CANADIAN APPLIED
MATHEMATICS QUARTERLY
Volume 20, Number 4, Winter 2012

WEST NILE DYNAMICS: VIRUS TRANSMISSION BETWEEN DOMESTIC AND WILD BIRD POPULATIONS THROUGH VECTORS

TUFAIL MALIK, PAUL SALCEANU, ANUJ MUBAYI,
ABDESSAMAD TRIDANE AND MUDASSAR IMRAN

ABSTRACT. A deterministic model is constructed and analyzed to study the dynamics of West Nile Virus (WNV) transmission between two bird populations—domestic and wild—through vectors (mosquitoes). Different effective contact rates are assumed between vectors and each of the two bird populations. The analysis of the model, which is based on a system of ordinary differential equations, has a globally asymptotically stable disease-free equilibrium whenever a certain epidemiological threshold, known as the reproduction number, is less than unity. The disease persists uniformly with respect to small changes in the model parameters when the reproduction number is larger than unity. Furthermore, in the later case, the model is shown to have a unique endemic equilibrium under a certain condition. Numerical simulations suggest that this endemic equilibrium is asymptotically stable. Our results suggest that intervention programs aimed at the reduction of mosquito density—rather than those associated with birds—have a higher impact in controlling WNV in the two bird populations.

1 Introduction The natural transmission cycle of West Nile Virus (WNV) primarily involves *Culex* species mosquitoes and birds, with humans being incidental (dead end) hosts [**22**]. There is a significant strain variation among WNVs [**22**]. Two major phylogenetic lineages of WNV are known; the first includes viruses from North Africa, Europe, Asia, the Americas, and Australia, the second from southern Africa and Mada-

AMS subject classification (2010): 92.
Keywords: West Nile virus, disease-free equilibrium, rekproduction number, global asymptotic stability, endemic equilibrium.

gascar [**1, 4, 22**]. A lineage 2 strain was identified to be responsible for the death of a goshawk fledgling in Hungary suggesting that some strains may also be spread by migratory birds [**1, 4**].

West Nile Virus has been detected in numerous non-migratory (or resident) birds, including crows and songbirds [**3**], House Sparrow and Common Magpie (Pica pica) [**18**] and other domestic birds [**13**]. Evidence of exposure of and transmission by migratory birds has been well documented in literature [**3, 33**]. The human WNV outbreaks are known to occur in wetlands in urban areas where humans, migratory birds and ornithophilic mosquitoes are concentrated [**33**]. Furthermore, a large number of wild and captive birds coincides with increased number of human WNV cases [**33**]. It is therefore important that the transmission dynamics of WNV among the migratory birds, resident birds and the vectors, is studied. In another study [**29**], birds were found to predict the highest proportion of human cases, followed by mosquito pools and equines.

To the authors' knowledge, co-infection of birds with two different WNV strains has not been reported. However there is documented evidence of co-infection of WNV with two other closely related yet distinct encephalitis viruses (also transmitted by *Culex* mosquitoes), the St. Louis Encephalitis and the Western equine encephalomyelitis virus [**34**].

Different mathematical models have studied various aspects of WNV, its spread in human populations and the effect of the ecosystem on possible scenarios of the dynamics of this spread. For example Wonham *et al.* [**44**] introduce a mathematical model of WNV dynamics in a mosquito-bird community. They investigate the factors leading to the disease outbreak and present a graphical and analytical method to determine a necessary public health measure, such as mosquito control, that could help to avoid an outbreak. Bowman *et al.* [**5**] investigate two possible preventive controls of WNV, namely mosquito reduction strategies and human protection, in a mathematical model that includes a mosquito-bird-human community. The model shows the possible eradication of the disease if the mosquito reduction control is used efficiently. Using the theory of monotone dynamical systems the authors show the possibility of persistence of the disease.

Chatterjee *et al.* [**9**] study an extended model of migratory birds and their predators. These birds could recover from an infection and gain permanent immunity, which is the case in the WNV spread by the migratory birds. In this paper, the authors show that the only way to control the disease is vaccination and an increase in the predation of the

birds.

The study by Jiang *et al.* [**15**] focuses on the global dynamics of a similar model, showing the possible global asymptotic stability of the endemic equilibrium if the reproduction number is below one and the death rate of birds is below a certain threshold. But the numerical simulations showed that even with big values of death rate, the endemic equilibrium is still globally asymptotically stable. As a result, Jiang *et al.* [**16**] show, using the criterion for the orbital stability of periodic orbits associated with higher-dimensional nonlinear autonomous systems, the global stability of the same endemic equilibrium in the interior of a feasible region. Moreover, using K-competitive and index theory of dynamical systems on a surface, Jiang *et al.* [**17**] show that the reproduction number cannot be the only factor that characterizes the level of transmission of the pandemics such as WNV. The authors suggest that the initial level of the infection is a big factor in the infection spread.

Maidana and Yang [**26**] study the effect of variation of the birds species and populations, involved in WNV transmission, on the diffusion of the disease. By considering a model of different avian species and a single type of vectors, the authors consider different values for the biting rates and show that the information of the relative abundance of these bird species would permit to give an accurate estimation of the overall transmission risk of the infection.

The work by Wan and Zhu [**43**] focuses on the backward bifurcation in a comparison study of four existing WNV compartmental models [**5, 12, 24, 25, 44**]. The authors show that backward bifurcation is due to the high mortality of the avian host and not the variation of the mosquito population regardless of the nature of growth rate of the birds and mosquitoes (logistic or constant).

Qiu [**32**] studies a two-patches model that can be applied to the spread of a vector-host disease such as WNV. The author gives conditions of uniform persistence and extinction of each patch via the reproduction numbers. This result is very important for investigating the impact of host dispersal on final size of a pandemic such as WNV.

In this work, we present a mathematical model of the WNV transmission between two types of birds population (domestic and wild) via mosquitoes. The aim is to investigate the effects of different contact rates of the mosquitoes and each bird population. The model results suggest mechanisms responsible for the frequent transmission and shed some light on the possible control strategies with regard to different birds' population.

The paper is organized as follows. In Section 2, we introduce the

model and describe relevant assumptions. The boundedness properties of the model is presented in Section 3. In Section 4, we study the local stability of the disease free equilibria whereas introduce the persistence result in Section 5. The sensitivity analysis of the parameter of our model is presented in Section 6 and we conclude our work in Section 7.

2 Model formulation The model monitors the temporal dynamics of susceptible (uninfected) female mosquitoes $S_v(t)$, infected female mosquitoes $I_v(t)$, and susceptible, infected and recovered wild (respectively, domestic) birds $S_w(t)$, $I_w(t)$ and $R_w(t)$ (respectively, $S_d(t)$, $I_d(t)$ and $R_d(t)$).

$$
\begin{aligned}
S_v' &= \Lambda_v - b\beta_{vw} S_v I_w - b\beta_{vd} S_v I_d - \mu_v S_v \\
I_v' &= b\beta_{vw} S_v I_w + b\beta_{vd} S_v I_d - (\mu_v + \delta_v) I_v \\
S_w' &= \Lambda_w - b\beta_{wv} S_w I_v - \mu_w S_w \\
I_w' &= b\beta_{wv} S_w I_v - (\mu_w + \gamma_w + \delta_w) I_w \\
R_w' &= \gamma_w I_w - \mu_w R_w \\
S_d' &= \Lambda_d - b\beta_{dv} S_d I_v - \mu_d S_d \\
I_d' &= b\beta_{dv} S_d I_v - (\mu_d + \gamma_d + \delta_d) I_d \\
R_d' &= \gamma_d I_d - \mu_d R_d.
\end{aligned} \tag{1}
$$

The population of susceptible mosquitoes is generated by birth (recruitment) at per capita rate Λ_v. It is reduced by infection, following contact, either with infectious wild birds at a rate of infection $b\beta_{vw}$, where b is the biting rate (bites given by a mosquito per unit time) and β_{vw} is the effective contact rate (contact with an infectious wild bird, sufficient for infection of the vector), or with infectious domestic birds (at a rate of infection $b\beta_{vd}$), and by natural death (at a rate μ_v). We point out that even though standard incidence is more popular in epidemiological modeling, we chose mass-action incidence for mathematical tractability. The reader is referred to [**23**] for a discussion of the use of standard incidence and mass action rates in WNV models. The population of infected mosquitoes is generated by infection of the susceptible mosquitoes following contact with wild or domestic birds (at rates $b\beta_{vw}$ and $b\beta_{vd}$ respectively) and is reduced by natural death (at a rate μ_v) and disease-induced mortality (at a rate δ_v). We assume that a mosquito never recovers from infection due to its short life [**12**].

