} - 2\right) + b\beta_{2} \sum_{k=1}^{n} p_{jk} a_{kj} M_{ki}^{*} B_{js}^{*} \left(2 + \frac{M_{ki}}{M_{ki}^{*}} - \frac{B_{js}^{*}}{B_{js}} - \frac{M_{ki} B_{js} B_{ji}^{*}}{M_{ki}^{*} B_{js}^{*} B_{ji}} - \frac{B_{ji}}{B_{ji}^{*}}\right) \\ & b\beta_{1} \sum_{k=1}^{n} p_{kj} B_{ki}^{*} (1 - M_{ji}^{*}) \left(1 + \frac{B_{ki} (1 - M_{ji})}{B_{ki}^{*} (1 - M_{ji}^{*})} - \frac{B_{ki} (1 - M_{ji}) M_{ji}}{B_{ki}^{*} (1 - M_{ji}^{*}) M_{ji}} - \frac{M_{ji}}{M_{ji}^{*}}\right), \end{split}$$

Let  $b_{jk} = b\beta_2 p_{jk} a_{kj} M_{ki}^* B_{js}^*$ ,  $G_j(M_{ji}, B_{ji}) = \frac{\beta_1 p_{kj} B_{ki}^* (1 - M_{ji}^*)}{\beta_2 p_{jk} a_{kj} M_{ki}^* B_{js}^*} \left( -\frac{M_{ji}}{M_{ji}^*} + \ln \frac{M_{ji}}{M_{ji}^*} \right) - \frac{B_{ji}}{B_{ji}^*} + \ln \frac{B_{ji}}{B_{ji}^*}$ , and  $F_{jk}(B_{js}, M_{ji}, B_{ji}, M_{ki}, B_{ki}) = 2 + \frac{M_{ki}}{M_{ki}^*} - \frac{B_{js}}{B_{js}} - \frac{M_{ki} B_{js} B_{ji}^*}{M_{ki}^* B_{js}^* B_{ji}} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{ji}^*} + \frac{B_{ji}}{B_{ji}^*} - \frac{B_{ji}}{B_{$ 

 $\frac{\beta_1 p_{kj} B_{ki}^* (1-M_{ji}^*)}{\beta_2 p_{jk} a_{kj} M_{ki}^* B_{js}^*} \left(1 + \frac{B_{ki} (1-M_{ji})}{B_{ki}^* (1-M_{ji}^*)} - \frac{B_{ki} (1-M_{ji}) M_{ji}^*}{B_{ki}^* (1-M_{ji}^*) M_{ji}} - \frac{M_{ji}}{M_{ji}^*}\right). \text{ Then, by } \frac{B_{js}^*}{B_{js}} + \frac{B_{js}}{B_{js}^*} - 2 \ge 0, \text{ we have}$  $\dot{U}_j \le \sum_{k=1}^n b_{jk} F_{jk}(B_{js}, M_{ji}, B_{ji}, M_{ki}, B_{ki}),$ 

Let  $\phi(a) = 1 - a + \ln a$ . Then  $\phi(a) \le 0$  for a > 0 and equality holds only at a = 1. Furthermore, we have

$$\begin{split} F_{jk} = & G_j(M_{ji}, B_{ji}) - G_k(M_{ki}, B_{ki}) + \phi\left(\frac{B_{js}^*}{B_{js}}\right) + \phi\left(\frac{M_{ki}B_{js}B_{ji}^*}{M_{ki}^*B_{js}^*B_{ji}}\right) + \phi\left(\frac{B_{ki}M_{ki}^*}{B_{ki}^*M_{ki}}\right) \\ & + \frac{\beta_1 p_{kj}B_{ki}^*(1 - M_{ji}^*)}{\beta_2 p_{jk}a_{kj}M_{ki}^*B_{js}^*} \left(\phi(\frac{B_{ki}(1 - M_{ji})M_{ji}^*}{B_{ki}^*(1 - M_{ji}^*)M_{ji}}) + \phi\left(\frac{M_{ki}(1 - M_{ji})B_{ki}^*}{M_{ki}^*(1 - M_{ji})B_{ki}}\right)\right) \\ & + \left(\frac{M_{ki}}{M_{ki}^*} - 1\right) \left(1 - \frac{M_{ki}^*B_{ki}}{M_{ki}B_{ki}^*}\right) + \frac{\beta_1 p_{kj}B_{ki}^*(1 - M_{ji}^*)}{\beta_2 p_{jk}a_{kj}M_{ki}^*B_{js}^*} \left(\frac{B_{ki}(1 - M_{ji})}{B_{ki}^*(1 - M_{ji}^*)} - 1\right) \\ & \left(1 - \frac{M_{ki}B_{ki}^*(1 - M_{ji}^*)}{M_{ki}^*B_{ki}(1 - M_{ji})}\right) \\ & \leq G_j(M_{ji}, B_{ji}) - G_k(M_{ki}, B_{ki}) + \left(\frac{M_{ki}}{M_{ki}^*} - 1\right) \left(1 - \frac{M_{ki}^*B_{ki}}{M_{ki}B_{ki}^*}\right) + \frac{\beta_1 p_{kj}B_{ki}^*(1 - M_{ji}^*)}{\beta_2 p_{jk}a_{kj}M_{ki}^*B_{js}^*} \\ & \left(\frac{B_{ki}(1 - M_{ji})}{B_{ki}^*(1 - M_{ji}^*)} - 1\right) \left(1 - \frac{M_{ki}B_{ki}^*(1 - M_{ji}^*)}{M_{ki}^*B_{ki}(1 - M_{ji})}\right). \end{split}$$

