A MATHEMATICAL MODEL FOR THE DYNAMICS OF WEST NILE VIRUS

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Abstract: In this paper a mathematical model is formulated to study the dynamics of West Nile Virus (WNV) infection between mosquito and bird population. A qualitative analysis as well as some numerical examples are given for the model.

Keywords: Dynamics, Ecology, Lyapunov, modelling, nonlinear, simulation, stability.

1. INTRODUCTION

West Nile virus (WNV) is a mosquito-borne flavivirus and human, equine, and bird pathogen. It is believed that birds are the natural reservoir, and humans, equines and probably other vertebrates are circunstantial hosts; that is, they can be infected by an infectious mosquito but they do not transmit the infection. Then, WN virus is maintained in nature in a mosquito-bird-mosquito transmission cycle (Lanciotti et al., 1999; Kramer and Bernard, 2001; Campbell et al., 2002). The primary vectors of WNV are *Culex* spp. mosquitoes (Campbell et al., 2002), although the virus has been isolated from at least 29 more species of ten genera. The virus can also be passed via vertical transmission between a mosquito and her offspring (Baqar et al., 1993; Swayne et al., 2000), and that increases the survival of WNV in nature. It has been found (Komar et al., 2003) that certain bird species may become infectious by WNV after ingesting it in an infected dead animal and infected mosquitoes, both natural food items of some species .

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 in humans, some other mammals, birds and mosquitoes (Montaño, 2002)in countries of Africa, Asia and Europe. WNV was detected for the first time in North America in 1999, during an outbreak in New York City (CDC, 1999). Since then it has been spread rapidily to most of 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 bird population. During 2003, 9858 human cases and 14 deaths were reported (CDC, 2004).

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In Mexico WNV activity is rather mild (Blitvich et al, 2003) compared with United States. Some studies (Tesh et al., 2002) point to the hypothesis that the presence in the region of other arbovirus infections as Dengue and Saint Louis Encephalitis can cause cross immunization to WNV.

In this paper a model is presented to explore the temporal mosquito-bird cycle transmission of the 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 mosquito population. Birds that arrive to the community by birth or migration are all susceptible.

2. FORMULATION OF THE MODEL

Let $\bar{N}_a$ and $\bar{N}_v$ be the bird and mosquito population sizes, respectively. It is assumed that the mosquito population has constant size with birth and death rate constant equal to μ_v. For the bird population it is assumed a constant recruitment rate Λ_a due to births and immigrations; total deaths occur at a rate $\mu_a \bar{N}_a$ where μ_a is the per capita mortality rates of birds. Thus, the differential equation that governs the disease free bird population dynamics is

$$\bar{N}_a' = \Lambda_a - \mu_a \bar{N}_a.$$

The solutions $\bar{N}_a$ of this equation approach the equilibrium Λ_a/μ_a as $t \to \infty$.

Let $\bar{S}_a(t)$, $\bar{I}_a(t)$, and $\bar{R}_a(t)$ the number of susceptibles, infectives and recovered in the bird population; and $\bar{S}_v$, $\bar{I}_v$ the number of susceptibles and infectives in the mosquito population. Due to its short life, a mosquito never recovers from the infection (Gubler, 1986), and the recovered class is not considered in this population.

The infection rates for each species depend on the biting rate of mosquitos, the transmission probabilities, as well as the number of infectives and susceptibles of each species. The biting rate b of mosquitoes is the average number of bites per mosquito per day. The transmission probability is the probability that an infectious bite produces a new case in a susceptible member of the other species.

