Modelling the dynamics of West Nile Virus

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Abstract

In this work we formulate and analyze a mathematical model for the transmission of West Nile Virus (WNV) infection between vector (mosquito) and avian population. We find the Basic Reproductive Number $\tilde{R}_0$ in terms of measurable epidemiological and demographic parameters. $\tilde{R}_0$ is the threshold condition that determines the dynamics of WNV infection: if $\tilde{R}_0 \le 1$ the disease fades out, and for $\tilde{R}_0 > 1$ the disease remains endemic. Using experimental and field data we estimate $\tilde{R}_0$ for several species of birds. Numerical simulations of the temporal course of the infected bird proportion show damped oscillations approaching the endemic value.
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1. Introduction

West Nile virus (WNV) is a mosquito-borne flavivirus and human, equine, and avian pathogen. It is believed that birds are its natural reservoir. Humans, horses and probably other vertebrates are circumstantial hosts; that is, they can be infected by an infectious mosquito but they do not transmit the disease. Then, WNV is maintained in nature in a

mosquito–bird–mosquito transmission cycle (Campbell et al., 2002; Hayes, 1989; Komar et al., 2003; Lanciotti et al., 1999; Montaño-Hirose, 2002).

The primary vectors of WNV are *Culex* spp. mosquitoes, although the virus has been isolated from at least 29 more species of ten genera (Campbell et al., 2002).

When an infected mosquito bites a bird, it transmits the virus; the birds may then develop sufficiently high viral titers during three to five days to infect another mosquito.

The virus can also be passed via vertical transmission from a mosquito to its offspring (Baqar et al., 1993; Swayne et al., 2000) and this increases the survival of WNV in nature. It is believed that this was the mechanism responsible for the persistence of the epidemics in New York after the winter of 1999.

It has been found that birds from certain species may become infected by WNV after ingesting it from an infected dead animal or infected mosquitoes, both natural food items of some species (Komar et al., 2003).

Most WN viral infections are subclinical, but clinical infections can range in severity from uncomplicated WN fever to fatal meningoencephalitis (Campbell et al., 2002).

The virus has been isolated from blood samples of humans, some other mammals, birds and mosquitoes only in countries of Africa, Asia and Europe. In the 50s, 40% of the human population in the Egyptian Delta Nile was seropositive, and around 3000 clinical cases were registered in South Africa (Vargas-García and Cárdenas Lara, 2002). WNV was detected for the first time in North America in 1999, during an outbreak involving humans, horses, and birds in New York City (CDC, 1999). Since then it has spread rapidly to most of the United States (CDC, 2001). In this country between 1999 and 2001, WNV was associated with 149 cases of neurological diseases in humans, 814 cases of equine encephalitis and 11,932 deaths in the avian population. During 2003, 9858 human cases and 14 deaths were reported (CDC, 2004).

In this paper we use a system of nonlinear differential equations to explore the temporal mosquito–bird cycle transmission of WNV. It consists of the interactions among susceptible and infective individuals of the two species assuming that the transmission of the disease is only by mosquito bites and vertical transmission in the vector population. Birds that arrive in the community by birth or immigration are all susceptible. We study the stability of the steady states of the system and we find the basic reproductive number $\tilde{R}_0$ which controls the dynamics of the infection. We conclude that this basic reproductive number is a better measure of the capacity of the species to transmit the infection than the competence index c_j used in the literature.

We show that the infection presents oscillations intrinsic to its dynamics, which should not be confused with seasonal oscillations. The model also shows that after the outbreak the infection seems to disappear for a period of time, but one should be aware that under certain circumstances a second peak may appear.

In this paper we are concerned with the importance of the vector vertical transmission to the dynamics of the infection. As was mentioned above, this mechanism of transmission is believed to be responsible for the permanence of the infection even with scarce avian population. Our model supports this thesis. We find that if the coefficient of vertical transmission is high enough, the dynamics of the infection depends more strongly on the vectors than on the host population. Also the endemic proportion of infected birds grows with the vertical transmission.

Another important issue which we address is the impact of the epidemics on the host population. The model predicts that during the first two or three years after the outbreak the impact on the host population is very important. After this period of time the epidemic tends to an endemic steady state with an infected proportion that depends on R_0. In our simulations this proportion is not bigger than 0.05% of the avian population.

In the next section we formulate the model; in Sections 3 and 4 we find and analyze the equilibrium points of the model. Section 5 contains numerical results and applications, and in Section 6 we present the conclusions.

2. Formulation of the model

Let $N_a(t)$ and $N_v(t)$ be the avian and vector populations at time t, respectively. We assume that, in a given period of time, the mosquito population is constant and equal to N_v, with birth and death rate constants equal to μ_v. For the avian population we assume a constant recruitment rate Λ_a due to births and immigration; total deaths occur at a rate $\mu_a N_a$ where μ_a is the per capita mortality rate of birds. Thus, the differential equation which governs the disease-free avian population dynamics is

$$\frac{\mathrm{d}N_a}{\mathrm{d}t} = \Lambda_a - \mu_a N_a.$$

It is well known that the solutions of this equation approach the equilibrium Λ_a/μ_a as $t \to \infty$.

Let $S_a(t)$, $I_a(t)$, and $R_a(t)$ denote the number of susceptible, infective, and recovered in the avian population; and $S_v(t)$, $I_v(t)$ the number of susceptible and infective in the vector population. Due to its short life, a mosquito never recovers from the infection (Gubler, 1986), and we do not consider the recovered class in this population.

The infection rate for each species depends on the biting rate of mosquitoes, the transmission probabilities, as well as on the number of infective and susceptible of each species.

