A COMPARISON OF CONTINUOUS AND DISCRETE TIME WEST NILE VIRUS MODELS

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Abstract. The first recorded North American epidemic of West Nile virus was detected in New York state in 1999, and since then the virus has spread and become established in much of North America. Mathematical models for this vector transmitted disease with cross-infection between mosquitoes and birds have recently been formulated with the aim of predicting disease dynamics and evaluating possible control methods. We consider discrete and continuous time versions of the West Nile virus models proposed by Wonham et al. [WCL] and by Thomas and Urena [TU], and evaluate the basic reproduction number as the spectral radius of the next generation matrix in each case. The assumptions on mosquito feeding efficiency are crucial for the basic reproduction number calculation. Differing assumptions lead to the conclusion from one model [WCL] that a reduction in bird density would exacerbate the epidemic, while the other model [TU] predicts the opposite: a reduction in bird density would help control the epidemic.

1. Introduction

Although West Nile (WN) virus is endemic in Africa, the Middle East and western Asia, the first recorded North American epidemic of WN virus was detected in New York state in 1999. Since 1999 WN virus has spread and has become established in much of North America. Recent mathematical models for this disease have been proposed in an attempt to predict disease dynamics and elucidate control methods [B, LD2, TU, WCL].

The temporal spread of WN virus involves an interplay of the transmission between birds via female mosquitoes as disease vectors and of the disease dynamics within a reservoir of birds. While birds can die quickly from the virus (especially corvids, such as crows and jays), the mosquito disease vectors do not appear to be affected adversely by the disease. The interaction of WN virus with secondary hosts (mainly humans and horses) is dead-end in the sense that there is no evidence that these secondary hosts can infect feeding mosquitoes (although see [] for very recent evidence that some mammals may not be dead-end hosts). Thus a cross-infection model involving susceptible-infectious interactions between birds (reservoirs) and mosquitoes (vectors) is the central starting point for formulating WN virus dynamics, and is the basis for all models considered here.

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Given a mathematical model for disease spread, the *basic reproduction number*, $\mathcal{R}_0$, is an essential summary parameter. It is defined as the expected number of secondary cases caused by a single infected individual introduced into an otherwise susceptible population. If $\mathcal{R}_0$ is greater than one, a local disease outbreak is possible. Control methods can be designed to bring the control basic reproduction number, which we also denote by $\mathcal{R}_0$, to a value less than one. The dependence of $\mathcal{R}_0$ on model parameters can be used (through the modification of parameter values) to evaluate efficacy of such control methods.

However, different models for a given disease may not deliver the same $\mathcal{R}_0$. Models for the same disease can be based on differing assumptions about disease transmission and dynamics. These, in turn, result in different $\mathcal{R}_0$ expressions. In evaluating the effect of control methods by using $\mathcal{R}_0$, the conclusions that are drawn depend crucially on the model assumptions. Thus, if $\mathcal{R}_0$ is to be used to evaluate the effect of control measures on disease outbreak, it is necessary to link the $\mathcal{R}_0$ value directly to model assumptions. This is the primary purpose of the paper. Indeed, we will show that the assumptions on mosquito feeding efficiency are crucial for the basic reproduction number calculation. Differing assumptions lead one model [WCL] to conclude that a reduction in bird density would exacerbate the epidemic, while the other model [TU] predicts the opposite: a reduction in bird density would help control the epidemic.

Our approach is to consider how both biological and temporal model structure influence $\mathcal{R}_0$. We proceed by example, analyzing, in depth, continuous and discrete time versions of models by Wonham et al. [WCL] and Thomas and Urena [TU]. Both consider the North American WN virus epidemic, but are formulated with different biological assumptions and time structures. The first model [WCL] treats time as a continuous variable, and is an 8-dimensional system of ordinary differential equations for vector and reservoir compartments. For comparison, we also formulate two different discrete time versions of this model. The second model [TU] is in discrete time and consists of a 9-dimensional difference equation system for vector, reservoir and human compartments. We also formulate a continuous time version of this model. For the resulting five models, we calculate and compare $\mathcal{R}_0$ expressions and consider their differing implications for disease control.