The probability that a mosquito chooses a particular bird as a host is given by $\bar{N}_v/\bar{N}_a$. Thus a bird receives in average $b(\bar{N}_v/\bar{N}_a)$ bites per unit of time. Then, the infection rates per susceptible bird and susceptible mosquito are given by

$$\beta_a b \frac{\bar{N}_v}{\bar{N}_a}\frac{\bar{I}_v}{\bar{N}_v} = \frac{\beta_a b}{\bar{N}_a}\bar{I}_v \quad \text{and} \quad \beta_v b \frac{\bar{I}_a}{\bar{N}_a}.$$

The infected birds recover at a constant rate γ_a, and α_a denotes the specific death rate associated with WNV in the bird population. Then, the adjusted infectious period taking into account mortality rates is given by $1/(\gamma_a+\mu_a+\alpha_a)$. In this paper it is assumed $\alpha_a \leq \gamma_a$, which is consistent with the observations as it can be seen in Table 1 of section 5, where $\gamma = 1/d$.

As was mentioned in the Introduction, some species of mosquitoes can transmit WN virus vertically. Here, it is assumed that a fraction $0 \leq p \leq 1$ of the progeny of infectious mosquitos is infectious.

Combining the elements above, the following system of differential equations is obtained:

$$\begin{aligned}
\bar{S}_a' &= \Lambda_a - \frac{b\beta_a}{\bar{N}_a}\bar{I}_v\bar{S}_a - \mu_a\bar{S}_a \\
\bar{I}_a' &= \frac{b\beta_a}{\bar{N}_a}\bar{I}_v\bar{S}_a - (\gamma_a+\mu_a+\alpha_a)\bar{I}_a \\
\bar{R}_a' &= \gamma_a\bar{I}_a - \mu_a\bar{R}_a \\
\bar{S}_v' &= \mu_v\bar{S}_v + (1-p)\mu_v\bar{I}_v - \frac{b\beta_v}{\bar{N}_a}\bar{I}_a\bar{S}_v - \mu_v\bar{S}_v \\
\bar{I}_v' &= p\mu_v\bar{I}_v + \frac{b\beta_v}{\bar{N}_a}\bar{I}_a\bar{S}_v - \mu_v\bar{I}_v \\
\bar{N}_a' &= \Lambda_a - \mu_a\bar{N}_a - \alpha_a\bar{I}_a
\end{aligned} \tag{1}$$

with the conditions $\bar{S}_a + \bar{I}_a + \bar{R}_a = \bar{N}_a$ and $\bar{S}_v + \bar{I}_v = \bar{N}_v$.

The first orthant in the $\bar{S}_a\bar{I}_a\bar{R}_a\bar{S}_v\bar{I}_v\bar{N}_a$ space is positively invariant for system (1), since the vector field on the boundary does not point to the exterior. Furthermore, since $\bar{N}_a' < 0$ for $\bar{N}_a > \dfrac{\Lambda_a}{\mu_a}$ and $\bar{N}_v$ is constant, all trajectories in the first orthant enter or stay inside the region

$$T_+ = \{\bar{S}_a + \bar{I}_a + \bar{R}_a = \bar{N}_a \leq \frac{\Lambda_a}{\mu_a}, \bar{S}_v + \bar{I}_v = \bar{N}_v\}.$$

The right-hand side of (1) is Lipschitz continous which 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 (1) is mathematically well posed and biologically reasonable since all variables remain nonnegative.

In order to reduce the number of parameters and simplify (1), the bird and mosquito populations are normalized: $S_a = \dfrac{\bar{S}_a}{\Lambda/\mu_a}, I_a = \dfrac{\bar{I}_a}{\Lambda/\mu_a}, R_a = \dfrac{\bar{R}_a}{\Lambda/\mu_a}, N_a = \dfrac{\bar{N}_a}{\Lambda/\mu_a}, S_v = \dfrac{\bar{S}_v}{\bar{N}_v}, I_v = \dfrac{\bar{I}_v}{\bar{N}_v}$. Since $R_a = N_a - S_a - I_a$ and $S_v = 1 - I_v$, system (1) is equivalent to the four dimensional non-linear system of ODEs for the proportions:

$$S_a' = \mu_a - \frac{b\beta_a m}{N_a} I_v S_a - \mu_a S_a$$

$$
\begin{aligned}
I_a' &= \frac{b\beta_a m}{N_a} I_v S_a - (\gamma_a + \mu_a + \alpha_a) I_a \\
I_v' &= \frac{b\beta_v}{N_a} I_a (1 - I_v) - (1-p)\mu_v I_v \\
N_a' &= \mu_a - \mu_a N_a - \alpha_a I_a
\end{aligned} \tag{2}
$$

in the subset of R^4 given by

$$\Omega = \{0 \leq S_a, 0 \leq I_a, S_a + I_a \leq N_a \leq 1, 0 \leq I_v \leq 1\}.$$

Here $m = \dfrac{\bar{N}_v}{\Lambda_a/\mu_a}$ is the ratio between the mosquito and bird populations.

