

A Theoretical Framework for the Analysis of the West Nile Virus Epidemic

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We present a model for the growth of West Nile virus in mosquito and bird populations based on observations of the initial epidemic in the U.S. Increase of bird mortality as a result of infection, which is a feature of the epidemic, is found to yield an effect which is observable in principle, viz., periodic variations in the extent of infection. The vast difference between mosquito and bird lifespans, another peculiarity of the system, is shown to lead to interesting consequences regarding delay in the onset of the steady-state infection. An outline of a framework is provided to treat mosquito diffusion and bird migration.

Keywords: Mathematical Epidemiology; West Nile Virus; Spread of an Infection

I. INTRODUCTION

Mathematical modeling of the spread of epidemics poses intriguing challenges and promises useful insights and possibly predictive capabilities. Recent work by some of the present coauthors [1, 2, 3] has led to the understanding of observed features, particularly spatiotemporal patterns, in the Hantavirus infection [4]. It is the purpose of the present paper to initiate a formalism for the understanding of the West Nile virus epidemic, which bears some similarities, but possesses some distinguishing characteristics, relative to the Hantavirus. The paper is laid out as follows. In the rest of the section we describe some essential characteristics of the West Nile virus epidemic and comment on how they may be folded into a model of differential equations similar to the Abramson-Kenkre (AK) model of the Hantavirus [1]. In Section II, we modify the Hantavirus model equations to incorporate cross-infection of two taxa, a characteristic of the West Nile virus epidemic. We comment on general features expected on the grounds of simple intuition based on nonlinear dynamics. In Section III, we augment the generalized cross-infection model to include three realistic features of the West Nile virus epidemic and present scenarios for time evolution of the populations of mosquitoes and birds, the two taxa which appear central to the West Nile virus problem. The present paucity of field data prevents us from attempting to explain specific observations. However, interesting predictions of the ‘what-if’ type are possible as will be seen below. Concluding remarks form Section IV.

West Nile virus is a mosquito-borne virus that infects primarily birds, but also a wide range of other species, including horses, dogs and cats, and occasionally humans. The first outbreak of West Nile virus encephalitis on the North American continent occurred in New York in 1999. Successive outbreaks in humans have occurred annually in the USA since then. West Nile virus is fatal in many species of birds, and is sometimes fatal in humans. It is unusual among mosquito-borne diseases in that “vertical transmission”, where the virus is passed from the mother to her eggs, may occur in the wild. This has potentially serious consequences, because once an area is infected it may remain so indefinitely, because the virus may survive the winter in infected mosquito larvae and reemerge to infect human and animal populations in the spring [5].

Previous work has shown that the virus travels along watershed areas through avian and mosquito host populations [6]. Extensive field studies have led to attention being focused on birds as well as mosquitoes in the dynamics of the West Nile virus epidemic. During their migration, infected birds arrive at a location and transmit the virus to female mosquitoes that feed upon them. The mosquitoes in turn transmit the virus to other birds, not originally infected, and to other animals including horses and humans. Collection of field data consists, therefore, of testing mosquitoes and birds. Mosquitoes are trapped with CO₂-releasing boxes with organic-rich water at their base. In addition, surveillance systems for reporting dead birds and testing them for infection, as well as trapping live birds and testing them for seroconversion (a symptom of recent West Nile virus infection) are in place in centers of infection [7, 8].

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The model that we develop for studies of the West Nile virus epidemic is similar to the AK analysis of the Hantavirus [1] but incorporates the above as important additional features. From the point of view of modeling, three additions to the AK model are crucial. The first is that there are *two* taxa in the West Nile virus system (mosquitoes and birds, as compared to the single rodent species in the Hantavirus), that cross-infect each other. The second is that these two taxa have vastly different (natural) lifespans: the characteristic times are on the order of a few weeks for mosquitoes but on the order of a year or two for birds. The third is that while mice are never born infected with Hantavirus, mosquitoes may be hatched West Nile virus; and while mice do not die from hanta infection, birds often do die from West Nile virus infection. The first addition is treated in Section II where we find obtain the generalized condition for steady state infection to exist for a system with two populations. The second addition is shown in Section III to lead to interesting consequences in the onset of the steady-state infection. The treatment of the third addition, also in Section III, shows that the increase in bird mortality due to infection can lead to oscillations in infected populations.

II. GENERALIZATION OF THE HANTAVIRUS MODEL TO INCLUDE CROSS-INFECTON

A. Recasting the Hantavirus Equations

The AK model equations, at the mean-field level (at which diffusion is not shown explicitly), are

$$\frac{dm_s}{dt} = b m - c m_s - \frac{m_s m}{K} - a m_s m_i, \quad (1a)$$

$$\frac{dm_i}{dt} = -c m_i - \frac{m_i m}{K} + a m_s m_i, \quad (1b)$$

where the subscripts i and s refer to infected and susceptible animals (in this case, mice) respectively, $m = m_s + m_i$ is the total population, b the birth rate, c the natural death rate, K the environmental parameter, and a is the transmission rate responsible for infection.

The fact, well-known to Hantavirus biologists [9, 10], that infection does not affect the lifespan of the infected mice, is naturally reflected in the mathematical observation that the total population m is independent of all information regarding the infection process. The total population m obeys a logistic equation, whose solution is known. Noticing this, the solution of system (1) can be easily obtained analytically as shown by one of the present authors in Ref. [11].

This suggests that we first recast the Abramson-Kenkre (AK) equations (1), changing the variables m_i and m_s to the total population m and the infected fraction $\chi \equiv m_i/m$. It is straightforward to write

$$\frac{dm}{dt} = (b - c) m - \frac{m^2}{K}, \quad (2a)$$

$$\frac{d\chi}{dt} = -b \chi + a m \chi (1 - \chi). \quad (2b)$$

The first of these equations merely describes the logistic evolution of the total population. The second equation has an interesting structure. The two terms on the right side have opposite signs. The first term, $-b\chi$, plays a role against infection because birth of new individuals always decreases the infected fraction χ : the offspring are always susceptible (not infected). The second term, $am\chi(1-\chi)$, represents the flux of individuals from susceptible to infected. This flux occurs as a result of transmission of infection between a susceptible individual and an infected one by direct contact. The transmission is represented by the product of the infected fraction, the susceptible fraction, and, of course, the total population.