The biting rate b of mosquitoes is the average number of bites per mosquito per day. This rate depends on a number of factors, in particular, climatic ones, but for simplicity in this paper we assume b constant, typical values are once every two or three days (Gubler, 1986). The number of vectors per bird is given by N_v/N_a, thus a particular bird receives on average $b(N_v/N_a)$ bites per unit of time.

The transmission probability is the probability that an infectious bite produces a new case in a susceptible member of the other species. Here, the transmission probabilities from vectors to birds and from birds to vectors are denoted by β_a and β_v, respectively.

Then, the infection rates per susceptible bird and susceptible vector are given by

$$b\beta_a \frac{N_v}{N_a} \frac{I_v}{N_v} = \frac{b\beta_a}{N_a} I_v$$

and

$$b\beta_v \frac{I_a}{N_a}.$$

We assume that the infected birds recover at a constant rate γ_a, and we denote by α_a the specific death rate associated with WNV in the avian population. Then, the adjusted infectious period taking into account mortality rates is given by $1/(\gamma_a + \mu_a + \alpha_a)$. We shall assume that $\alpha_a \leq \gamma_a$; this is consistent with observations as can be seen in Table 2 of Section 5.

As mentioned in the Introduction, some species of mosquitoes can transmit WNV vertically. Here, we assume that a fraction $0 \leq p \leq 1$ of the progeny of infectious mosquitos is infectious.

Combining the elements above, we arrive at the following system of differential equations:

$$
\begin{aligned}
\frac{dS_a}{dt} &= \Lambda_a - \frac{b\beta_a}{N_a} I_v S_a - \mu_a S_a \\
\frac{dI_a}{dt} &= \frac{b\beta_a}{N_a} I_v S_a - (\gamma_a + \mu_a + \alpha_a) I_a \\
\frac{dR_a}{dt} &= \gamma_a I_a - \mu_a R_a \\
\frac{dS_v}{dt} &= \mu_v S_v + (1-p)\mu_v I_v - \frac{b\beta_v}{N_a} I_a S_v - \mu_v S_v \\
\frac{dI_v}{dt} &= p\mu_v I_v + \frac{b\beta_v}{N_a} I_a S_v - \mu_v I_v \\
\frac{dN_a}{dt} &= \Lambda_a - \mu_a N_a - \alpha_a I_a
\end{aligned}
\tag{2.1}
$$

with the conditions $S_a + I_a + R_a = N_a$ and $S_v + I_v = N_v$.

The first orthant in the $S_a I_a R_a S_v I_v N_a$ space is positively invariant for system (2.1) since the vector field on the boundary does not point to the exterior. Furthermore, since $dN_a/dt < 0$ for $N_a > \frac{\Lambda_a}{\mu_a}$ and N_v is constant, all trajectories in the first orthant enter or stay inside the region

$$
T_+ = \left\{ S_a + I_a + R_a = N_a \leq \frac{\Lambda_a}{\mu_a}, \, S_v + I_v = N_v \right\}.
$$

The continuity of the right-hand side of (2.1) implies that unique solutions exist on a maximal interval. Since solutions approach, enter or stay in T_+, they are eventually bounded and hence exist for $t > 0$, (Coddington and Levinson, 1955). Therefore, the initial value problem for system (2.1) is mathematically well posed and biologically reasonable since all variables remain nonnegative.

In order to reduce the number of parameters and simplify system (2.1) we normalize the bird and vector population

$$
s_a = \frac{S_a}{\Lambda/\mu_a}, \qquad i_a = \frac{I_a}{\Lambda/\mu_a}, \qquad r_a = \frac{R_a}{\Lambda/\mu_a}, \qquad n_a = \frac{N_a}{\Lambda/\mu_a},
$$
$$
s_v = \frac{S_v}{N_v}, \qquad i_v = \frac{I_v}{N_v}.
$$

Since $r_a = n_a - s_a - i_a$ and $s_v = 1 - i_v$, we can omit the equations for r_a and s_v. Then, system (2.1) is equivalent to the four dimensional non-linear system of ODEs for the proportions:

$$\frac{ds_a}{dt} = \mu_a - \frac{b\beta_a m}{n_a} i_v s_a - \mu_a s_a$$

$$\frac{di_a}{dt} = \frac{b\beta_a m}{n_a} i_v s_a - (\gamma_a + \mu_a + \alpha_a) i_a$$

$$\frac{di_v}{dt} = \frac{b\beta_v}{n_a} i_a (1 - i_v) - (1 - p)\mu_v i_v \qquad (2.2)$$

$$\frac{dn_a}{dt} = \mu_a - \mu_a n_a - \alpha_a i_a,$$

in the subset $\Omega = \{0 \leq s_a, 0 \leq i_a, s_a + i_a \leq n_a \leq 1, 0 \leq i_v \leq 1\}$. Here $m = \frac{N_v}{\Lambda_a/\mu_a}$ is the ratio between the vector population and the disease-free equilibrium bird population.