Under conditions (4.6) and (4.7), we can show that  $U_j$ ,  $F_{jk}$ ,  $G_j$ ,  $b_{jk}$  satisfy the assumptions of Lemma 4.2. Therefore, the function  $U = \sum_{j=1}^{n} r_j U_j(M_{ji}, B_{js}, B_{ji})$ as defined in Lemma 4.2 is a Lyapunov function for (2.2), namely,  $U \leq 0$  for all  $(M_{1i}, B_{1s}, B_{1i}, \cdots, M_{ni}, B_{ns}, B_{ni}) \in \Omega - \{0\}$ . It can be verified that the only compact invariant set where  $\dot{U} = 0$  is the singleton  $\{E^*\}$ . By the LaSalle Invariance Principle [11],  $E^*$  is globally asymptotically stable in  $\Omega - \{0\}$ . 

**Remark 4.1.** For a unique endemic equilibrium  $E^*$  of the system (2.2), it is globally asymptotically stable when (4.6) and (4.7) are satisfied. We further analyze the relationship between them.

Let  $\frac{M_{ki}}{M_{ki}^*} = \theta_k$ ,  $\frac{B_{ki}}{B_{ki}^*} = \rho_k$ . Then the deformation of formula (4.6) is  $M_{ki}^* B_{ki}^*(\theta_k - 1)(\theta_k - \rho_k) \leq 0$ . Further, we have

Case 1:  $\theta_k \ge 1$ ,  $\theta_k \le \rho_k$ , i.e.,  $1 \le \theta_k \le \rho_k$ , or Case 2:  $\theta_k \leq 1, \, \theta_k \geq \rho_k, \, \text{i.e.}, \, \rho_k \leq \theta_k \leq 1.$ 

A similar method can be used to deform (4.7).  $M_{ki}^* B_{ki}^{*2} (1 - M_{ji}^*)^2 (\rho_k \frac{1 - M_{ji}}{1 - M_{ji}^*})^2$  $1)(\rho_k \frac{1-M_{ji}}{1-M_{ii}^*}-\theta_k) \leq 0$  is obtained. So, we have

Case 3:  $\rho_k \leq \frac{1-M_{ji}^*}{1-M_{ji}^*}, \ \rho_k \geq \theta_k \frac{1-M_{ji}^*}{1-M_{ji}^*}, \ \text{i.e.}, \ \theta_k \frac{1-M_{ji}^*}{1-M_{ji}} \leq \rho_k \leq \frac{1-M_{ji}^*}{1-M_{ji}}, \ \text{or}$ Case 4:  $\rho_k \geq \frac{1-M_{ji}^*}{1-M_{ji}}, \ \rho_k \leq \theta_k \frac{1-M_{ji}^*}{1-M_{ji}}, \ \text{i.e.,} \ \frac{1-M_{ji}^*}{1-M_{ji}} \leq \rho_k \leq \theta_k \frac{1-M_{ji}^*}{1-M_{ji}}.$ If Case 1 and Case 3 are satisfied, this is null set. If Case 2 and Case 3 are satisfied, we obtain  $\theta_k \leq 1$ ,  $\frac{\theta_k(1-M_{ji}^*)}{1-\theta_j M_{ji}^*} \leq \rho_k \leq \frac{1-M_{ji}^*}{1-\theta_j M_{ji}^*}$ If Case 1 and Case 4 are satisfied, we obtain  $\theta_k \geq 1$ ,  $\frac{1-M_{ji}^*}{1-\theta_j M_{ji}^*} \leq \rho_k \leq \frac{\theta_k(1-M_{ji}^*)}{1-\theta_j M_{ji}^*}$ 

If Case 2 and Case 4 are satisfied, this is null set.