The system (2) has four equilibria. Two of them are irrelevant because one is the null state and the other has negative population for all values of the parameters. With the help of a linear stability analysis [12, 13] of the equilibria, it can be shown that the other two equilibria interchange their stability character at a critical value of the parameter set. The state with infection different from zero ($\chi > 0$) is stable only if, as given in Ref.[1],

$$K(b - c) > b/a. \quad (3)$$

If this condition is not fulfilled, then the steady state has no infection, i.e. $\chi = 0$. Equation (2b) also suggests an intuitive graphical procedure to ascertain the presence and magnitude of the infection. A plot of the χ -dependence of each of the terms, $b\chi$ (straight line), and $am\chi(1-\chi)$ (inverted shifted parabola) indicates in the steady state the presence (absence) of infection if the two curves do (do not) intersect each other at a χ value other than 0. See Fig. 1. As $m(t)$ evolves in time via its logistic equation, the term $am\chi(1-\chi)$ changes. If its initial and final values are small and large respectively, the nontrivial intersection of the curves (nonzero χ) will be absent at first, but present later

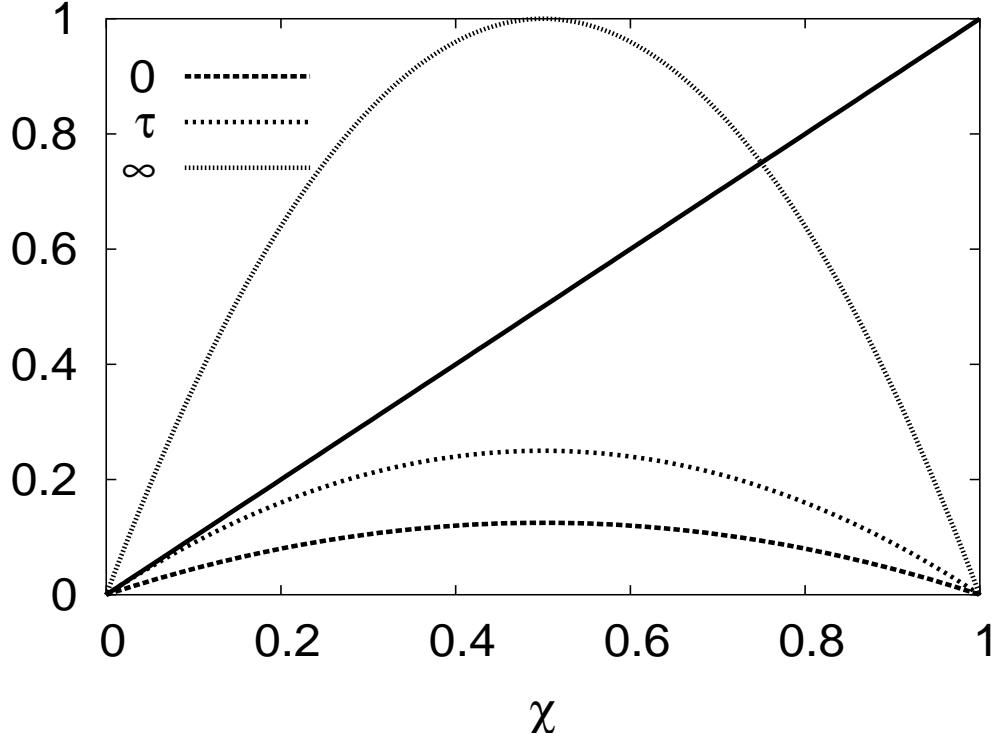


FIG. 1: Balance between the two terms in the infection evolution. The straight line has slope b and represents the decrease of the infected fraction due to birth of susceptible individuals. The parabolas represent the transfer of infection and their strength is proportional to the total population m . Three cases are shown. The lowermost parabola describes the initial time $t = 0$ at which no nonzero intersection exists. The topmost parabola describes the eventual situation at $t = \infty$, i.e., the steady state. The central parabola for which the straight line is a tangent describes the time at which the nontrivial intersection appears. This is the time τ at which the infection turns from its tendency to vanish and begins to rise to the nonzero steady state value.

on. Fig. 1 shows such a case. We have labeled the initial and final situations by 0 and ∞ respectively, and the critical situation, wherein the nontrivial intersections just begins to appear, by the time τ taken for it to occur. The time dependence of the infected fraction and the total population is plotted in Fig. 2. We notice the clear tendency of the infection first to disappear (corresponding to the fact that the nontrivial intersection does not yet exist in Fig. 1) followed by evolution to the eventual steady-state value (corresponding to the intersection with the top curve in Fig. 1).

The delay τ in the onset of infection is plotted in Fig. 3 versus the dimensionless ratio of the two rates that enter into the balance as clear from equation (2b). An initial increase, a point of inflection, and an eventual blow-up at the point the rate ratio equals 1, are to be noted in the τ curve. The blow-up signals that the nontrivial intersection is always absent: the steady-state infection vanishes.

B. Incorporating cross-infection

Unlike with the Hantavirus, the spread of the West Nile virus requires the presence of both mosquitoes and birds: the West Nile virus is transmitted through cross-infection. This means that an infected individual infects a susceptible individual of the *other* taxon. A mosquito infects a bird and vice-versa.