The population of susceptible wild (respectively domestic) birds is generated by birth (at a rate $\Lambda_w(\Lambda_d)$), and is reduced by infection, after susceptible birds are bitten by infectious mosquitoes, at a rate of infection $b\beta_{wv}$ (respectively, $b\beta_{dv}$), where b is the biting rate (bites received by a wild (domestic) bird per unit time), and $\beta_{wv}(\beta_{dv})$ is the effective contact rate (contact with an infectious mosquito, sufficient for infection of the wild (domestic) bird), and by natural death (at a rate μ_w (respectively, μ_d)). The infectious wild (domestic) birds population is generated by infection of the susceptible birds after susceptible birds are bitten by infectious mosquitoes (at a rate $b\beta_{wv}$ (respectively, $b\beta_{wd}$)). It is diminished by natural death (at a rate μ_w (respectively, μ_d)), by recovery (at a rate γ_w (respectively, γ_d)), and by disease-induced mortality (at the rate δ_w (respectively, δ_d)).

Finally, the population of the recovered birds is generated by recovery of infectious birds of the respective population. It is decreased by natural death (at a rate μ_w and μ_d respectively). Recovered birds are assumed to have acquired immunity against the virus.

3 Basic properties The model (1) monitors bird and mosquito populations, therefore the associated parameters and state variables are nonnegative for $t \geq 0$. The positive octant in $\mathbb{R}^8$ is positively invariant, since the vector field on the boundary does not point to the exterior (Theorem B.1. in [**38**]).

For $N_v(t) = S_v(t) + I_v(t)$, $N_w(t) = S_w(t) + I_w(t) + R_w(t)$ and $N_d(t) = S_d(t) + I_d(t) + R_d(t)$, which give the total population of mosquitoes, wild birds and domestic birds at time t, respectively, consider the biologically feasible region

$$\mathcal{D} = \left\{ (S_v, I_v, S_w, I_w, R_w, S_d, I_d, R_d) \in \mathbb{R}^8_+ : \right.$$
$$\left. N_w \leq \frac{\Lambda_w}{\mu_w}, N_d \leq \frac{\Lambda_d}{\mu_d}, N_v \leq \frac{\Lambda_v}{\mu_v} \right\}.$$

Lemma 1. *The closed set $\mathcal{D}$ is positively invariant and attracting.*

Proof. Adding the first two equations of the model (1) gives the rate of change of the mosquito population:

$$N_v' = \Lambda_v - \mu_v N_v - \delta_v I_v.$$

Thus, $N_v' \leq \Lambda_v - \mu_v N_v$ from which it follows that

$$N_v(t) \leq N_v(0)e^{-\mu_v t} + \frac{\Lambda_v}{\mu_v}(1 - e^{-\mu_v t}). \tag{2}$$

From (2) it is clear that $\limsup_{t\to\infty} N_v(t) \leq \Lambda_v/\mu_v$ and also that $0 \leq N_v(0) \leq \Lambda_v/\mu_v$ implies $0 \leq N_v(0) \leq \Lambda_v/\mu_v$, for all $t \geq 0$.

Similar arguments can be used in regard to N_w and N_d, by adding the S, I, R equations of the individual bird populations. □

The right side of the model (1) is Lipschitz continuous. Therefore the usual existence, uniqueness and continuation properties hold in $\mathcal{D}$ (i.e. the model (1) is mathematically and biologically well-posed in $\mathcal{D}$).

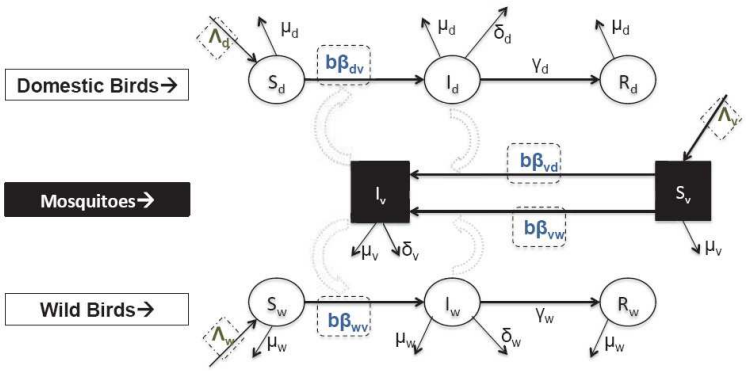

FIGURE 1: Flow chart of the model (1).

4 The disease free equilibrium (DFE) and basic reproduction number The model (1) has a DFE (obtained by setting the right side to zero), given by

$$E_0 : (S_v^*, I_v^*, S_w^*, I_w^*, R_w^*, S_d^*, I_d^*, R_d^*) = \left(\frac{\Lambda_v}{\mu_v}, 0, \frac{\Lambda_w}{\mu_w}, 0, 0, \frac{\Lambda_d}{\mu_d}, 0, 0\right) \tag{3}$$

We prove the local stability of E_0 using the basic reproductive number R_0, which is computed using the next generation operator method

[**11, 42**]. Lewis *et al.* [**23**] highlight the relevance of the method to both discrete and continuous time models and point out contrasting implications of the basic reproductive number R_0 (in relation with the control strategies of disease elimination) of [**41**] and [**44**] owing to the underlying model assumptions. The continuous time model of the former includes virus-induced death of birds, while the discrete time model of the later does not, but incorporates the vertical transmission of the virus in vectors.

The model (1) has three infected populations, I_v, I_w and I_d, therefore the matrix F (of the new infection terms) and the matrix V (of the transmission terms) are given by

$$F = \begin{pmatrix} 0 & b\beta_{vw}S_v^* & b\beta_{vd}S_v^* \\ b\beta_{wv}S_w^* & 0 & 0 \\ b\beta_{dv}S_d^* & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ 0 & k_2 & 0 \\ 0 & 0 & k_3 \end{pmatrix},$$

where $k_1 = \mu_v + \delta_v$, $k_2 = \mu_w + \gamma_w + \delta_w$, and $k_3 = \mu_d + \gamma_d + \delta_d$.

It follows that the *basic reproduction number* of the model (1), denoted by $\mathcal{R}_0$, is given by (where ρ denotes the spectral radius)

(4)
$$\begin{aligned} \mathcal{R}_0 &= \rho(FV^{-1}) \\ &= \sqrt{\frac{b\beta_{vw}\Lambda_v}{\mu_v(\mu_w+\gamma_w+\delta_w)}\frac{b\beta_{wv}\Lambda_w}{\mu_w(\mu_v+\delta_v)} + \frac{b\beta_{vd}\Lambda_v}{\mu_v(\mu_d+\gamma_d+\delta_d)}\frac{b\beta_{dv}\Lambda_d}{\mu_d(\mu_v+\delta_v)}}. \end{aligned}$$

Using Theorem 2 in [**42**], the following result is established.