3. STEADY STATES OF THE MODEL

Suppose first $0 \leq p < 1$. In this case the steady states of equation (2) satisfy the following relations

$$
\begin{aligned}
\hat{S}_a &= \frac{\mu_a \hat{N}_a}{b\beta_a m \hat{I}_v + \mu_a \hat{N}_a} \\
\hat{I}_a &= \frac{\mu_a (1 - \hat{N}_a)}{\alpha_a} \\
\hat{I}_v &= \frac{b\beta_v \hat{I}_a}{b\beta_v \hat{I}_a + (1-p)\mu_v \hat{N}_a}.
\end{aligned} \tag{3}
$$

Substituting (3) in the corresponding equilibrium second equation of (2) it can be seen that $\hat{N}_a$ is a solution of the following equation

$$(1 - N_a)\, r(N_a) = 0 \tag{4}$$

where $r(N_a) = AN_a^2 + BN_a + C$ is a polynomial of second degree with coefficients

$$
\begin{aligned}
A &= \mu_a b\beta_v - \alpha_a (1-p)\mu_v, \\
B &= (1-p)\mu_v(\gamma_a + \mu_a + \alpha_a) R_0 - \mu_a b\beta_v, \\
C &= -(\gamma_a + \mu_a)(1-p)\mu_v R_0,
\end{aligned}
$$

and

$$R_0 = \frac{mb^2 \beta_a \beta_v}{(1-p)\mu_v(\gamma_a + \mu_a + \alpha_a)}. \tag{5}$$

The solution $\hat{N}_a = 1$ gives the disease free equilibrium point $\mathbf{P_0}$ whose coordinates are $\hat{S}_a = 1, \hat{I}_a = 0, \hat{I}_v = 0$, and $\hat{N}_a = 1$.

The nontrivial equilibrium solutions in the interior of Ω satisfy $0 < \hat{I}_a < \hat{N}_a < 1$. From the second equation of (3) it can be seen that this implies $\dfrac{\mu_a}{\mu_a + \alpha_a} < \hat{N}_a < 1$, therefore it is enough to restrict the searching to that interval. It is straightforward to confirm that $r(\dfrac{\mu_a}{\mu_a + \alpha_a}) < 0$ and

$$r(1) = (1-p)\mu_v(R_0 - 1).$$

When $R_0 = 1$, $N_a = 1$ is a root of of $r(N_a)$, and it can be seen that the other root is bigger than one. If $R_0 < 1$, the value of the polynomial $r(N_a)$ is negative at the endpoints of the interval. In this case, either there are two, one or none roots in such interval. The conditions for having one or two roots are a) $A < 0$; b) $\dfrac{\mu_a}{\mu_a + \alpha_a} < -\dfrac{B}{2A} < 1$; c) $B^2 - 4AC \geq 0$.

In this case b) and c) are not compatible, and therefore there are no roots in the mentioned interval.

If $R_0 > 1$ then $r(1) > 0$, therefore there exists a unique root in the interval $(\dfrac{\mu_a}{\mu_a + \alpha_a}, 1)$ which implies the existence of a unique equilibrium point $\mathbf{P_1} = (\hat{S}_a, \hat{I}_a, \hat{I}_v, \hat{N}_v)$ in the interior of Ω. $\mathbf{P_0}$ is the disease-free equilibrium, and $\mathbf{P_1}$ corresponds to the endemic value. Thus, it was proved that for $R_0 \leq 1$, $\mathbf{P_0}$ is the only equilibrium point in Ω, but in the case $R_0 > 1$ the endemic equlibrium $\mathbf{P_1}$ will also lie in Ω.