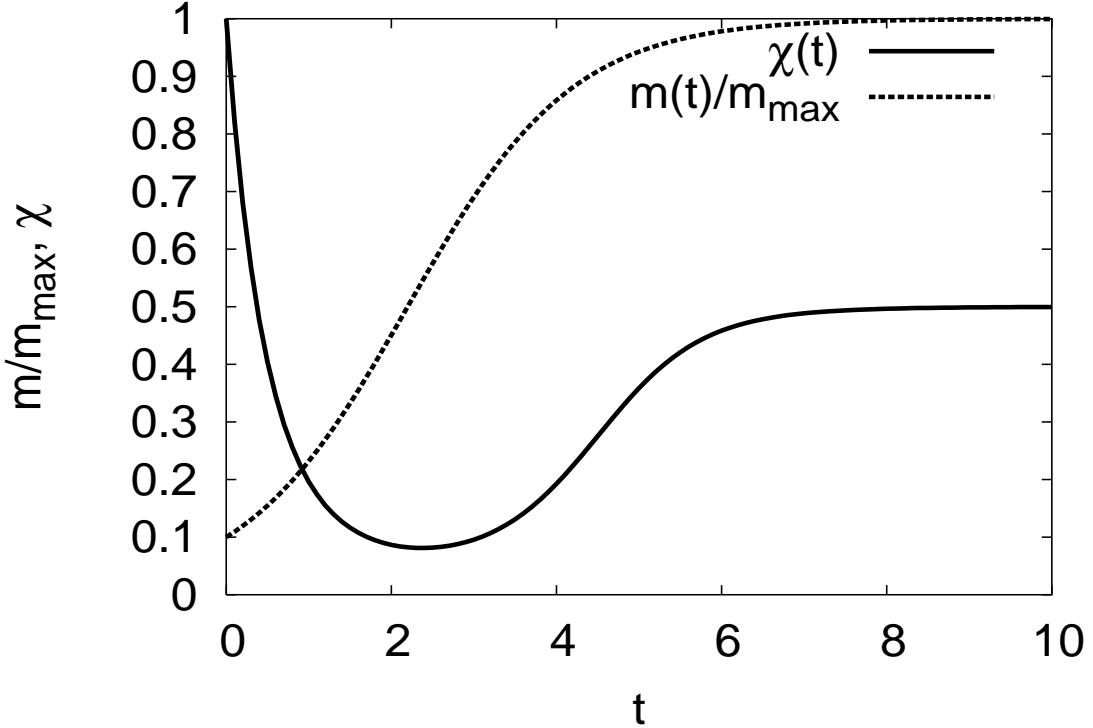


FIG. 2: Time dependence of the infected fraction χ and the total population m corresponding to the situations depicted in Fig. 1. Parameters and initial conditions are arbitrary: $a = 4, b = 2, c = 1, K = 1, m(0) = 0.1$ and $\chi(0) = 1$. We see that χ first tends to vanish and then turns to its eventual steady state value. Time t is plotted in units of $\frac{1}{b-c}$.

Therefore, in place of the AK equations (1), we write

$$\frac{dm_s}{dt} = b m - c m_s - \frac{m_s m}{K} - a m_s A_i, \quad (4a)$$

$$\frac{dm_i}{dt} = -c m_i - \frac{m_i m}{K} + a m_s A_i, \quad (4b)$$

$$\frac{dA_s}{dt} = \beta A - \gamma A_s - \frac{A_s A}{\kappa} - \alpha A_s m_i, \quad (4c)$$

$$\frac{dA_i}{dt} = -\gamma A_i - \frac{A_i A}{\kappa} + \alpha A_s m_i, \quad (4d)$$

where the subscripts i and s refer, as before, to the infected and susceptible state respectively. The symbol m now represents mosquitoes rather than mice. The symbol A (after Latin *avis*) represents birds with β as the birth rate, γ as the death rate, and κ as the environmental parameter. The cross-infection rates are a and α . Equations (4) are formally symmetric in the two taxa. In each case, infected as well as susceptible individuals breed susceptible individuals of their own species. Also, in each case, infected individuals infect susceptible members of the *other* species. In addition, each species has its own vital dynamics—each modeled by a logistic equation—via its own birth rate, death rate, and environmental parameter. For the sake of explanation let us call one of the taxa (i.e., the birds) the host population and the other (i.e., the mosquitoes) the vector. An infected individual of the host population, A_i , transmits the disease to a susceptible member of the vector taxon, m_s . This member becomes infected, increasing the infected population of the vector taxon, m_i . Only then is this newly infected individual able to transmit the disease to a susceptible member of the original host population, A_s . As a result of the last interaction, an individual of the host species will finally join the infected population A_i . There is thus an underlying cyclic process. In this way, the infection process can be thought of in two stages. One is the acquisition of the infection by the ‘vector’. The other is the transmission to the ‘host’ population. Each of these requires direct contact between transmitter and receptor. Therefore, the magnitude of each infection process depends on the number of receptors, the number of transmitters, and the respective infection rates.

How is the equation set (2) augmented by the incorporation of cross-infection? To answer this we rewrite equations