Lemma 2. *The DFE of the model* (1)*, given by* E_0*, is locally asymptotically stable (LAS) if* $\mathcal{R}_0 < 1$*, and unstable if* $\mathcal{R}_0 > 1$*.*

The basic reproduction number ($\mathcal{R}_0$) measures the average number of new infections that one infectious individual (vector or bird) can produce if introduced into a population composed of susceptible vectors and birds. The first term under the radical sign, $(b\beta_{vw}\Lambda_v/(\mu_v(\mu_w + \gamma_w + \delta_w)))(b\beta_{wv}\Lambda_w/(\mu_w(\mu_v + \delta_v)))$, is related to the virus transmission

between the vector and the wild bird populations. The first factor, $b\beta_{vw}\Lambda_v/(\mu_v(\mu_w+\gamma_w+\delta_w))$, can be interpreted as follows: The effective contact rate for virus transmission from a wild bird to a vector is $b\beta_{vw}$. Total effective contacts made by an infectious wild bird, in a population of Λ_v/μ_v susceptible vectors, is $b\beta_{vw}(\Lambda_v/\mu_v)$. The lifespan of an infectious wild bird is $1/(\mu_w+\gamma_w+\delta_w)$. Thus $b\beta_{vw}\Lambda_v/(\mu_v(\mu_w+\gamma_w+\delta_w))$ determines the total infectious mosquitoes produced by the introduction of an infectious wild bird into a susceptible vector population, during its infectious lifespan. Likewise, the second factor, $b\beta_{wv}\Lambda_w/(\mu_w(\mu_v+\delta_v))$, determines the total infectious wild birds produced by the introduction of an infectious mosquito (with an effective biting rate $b\beta_{wv}$), into a susceptible wild birds population (of size Λ_w/μ_w), during its infectious lifespan $(1/(\mu_v+\delta_v))$.

Virus transmission can either occur between vectors and wild birds (first term under the radical sign), or between vectors and domestic birds (second term under the radical sign). Note that the reproduction number of the vector population and the wild bird population ($\mathcal{R}_w$), in the absence of the domestic bird population, $(S_d^*=\Lambda_d/\mu_d=0)$, is the geometric mean of the number of secondary infections in the vectors and that in wild birds populations:

$$\mathcal{R}_w=\sqrt{\frac{b\beta_{vw}\Lambda_v}{\mu_v(\mu_w+\gamma_w+\delta_w)}\frac{b\beta_{wv}\Lambda_w}{\mu_w(\mu_v+\delta_v)}}.$$

Similarly in the absence of the wild birds, $(S_w^*=\Lambda_w/\mu_w=0)$, the reproduction number of the vector population and the domestic birds population ($\mathcal{R}_d$), is given by the geometric mean of the number of secondary infections in the vectors and that in the domestic birds populations:

$$\mathcal{R}_d=\sqrt{\frac{b\beta_{vd}\Lambda_v}{\mu_v(\mu_d+\gamma_d+\delta_d)}\frac{b\beta_{dv}\Lambda_d}{\mu_d(\mu_v+\delta_v)}}.$$

Note that $\mathcal{R}_0<1\Rightarrow\mathcal{R}_w<1$ and $\mathcal{R}_d<1$. Thus if either of the two reproduction numbers, $\mathcal{R}_w$ or $\mathcal{R}_d$, is greater than unity, then $\mathcal{R}_0>1$.

Lemma 2 implies that the virus can be eliminated from the two bird populations as well as the mosquito population (when $\mathcal{R}_0<1$), if the initial sizes of the three subpopulations are in the basin of attraction of the DFE, E_0. In order to ensure that the elimination of the virus is independent of the initial population sizes, it is necessary to show that the DFE E_0 is globally asymptotically stable (GAS). To accomplish this we proceed as follows.

5 Global dynamics of the model In this section, we show that the basic reproduction number $\mathcal{R}_0$ determines when the disease gets extinct, or else, when it persists. More exactly, we show that if $\mathcal{R}_0 < 1$ then the disease free equilibrium is globally asymptotically stable (hence the disease gets extinct), while if $\mathcal{R}_0 > 1$ the disease persists in the model and this persistence is uniform with respect to small changes in the parameters, that is, the persistence is robust (see Theorem 1 part (b) below).

Define the region

$$\mathcal{D}_* = \{(S_v, I_v, S_w, I_w, R_w, S_d, I_d, R_d) \in \mathcal{D} : \\ S_v \leq S_v^*, S_w \leq S_w^*, S_d \leq S_d^*\}.$$

Lemma 3. *The region $\mathcal{D}_*$ is positively invariant and attracting.*

Proof. Recall that in Lemma 1, $\mathcal{D}$ was shown to be positively invariant and attracting. The equation for S_v' of (1) can be simplified to

$$\begin{aligned} S_v' &= \Lambda_v - b\beta_{vw} S_v I_w - b\beta_{vd} S_v I_d - \mu_v S_v \\ &\leq \Lambda_v - \mu_v S_v. \end{aligned}$$

Therefore, as in Lemma 1, it follows that $\limsup_{t\to\infty} S_v(t) \leq \Lambda_v/\mu_v = S_v^*$ and also that $0 \leq S_v(0) \leq S_v^*$ implies $S(t) \leq S_v^*$ for all $t \geq 0$.

Similar arguments can be used in regard to $S_w(t)$ and $S_d(t)$. Thus, the set $\mathcal{D}_*$ is attracting and positively invariant. □

Theorem 1. *The following hold:*

(a) *If $\mathcal{R}_0 < 1$, the DFE, E_0, of the model* (1)*, is GAS in $\mathcal{D}_*$.*
(b) *If $\mathcal{R}_0 > 1$, then the disease is robustly uniformly persistent: for every parameter vector ξ_0 in* (1)*, there exists a neighborhood Δ of ξ_0, $\exists\, \varepsilon > 0$ such that*

$$\liminf_{t\to\infty} \min\{I_v^\xi(t), I_w^\xi(t), I_d^\xi(t)\} > \varepsilon, \quad \xi \in \Delta \tag{5}$$

for all solutions $(S_v^\xi(t), I_v^\xi(t), S_w^\xi(t), I_w^\xi(t), R_w^\xi(t), S_d^\xi(t), I_d^\xi(t), R_d^\xi(t))$ of (1)*, corresponding to ξ, with $I_v^\xi(0) + I_w^\xi(0) + I_d^\xi(0) > 0$.*

Proof. (a) Assume that $\mathcal{R}_0 < 1$. From Lemma 3, the omega limit set of any initial condition in $\mathcal{D}$ is contained in $\mathcal{D}_*$. Since E_0 is asymptotically stable (Lemma 2), it suffices to show that every solution starting in $\mathcal{D}_*$ converges to E_0. So, let $x(t) = (S_v(t), I_v(t), S_w(t), I_w(t),$

$R_w(t), S_d(t), I_d(t), R_d(t))$ be a solution of (1) with $x_0 = x(0) = (S_v(0), I_v(0), S_w(0), I_w(0), R_w(0), S_d(0), I_d(0), R_d(0)) \in \mathcal{D}_*$. From (1) we have

$$\begin{pmatrix} I_v' \\ I_w' \\ I_d' \end{pmatrix} = \begin{pmatrix} -(\mu_v + \delta_v) & b\beta_{vw}S_v & b\beta_{vd}Sv \\ b\beta_{wv}S_w & -(\mu_w + \gamma_w + \delta_w) & 0 \\ b\beta_{dv}S_d & 0 & -(\mu_d + \gamma_d + \delta_d) \end{pmatrix} \begin{pmatrix} I_v \\ I_w \\ I_d \end{pmatrix} \tag{6}$$