3. Steady states of the model

We find the steady states of Eq. (2.2) by equating the derivatives on the left-hand side to zero and solving the resulting algebraic equations. We first analyze the case $0 \leq p < 1$. The points of equilibrium $(\hat{s}_a, \hat{i}_a, \hat{i}_v, \hat{n}_a)$ satisfy the following relations

$$\hat{s}_a = \frac{\mu_a - (\gamma_a + \mu_a + \alpha_a)\hat{i}_a}{\mu_a}$$

$$\hat{n}_a = \frac{\mu_a - \alpha_a \hat{i}_a}{\mu_a} \qquad (3.3)$$

$$\hat{i}_v = \frac{\mu_a b\beta_v \hat{i}_a}{(b\beta_v \mu_a - \alpha_a(1 - p)\mu_v)\hat{i}_a + (1 - p)\mu_v \mu_a}.$$

Substituting (3.3) in the corresponding second equilibrium equation of (2.2), we obtain that the solutions are $\hat{i}_a = 0$, and the roots of the equation

$$r(i_a) = A i_a^2 + B i_a + C \qquad (3.4)$$

where

$$A = [b\beta_v \mu_a - \alpha_a(1 - p)\mu_v]\frac{\alpha_a}{\mu_a},$$

$$B = 2\alpha_a(1 - p)\mu_v - b\beta_v \mu_a - (1 - p)\mu_v(\gamma_a + \mu_a + \alpha_a)R_0,$$

$$C = \mu_a(1 - p)\mu_v(R_0 - 1),$$

and

$$R_0 = \frac{mb^2 \beta_a \beta_v}{(1 - p)\mu_v(\gamma_a + \mu_a + \alpha_a)}. \qquad (3.5)$$

The solution $\hat{i}_a = 0$ gives the disease-free equilibrium point P_0, whose coordinates are $\hat{s}_a = 1, \hat{i}_a = 0, \hat{i}_v = 0$, and $\hat{n}_a = 1$.

We are looking for nontrivial equilibrium solutions in the interior of Ω. From (3.3) it can be seen that this implies $\hat{i}_a \in (0, \frac{\mu_a}{\mu_a + \gamma_a + \alpha_a})$. Evaluating $r(i_a)$ at the end points of the interval we obtain

$$r(0) = \mu_a(1 - p)\mu_v(R_0 - 1)$$

$$r\left(\frac{\mu_a}{\gamma_a + \mu_a + \alpha_a}\right) = -\frac{\mu_a^2 b \beta_v(\gamma_a + \mu_a)}{(\gamma_a + \mu_a + \alpha_a)^2} - \frac{(1 - p)\mu_v \mu_a(\gamma_a + \mu_a)^2}{(\gamma_a + \mu_a + \alpha_a)^2} < 0.$$

When $R_0 = 1$, the roots of $r(i_a)$ are 0 and $\hat{i}_a = -\frac{B}{A}$ with $B = -b\beta_v\mu_a - (1-p)\mu_v(\gamma_a + \mu_a - \alpha_a) < 0$ since by assumption $\alpha_a \leq \gamma_a$. Then, $\hat{i}_a$ is less than zero if $A < 0$. For $A > 0$ it can be seen easily that $\hat{i}_a > \frac{\mu_a}{\gamma_a + \mu_a + \alpha_a}$.

When $R_0 < 1$, the value of the polynomial $r(i_a)$ is negative at the end points of the interval. The conditions to have at least one root in the mentioned interval are

(a) $A < 0$

(b) $0 < -\frac{B}{2A} < \frac{\mu_a}{\mu_a + \gamma_a + \alpha_a}$

(c) $B^2 - 4AC \geq 0$,

but it can be seen that in this case (b) and (c) are not compatible, therefore there are no roots in the interval.

If $R_0 > 1$ then $r(0) > 0$, therefore there exists a unique root in the interval, which implies the existence of a unique equilibrium point $P_1 = (\hat{s}_a, \hat{i}_a, \hat{i}_v, \hat{n}_a)$ in the interior of Ω.

Thus, we have proved that for $R_0 \leq 1$, P_0 is the only equilibrium point in Ω, but in the case $R_0 > 1$ the endemic equilibrium P_1 will also lie in Ω.

The *Basic Reproductive Number* of a disease is the average number of secondary cases that one infectious individual produces during its infectious period in a totally susceptible population.

For the WNV infection, the number of infections produced by a single bird during its infectious period in a susceptible mosquito population is given by

$$\frac{mb}{\gamma_a + \mu_a + \alpha_a}\beta_v.$$

Analogously, the number of infections in a susceptible avian population produced by a single infectious mosquito during its lifespan is given by

$$\frac{b}{(1 - p)\mu_v}\beta_a.$$

The geometric mean of these quantities, $\sqrt{R_0}$, represents the average number of secondary infections produced by a single infectious bird or mosquito during its infectious period. Therefore $\tilde{R}_0 = \sqrt{R_0}$ is the Basic Reproductive Number of WNV disease.

Now, we proceed to analyze the case $p = 1$. The reported values of p are rather small as can be seen in Table 2. However, we present this limit case to illustrate the impact of vertical transmission on the permanence of the infection. Notice that $R_0 \to \infty$ when $p \to 1$.

The equilibrium points of (2.2) in this case, are P_0 and the solution of the equations

$$\hat{s}_a = \frac{\mu_a - (\gamma_a + \mu_a + \alpha_a)\hat{i}_a}{\mu_a} \tag{3.6}$$

$$\hat{n}_a = \frac{\mu_a - \alpha_a\hat{i}_a}{\mu_a} \tag{3.7}$$

$$\hat{i}_v = 1$$

where $\hat{i}_a$ is a root of the polynomial

$$q(i_a) = \alpha_a(\gamma_a + \mu_a + \alpha_a)i_a^2 - (b\beta_a m + \mu_a)(\gamma_a + \mu_a + \alpha_a)i_a + b\beta_a m \mu_a \tag{3.8}$$

in the interval $(0, \frac{\mu_a}{\gamma_a + \mu_a + \alpha_a})$. Evaluating $q(i_a)$ at the end points, we obtain

$$q(0) = b\beta_a m \mu_a > 0$$

$$q\left(\frac{\mu_a}{\gamma_a + \mu_a + \alpha_a}\right) = -\frac{\mu_a^2(\mu_a + \gamma_a)}{\gamma_a + \mu_a + \alpha_a} < 0$$

so that (3.8) has a unique root $\hat{i}_a$ in $(0, \frac{\mu_a}{\gamma_a + \mu_a + \alpha_a})$. Therefore, if $p = 1$, the endemic equilibrium state P_1 is in Ω independently of the values of the rest of the parameters. In this case, as we will show in the next section, the infection will remain endemic independently of the other parameters.