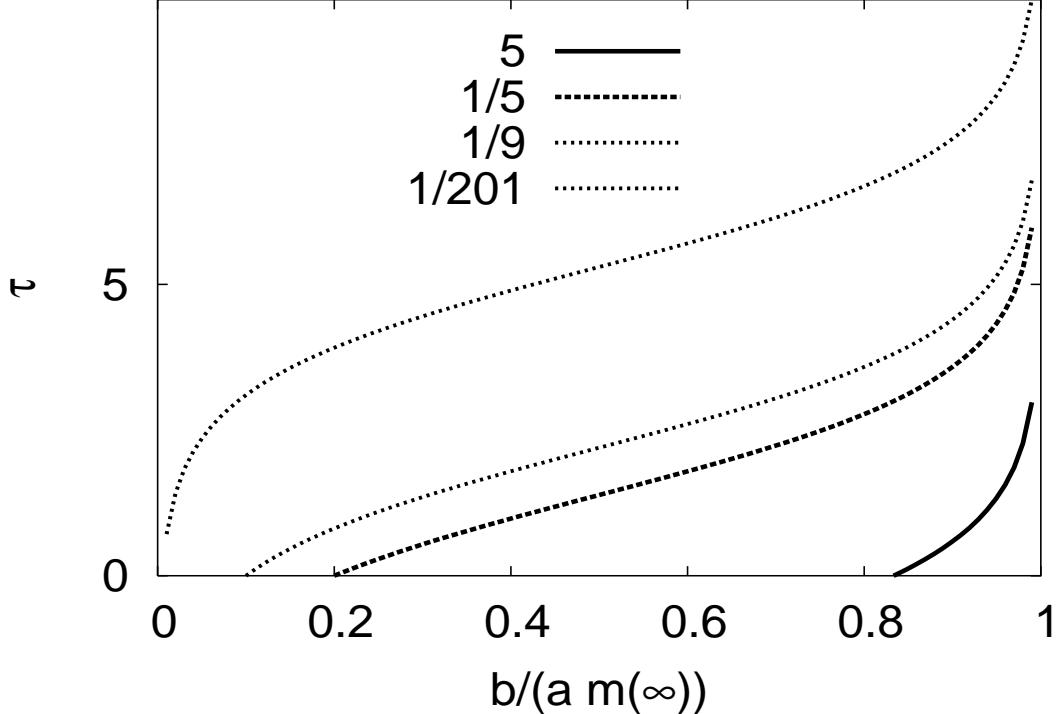


FIG. 3: The delay time τ taken by the infected fraction to change its vanishing tendency and turn towards the nonzero steady state value plotted as a function of the ratio of the opposing rates b and $am(\infty)$. In this simple case τ is given by the analytic expression $(b - c)\tau = \ln\left(\frac{1-m(\infty)/m(0)}{1-m(\infty)/m_c}\right)$ where $m_c = b/a$ is the critical carrying capacity. To be noted are the initial rise of τ , the point of inflection and the blow up at the right extreme which signifies that a nonzero infected fraction cannot be supported at higher values of the abscissa. The delay time is plotted in units of $\frac{1}{b-c}$.

(4) in terms of total populations and infected fractions

$$\frac{dm}{dt} = (b - c)m - \frac{m^2}{K}, \quad (5a)$$

$$\frac{d\chi_m}{dt} = -b\chi_m + aA\chi_A(1 - \chi_m), \quad (5b)$$

$$\frac{dA}{dt} = (\beta - \gamma)A - \frac{A^2}{\kappa}, \quad (5c)$$

$$\frac{d\chi_A}{dt} = -\beta\chi_A + \alpha m\chi_m(1 - \chi_A). \quad (5d)$$

We see that the evolution of the total population of the mosquitoes, $m = m_i + m_s$, and of the birds, $A = A_i + A_s$, is formally unchanged. The respective infected fractions $\chi_m = m_i/m$ and $\chi_A = A_i/A$ clearly show the effect of cross-infection. As in equations (2), we see that the infected fraction of either taxon decreases as the result of births in that taxon because only susceptible individuals are born (represented by the terms $-b\chi_m$ and $-\beta\chi_A$ in (5b) and (5d) respectively).

Linear stability analysis [12, 13] of (5) along the lines of (1) shows that the equilibria when infection is different from zero ($\chi_m \neq 0, \chi_A \neq 0$) are stable only when

$$K(b - c)\kappa(\beta - \gamma) > (b/a)(\beta/\alpha). \quad (6)$$

Equation (6) represents a generalization of (3) to the cross-infection case. Threshold values for infection survival depend on products of quantities characteristic of the two taxa.

III. INCORPORATING REALISTIC FEATURES OF THE WEST NILE VIRUS

The preceding analysis has focused on the consequence of replacing same-taxon infection by cross-infection typical of West Nile virus and has relied on a highly simplified and symmetrical model. We now include three realistic features of the West Nile virus: (i) the possibility of vertical transmission in mosquitoes, (ii) the possibility of infection-caused mortality of birds, and (iii) time scale disparity between mosquitoes and birds.

A. Partial Heritage of the Infection: Vertical Transmission

Vertical transmission has been strongly suspected in the West Nile virus [5]. By this term is meant the passage of virus from infected individuals to their offspring via the process of birth. Therefore, we now consider that some of the offspring of infected mosquitoes are infected during the formation of eggs. We take the rate of mosquitoes being “born” (hatched) already infected as b_i . Infected mosquitoes can only be born from infected mosquitoes. The total rate of mosquitoes born from infected ones is still b . To incorporate this effect, we subtract the term $b_i m_i$ from equation (4a) and add it to the right-hand side of (4b).

While vertical transmission could lead to the survival of infection within mosquito larvae and reemerge in the spring, within the framework of equations we have adopted here, this modification has no important qualitative effect if $b_i \neq b$, i.e., if not all offspring of infected mosquitoes are hatched infected. It only changes the critical values and the evolution times in a straightforward way. Linear stability analysis [12, 13] shows that now the condition for the steady infected state is

$$K(b - c)\kappa(\beta - \gamma) > \left(\frac{b - b_i}{a}\right) \left(\frac{\beta}{\alpha}\right). \quad (7)$$

The asymmetry between the susceptible and infected populations, which favors the former as in Eq. (6), still holds provided $b_i \neq b$. The modification in the bifurcation point is changed only quantitatively.

B. Mortality Increase due to Infection

By contrast to vertical transmission discussed above, the increase of the mortality rate in the bird population due to infection can have substantial consequences within our analytical framework. In addition to the vertical transmission modifications involving b_i , we now replace γA_i in equation (4d) by $(\gamma + \delta)A_i$ where δ represents the infection-based contribution to the bird mortality rate. The generalization of equations (5) is now

$$\frac{dm}{dt} = (b - c)m - \frac{m^2}{K}, \quad (8a)$$

$$\frac{d\chi_m}{dt} = -(b - b_i)\chi_m + aA\chi_A(1 - \chi_m), \quad (8b)$$

$$\frac{dA}{dt} = (\beta - \gamma - \delta\chi_A)A - \frac{A^2}{\kappa} \quad (8c)$$

$$\frac{d\chi_A}{dt} = -\beta\chi_A + (\alpha m\chi_m - \delta\chi_A)(1 - \chi_A). \quad (8d)$$

The condition for the stability of the state with nonzero infection is,

$$K(b - c)\kappa(\beta - \gamma) > \left(\frac{b - b_i}{a}\right) \left(\frac{\beta + \delta}{\alpha}\right). \quad (9)$$

The difference with the previous condition (7) for linear stability of the infected state is the replacement of β in the right side by $\beta + \delta$. This result may appear puzzling. That a death rate contribution δ should *add* to, rather than subtract from, the birth rate β may look counterintuitive. However, it arises simply from the fact that the relative increase of the number of susceptible over infected birds is a consequence of increased infected mortality as well as of the birth process for susceptible birds.

A noteworthy outcome of the increase of mortality in birds due to infection is damped oscillations in A, χ_m, χ_A as they approach their steady values. The mosquito population m is not affected. We show in Fig. 4 this damped approach to equilibrium of the infected fractions χ_m and χ_A .