Since $\mathcal{D}^*$ is positively invariant, $S_v^*(t) \leq S_v^*$, $S_w^*(t) \leq S_w^*$ and $S_d^*(t) \leq S_d^*$, for all $t \geq 0$. Hence,

$$\begin{pmatrix} I_v' \\ I_w' \\ I_d' \end{pmatrix} \leq \begin{pmatrix} -(\mu_v + \delta_v) & b\beta_{vw}S_v^* & b\beta_{vd}Sv^* \\ b\beta_{wv}S_w^* & -(\mu_w + \gamma_w + \delta_w) & 0 \\ b\beta_{dv}S_d^* & 0 & -(\mu_d + \gamma_d + \delta_d) \end{pmatrix} \begin{pmatrix} I_v \\ I_w \\ I_d \end{pmatrix}, \tag{7}$$

where the vector inequality above is considered to be componentwise. The 3×3 matrix in (7) equals $F - V$, hence its spectral radius is less than zero, because $\mathcal{R}_0 < 1$ (see [**42**]). Also, $F - V$ has all off-diagonal entries non negative. Then $I_v(t), I_w(t), I_d(t) \to 0$ as $t \to \infty$ (see Corollary B.2. in [**38**]). This means that the omega limit set of x_0, $\omega(x_0)$, is contained in the disease-free space. But, on the other hand, it is straight forward to check that every solution with initial condition in the disease-free space converges to E_0. Hence $E_0 \in \omega(x_0)$. This implies that, in fact, $\omega(x_0) = E_0$ (because E_0 is asymptotically stable). Thus, $x(t) \to E_0$ as $t \to \infty$, which completes the proof.

(*b*) Let ξ_0 be a fixed parameter. First we show that

$$\liminf_{t\to\infty} \{I_v^\xi(t) + I_w^\xi(t) + I_d^\xi(t)\} > \varepsilon, \tag{8}$$

for every solution with $I_v^\xi(0) + I_w^\xi(0) + I_d^\xi(0) > 0$, $\xi \in \Delta$, for some neighborhood Δ of ξ_0.

Let $X = \{x \in \mathbb{R}_+^8 \mid I_v = I_w = I_d = 0\}$. Let $M = \mathcal{D}_* \cap X$. Then note that both X and M are positively invariant, $E_0 \in M$ and E_0 attracts all trajectories in X. Considering E_0 as a periodic orbit (of period $T = 1$, for example), we will apply Corollary 4.7 in [**35**]. Then (8) follows from Proposition 4.1 and Theorem 3.2 in [**35**]. Thus, denote the 3×3 matrix in (6) by $A(x)$ and notice that $A(E_0) = F - V$. Hence the spectral radius of this matrix is greater than zero, because $\mathcal{R}_0 > 1$ (see [**42**]). So condition 2) of Corollary 4.7 in [**35**] is satisfied (where $\Omega(M) = \{E_0\}$). Also, $A(E_0)$ is irreducible which, using Theorem 1.1 in [**37**], implies that $e^{tA(E_0)}$ has all entries positive, for all $t > 0$. This implies that condition 1) of the above mentioned corollary is satisfied.

Now we show that (8) implies (5) arguing by contradiction. Thus, suppose (5) does not hold. Then for every $\varepsilon > 0$ and every Δ neighborhood of ξ_0 there exists a solution of (1) corresponding to some $\xi \in \Delta$, satisfying

$$\liminf_{t\to\infty} \min\{I_v^\xi(t), I_w^\xi(t), I_d^\xi(t)\} \le \varepsilon. \tag{9}$$

Let us assume that $\liminf_{t\to\infty} I_v^\xi(t) \le \varepsilon$ (the other two cases can be treated analogously). From the equation for S_v in (1) and using Lemma 1 it follows that there exist positive numbers C and t_0 such that $S_v^\xi(t) \ge \Lambda_v - CS_v$ for all $t \ge t_0$ and C depends continuously on ξ. Hence there exist a neighborhood Ξ_0 of ξ_0 and $\alpha > 0$ such that

$$\liminf_{t\to\infty} S_v^\xi(t) \ge \alpha, \ \forall\, \xi \in \Xi_0. \tag{10}$$

Let $\Delta_1 \subseteq \Xi_0$ for which (8) holds. The Fluctuation Method (Proposition A.14 in [**39**]) implies that, for every parameter ξ, there exists a sequence $t_n \to \infty$ such that $I_v^\xi(t_n) \to \liminf_{t\to\infty} I_v^\xi(t)$ and $(I_v^\xi)'(t_n) \to 0$ as $n \to \infty$. On the other hand, there exists $\Delta_2 \subseteq \Delta_1$ a neighborhood of ξ_0 and $c > 0$ such that

$$\begin{aligned}(I_v^\xi)'(t_n) \ge cS_v^\xi(t_n)(I_v^\xi(t_n) + I_w^\xi(t_n) + I_d^\xi(t_n)) \\ - (\mu_v + \delta_v + 1)I_v^\xi(t_n), \quad \forall\, \xi \in \Delta_2.\end{aligned} \tag{11}$$

Let $\Delta_3 \subseteq \Delta_2$ and $\tilde{\xi} \in \Delta_3$ such that

$$\liminf_{t\to\infty} I_v^{\tilde{\xi}}(t) \le \frac{c\varepsilon\alpha}{2(\mu_v + \delta_v + 1)}. \tag{12}$$

Then (8), (10), (11) and (12) lead to a contradiction and this competes our proof.

□

Figure 2 (left column) depicts solution profiles of the model (1), for various initial conditions, showing convergence to the DFE (E_0) for $R_0 < 1$ (in line with Theorem 1).

5.1 Existence and uniqueness of endemic equilibrium point (EEP) Let $E_1 : (S_v^{**}, I_v^{**}, S_w^{**}, I_w^{**}, R_w^{**}, S_d^{**}, I_d^{**}, R_d^{**})$ represent an arbitrary endemic equilibrium of the model (1), where at least one of I_v^{**},