4. Stability analysis

In this section we study the stability of the steady states of system (2.2). We start with the case $0 \leq p < 1$. Linearizing around the disease-free equilibrium P_0, we obtain the matrix

$$DF(P_0) = \begin{pmatrix} -\mu_a & 0 & -mb\beta_a & 0 \\ 0 & -(\gamma_a + \mu_a + \alpha_a) & mb\beta_a & 0 \\ 0 & b\beta_v & -(1-p)\mu_v & 0 \\ 0 & -\alpha_a & 0 & -\mu_a \end{pmatrix} \tag{4.9}$$

where DF denotes the derivative of the vector field F given by the right-hand side of Eq. (2.2). The eigenvalues of (4.9) are $-\mu_a$ of multiplicity two, and the roots of the polynomial

$$\lambda^2 + (\gamma_a + \mu_a + \alpha_a + (1-p)\mu_v)\lambda + (\gamma_a + \mu_a + \alpha_a)(1-p)\mu_v(1-R_0).$$

The roots of a polynomial of order two have negative real parts if and only if its coefficients are positive. In our case, both coefficients are positive if and only if $R_0 < 1$. Therefore, the disease-free equilibrium P_0 is locally asymptotically stable for $R_0 < 1$, and unstable for $R_0 > 1$.

When $R_0 > 1$, P_0 becomes an unstable equilibrium point, and the endemic equilibrium P_1 emerges in Ω. The local stability of this point is governed by the roots of the

characteristic equation $\text{Det}(\lambda I - DF(P_1)) = 0$ where $\lambda I - DF(P_1)$ is given by

$$
\begin{pmatrix}
\lambda + \dfrac{mb\beta_a \hat{i}_v}{\hat{n}_a} + \mu_a & 0 & \dfrac{mb\beta_a \hat{s}_a}{\hat{n}_a} & -\dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{n}_a^2} \\[3mm]
-\dfrac{mb\beta_a \hat{i}_v}{\hat{n}_a} & \lambda + \gamma_a + \mu_a + \alpha_a & -\dfrac{mb\beta_a \hat{s}_a}{\hat{n}_a} & \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{n}_a^2} \\[3mm]
0 & -\dfrac{b\beta_v(1 - \hat{i}_v)}{\hat{n}_a} & \lambda + \dfrac{b\beta_v \hat{i}_a}{\hat{n}_a} + (1 - p)\mu_v & \dfrac{b\beta_v \hat{i}_a(1 - \hat{i}_v)}{\hat{n}_a^2} \\[3mm]
0 & \alpha_a & 0 & \lambda + \mu_a
\end{pmatrix}.
$$

Adding the second row of $\lambda I - DF(P_1)$ to the first one, and using Eq. (2.2) in equilibrium

$$
\begin{aligned}
\gamma_a + \mu_a + \alpha_a &= \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{i}_a \hat{n}_a} \\[3mm]
\dfrac{b\beta_v \hat{i}_a}{\hat{n}_a} + (1 - p)\mu_v &= \dfrac{b\beta_v \hat{i}_a}{\hat{i}_v \hat{n}_a}
\end{aligned}
\tag{4.10}
$$

we obtain the equivalent matrix

$$
\begin{pmatrix}
\lambda + \mu_a & \lambda + \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{i}_a \hat{n}_a} & 0 & 0 \\[3mm]
-\dfrac{mb\beta_a \hat{i}_v}{\hat{n}_a} & \lambda + \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{i}_a \hat{n}_a} & -\dfrac{mb\beta_a \hat{s}_a}{\hat{n}_a} & \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{n}_a^2} \\[3mm]
0 & -\dfrac{b\beta_v(1 - \hat{i}_v)}{\hat{n}_a} & \lambda + \dfrac{b\beta_v \hat{i}_a}{\hat{i}_v \hat{n}_a} & \dfrac{b\beta_v \hat{i}_a(1 - \hat{i}_v)}{\hat{n}_a^2} \\[3mm]
0 & \alpha_a & 0 & \lambda + \mu_a
\end{pmatrix}.
$$

Expanding the determinant of this matrix by the last row, it can be seen that the roots of $\text{Det}(\lambda I - DF(P_1))$ are $-\mu_a$ and the roots of

$$
\lambda^3 + P\lambda^2 + Q\lambda + R = 0
\tag{4.11}
$$

where

$$
\begin{aligned}
P &= \dfrac{\mu_a}{\hat{s}_a} + \dfrac{mb\beta_a \hat{i}_v \hat{s}_a}{\hat{i}_a \hat{n}_a} + \dfrac{b\beta_v \hat{i}_a}{\hat{i}_v \hat{n}_a} \\[3mm]
Q &= \dfrac{\mu_a b\beta_v \hat{i}_a}{\hat{s}_a \hat{i}_v \hat{n}_a} + \dfrac{mb^2 \beta_a \beta_v \hat{s}_a \hat{i}_v}{\hat{n}_a^2} + \dfrac{mb\beta_a i_v(\mu_a \hat{n}_a - \alpha_a \hat{i}_a \hat{s}_a)}{i_a n_a^2} \\[3mm]
R &= \dfrac{\mu_a mb^2 \beta_a \beta_v((\hat{n}_a - \hat{s}_a) + \hat{s}_a \hat{i}_v)}{\hat{n}_a^3} - \dfrac{\alpha_a mb^2 \beta_a \beta_v \hat{s}_a \hat{i}_a}{\hat{n}_a^3}.
\end{aligned}
\tag{4.12}
$$