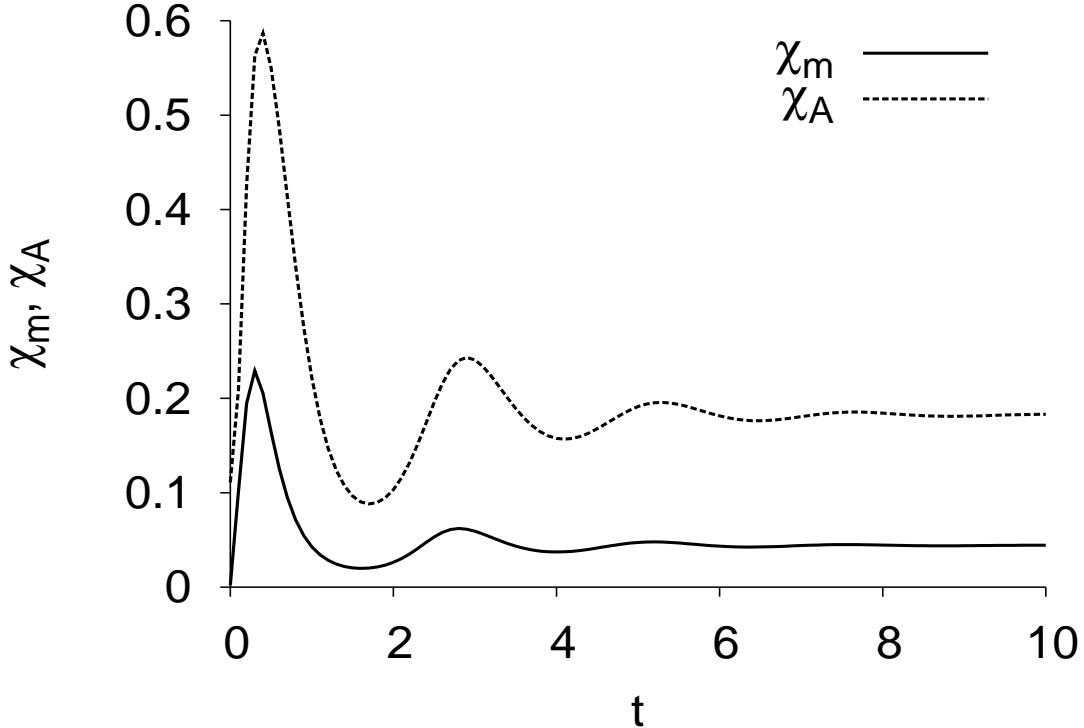


FIG. 4: Damped oscillations in the infected fractions during their approach to steady state, arising from the presence of the extra bird mortality (the additional death rate is δ) from infection. Time is plotted in units of $1/(b - c)$. Parameters are arbitrary: $b = 20, c = 19, b_i = 0, a = 0.25, \beta = 5, \gamma = 3.5, \alpha = 1, \delta = 6, K = 50, \kappa = 50$

The damped oscillations behavior does not require cross-infection as long as the extra death rate of infected individuals is present. Indeed, the phenomenon can be understood more easily in the simpler case of direct infection. Consider for this case

$$\frac{dA}{dt} = (\beta - \gamma - \delta\chi) A - \frac{A^2}{\kappa} \quad (10a)$$

$$\frac{d\chi}{dt} = -\beta\chi + (\alpha A - \delta)\chi(1 - \chi). \quad (10b)$$

Whereas the coefficient of the negative term $-\beta\chi$ in equation (10b) is fixed for all time t , the transmission term is time dependent because of the time dependence of A . That dependence is influenced, as (10a) shows, by the evolution of the infected fraction χ through the extra death rate via the term $(-\delta\chi A)$. Suppose that we are in a state in which the infected population is growing. While the amount of infected individuals increases, the number of deaths in population A increases. Thus, A decreases. If the decay of the value of the total population A is such that $(\alpha A - \delta) < \beta$ at some moment, the system will be in a situation wherein the infection is more likely to disappear than not. So, the infection decreases and the population recovers. This might be repeated several times until the system reaches the steady state. If, however, the extra death rate is high enough, oscillations might be completely suppressed and the infection may go directly to a steady state or disappear entirely.

The oscillations arise, thus, from the need of the presence of infection from transmission to occur, combined with the decrease in the population due to mortality from infection. This combination has another effect: the extra death rate parameter δ has an optimal value (see Fig. 5). If δ is too large, the infection is eliminated completely because the indispensable elements for transmission (infected individuals) are killed very quickly (see Fig. 5 B). Continuity of the infection requires a value for δ which provides the proper balance between the infection process and the death of infected birds.