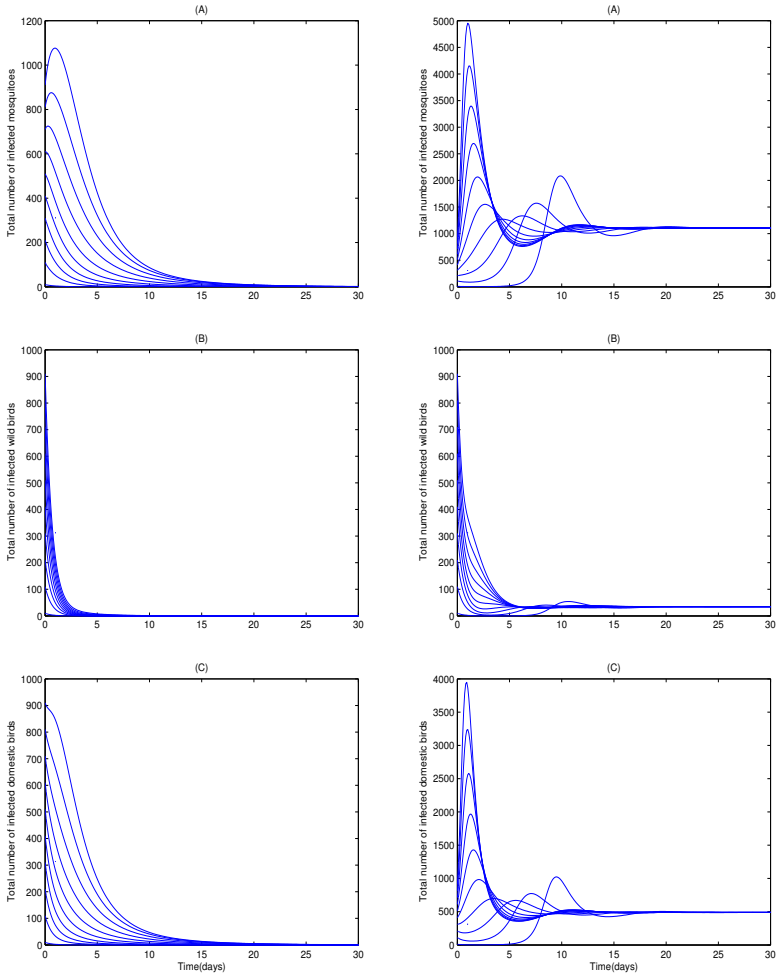

FIGURE 2: Numerical simulations of the model (1) showing the total number of infected individuals as a function of time, using various initial conditions. (A) Infected mosquitoes, (B) Infected wild birds, and (C) Infected domestic birds. Parameter values are: $\Lambda_w = 1000; \Lambda_d = 1000; \Lambda_v = 2000; \beta_{wv} = 0.01; \beta_{dv} = 0.5; \beta_{vw} = 0.05; \beta_{vd} = 0.5; \mu_v = 1/5; \mu_w = 1/5; \mu_d = 1/5; \gamma_w = 0.5; \gamma_d = 0.5; \delta_v = 0.8; \delta_w = 0.8; \delta_d = 0.8;$ Left column (convergence to the DFE): $b = 0.0003$ (so that $\mathcal{R}_0 = 0.87$). Right column (convergence to the EEP): $b = 0.001$ so that $\mathcal{R}_0 = 2.89$).

I_w^{**} and I_d^{**} is nonzero. In order to find the conditions for the existence of an endemic equilibrium for which the disease is endemic in the vector-avian population, the equations in the model (1) are solved at steady-state, giving

$$
\begin{aligned}
S_v^{**} &= \frac{\Lambda_v}{b\beta_{vw}I_w^{**} + b\beta_{vd}I_d^{**} + \mu_v},\\
S_w^{**} &= \frac{\Lambda_w}{b\beta_{wv}I_v^{**} + \mu_w},\ I_w^{**} = \frac{b\beta_{wv}\Lambda_w I_v^{**}}{k_2(b\beta_{wv}I_v^{**} + \mu_w)},\quad R_w^{**} = \frac{\gamma_w I_w^{**}}{\mu_w},\\
S_d^{**} &= \frac{\Lambda_d}{b\beta_{dv}I_v^{**} + \mu_d},\ \ I_d^{**} = \frac{b\beta_{dv}\Lambda_d I_v^{**}}{k_3(b\beta_{dv}I_v^{**} + \mu_d)},\quad R_d^{**} = \frac{\gamma_d I_d^{**}}{\mu_d}.
\end{aligned}
\tag{13}
$$

Numerical simulation results, depicted in Figure 2 (right column), show convergence of solutions to this EEP when $R_0 > 1$ suggesting that the EEP is unique and asymptotically-stable when it exists. We investigate the existence and uniqueness properties of the EEP (E_1) under a certain condition, as follows.

Substituting the expression for S_v^{**} in the second equation of the model (1) at steady state, simplifies to

$$
b\beta_{vw}I_w^{**} + b\beta_{vd}I_d^{**} = \frac{(\mu_v + \delta_v)\mu_v I_v^{**}}{(\mu_v + \delta_v)I_v^{**} - \Lambda_v}.
$$

Using the values of I_w^{**} and I_d^{**} now gives

$$
a_0(I_v^{**})^2 + b_0(I_v^{**}) + c_0 = 0, \tag{14}
$$

where

$$
\begin{aligned}
a_0 &= k_1k_3b^3\beta_{vw}\beta_{wv}\beta_{dv}\Lambda_w + k_1k_2b^3\beta_{vd}\beta_{wv}\beta_{dv}\Lambda_d - k_1k_2k_3b^2\beta_{wv}\beta_{dv}\mu_v,\\
b_0 &= k_1k_3b^2\beta_{vw}\beta_{wv}\mu_d\Lambda_w - k_3b^3\beta_{vw}\beta_{wv}\beta_{dv}\Lambda_v\Lambda_w + k_1k_2b^2\beta_{vd}\beta_{dv}\mu_w\Lambda_d\\
&\quad - k_2b^3\beta_{vd}\beta_{wv}\beta_{dv}\Lambda_v\Lambda_d - k_1k_2k_3b\beta_{wv}\mu_v\mu_d - k_1k_2k_3b\beta_{dv}\mu_v\mu_w,\\
c_0 &= -k_3b^2\beta_{vw}\beta_{wv}\mu_d\Lambda_v\Lambda_w - k_2b^2\beta_{vd}\beta_{dv}\mu_w\Lambda_v\Lambda_d - k_1k_2k_3\mu_v\mu_w\mu_d.
\end{aligned}
$$

Note that $c_0 < 0$. Clearly, $I_v^{**} = 0 \Rightarrow c_0 = 0$ (from (14)), and is therefore impossible. In other words, the endemic equilibrium E_1 will never have $I_v^{**} = 0$. Furthermore, $I_v^{**} = 0 \Leftrightarrow I_w^{**} = 0 \Leftrightarrow I_d^{**} = 0$, also yielding $R_w^{**} = R_d^{**} = 0$. Thus the EEP has infectious individuals

present from each subpopulation ($I_v^{**} > 0,\ I_w^{**} > 0,\ I_d^{**} > 0$). Otherwise the equilibrium corresponds to the DFE.

Suppose that

$$\mu_v + \delta_v = \Lambda_v, \quad b\beta_{wv} = \mu_w, \quad b\beta_{dv} = \mu_d. \tag{15}$$

Then

$$a_0 = k_1k_2k_3\mu_v\mu_w\mu_d(\mathcal{R}_0^2 - 1) \quad \text{and} \quad b_0 = -2k_1k_2k_3b\beta_{wv}\mu_v\mu_d < 0.$$

Thus, (14) has a unique zero whenever $a_0 > 0$. Then (13) yields unique corresponding values for the rest of the components of the EEP E_1.

We have established the following:

Theorem 2. *Suppose* (15) *holds. The model* (1) *has:*

1. *a unique endemic equilibrium whenever* $\mathcal{R}_0 > 1$ *(*$\Leftrightarrow a_0 > 0$*),*
2. *no endemic equilibrium otherwise.*

It is worth emphasizing that Theorem 2 asserts the existence of a unique EEP only if $R_0 > 1$. On the other hand, no endemic equilibrium exists if $R_0 < 1$ (because (14) has no solution). It was shown in Theorem 1 that the DFE is GAS whenever $R_0 < 1$. This is in contrast to some other works (see, for example, [**3, 15, 43**]) which show the existence of a backward bifurcation in the case $R_0 < 1$.

6 Sensitivity analysis Since the model's asymptotic analysis is completely based on the reproduction number, we perform parameters-related global uncertainty and sensitivity analyses on the $\mathcal{R}_0$. Parameters estimates are not always known with precision because of many reasons including natural variation, error in measurements, or a lack of measuring techniques. Uncertainty analysis is the study of how the uncertainty in the input model parameters affects the uncertainty in the output measure ($\mathcal{R}_0$ in our case). This analysis quantifies the degree of confidence in the existing data and parameter estimates. On the other hand, the sensitivity analysis identifies critical model parameters and quantifies the impact of each input parameter on the value of an output, in presence of other input parameters [**36**].