From the Routh–Hurwitz criterion, it follows that all eigenvalues of Eq. (4.11) have negative real parts if and only if $P > 0$, $R > 0$ and $PQ > R$. Now, it is clear that $P > 0$. Using the relation $\hat{n}_a - \hat{s}_a = \hat{i}_a + \hat{r}_a = \frac{(\gamma_a + \mu_a)}{\mu_a}\hat{i}_a$, R becomes

$$
R = \dfrac{mb^2 \beta_a \beta_v \hat{i}_a((\gamma_a + \mu_a) - \alpha_a \hat{s}_a)}{\hat{n}_a^3} + \dfrac{\mu_a mb^2 \beta_a \beta_v \hat{s}_a \hat{i}_v}{\hat{n}_a^3}.
$$

Since we have assumed $\alpha_a \leq \gamma_a$ then $\alpha_a \leq \gamma_a + \mu_a$, also $\hat{s}_a < 1$ therefore $R > 0$. Finally, the inequality $PQ > R$ can be proved easily. Thus, we have proved that P_1 is locally asymptotically stable.

Now, we analyze the stability of the equilibria in the case $p = 1$. The eigenvalues of the Jacobian around the disease-free equilibrium P_0 are $-\mu_a$ and the roots of the polynomial

$$\lambda^2 + (\gamma_a + \mu_a + \alpha_a)\lambda - mb^2\beta_a\beta_v.$$

Since the last coefficient of this equation is negative, then P_0 is always unstable.

On the other hand, for the endemic equilibrium P_1, the eigenvalues are $-\mu_a$, $-\frac{b\beta_v\hat{i}_a}{\hat{n}_a}$ besides the roots of the polynomial

$$\lambda^2 + \left(\frac{mb\beta_a}{\hat{n}_a} + \gamma_a + 2\mu_a + \alpha_a\right)\lambda + (\gamma_a + \mu_a + \alpha_a)\left(\frac{mb\beta_a}{\hat{n}_a} + \mu_a\right) + \alpha_a\frac{mb\beta_a\hat{s}_a}{\hat{n}_a^2},$$

which have negative real part since the coefficients are positive. Therefore, P_1 is locally asymptotically stable.

The above results can be summarized in the following theorem.

Theorem 1. *If $0 \leq p < 1$, then the disease-free equilibrium P_0 is unique and locally asymptotically stable for $R_0 < 1$. When $R_0 > 1$, P_0 becomes unstable, and there appears a new endemic equilibrium P_1 locally asymptotically stable. If $p = 1$, P_0 is always unstable, and P_1 is locally asymptotically stable.*

In the case $\alpha_a = 0$, we can be more precise about the kind of stability of the equilibrium points P_0 and P_1.

When $\alpha_a = 0$ and $R_0 \leq 1$, we can actually prove global stability of P_0. In this case $n_a(t) \rightarrow 1$, and system (2.2) becomes

$$\frac{ds_a}{dt} = \mu_a - mb\beta_a i_v s_a - \mu_a s_a$$
$$\frac{di_a}{dt} = mb\beta_a i_v s_a - (\gamma_a + \mu_a)i_a \qquad (4.13)$$
$$\frac{di_v}{dt} = b\beta_v i_a(1 - i_v) - (1 - p)\mu_v i_v.$$

We define the following Lyapunov function in Ω

$$L = i_a + \frac{mb\beta_a}{(1-p)\mu_v}i_v. \qquad (4.14)$$

The orbital derivative of L is given by

$$\dot{L} = -mb\beta_a(1 - s_a)i_v - (\gamma_a + \mu_a)(1 - R_0(1 - i_v))i_a \qquad (4.15)$$

which is less than or equal to zero for $R_0 \leq 1$. The maximal invariant subset contained in $\dot{L} = 0$ consists of the s_a-axis. In this set, system (4.13) reduces to

$$\frac{ds_a}{dt} = \mu_a - \mu_a s_a$$

Table 1
West Nile Virus competence index for eight species of birds

Common name	s	i	d (days)	c_j
Blue jay	1.0	0.68	3.75	2.55
Common grackle	1.0	0.68	3	2.04
House finch	1.0	0.32	5.5	1.76
American crow	1.0	0.50	3.25	1.62
House sparrow	1.0	0.53	3	1.59
Ring-billed gull	1.0	0.28	4.5	1.26
Black-billed magpie	1.0	0.36	3	1.08
Fish crow	1.0	0.26	2.8	0.73

Data taken from Komar et al. (2003).

$$\frac{\mathrm{d}i_a}{\mathrm{d}t} = 0$$
$$\frac{\mathrm{d}i_v}{\mathrm{d}t} = 0.$$

From these equations we see that $s_a(t) \to 1$, $i_a(t) = 0$, $i_v(t) = 0$ for $t \geq 0$. Therefore, from the LaSalle–Lyapunov theorem (Hale, 1969) it follows that P_0 is locally stable and all trajectories starting in Ω approach P_0 for $R_0 \leq 1$.

Thus, we have proved the global asymptotical stability of P_0 for $R_0 \leq 1$ and $\alpha_a = 0$.

When $\alpha_a = 0$ and $R_0 > 1$, (2.2) becomes a three-dimensional system. In this case, global stability of P_1 can be proved using results of competitive systems and compound matrices as in Esteva and Vargas (1998).