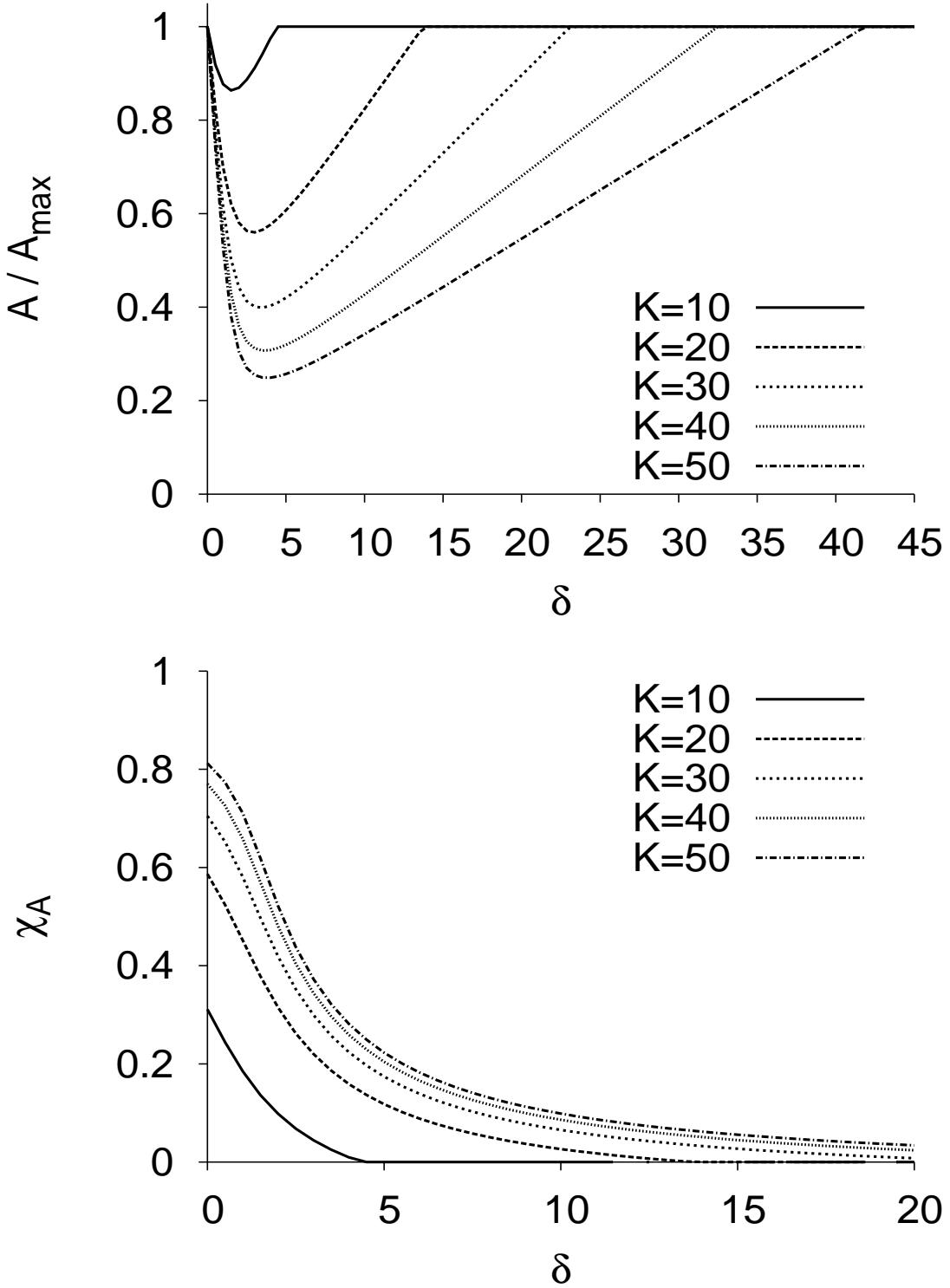


FIG. 5: Effect of the increased mortality rate δ on the steady state values of the total bird population and the infected fraction. The total population is expressed relative to the carrying capacity A_{\max} while the increased mortality rate δ is expressed relative to the rate difference $b - c$. Parameters are arbitrary: $b = 20, c = 19, b_i = 0, a = 0.25, \beta = 5, \gamma = 3.5, \alpha = 1, \kappa = 50$. The dependence of the infected fraction is monotonic while that of the total population has a minimum.

C. Time Scale Disparity

A peculiarity of the West Nile virus epidemic is that processes involving mosquitoes and birds occur on quite different time scales. The natural lifespan of a mosquito is not generally longer than a month while that of a bird might be several years. The birth and death rates of one taxon are thus very different from those of the other taxon. In order to understand what effect this might produce on the dynamics of infection, we first make the simple assumption that the corresponding rates for the two taxa differ by a factor ξ , i.e.,

$$\xi = \frac{b}{\beta} = \frac{c}{\gamma}. \quad (11)$$

We have followed several time scale separation schemes based on recasting the terms appearing in the evolution equations into those divided by ξ and those independent of ξ . For large time scale disparity, the former drop out of the evolution effectively. Our studies of time scale disparity along these lines are of interest to the nonlinear dynamics of the system but appear so far to be of much less value to the understanding of the epidemic. Therefore, we display here only the temporal evolution for two different values of ξ (see Fig. 6), and the corresponding interesting nonlinear dependence of the delay τ in the onset of steady state infection on ξ (see Fig. 7).

Figure 7 is the generalization of Fig. 3 to the cross-infection situation present in the West Nile virus epidemic. If the condition (6) is fulfilled, the variables $\chi_{m,A}$ attain their limiting nonzero values as $t \rightarrow +\infty$. However, as seen in Fig. 6, for some initial amount of time, they could appear to be attracted to a state with no infection. A measure of this transient time can be estimated by noticing that the condition (6) for the stability of the state with infection can be written as

$$\lim_{t \rightarrow \infty} A(t)m(t) > (b/a)(\beta/\alpha), \quad (12)$$

and asking for the value τ of the time t at which this condition starts to be satisfied by the system. In other words, a measure of the delay τ can be obtained from

$$A(\tau)m(\tau) = (b/a)(\beta/\alpha). \quad (13)$$

Because the functions $A(t)$ and $m(t)$ are known solutions of the logistic equation, we can solve this equation numerically. In this way, we can obtain the delay time τ as a function of ξ for any initial conditions and parameter values. An example of the dependence of the delay time τ on the time scale disparity factor ξ is shown in Fig. 7. In changing ξ we keep the bird parameters constant and change only the mosquito rates. Additionally, the environmental parameter of the mosquitoes, K , is reduced by the same factor as ξ is increased, in order to keep the carrying capacity $K(b - c)$ constant.

The value of τ plotted in Fig. 3 is also obtained in this fashion although only a single population (ratio) enters in that case into the left (right) hand side of the criterion equation. The blow-up feature at the limiting values of the rate ratio is common to both the direct and the cross infection cases (Fig. 3 and Fig. 7). The strong non-monotonic behaviour of τ in the cross-infection case is interesting and merits further study.

Further time scale disparity conclusions cannot be drawn until the relative values of the cross-infection parameters are known. Thus, it is possible to recast the equations of the mosquito and bird populations in dimensionless form to make clear the time scale disparity obvious from the numerical solutions displayed in the figures. Defining $\mu = m/m_{max}$ and $\mathcal{A} = A/A_{max}$, where m_{max} is the mosquito carrying capacity $K(b - c)$ and A_{max} is the bird carrying capacity $\kappa(\beta - \gamma)$, evolution equations for the total populations in terms of the dimensionless time $t' = t(b - c)$ take the form

$$\frac{d\mu}{dt'} = \mu - \mu^2 \quad (14a)$$

$$\frac{d\mathcal{A}}{dt'} = \frac{1}{\xi}(\mathcal{A} - \mathcal{A}^2) \quad (14b)$$

The time rate of change of the bird population is clearly slower than of the mosquito population by the disparity factor. This agrees with the quick rise of the mosquito population displayed in the figures. However, whether the infection ratios change on the same or disparate time scales depends on the relative values of the parameters $\frac{aA_{max}}{b-c}$ and $\frac{am_{max}}{\beta-\gamma}$. If they are of the same order as each other, bird infection will involve on a slower time scale than mosquito infection. For values we have taken to draw the plots displayed, both infected fractions appear to evolve on the same time scale.