Therefore, we obtain the specific range (i.e.,  $\theta_k \leq 1$ ,  $\frac{\theta_k(1-M_{j_i}^*)}{1-\theta_j M_{j_i}^*} \leq \rho_k \leq \frac{1-M_{j_i}^*}{1-\theta_j M_{j_i}^*}$ , and  $\theta_k \geq 1$ ,  $\frac{1-M_{j_i}^*}{1-\theta_j M_{j_i}^*} \leq \rho_k \leq \frac{\theta_k(1-M_{j_i}^*)}{1-\theta_j M_{j_i}^*}$ ) of the globally asymptotically stability of the endemic equilibrium.

## 5. Application to two patches

In this section, we will perform a series of numerical simulations to verify the mathematical analysis. Specially, we want to present some numerical simulation for two patches to illustrate how  $R_0$  changes with  $p_{ij}$  which is the fraction of time a bird resident in patch *i* spends visiting patch *j*. For simulation purpose, we consider some parameters are chosen in Table 1. The other parameters are changed.

Following, we first give the basic reproduction number of the special case of the system (2.1) with only two patches. Thus, we have the system

$$\frac{dM_{1i}}{dt} = b\beta_1(p_{11}B_{1i} + p_{21}B_{2i})(1 - M_{1i}) - (1 - q)d_m M_{1i}, 
\frac{dB_{1s}}{dt} = d_b - d_b B_{1s} - b\beta_2(p_{11}a_{11}M_{1i} + p_{12}a_{21}M_{2i})B_{1s}, 
\frac{dB_{1i}}{dt} = b\beta_2(p_{11}a_{11}M_{1i} + p_{12}a_{21}M_{2i})B_{1s} - (d_b + \gamma_b)B_{1i}, 
\frac{dM_{2i}}{dt} = b\beta_1(p_{12}B_{1i} + p_{22}B_{2i})(1 - M_{2i}) - (1 - q)d_m M_{2i}, 
\frac{dB_{2s}}{dt} = d_b - d_b B_{2s} - b\beta_2(p_{21}a_{12}M_{1i} + p_{22}a_{22}M_{2i})B_{2s}, 
\frac{dB_{2i}}{dt} = b\beta_2(p_{21}a_{12}M_{1i} + p_{22}a_{22}M_{2i})B_{2s} - (d_b + \gamma_b)B_{2i}.$$
(5.1)

Therefore, we have

$$\mathcal{A} = b\beta_1 \begin{pmatrix} p_{11} & p_{21} \\ p_{12} & p_{22} \end{pmatrix}, \mathcal{B} = b\beta_2 \begin{pmatrix} p_{11}a_{11} & p_{12}a_{21} \\ p_{21}a_{12} & p_{22}a_{22} \end{pmatrix},$$
$$\mathcal{C} = \begin{pmatrix} (1-p)d_m & 0 \\ 0 & (1-p)d_m \end{pmatrix}, \mathcal{D} = \begin{pmatrix} d_b + \gamma_b & 0 \\ 0 & d_b + \gamma_b \end{pmatrix}.$$

Hence,  $R_0^2 = \rho(\mathcal{BC}^{-1}\mathcal{AD}^{-1})$ , where

$$\mathcal{BC}^{-1}\mathcal{AD}^{-1} = \frac{b^2\beta_1\beta_2}{(d_b+\gamma_b)(1-p)d_m} \begin{pmatrix} p_{11}^2a_{11}+p_{12}^2a_{21} & p_{11}p_{21}a_{11}+p_{12}p_{22}a_{21} \\ p_{11}p_{21}a_{12}+p_{12}p_{22}a_{22} & p_{21}^2a_{12}+p_{22}^2a_{22} \end{pmatrix}$$
$$R_0^2 = \frac{b^2\beta_1\beta_2}{2(d_b+\gamma_b)(1-p)d_m} \left(p_{11}^2a_{11}+p_{12}^2a_{21}+p_{21}^2a_{12}+p_{22}^2a_{22}+\sqrt{(p_{11}^2a_{11}+p_{12}^2a_{21}-p_{21}^2a_{12}-p_{22}^2a_{22})^2+4(p_{11}p_{21}a_{11}+p_{12}p_{22}a_{21})(p_{11}p_{21}a_{12}+p_{12}p_{22}a_{22})}\right)$$