Ideally, uncertainty and sensitivity analysis should be run in tandem. Here we use latin-hypercube sampling based method to quantify uncertainty and sensitivity of $\mathcal{R}_0$ as a function of 13 model parameters (b, μ_v,

μ_d, μ_w, β_{vd}, β_{vw}, β_{dv}, β_{wv}, δ_v, δ_d, δ_w, γ_d, and γ_w). The sensitivity indexes which measure the impact of the parameters on the output variable in the sensitivity analysis were computed via Partial Rank Correlation Coefficient (PRCC) [**28**]. The PRCC measures the linear relationship between the two variables. However, it is possible that the variables may be nonlinear and monotonic. Hence, PRCC method uses the rank transformation of the data (that is, replacing the values with their ranks) to reduce the effects of nonlinearity. The rank correlation coefficient (RCC) indicates the degree of monotonicity between the input and output variables. The resultant data are considered partially in some sense, that is, "Partial" Rank Correlation Coefficients ('P'RCC) are computed that takes account for correlations among other input variables. The expression of *basic reproduction number* of the model (Figure 1), which is the output measure in the uncertainty and sensitivity analyses, is given in Equation 4.

6.1 Estimating model parameters Various studies in the literature were reviewed to estimate distributions of the parameters. We use data from [**12**] and [**20**] as well as other information from literature to stratify the data, consisting of various species of birds, into two categories (domestic and wild) via average migration patterns of the species (Table 2). These all studies included data from US. The details of parameters estimates are described below:

- **b** :The average **rate of biting** of birds by mosquitoes are found to be 0.1 per day [**5, 44**] or 0.4 per day (that is, once every 2 or 3 days; [**12**]). We took $b = 0.4$ with a range of (0.1, 0.5), which includes the estimates from the literature.
- $\mu_{\mathbf{v}}$: The averate **lifespan of a mosquito** is around 14 days [**5**] with range (10, 21) days (with average natural mortality rate of vectors of 0.07 per day [**14**]). We estimated the per capita mortality rate of the mosquito as $\mu_v = 0.07$.
- $\mathbf{\Lambda_v}$: Teboh-Ewungkem *et al.* 2009 [**40**] provided estimates of recruitment rate among vectors as $\Lambda_v = 10^4$ per day whereas the maximum number of recruitment in mosquitoes was assumed to be in the range 4.0×10^3 and 3.6×10^5 per day (with a mean of 2.2×10^4 per day) by Bowman *et al.* [**5**]. We estimated the **mosquitoes' recruitment rate** in their susceptible class as 10^4 per day.
- $\mu_{\mathbf{d}}$, $\mu_{\mathbf{w}}$: Cruz-Pacheco *et al.* [**12**] estimated the **lifespan of birds** in the range of 3 to 10 years with a mean of 6 years for all species (that is, mean natural mortality rate of $\mu_d = \mu_w = 0.0004$ per day). We estimated mean natural mortality rate of domestic and wild birds

Param.	**Definition**	**Mean (std.)**	**Dist. (Ref.)**
Mosquitoes related parameters			
Λ_v	Per day recruitment rate in vectors	10^4	Fixed ([**40**])
β_{vd}	Effective contact rate between susceptible mosquitos and infectious domestic birds	1.95E-7 (0.6E-7)	$\mathcal{G}$ ([**20**])
β_{vw}	Effective contact rate between susceptible mosquitos and infectious wild birds	2.2E-7 (0.9E-7)	$\mathcal{G}$ ([**20**])
μ_v	Per-capita natural mortality rate in vectors	0.07 (0.015)	$\mathcal{N}$ ([**14**])
δ_v	Per-capita disease-induced death rate in vectors	1.0E-6 (0.2E-6)	$\mathcal{U}$ (Estm.)
b	Average number of bites by a mosquito per day	0.4 (0.15)	$\mathcal{U}$ ([**44**])
Domestic birds related parameters			
Λ_d	Per day recruitment rate in domestic birds	10^3	Fixed ([**5**])
β_{dv}	Effective contact rate between infectious mosquito and susceptible domestic birds	1 (0.06)	$\mathcal{G}$ ([**12**])
μ_d	Per-capita natural mortality rate in domestic birds	5.0E-4 (3.0E-4)	$\mathcal{N}$ ([**19**])
γ_d	Per-capita recovery rate of domestic birds (per day)	0.30 (0.07)	$\mathcal{N}$ ([**20**])
δ_d	Per-capita disease-induced death rate in domestic birds	0.24 (0.21)	$\mathcal{E}$ ([**5**])
Wild birds related parameters			
Λ_w	Per day recruitment rate in wild birds	10^3	Fixed ([**5**])
β_{wv}	Effective contact rate between infectious mosquito and susceptible wild birds	1 (0.04)	$\mathcal{G}$ ([**12**])
μ_w	Per-capita natural mortality rate in wild birds	4.0E-4 (2.0E-4)	$\mathcal{N}$ ([**2**])
γ_w	Per-capita recovery rate of wild birds (per day)	0.27 (0.06)	$\mathcal{N}$ ([**20**])
δ_w	Per-capita disease-induced death rate in wild birds	0.11 (0.04)	$\mathcal{E}$ ([**5**])

TABLE 1: Description and estimates of the parameters of the model (1). $\mathcal{E}$, $\mathcal{G}$, $\mathcal{N}$, & $\mathcal{U}$ represent Exponential, Gamma, Normal, & Uniform, respectively.

Species Name		(per day)	(per day)	(per day)
Domestic birds	$\beta_{vd}N_d$	γ_d	δ_d	μ_d
House Finch	0.32	1/5.5	0.14	1/(9*365)
American Crow	0.50	1/3.3	0.19	1/(7*365)
House Sparrow	0.53	1/3.0	0.10	1/(3*365)
Black-billed Magpie	0.36	1/3.0	0.16	1/(5*365)
Fish Crow	0.26	1/2.8	0.60	1/(10*365)
Mean (std)	0.39	0.30	0.24	0.0005
	(0.12)	(0.07)	(0.21)	(0.0003)
Wild birds	$\beta_{vw}N_w$	γ_w	δ_w	μ_w
Blue Jay	0.68	1/3.8	0.15	1/(7*365)
Common Grackle	0.68	1/3.0	0.07	1/(10*365)
Ring-billed Gull	0.28	1/4.5	0.10	1/(4*365)
Mean (std)	0.55	0.27	0.11	0.0004
	(0.23)	(0.06)	(0.04)	(0.0002)

TABLE 2: Parameters related to domestic and wild birds affected by WNV. Data was obtained from [**2, 12, 19, 20**] and literature review.

as 0.0005 and 0.0004 per day, respectively (Table 2).