There is another time scale disparity comment worth making. The short-time part of any logistic evolution is exponential increase. Observation times of interest in the kind of West Nile virus studies we have discussed in this paper are typically short on bird time scales. They are, however, not short on mosquito time scales. Therefore, the total bird population $A(t)$ might be taken to be $A(0)e^{(\beta-\gamma)t}$ to a good approximation. The total mosquito population $m(t)$ should not be approximated in this manner.

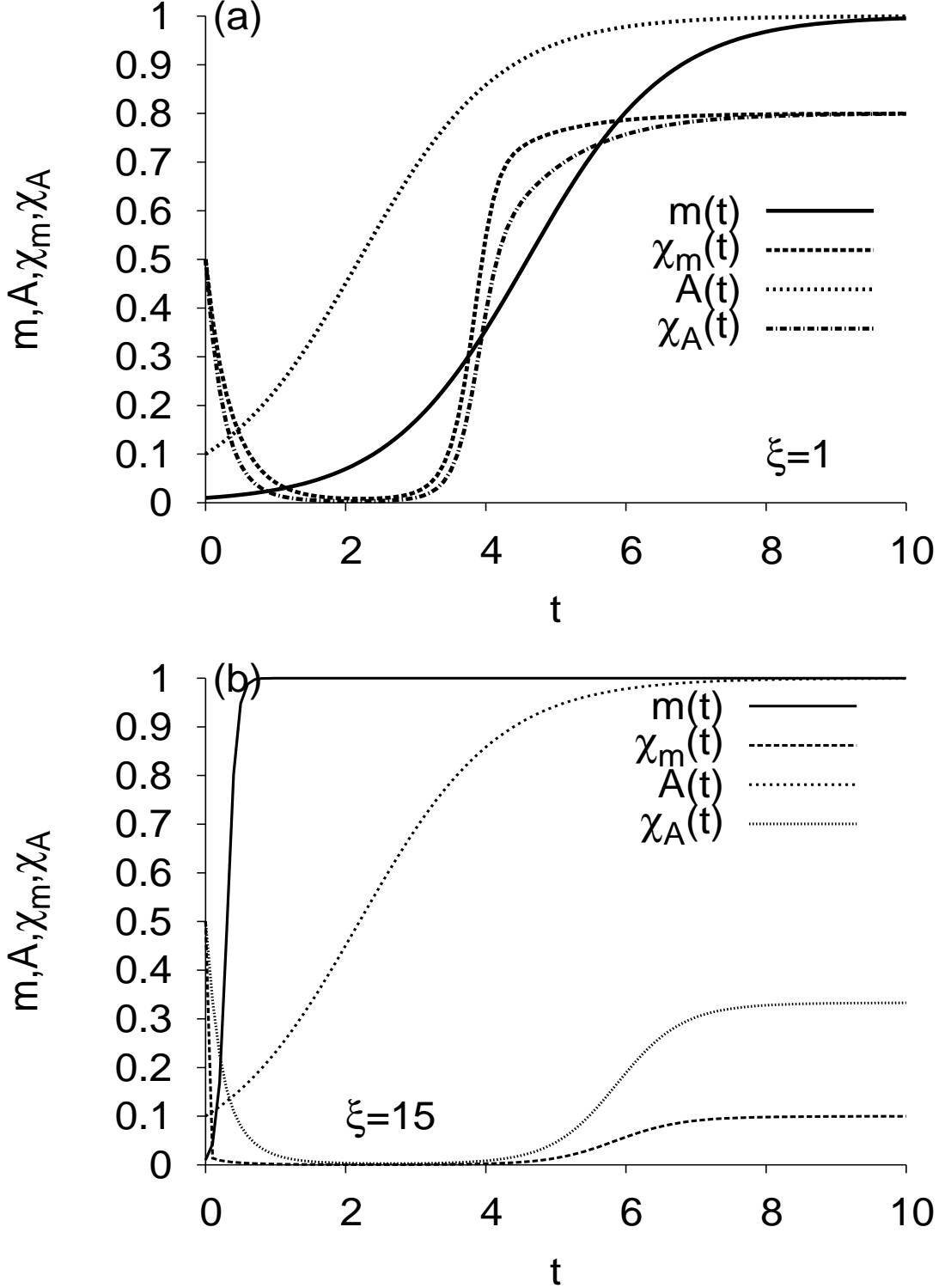


FIG. 6: Effect of time scale disparity on the time evolution of the mosquito and bird populations m and A , and their respective infected fractions χ_m and χ_A . Time is plotted in units of the rate difference $1/(\beta - \gamma)$. The disparity factor ξ (see text) is 1 in (a) and 15 in (b). This means that $b = \xi\beta$ and $c = \xi\gamma$. Other parameters are arbitrary: $\beta = 4, \gamma = 3, \kappa = 1, a = 20, \alpha = 20, K = \frac{1}{b-c}$. The initial conditions have been taken to be $A(0) = 0.1, m(0) = 0.01, \chi_m(0) = 0.5, \chi_A(0) = 0.5$.