The quantity $\tilde{R}_0 = \sqrt{R_0}$ is called the *Basic Reproductive Number* of the disease, and it represents the average number of secondary cases that one infectious can produce if introduced into a susceptible population. This can be seen as follows: an infective bird introduced into the suceptible population is biten during his infective period by $mb/(\gamma_a + \mu_a + \alpha_a)$ mosquitoes; a proportion $m\beta_v b/(\gamma_a + \mu_a + \alpha_a)$ of these mosquitoes becomes infectious. Similarly, an infective mosquito distributes $b/(1-p)\mu_v$ bites in the bird population during the rest of its life and a proportion $\beta_a b/(1-p)\mu_v$ of these bites becomes new infections. Therefore, the geometric mean of these quantities, $\tilde{R}_0$, gives the number of secondary infections.

In the case $p = 1$ the equilibrium points of (2) are $\mathbf{P_0}$ and the solution of the equations:

$$
\begin{aligned}
\hat{S}_a &= \frac{\mu_a \bar{N}_a}{mb\beta_a N_v + \mu_a \bar{N}_a} \\
\hat{I}_a &= \frac{\mu_a (1 - \bar{N}_a)}{\alpha_a} \\
\hat{I}_v &= 1
\end{aligned} \tag{6}
$$

where $\hat{N}_a$ is a root of the equation

$$
\begin{aligned}
q(N_a) = \mu_a N_a^2 + (mb\beta_a - \mu_a) N_a - \\
mb\beta_a \frac{\gamma_a + \mu_a}{\gamma_a + \mu_a + \alpha_a} = 0
\end{aligned} \tag{7}
$$

in the interval $\left(\dfrac{\mu_a}{\alpha_a + \mu_a}, 1\right)$. Evaluating $q(N_a)$ at the endpoints,

$$
\begin{aligned}
q(\frac{\mu_a}{\alpha_a + \mu_a}) &< 0 \\
q(1) &= mb\beta_a \frac{\alpha_a}{\gamma_a + \mu_a + \alpha_a} > 0,
\end{aligned}
$$

then (7) always has a unique root $\hat{N}_a$. Therefore, the endemic equilibrium state $\mathbf{P_1}$ is in Ω independently of the values of the parameters. Notice that, as $p \to 1$, $R_0 \to \infty$.

4. STABILITY ANALYSIS

In this section the stability of the steady states of system (2) is investigated. Consider first the case $0 \leq p < 1$. The Jacobian $\mathbf{DF}(\mathbf{P_0})$ of Eq. (2) evaluated in the disease-free equilibrium is given by

$$\begin{pmatrix} -\mu_a & 0 & -mb\beta_a & 0 \\ 0 & -(\gamma_a+\mu_a+\alpha_a) & mb\beta_a & 0 \\ 0 & b\beta_v & -(1-p)\mu_v & 0 \\ 0 & -\alpha_a & 0 & -\mu_a \end{pmatrix}. \quad (8)$$

The eigenvalues of (8) 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 this case, both coefficients are positive if and only if $R_0 < 1$. Therefore, the disease-free equilibrium $\mathbf{P_0}$ is locally asymptotically stable for $R_0 < 1$, and unstable for $R_0 > 1$.

When $\alpha_a = 0$, global stability of $\mathbf{P_0}$ for $R_0 \leq 1$ can be proved. In this case $N_a(t) \to 1$, and system (2) becomes

$$\begin{aligned} S_a' &= \mu_a - mb\beta_a I_v S_a - \mu_a S_a \\ I_a' &= mb\beta_a I_v S_a - (\gamma_a+\mu_a) I_a \\ I_v' &= b\beta_v I_a (1-I_v) - (1-p)\mu_v I_v. \end{aligned} \quad (9)$$