Let  $R_{0i}^2 = \frac{b^2 \beta_1 \beta_2 a_{ii}}{(d_b + \gamma_b)(1-p)d_m}$  be the basic reproduction number for patch i(i = 1, 2) in isolation (i.e., birds don't move back and forth between patch 1 and patch 2). If it

exists the fraction of time a bird in patch *i* spends visiting patch *j*, then the modified reproduction numbers are  $\widetilde{R}_{01}^2 = \frac{b^2 \beta_1 \beta_2 p_{11}^2 a_{11}}{(d_b + \gamma_b)(1-p)d_m}$ , and  $\widetilde{R}_{02}^2 = \frac{b^2 \beta_1 \beta_2 p_{22}^2 a_{22}}{(d_b + \gamma_b)(1-p)d_m}$ . Their relations are not very illuminating. In addition, we know the endemic equilibrium is existed. Since the endemic equilibrium satisfies the bivariate cubic equations, we can not point out the concrete expression of the endemic equilibrium. Based upon these results, we can present two examples that illustrate the effect of bird populations migration among regions on the spread of WNV.

In the first and second patches, fix  $d_m = 0.7, d_b = 0.003$ . This means that we assume that two patches have the same demographic structure and the same recovery rate for WNV. But the ratio of the total number of mosquitoes and birds is different in two patches.

**Example 5.1.** We fix  $N_{1M}/N_{1B} = 3.5$ ,  $N_{2M}/N_{2B} = 2.7$ ,  $N_{1M}/N_{2B} = 2.25$ . If the two patches are isolated, in the first patch,  $R_{01} = 0.9495 < 1$ , the disease will disappear. Further, in the second patch,  $R_{02} = 0.8340 < 1$ , the disease will disappear. Although the disease cannot spread in any patch, the disease will be persistent in the two patches when bird populations dispersal occurs(see Fig.2).



Figure 2.  $R_{0i} < 1$ , the disease will be persistent in the two patches when bird populations dispersal occurs.

**Example 5.2.** We fix  $N_{1M}/N_{1B} = 3.7$ ,  $N_{2M}/N_{2B} = 4.5$ ,  $N_{1M}/N_{2B} = 2.25$ . If the two patches are isolated, in the first patch,  $R_{01} = 0.9763 < 1$ , the disease will disappear. Further, in the second patch,  $R_{02} = 1.0767 > 1$ , the disease will be persistent. The disease will be persistent in the two patches when bird populations dispersal occurs(see Fig.3).



Figure 3.  $R_{01} < 1$ , and  $R_{02} > 1$ , the disease will be persistent in the two patches when bird populations dispersal occurs.

Numerical simulation results show that disease can be maintained in a zero or low endemic area from a high endemic area. And they can also show the fraction of time a bird in patch *i* spends visiting patch j(i, j = 1, 2) impact the spread of the infectious diseases. When the fraction of time a bird in patch *i* spends visiting patch j(i, j = 1, 2) is very small or large, the diseases are more likely to outbreak.

### 6. Discussions

In this paper, based on the living habits of birds, we assume that the birds can move back and forth between patches, but not the mosquitoes. The fractions of their time that birds spend in each patch affect the transmission rate. Hence, we have proposed and analyzed a multi-patch WNV model with birds movment. In the model, epidemiological parameters are the same for n patches. In fact, the analysis here can be extended when these parameters differ from patch to patch. We obtain for  $R_0$  a formula, which although intricate, can be used to explore the effects of the parameters on the basic reproduction number. The relation between  $R_0$  and the biological parameters is involved. The formula will permit to explore the efficiency of controlling WNV. We obtain that either the disease will disappear or that it will become established at a unique stable equilibrium, depending on the parameters and the basic reproduction number  $R_0$ . We also see that the disease becomes endemic in both patches when birds move back and forth between these two patches.

## Acknowledgments

We would like to thank the referee for his/her helpful comments.