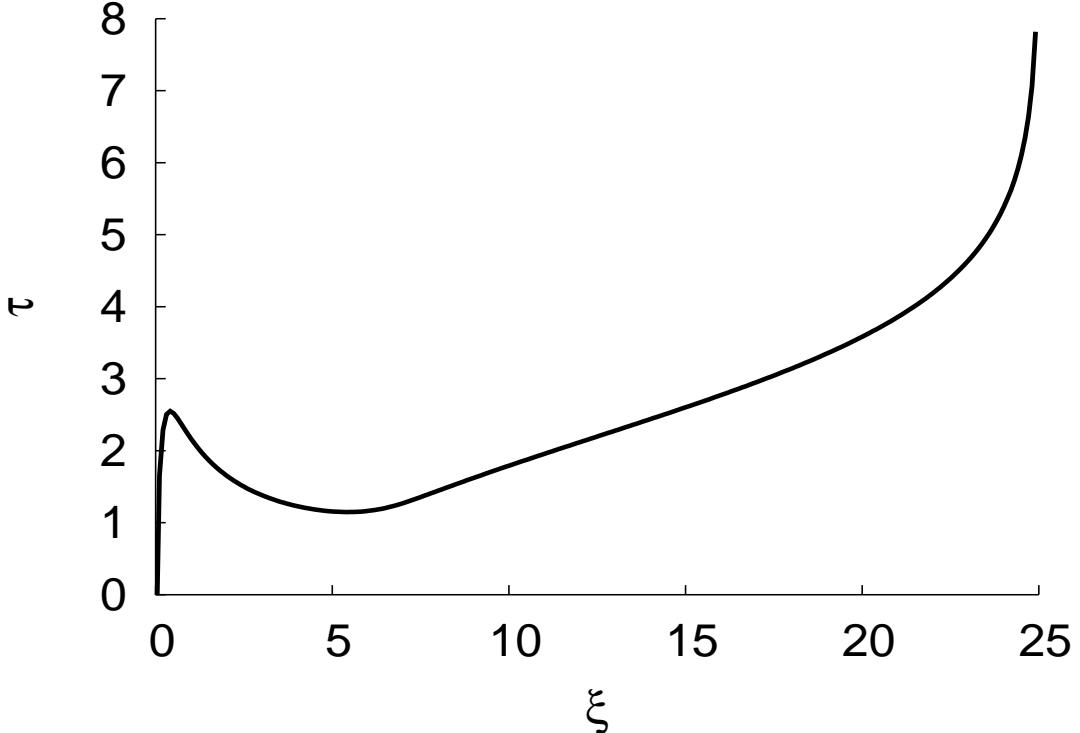


FIG. 7: Strongly nonmonotonic dependence of the delay τ (see text) in the appearance of the infection as a function of disparity factor ξ . The delay is plotted in units of the rate difference $1/(\beta - \gamma)$. Parameters are as in Fig. 6. Sharply different rates of increase of the delay with the disparity factor are seen, along with a blow-up at the extreme right. The blow-up occurs at the critical value of $\xi_c = \frac{\beta}{\alpha m(\infty)} \frac{b}{\alpha A(\infty)}$, beyond which the state with nonzero infection cannot be supported.

IV. CONCLUSIONS

In this paper we have presented some essential features of a theoretical framework to analyze the spread of the West Nile virus epidemic. Explaining existing data has not been our aim because such data are rather scarce. Starting with the AK equations given earlier for Hantavirus investigations [1], we have developed the West Nile virus theory in three stages.

First, we modified the Hantavirus equations (1) by replacing same-taxon infection by cross-infection, peculiar to the West Nile virus. At this stage, we retained Hantavirus features by assuming that all susceptible organisms have comparable lifespans, no organisms are born infected, and the infection does not affect the death rate of members of either taxon. Our results (equations 4 and 5) showed that the condition for steady-state infection is a generalization from the one obtained for the Hantavirus (no cross-infection), involving a combination of the parameter of the two taxa.

In the second stage, we studied realistic features of the West Nile virus by including an analysis of vertical transmission for the mosquitoes, and increased mortality rate in birds due to infection. We found that vertical transmission does not affect the qualitative behavior of the system within the framework of equations we have adopted. However, we found that damped oscillations in the evolution emerge from the increased mortality rate in birds due to infection. Furthermore, we saw that, while the dependence of the infected fraction of birds on the increased mortality rate is monotonic, the dependence of the total bird population is not, there being a characteristic value at which the maximum number of birds are killed. For lower as well as higher values of the mortality rate, the bird population is larger. Finally, we found that disparate lifespans of mosquitoes and birds lead to the effect that the delay in the onset of steady-state infection depends nonlinearly on the ratio of the characteristic times of the two taxa.

The third stage of our investigations addresses an important feature of the West Nile virus epidemic: the *movement* of mosquitoes and birds, particularly the *migration* of birds. Because this stage has not been completed, we have not presented our results in this paper. However we state here the basic idea and the equations we use for this purpose.

The equations are

$$\begin{aligned}\frac{\partial m_s}{\partial t} &= (b - c)m_s + (b - b_i)m_i - am_s A_i - \frac{m_s m}{K} + D_m \frac{\partial^2 m_s}{\partial x^2}, \\ \frac{\partial m_i}{\partial t} &= (b_i - c)m_i + am_s A_i - \frac{m_i m}{K} + D_m \frac{\partial^2 m_i}{\partial x^2}, \\ \frac{\partial A_s}{\partial t} &= \beta A - \gamma A_s - \alpha A_s m_i - \frac{A_s A}{\kappa} + \int dy f(x, y) A_s(y, t), \\ \frac{\partial A_i}{\partial t} &= -(\gamma + \delta)A_i + \alpha A_s m_i - \frac{A_i A}{\kappa} + \int dy f(x, y) A_i(y, t).\end{aligned}$$

The movement of the mosquitoes is considered diffusive and represented by the diffusion constant D_m while the long range movement of birds (including, particularly, migration) is represented by the integral terms involving $f(x, y)$. Information about the speed at which mosquitoes move, as well as their effectively enhanced mobility due to wind and related effects, is fed into D_m . An alternative to the integral description of the long-range motion of birds given above is a treatment through a partially systematic and partially stochastic term representing the appearance and disappearance of birds (and infection) at the site under investigation, as a result of their migration. Another important feature missing from the work reported in the present paper is the seasonal disappearance of mosquitoes, an essential part of the conduit of infection, when the temperature drops below that capable of sustaining them. Yet another is the possible of reemergence of infection in the spring from infected larvae. Work on all these aspects is under way and will be reported in a future publication. An alternative approach to the theory of the West Nile virus based on a difference equation model [14] that has appeared recently in the literature, has been brought to our attention. In future work we will report similarities, differences and domains of applicability of the two formalisms.

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