Defining the following Lyapunov function in Ω

$$L = I_a + \frac{b\beta_a}{(1-p)\mu_v} I_v, \quad (10)$$

the orbital derivative of L is given by

$$\begin{aligned} \dot{L} = &-mb\beta_a(1-S_a)I_v - \\ &(\gamma_a+\mu_a)(1-R_0(1-I_v))I_a. \end{aligned} \quad (11)$$

which is less 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 (9) reduces to $S_a' = \mu_a - \mu_a S_a$, $I_a' = 0$, $I_v' = 0$. From these equations it can be seen that $S_a(t) \to 1$, $I_a(t) = 0$, $I_v(t) = 0$ for $t > 0$. Therefore, from LaSalle -Lyapunov theorem (Hale, 1969) it follows that $\mathbf{P_0}$ is locally stable and all trajectories starting in Ω approach $\mathbf{P_0}$ for $R_0 \leq 1$. Thus, the global asymptotical stability of $\mathbf{P_0}$ for $R_0 \leq 1$ and $\alpha_a = 0$ has been established.

When $R_0 > 1$, $\mathbf{P_0}$ becomes an unstable equilibrium point, and the endemic equilibrium $\mathbf{P_1}$ emerges in Ω. The local stability of this point is governed by the Jacobian $\mathbf{DF}(\mathbf{P_1})$. From the equations of system (2) in equilibrium the following relations are obtained

$$\begin{aligned} \frac{mb\beta_a \hat{I}_v}{\hat{N}_a} + \mu_a &= \frac{\mu_a}{\hat{S}_a}, \\ \gamma_a + \mu_a + \alpha_a &= \frac{mb\beta_a \hat{I}_v S_a}{\hat{I}_a \hat{N}_a}, \\ \frac{b\beta_v \hat{I}_a}{\hat{N}_a} + (1-p)\mu_v &= \frac{b\beta_v \hat{I}_a}{\hat{I}_v \hat{N}_a}, \\ \mu_a &= \mu_a \hat{N}_a + \alpha_a \hat{I}_a, \end{aligned} \quad (12)$$

and $DF(\mathbf{P_1})$ can be written as the matrix

$$\begin{pmatrix} -\frac{\mu_a}{\hat{S}_a} & 0 & -\frac{mb\beta_a \hat{S}_a}{\hat{N}_a} & \frac{mb\beta_a \hat{I}_v \hat{S}_a}{\hat{N}_a^2} \\ \frac{mb\beta_a \hat{I}_v}{\hat{N}_a} & -\frac{mb\beta_a \hat{I}_v \hat{S}_a}{\hat{I}_a \hat{N}_a} & \frac{mb\beta_a \hat{S}_a}{\hat{N}_a} & -\frac{mb\beta_a \hat{I}_v \hat{S}_a}{\hat{N}_a^2} \\ 0 & \frac{b\beta_v(1-\hat{I}_v)}{\hat{N}_a} & -\frac{b\beta_v \hat{I}_a N_v}{\hat{I}_v \hat{N}_a} & -\frac{b\beta_v \hat{I}_a (1-\hat{I}_v)}{\hat{N}_a^2} \\ 0 & -\alpha_a & 0 & -\mu_a \end{pmatrix},$$

whose eigenvalues are $-\mu_a$ and the roots of the polynomial $\lambda^3 + P\lambda^2 + Q\lambda + R$, where

$$\begin{aligned} P &= \frac{\mu_a}{\hat{S}_a} + \frac{mb\beta_a \hat{I}_v \hat{S}_a}{\hat{I}_a \hat{N}_a} + \frac{b\beta_v \hat{I}_a}{\hat{I}_v \hat{N}_a} \\ Q &= \frac{\mu_a b\beta_v \hat{I}_a}{\hat{S}_a \hat{I}_v \hat{N}_a} + \frac{mb^2 \beta_a \beta_v \hat{S}_a \hat{I}_v}{\hat{N}_a^2} + \\ &\quad \frac{mb\beta_a \hat{I}_v (\mu_a \hat{N}_a - \alpha_a \hat{I}_a \hat{S}_a)}{\hat{I}_a \hat{N}_a^2} \\ R &= \frac{\mu_a mb^2 \beta_a \beta_v ((\hat{N}_a - \hat{S}_a) + \hat{S}_a \hat{I}_v)}{\hat{N}_a^3} - \\ &\quad \frac{\alpha_a mb^2 \beta_a \beta_v \hat{S}_a \hat{I}_a}{\hat{N}_a^3}. \end{aligned} \quad (13)$$