5. Numerical results

To estimate experimentally the transmission dynamics, Komar (Komar et al., 2003) exposed 25 bird species to WNV by infectious bites of *Culex tritaeniorhynchus*. He analyzed viremia data to determine values for susceptibility (s), mean daily infectiousness (i), duration of infectious viremia (d), and competence index (c_j) for each species

$$c_j = s \times i \times d.$$

Susceptibility is the proportion of birds that become infected as a result of the exposure; mean daily infectiousness is the proportion of exposed vectors that become infectious per day, and duration of infectious viremia is the number of days that birds maintain an infectious viremia.

The competence index is calculated as a function of the viremia that the bird species develops after mosquito-borne infection and it is a measure of the species efficiency as a transmitter. Table 1 shows the values of s, i, d and c_j obtained in Komar et al. (2003) for eight species.

In the context of our model $s = \beta_a$, $i = \beta_v$, $d = \frac{1}{\gamma}$ and $c_j = \frac{\beta_a \beta_v}{\gamma}$. The same authors estimated the proportion of fatal infections of birds exposed to WNV by mosquito bites and the mean number of days to death. From these data we calculate the daily disease mortality rate α_a as the proportion of deaths divided by the mean number of days to death.

Table 2
Epidemiological and demographic parameters of model (2.2)

Common name	β_a	β_v	γ_a (day^{-1})	α_a (day^{-1})	μ_a (day^{-1})	μ_v (day^{-1})	m	$\tilde{R}_0$
Blue jay	1.0	0.68	0.26	0.15	.0002	.06	5	5.89
Common grackle	1.0	0.68	0.33	.07	.0001	.06	5	6.97
House finch	1.0	0.32	0.18	0.14	.0003	.06	5	4.57
American crow	1.0	0.5	0.31	0.19	.0002	.06	5	4.58
House sparrow	1.0	0.53	0.33	0.1	.0002	.06	5	5.08
Ring-billed gull	1.0	0.28	0.22	0.1	.0003	.06	5	4.28
Black-billed magpie	1.0	0.36	0.33	0.16	.0001	.06	5	3.92
Fish crow	1.0	0.26	0.36	.06	.0002	.06	5	3.60

Approximated values of μ_a for each bird species were obtained from Oliver Jr. (1961a,b), The University of Michigan Museum of Zoology (2004), and are given in Table 2. As mentioned before, typical values of the biting rate b are once every two or three days. Here we assumed $b = .5$ day^{-1}. The reported values for the lifespan of mosquitoes vary from weeks to months. An average value is two or three weeks for females (Gubler, 1986).

Using the values of the parameters in Table 1, we estimated $\tilde{R}_0$ for each bird species. We assumed in all of the cases that the ratio $m = \frac{N_v}{\Lambda_a/\mu_a} = 5$. In Komar et al. (2003) the value of m varies from ten to fifteen, but since the experiments consisted of caged birds with a predeterminate amount of mosquitoes that were allowed to bite as many times as wanted, we believe that this situation increased m artificially. The probability of vertical transmission was taken as $p = 0.007$ according to Dohm et al. (2002), Goddard et al. (2003).

According to Table 1, the American crow and the house finch are more competent than the house sparrow, however the number of secondary infections produced by individuals of these species is less than the corresponding number produced by the house sparrows. The same phenomenon is observed between the blue jay and the common grackle.

We noticed that the disease mortality rates of the American crow, the house finch and the blue jay are significantly greater than the corresponding ones for the house sparrow, and the common grackle. The role of disease-related mortality in the dynamics of the disease is reflected in $\tilde{R}_0$ but not in the competence index c_j. The disease-related death rate α_a reduces the average infectious period, and consequently the number of infection transmissions per infective. Thus, a high disease mortality is likely to diminish the efficiency of a species as a transmitter. This suggests that $\tilde{R}_0$ is a better measure of the epidemiological importance of a given species.

Fig. 1 illustrates the time course of the infected bird proportion for the blue jay, the American crow and the house sparrow. In this figure we only present the first epidemic peak.