- $\mathbf{\Lambda_d}$, $\mathbf{\Lambda_w}$: The **recruitment rate in birds** was taken as 1000 per day [**5**].
- $\delta_\mathbf{d}$, $\delta_\mathbf{w}$: The virus has been found in more than 326 bird species [**8**], though only some species get critically infected and may die. The range of birds' daily per capita mortality rate from West Nile virus is (0.125, 0.300) with a mean of 0.143 [**44**]. Our estimated mean duration of infectious viremia in domestic and wild birds are 3.51 and 3.75 days, respectively (Table 2), which falls in this range.
- $\gamma_\mathbf{d}$, $\gamma_\mathbf{w}$: Bowman *et al.* [**5**] mention that after about four days, the bird hosts develop life-long immunity to further West Nile infection (although a small number will succumb to the disease and die). That is, **recovery rate** of 0.25 per day in birds with wide range. We estimated recovery rate of 0.30 and 0.27 per day for domestic and wild birds, respectively (Table 2).
- $\mathbf{N_v^*}$, $\mathbf{N_d^*}$, $\mathbf{N_w^*}$: The equilibrium population sizes of vectors, domestic birds and wild birds, in the absence of WNV-related deaths, are computed using ratio of recruitment rate and natural mortality rate and hence, are estimated as 1.43×10^5, 2.0×10^6 and 2.5×10^6, respectively.
- $\beta_\mathbf{vd}$, $\beta_\mathbf{vw}$: Wonham *et al.* [**44**] and Bowman *et al.* [**5**] provide the

mean estimates of the **transmission probabilities from bird to mosquito** as 0.16 (with range of (0.02, 0.24)) (equivalent to estimates of parameters $\beta_{vd}N_d$ and $\beta_{vw}N_w$ in this work). Cruz-Pacheco *et al.* [**12**] also estimated such transmission probabilities from birds to vectors (Table 2) using data from Komar *et al.* [**20**], who exposed some bird species to WNV by infectious bite of Culex mosquitoes. Using stratification of domestic and wild birds (Table 2), the mean estimates of $\beta_{vd}N_d$, and $\beta_{vw}N_w$ were 0.39 and 0.55, respectively, the computation of which requires the stationary population size of the bird species.

- $\beta_{\mathbf{dv}}$, $\beta_{\mathbf{wv}}$: Wonham *et al.* [**44**] and Bowman *et al.* [**5**] also provide the mean estimates of the **transmission probabilities from mosquito to bird** as 0.88 (with a range of (0.8, 1.0)) (equivalent to estimates of parameters $\beta_{dv}N_d$ and $\beta_{wv}N_w$, respectively, in this work). Using data from Komar *et al.* [**20**], the mean estimates of $\beta_{dv}N_d$ or $\beta_{wv}N_w$ are found to be almost same and are taken to be equal to 1.

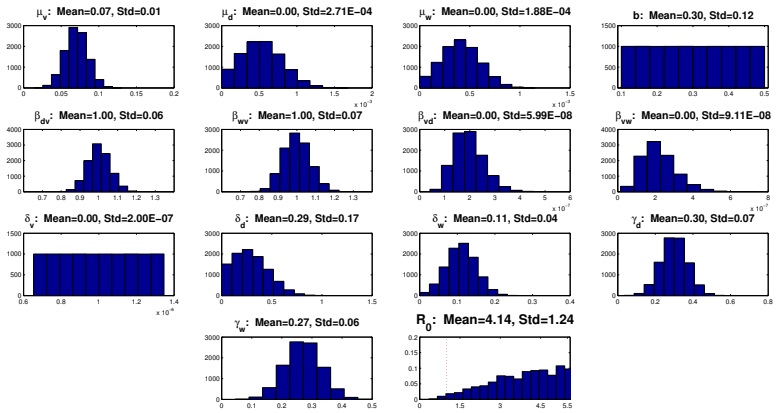

FIGURE 3: Model parameters distribution and estimated distribution of $\mathcal{R}_0$ from uncertainty analysis.

The assumed distribution of the model parameters used in the two analyses are mentioned in Table 6 (similar to ones mentioned in [**27**]). Our estimates of $\mathcal{R}_0$ for WNV from uncertainty analysis is 4.1 with 95%

CI $(1.66, 6.54)$ (Figure 3). The probability that $\mathcal{R}_0 > 1$ is 89%. Hence, under present conditions, WNV disease is most likely to become endemic in the U.S. However, the time to reach such state could be large.

The sensitivity analysis suggest that the most significant (PRCC values above 0.5 or below -0.5 in Figure 4) sensitivity parameters to $\mathcal{R}_0$ are b, μ_v, μ_w, μ_d, and β_{vw}. This suggests that these parameters need to be estimated with precision to capture the transmission dynamics of the WNV in US. The analyses further suggest the intervention programs that aim for reducing mosquitoes' density and biting rate, will have higher impact on controlling the WNV than control programs associated with birds.

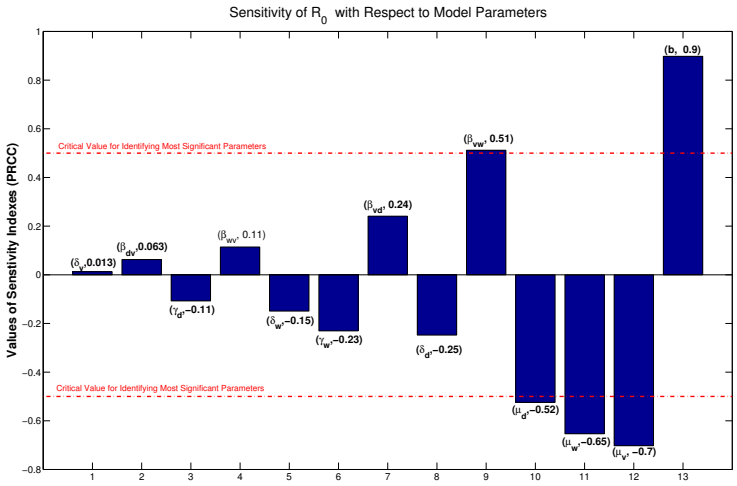

FIGURE 4: Sensitivity indexes of $\mathcal{R}_0$ with respect to the model parameters arranged in ascending order of their magnitude.

Conclusions A deterministic model is designed and used to study the transmission dynamics of WNV in a domestic and a wild bird population, via vectors. The main results are summarized below:

(i) The disease-free equilibrium of the model is globally-asymptotically stable whenever the reproduction number ($\mathcal{R}_0$) is less than unity;
(ii) The disease persists uniformly with respect to small changes in the model parameters, when $\mathcal{R}_0 > 1$;

(iii) When $\mathcal{R}_0 > 1$, the model has a unique endemic equilibrium under certain condition;
(iv) Numerical simulations suggest that the endemic equilibrium, when it exists, is asymptotically stable;
(v) Sensitivity analysis of the model parameters suggests that WNV intervention programs aimed at the reduction of mosquito density will have a higher impact in controlling the disease, than control programs associated with birds.

Acknowledgement Tufail Malik is grateful to Professor Abba Gumel of the University of Manitoba for useful suggestions on the design of the model.