By the Routh-Hurwitz criterion, it follows that all roots of the above polynomial have negative real parts if and only if $P > 0$, $Q > 0$, and $PQ > R$. It is clear that $P > 0$. From the inequalities $\hat{S}_a < \hat{N}_a$ and $\alpha_a \hat{I}_a < \mu_a$, $Q > 0$. Now, using the relation $\hat{N}_a - \hat{S}_a = \hat{I}_a + \hat{R}_a = \frac{(\gamma_a+\mu_a)}{\mu_a}\hat{I}_a$,

$$\begin{aligned} R &= \frac{mb^2 \beta_a \beta_v \hat{I}_a ((\gamma_a+\mu_a) - \alpha_a \hat{S}_a)}{\hat{N}_a^3} + \\ &\quad \frac{\mu_a mb^2 \beta_a \beta_v \hat{S}_a \hat{I}_v}{\hat{N}_a^3}. \end{aligned}$$

Since $\hat{S}_a < 1$ and $\alpha_a \leq \gamma_a + \mu_a$ then $R > 0$. Finally, the inequality $PQ > R$ can be proved easily. Thus, it has been proved that $\mathbf{P_1}$ is locally asymptotically stable.

Now, the stability of the equilibria in case $p = 1$ is analyzed. The eigenvalues of the Jacobian

around the disease-free equilibrium $\mathbf{P_0}$ are $-\mu_a$ of multiplicty two, and the roots of the equation

$$\lambda^2 + (\gamma_a + \mu_a + \alpha_a)\lambda - mb^2\beta_a\beta_v = 0.$$

Since the last coefficient of this equation is negative, $\mathbf{P_0}$ is always unstable. On the other hand, for the endemic equilibrium $\mathbf{P_1}$, the eigenvalues are $-\mu_a$, $-\frac{b\beta_v\hat{I}_a}{\hat{N}_a}$, and the roots of the polynomial $\lambda^2 + (\frac{mb\beta_a}{\hat{N}_a} + \gamma_a + 2\mu_a + \alpha_a)\lambda + (\gamma_a + \mu_a + \alpha_a)(\frac{mb\beta_a}{\hat{N}_a}+\mu_a)+\alpha_a\frac{mb\beta_a\hat{S}_a}{\hat{N}_a^2}$, which have negative real part. Therefore $\mathbf{P_1}$ 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 $\mathbf{P_0}$ is locally asymptotically stable for $R_0 < 1$. When $R_0 > 1$, $\mathbf{P_0}$ becomes unstable, and the endemic equilibrium $\mathbf{P_1}$ is locally asymptotically stable. If $p = 1$, $\mathbf{P_0}$ is always unstable, and $\mathbf{P_1}$ is locally asymptotically stable.*

5. NUMERICAL RESULTS

To evaluate the transmission dynamics, Komer et al. (2003) exposed 25 bird species to WNV by infectious bite of *Culex tritaeniorhynchus*. They analyzed viremia data to determine values for susceptibility (s), mean daily infectiousness (i), duration of infectious viremia (d), and competence index $c_j = s \times i \times d$, for each species, where susceptibility is the proportion of the birds that become infected as a result of the exposure; mean daily infectiousness is the proportion of exposed mosquitoes 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 transmissor. Table 1 shows for eight species the values of s, i, d and c_j obtained in (Komar et al, 2003). 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 the daily disease mortality rate α_a was calculated as the proportion of deaths divided by the mean number of days to death.