In Fig. 2 we present how as t tends to infinity the solutions oscillate to the endemic value $\hat{i}_a$. This behavior can be explained in terms of R_0. The proportion of susceptible mosquitoes infected by one infectious bird is $(mb\beta_v/(\gamma_a + \mu_a + \alpha_a))s_v$, analogously the proportion of susceptible birds which are infected by one mosquito is $(b\beta_a/(1 - p)\mu_v)s_a$. Therefore,
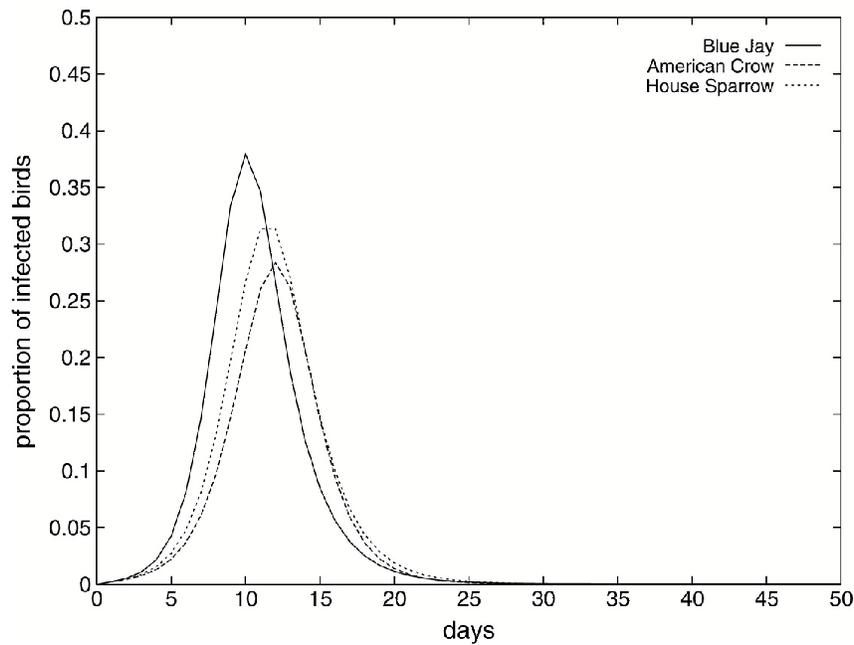
Fig. 1. Numerical simulation of system (2.2). The graphs show the temporal course of the proportion of infected birds. The parameters are given in Table 2 and the initial conditions are $s_a = 1$, $i_a = 0$, $i_v = 0.001$, $n_a = 1$.
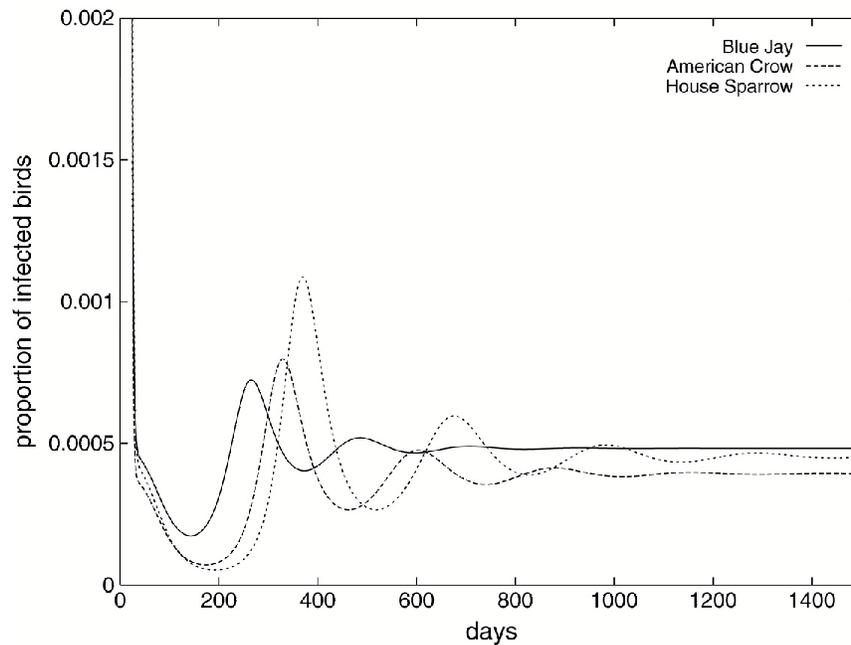

Fig. 2. Asymptotic behavior of the solutions of Fig. 1.

if in addition to $R_0 > 1$, we have $R_0 s_a s_v > 1$ then both fractions s_a and s_v decrease and the infectious proportions i_a and i_v first increase to a peak and then both decrease because there are not sufficient susceptibles to be infected and some of the infected ones
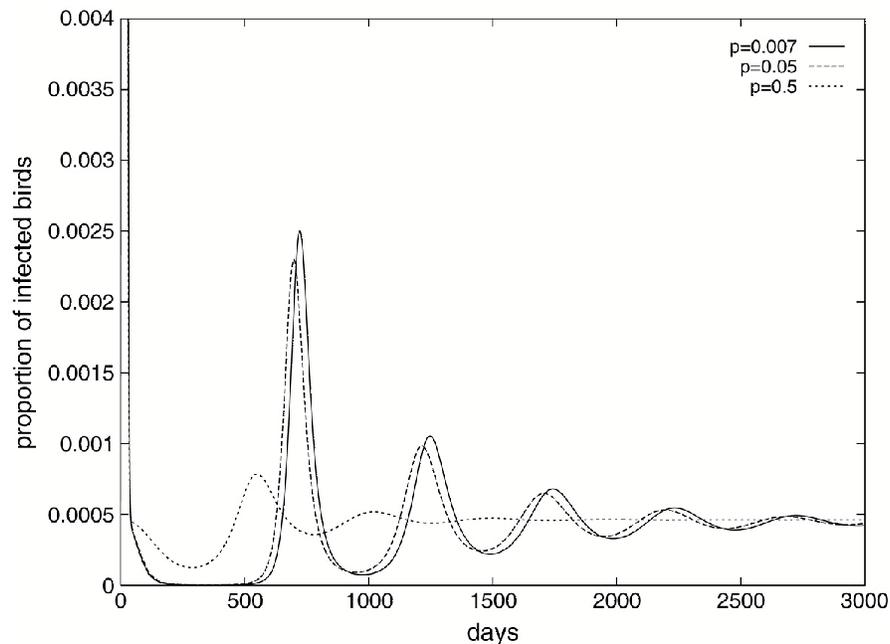
Fig. 3. Numerical solutions of system (2.2) for different values of p. The graphs show the temporal course of the proportion of infected fish crow when $p = 0.007, 0.05, 0.5$. The corresponding values of $\tilde{R}_0$ are 3.60, 3.68 and 5.07, respectively.

recover or die. When the susceptible fractions get large enough due to recruitment of new susceptibles, there are secondary smaller epidemics, and thus the solution oscillate to the endemic equilibrium.