REFERENCES

1. T. Bakonyi, *et al.*, *Lineage 1 and 2 strains of encephalitic West Nile virus, Central Europe*, Emerg. Inf. Dis. **12**(4) (2006), 618–623.
2. BirdLife International (2012), *"Quiscalus quiscula". IUCN Red List of Threatened Species*, Version 2012.1, International Union for Conservation of Nature, retrieved November 2012.
3. K. W. Blayneh, A. B. Gumel, S. Lenhart and T. Clayton, *Backward bifurcation and optimal control in transmission dynamics of West Nile virus,* Bull. Math. Biol. **72**(4) (2009), 1006–1028.
4. E. M. Botha *et al.*, *Genetic determinants of virulence in pathogenic lineage 2 West Nile virus strains,* Emerg. Inf. Dis. **14**(2) (2008), 222–230.
5. C. Bowman *et al.*, *A mathematical model for assessing control strategies against West Nile virus,* Bul. Math. Bio. **67** (2005), 1107–1133.
6. G. L. Campbell, A. A. Marfin, R. S. Lanciotti and D. J. Gubler, *West Nile virus: Reviews*, Lancet Infect. Dis. **2** (2002), 519–529.
7. Center for Disease Control and Prevention (CDC) website, *Malaria: Anopheles Mosquito*, `http://www.cdc.gov/malaria/biology/mosquito/index.htm` retrieved December 2007.
8. Center for Disease Control and Prevention (CDC) website, *West Nile Virus—Vertebrate Ecology*, `http://www.cdc.gov.`, retrieved March 2013.
9. S. Chatterjee, A. K. Ghosh and J. Chattopadhyay, *Controlling disease in migratory bird population: a probable solution through mathematical study*, Dyn. Sys. **21**(3) (2006), 265–288.
10. M. Y. Chowers *et al.*, *Clinical characteristics of the West Nile fever outbreak, Israel, 2000*, Emerg. Infect. Dis. **7** (2001), 686–691.
11. O. Diekmann and J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Dieases: Model Building, Analysis and Interpretation*, Wiley, New York, 1999.
12. G. Cruz-Pacheco, L. Esteva, J. A. Montano and C. Vargas, C. *Modelling the dynamics of West Nile virus*, Bull. Math. Biol. **67**(6) (2005), 1157–1172.
13. L.C. Glaser *et al.*, *West Nile virus infection among turkey breeder farm workers - Wisconsin, 2002*, JAMA **290**(21) (2003), 2793–2796.

14. D. J. Gubler, *Dengue,* in: The Arboviruses: Epidemiology and Ecology (T. P. Monath, ed.), Vol. II. (1986), CRC Press, Florida, 213–261.
15. J. F. Jiang, Z. P. Qiu, J. Wu and H. Zhu, *Threshold conditions for West Nile virus outbreaks,* Bull. Math. Biol. (2008), DOI 10.1007/s11538-008-9374-6.
16. J. Jiang and Z. Qiu, *The complete classification for dynamics in a nine-dimensional West Nile virus model*, SIAM J. Appl. Math. **69**(5) (2009), 1205–1227.
17. J. Jiang, Z. Qiu, J. Wu and H. Zhu, *Threshold conditions for West Nile virus outbreaks*, Bull. Math. Biol. **71** (2009), 627–647.
18. E. Jurdain *et al.*, *West Nile virus in wild resident birds, Southern France, 2004*, Vector Borne Zoonotic Dis. **7**(3) (2007), 448–452.
19. M. K. Klimkiewicz and A. G. Futcher, *Longevity records of North American birds: Supplement*, I. J. Field Ornithol. **60**(4) (1989), 469–494.
20. N. Komar *et al. Experimental infection of North American birds with the New York 1999 strain of West Nile virus*, Emerg. Infect. Dis. **9**(3) (2003), 311–323.
21. V. Lakshmikantham, S. Leela and A. A. Martynyuk, *Stability Analysis of Nonlinear Systems*, Marcel Dekker, Inc., New York and Basel, 1989.
22. R. S. Lanciotti *et al.*, *Complete genome sequences and phylogenetic analysis of West Nile virus strains isolated from the United States, Europe, and the Middle East*, Virology **298** (2002), 96–105.
23. M. A. Lewis, J. Rencławowicz, P. van den Driessche and M. Wonham, *A comparison of continuous and discrete-time west nile virus models*, Bull. Math. Biol. **68** (2006), 491–509.
24. C. C. Lord and J. F. Day, *Simulation studies of St. Louis encephalitis virus in South Florida*, Vector Borne Zoonotic Diseases **1**(4) (2001), 299–315.
25. C. C. Lord and J. F. Day, *Simulation studies of St. Louis encephalitis and West Nile viruses: the impact of bird mortality*, Vector Borne Zoonotic Diseases **1**(4) (2001), 317–329.
26. N. A. Maidana and H. M. Yang, *Dynamic of West Nile virus transmission considering several coexisting avian populations*, Math. Comp. Modelling **53** (2011), 1247–1260.
27. A. Mubayi *et al.*, *Transmission dynamics and underreporting of Kala-azar in the Indian state of Bihar*, J. Theor. Biol. **262**(1) (2010), 177–185.
28. A. Mubayi *et al.*, *Types of Drinkers and Drinking Settings: Application of a Mathematical Model*, Addiction **106**(4) (2011), 749–758.
29. J. L. Patnaik *et al.*, *Environmental predictors of human West Nile virus infections, Colorado*, Emerg. Inf. Dis. **13**(11) (2007), 1788–1790.
30. C. Pepperell *et al. West Nile virus infection in 2002: morbidity and mortality among patients admitted to hospital in southcentral Ontario*, CMAJ **168** (2003), 1399–1405.
31. L. R. Petersen, A. A. Marfin and D. J. Gubler, *West Nile virus*, JAMA **290**(4) (2003), 524–528.
32. Z. Qiu, *Dynamics of an epidemic model with host migration*, Appl. Math. and Comp. **218** (2011), 4614–4625.
33. J. H. Rappole, S. R. Derrickson and Z. Hubalek, *Migratory birds and spread of West Nile virus in the Western Hemisphere*, Emerg. Infect. Dis. **6** (2000), 319–328.
34. W. K. Reisen *et al.*, *Comparison of immune responses of brown-headed cowbird and related blackbirds to West Nile and other mosquito-borne encephalitis viruses*, J. Wildlife Dis. **43**(3) (2007), 439–449.
35. P. L. Salceanu, *Robust uniform persistence in discrete and continuous dynamical systems using Lyapunov exponents*, Math. Biosc. Eng. **8** (2011), 807–825.
36. M. A. Sanchez and S. M. Blower, *Uncertainty and sensitivity analysis of the basic reproductive rate: tuberculosis as an example*, Am. J. Epidemiol. **145**(12) (1997), 1127–1137.

37. H. L. Smith, *Monotone Dynamical Systems, an Introduction to the Theory of Competitive and Cooperative Systems*, American Mathematical Society, Mathematical Surveys and Monographs, 1995.
38. H. L. Smith and P. Waltman, *The Theory of the Chemostat. Dynamics of Microbial Competition*, Cambridge Studies in Mathematical Biology, Cambridge University Press, Cambridge, 1995.
39. H. L. Smith and H. R. Thieme, *Dynamical Systems and Population Persistence*, Graduate Studies in Mathematics, Amer. Math. Soc., Providence, RI, 2011.
40. M. I. Teboh-Ewungkem, *Malaria control: the role of local communities as seen through a mathematical model in a changing population-Cameroon*, in: Advances in Disease Epidemiology, Nova Science Publishers, New York, (J. M. Tchuenche and Z. Mukandavire, eds.), 2009, 101–138.
41. D. M. Thomas and B. Urena, *A model describing the evolution of West Nile-like encephalitis in Nyew York City*, Math. Comput. Modelling **34** (2001), 771–781.
42. P. van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Bios. **180** (2002), 29–48.
43. H. Wan and H. Zhu, *Backward bifurcation in compartmental models for West Nile virus*, Math. Biosc. **227** (2010), 20–28.
44. M. J. Wonham, T. de-Camino-Beck and M. Lewis, *An epidemiological model for West Nile virus: invasion analysis and control applications*, Proc. Roy. Soc. London, Series B **1538** (2004), 501–507.

Corresponding author: T. Malik
Department of Applied Mathematics and Sciences,
Khalifa University of Science, Technology and Research, Abu Dhabi, UAE.
E-mail address: tufail@asu.edu

Department of Mathematics, University of Louisiana, Lafayette, Louisiana, USA.

Department of Mathematics, Northeastern Illinois University, Chicago.

Department of Mathematical Sciences, United Arab Emirates University, Al Ain, Abu Dhabi, UAE.

Lahore University of Management Sciences, Lahore, Pakistan.