The lifespan of birds is difficult to estimate. Values reported range from three to ten years. Here, it is assumed an average lifespan of 6 years for all species which gives a mean value $\mu_a = .0004$. Since the time course of the infection is of a few days, the effect of this approximation on R_0 is negligible. Typical values of the biting rate b are once every two or three days, here $b = 0.5$. The reported values (Gubler, 1986) for the lifespan of mosquitoes vary from weeks to months. An average value is two or three weeks for females (Gubler, 1986) which gives $\mu_v = 0.06$.

Table 1. Competence index and $\tilde{R}_0$

Name	s	i	d	α_a	c_j	$\tilde{R}_0$
Blue Jay	1	.68	3.75	.15	2.55	2.62
Common Gackle	1	.68	3	.07	2.04	2.66
House Finch	1	.32	5.5	.14	1.76	2.04
American Crow	1	.5	3.25	.19	1.62	2.04
House Sparrow	1	.53	3	.1	1.59	2.26
Ring-billed Gull	1	.28	4.5	.1	1.26	1.9
Black-billed Magpie	1	.36	3	.16	1.08	1.74
Fish Crow	1	.26	2.8	.06	0.73	1.60

Using the values of the parameters above and the ones in Table 1, $\tilde{R}_0$ was estimated for each bird species. It is assumed in all of the cases, that the ratio $m = \frac{N_v}{\Lambda_a/\mu_a} = 1$, and that the probability of vertical transmission $p = 0$.

According to Table 1, American Crow and House Finch are more competent than House Sparrow, however the number of secondary infections produced by individuals of those species is less than the corresponding number produced by House Sparrow birds. The same phenomenum is observed between Blue Jay and Common Grackle.

Notice that disease mortality rates of American Crow, House Finch and Blue Jay are significatively greater than the corresponding ones for House Sparrow, and 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, high disease mortality is likely to diminish the efficiency of a species as a transmissor. This suggests that $\tilde{R}_0$ is a better measure of the epidemiological importance of a given species.

Figure 1 illustrates the time course of the infected bird proportion for Blue Jay, American Crow

and House Sparrow. In this figure only the first epidemic peak is presented.

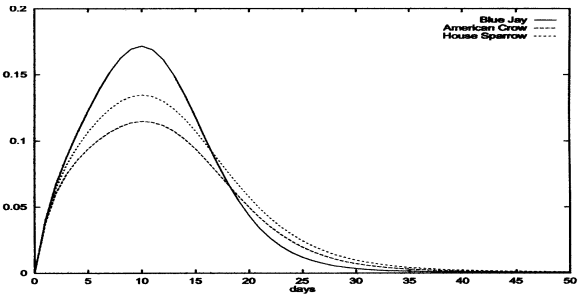

Fig. 1. Temporal course of the proportion of infectious birds.

6. CONCLUSIONS

The dramatic appearance of the epidemic of WNV in the northeast part of United States, is a reminder of the ability of virus to jump continents. The subsequent spread of WNV shows that arboviral virus that are introduced to new areas can become established if efficient mosquitoes and hosts are available. A condition for the maintenance of the disease when the virus is introduced in a certain region were obtained, finding 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 this disease. Then, if $\tilde{R}_0$ is less than one the disease will fade out since an infective will replace itself with less than one new infective. On the other hand, if $\tilde{R}_0$ is grater than one, the infected fraction of the mosquito and bird populations will tend to an endemic steady state.

R_0 is linear with respect the ratio, m, between mosquito and bird population. Then, disease spread is likely to be greater when birds migrates to regions with high density of mosquitos. Vertical transmission in the mosquito population is also a risk factor for WVN. The model predicts that if the probability of vertical transmission is sufficiently high, the disease can be maintained all of the time, even in regions with scarce bird population.

The effect of the disease on the population dynamics of bird population is simple and direct. When the disease remains endemic, the disease-related deaths decrease the population size. Low values of α_a would have a small effect in the population size. Since R_0 decreases when α_a increases, high values of α_a will cause the disease to die out, and eventually the population size will return to its original values. Intermediate values of α_a are the ones than can cause more damage to the population.

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