In Fig. 3 we observe the increment of WNV activity when vertical transmission increases. The values of p in these simulations are taken much larger than the values reported, in order to appreciate its effect on the dynamics of the epidemics. We notice that as p increases, the endemic equilibrium is reached faster and practically without oscillations. This means that WNV does not need a high recruitment of susceptible birds to remain in nature. In this case the dynamics of the infection is dominated by the vector population.

6. Conclusions

The dramatic appearance of the epidemic of WNV in the northeast part of the United States, is an unsettling remainder of the ability of viruses to jump continents. The subsequent spread of WNV shows that, although arboviral transmission cycles are usually very complex, the basic mechanisms for the introduction and maintenance of arbovirus in new areas are the movement pattern of birds, the existence of efficient vectors and susceptible hosts.

In this paper we developed and analyzed a mathematical model to understand the dynamics of WNV disease. We obtained conditions for the maintenance of the disease when the virus is introduced in a certain region.

We found that

$$\tilde{R}_0 = \sqrt{R_0} = \sqrt{\frac{mb^2 \beta_a \beta_v}{(\gamma_a + \mu_a + \alpha_a)(1 - p)\mu_v}}$$

is the basic reproductive number for the disease, that is, the mean number of secondary cases produced by a primary infective when introduced in a susceptible population. Then, if $\tilde{R}_0$ is less than one, the disease will fade out since an infective individual will be replaced with less than one new case. On the other hand, if $\tilde{R}_0$ is greater than one, the infected fraction of the mosquito and bird populations will approach an endemic steady state.

Usually, registers of WNV cases in the avian population are based on the number of dead birds found. Thus, epidemiological reports indicate high WNV prevalence in species with high disease mortality rate. This could lead to the idea that the most vulnerable species to the disease, like the American crow, are the best transmitters and consequently the main responsibles for the spread of the disease. Nevertheless, we found that in some cases, the basic reproductive number $\tilde{R}_0$ of such species is less than the corresponding one for species considered less competent. Thus, according to Table 2, the number of secondary infections derived from an infected house sparrow is bigger than the corresponding number derived from an American crow, even when the latter is considered a better transmitter. An explanation of this fact is that species with high disease mortality rate could not be such as good transmitters due to the fact that their infection period ends sooner. Then, statistics based on reported dead animals or laboratory experiments which do not take into account mortality are not sufficient to estimate the real importance of a bird species in the disease spread. Implementation of other mechanisms of surveillance such as sampling, would give a more accurate idea about disease prevalence in the bird population and the importance of the different species on the diffusion of the disease.

In Figs. 1 and 2, we observe that maximal prevalence in the avian population is very high when the disease is introduced for the first time. In the three cases analyzed, between two and four tenths of the avian population got infected during the first epidemic peak. This first outbreak fades out after around 30 days followed by a period of time where the infection seems to disappear. After this period a second epidemic peak appears. It is interesting to notice that the longer it takes for the second peak to appear, the higher it is. This is explained by the fact that when the period between the first two peaks is large, more susceptible birds are recruited by the end of this period, and their number becomes close to the initial data. Thus, the situation is more alike to the one of initial conditions. After the second peak, the infected population will approach the endemic prevalence value through damped oscillations. It is interesting to observe that in the three cases, even when the first outbreak is very high, the endemic prevalence is not: less than 0.05% of the total population size. Therefore, it seems that extinction of a bird population due to the disease could be possible only during the first two or three years after the first outbreak.

The quantity $\tilde{R}_0$ grows with the mosquito population; thus, the disease spreads more rapidly when birds migrate to a region with higher mosquito density. Vertical transmission in the vector population is also a risk factor for the spread of WNV. The model predicts that if vertical transmission is sufficiently high, the disease can be maintained forever, even in regions with scarce avian population. This is shown in Fig. 3. Here, as vertical

transmission increases, the proportion of endemic infected birds grows, and the epidemic peaks tend to disappear. Then the proportion of infected birds becomes less dependent upon the recruitment of susceptible birds.

The effect of the disease mortality associated with the infection on the dynamics of avian population is of great importance, and this depends upon α_a. Low values of α_a would have a small effect on the population size, while high values will cause the disease to fade out since $\tilde{R}_0$ decreases when α_a increases, and eventually the population size will return to its original values. Therefore, intermediate values of α_a are the ones which can cause more damage to the population.

In Mexico, WNV has circulated at least since 2002 (Blitvich et al., 2003; Estrada-Franco et al., 2003; Loroño-Pinto et al., 2003). However, the reports of the virus activity in birds show that the infection has caused a negligible epidemiological impact. In 2004 from January to October, among 4833 studied samples, 171 were serologically positive and only in two cases was the virus isolated (SSa, 2004). The main question is why WNV activity in Mexico is rather mild compared with that in the United States. Some studies point to the hypothesis that the presence in the region of other arboviruses such as dengue and Saint Louis encephalitis can cause cross immunity to WNV (Tesh et al., 2002). In our model, this hypothesis could be reflected on a reduction of the transmission probabilities β_a and β_v, that would drive the basic reproductive number $\tilde{R}_0$ to values less than one. However, this question should be a subject of further studies.

The control of WNV is very difficult to achieve due mainly to the large number of species of wild bird and mosquito susceptibles to it, and to the fact that some vectors present vertical transmission. For this reason, the most likely scenario for the spread of WNV is that it will remain endemic in the Americas. Even if the endemic prevalence is rather low, environmental changes (prolonged rains, droughts, hurricanes, etc.) could modify R_0 giving rise to more severe outbreaks, or in contrast, disappearance of the disease. Also, the immune response among bird species and cross-immunity due to exposure to other flaviviruses, together with the natural selection of the strongest individuals, could reduce the levels of endemicity.

Acknowledgments

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