#### References

- P. Auger, E. Kouokam, G. Sallet, M. Tchuente and B. Tsanou, *The Ross-Macdonald model in a patchy environment*, Math. Biosci., 2008, 216(2), 123–131.
- [2] C. H. Calisher, K. S. C. Maness, R. D. Lord and P. H. Coleman, Identification of two South American strains of eastern equine encephalomyelitis virus from migrant birds captured on the Mississippi Delta, Am. J. Epidemiol., 1971, 94(2), 172–178.
- [3] G. Cruz-Pacheco, L. Esteva, J. A. Montaño-Hirose and C. Vargas, Modelling the dynamics of West Nile virus, Bull. Math. Biol., 2005, 67(6), 1157–1172.
- [4] C. Cosner, J. C. Beier, R. S. Cantrell, D. Impoinvil, L. Kapitanski, M. D. Potts, A. Troyo and S. Ruan, *The effect of human movement on the persistence of vector-borne diseases*, J. Theor. Biol., 2009, 258(4), 550–560.
- [5] V. Capasso, Mathematical structures of epidemic systems, Springer, Berlin, 1993.
- [6] C. Dye and G. Hasibeder, *Population dynamics of mosquito-borne disease:* effects of flies which bite some people more frequently than others, Transactions of the Royal Society of Tropical Medicine and Hygiene, 1986, 80(1), 69–77.
- [7] D. Gao and S. Ruan, A multipatch malaria model with logistic growth populations, SIAM J. Appl. Math., 2012, 72(3), 819–841.

- [8] G. Hasibeder and C. Dye, Population dynamics of mosquito-borne disease: persistence in a completely heterogeneous environment, Theor. Popul. Biol., 1988, 33(1), 31–53.
- [9] N. Karabatsos, International catalogue of arboviruses including certain other viruses of vertebrates, American Society of Tropical Medicine and Hygiene, San Antonio, 1985.
- [10] M. Y. Li and Z. Shuai, Global-stability problem for coupled systems of differential equations on networks, J. Differ. Equations, 2010, 248(1), 1–20.
- [11] J. P. LaSalle, The Stability of Dynamical Systems (CBMS-NSF Regional Conference Series in Applied Mathematics) Paperback, Society for Industrial and Applied Mathematics, New York, 1987.
- [12] R. Liu, J. Shuai, J. Wu and H. Zhu, Modeling spatial spread of West Nile virus and impact of directional dispersal of birds, Math. Biosci. Eng., 2006, 3(1), 145–160.
- [13] R. D. Lord and C.H. Calisher, Further evidence of southward transport of arboviruses by migratory birds, Am. J. Epidemiol. 1970, 92(1), 73–78.
- [14] Z. Ma, Y. Zhou and J. Wu, Modeling and dynamics of infectious diseases, Higher Education Press, Beijing, 2009.
- [15] J. Owen, F. Moore, N. Panella, et al., Migrating birds as dispersal vehicles for West Nile virus, EcoHealth, 2006, 3(2),79–85.
- [16] J. H. Rappole, S. R. Derrickson and Z. Hubalek, Migratory birds and spread of West Nile virus in the Western Hemisphere, Emerg. Infect. Dis., 2000, 6(4), 319–328.
- [17] J. H. Rappole and Z. Hubalek, Migratory birds and West Nile virus, J. Appl. Microbiol., 2003, 94(s1), 47–58.
- [18] D. J. Rodriguez and L. Torres-Sorando, Models of infectious diseases in spatially heterogeneous environments, Bull. Math. Biol., 2001, 63(3), 547–571.
- [19] K. C. Smithburn, T. P. Hughes, A. W. Burke, and J. H. Paul, A neurotropic virus isolated from the blood of a native of Uganda. Am. J. Trop. Med. Hyg., 1940, 4, 471–492.
- [20] K. E. Steele, M. J. Linn, R. J. Schoepp, N. Komar, T. W. Geisbert, R. M. Manduca, et al., Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York, Vet Pathol., 2000, 37(3), 208–224.
- [21] D. L. Smith and F. E. McKenzie, Statics and dynamics of malaria infection in Anopheles mosquitoes, Malaria J., 2004, 3(1), 13–26.
- [22] L. Torres-Sorando and D. J. Rodriguez, Models of spatio-temporal dynamics in malaria, Ecol. Model., 1997, 104(2–3), 231–240.
- [23] Y. Takeuchi, K. Sato and Y. Iwasa, Mathematics for Life Science and Medicine, Springer, Berlin, 2007.
- [24] P. van den Driessche and J. Watmough, Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, Math. Biosci., 2002, 180(1), 29–48.

- [25] M. J. Wonham, T. de-Camino-Beck and M. Lewis, An epidemiological model for West Nile virus: invasion analysis and control applications, Proc. R. Soc. Lond. B, 2004, 271(1538), 501–507.
- [26] W. D. Wang and X. Q. Zhao, An epidemic model in a patchy environment, Math. Biosci., 2004, 190(1), 97–112.