The secondary purpose of the paper is to demonstrate, via our sample calculations, how the basic reproduction number can be calculated in a straightforward way for both discrete and continuous time models, even when the models become complicated, such as in the 8- and 9-dimensional cross-infection models for WN virus [WCL, TU]

2. Methods to calculate $\mathcal{R}_0$

2.1. **Continuous time models.** For an epidemic model with disease compartments that is formulated as an ordinary differential equation system, a precise mathematical definition of $\mathcal{R}_0$ is the spectral radius of the next generation matrix [DH, VW]. Provided that a disease free equilibrium (DFE) exists, and some other biologically realistic conditions are satisfied, then the next generation matrix can be determined from the system as follows. Write the equations for the infected compartments only as

$$\frac{dx}{dt} = (\mathcal{F} - \mathcal{V})(x),$$

where vector x gives the number in each infected compartment, $\mathcal{F}$ denotes the rate of new infections and $\mathcal{V}$ denotes the rate of transfer (by other means) between compartments. Let F and V denote the linearized matrices at the DFE from $\mathcal{F}$ and $\mathcal{V}$, respectively. Then FV^{-1} is the next generation matrix, with the (i, j) entry giving the expected number of new infections in compartment i produced by an infected individual introduced into compartment j. The basic reproduction number $\mathcal{R}_0$ is the spectral radius of FV^{-1}; see [DH, VW]. The DFE is locally asymptotically stable if the matrix $F - V$ has all eigenvalues with negative real parts. With the above definition of $\mathcal{R}_0$, this stability condition can be shown by using M-matrix theory to be equivalent to $\mathcal{R}_0 < 1$. In addition, $F - V$ is unstable if $\mathcal{R}_0 > 1$. Therefore, if introduced at a low level, the disease dies out when $\mathcal{R}_0 < 1$, but persists in the population when $\mathcal{R}_0 > 1$. The exact form of $\mathcal{R}_0$ is thus important in determining control strategies for the disease.

2.2. **Discrete time models.** For discrete time epidemic models, the equations for the infected compartments are written as

$$x(t + 1) = (\mathcal{F} + \mathcal{T})x(t)$$

where $x(t)$ is the number in each infected compartment at time step t, $\mathcal{F}$ represents the new infections and $\mathcal{T}$ represents other transitions between compartments. Linearization at the DFE (which is assumed to exist), gives rise to nonnegative matrices F and T, with the spectral radius of T less than 1. As in [C, LS], the discrete next generation matrix, which projects the infected compartments from one time step to the next, is given by $F(I - T)^{-1}$, where I denotes an identity matrix. For a discrete system, $\mathcal{R}_0$ is the spectral radius of $F(I - T)^{-1}$. It follows from Perron-Frobenius theory [C, LS], that the DFE is linearly stable or unstable according to whether $\mathcal{R}_0$ is less than or greater than one. Thus the exact formulation of a discrete model, which in turn gives an expression for $\mathcal{R}_0$, is important in determining whether or not the disease can persist, and in suggesting control measures.

2.3. **Common notation.** To compare different models, we introduce common notation for all state variables (numbers of mosquitoes, birds and humans in the different disease compartments) and parameters in the two original models. Exposed and infective compartments must both be considered as infected when calculating $\mathcal{R}_0$. Since West Nile virus is a vector transmitted disease, we expect a square root in the expression for $\mathcal{R}_0$, which arises as a geometric mean of the vector and reservoir variables [DH, VW].

State Variables	Vector	Reservoir	Humans
Larval	L_V		
Susceptible	S_V	S_R	S_H
Exposed	E_V		
Infectious	I_V	I_R	I_H
Recovered		R_R	R_H
Dead		X_R	
Total adults	A_V		
Total	N_V	N_R	N_H

Parameters	Vector	Reservoir	Humans
Birth	b_V	b_R	b_H
Proportion of births that are infected	ρ_V		
Maturation	m_V		
Death (natural)	d_L, d_V	d_R	
Death (from virus)		δ_R	
Vector biting on host		β_R	β_H
Virus transmission (to)	α_V	α_R	
Virus incubation	κ_V		
Recovery from virus		γ_R	γ_H

For continuous time models, all parameters are per capita rates per unit time, except for the proportion ρ_V and the probabilities α_V and α_R. For discrete time models, all parameters except ρ_V are probabilities or numbers per unit time. In the analysis that follows a superscript $\hat{}$ is used to distinguish parameters for discrete time models from parameters for continuous time models.

The models we consider here are for a single season under constant environmental conditions (but see [WCL] for extensions to a variable environment). Hence there is birth and death of mosquitoes (vectors), but only death of birds (reservoirs), as bird reproduction is assumed to have occurred before the mosquito season. The [WCL] model allows for virus-induced death of birds (which is necessary for birds, such as corvids, that die quickly from WNV), while the [TU] model does not allow for virus-induced death of birds (which may be a good approximation for birds, such as passerines, that do not die quickly from WNV) but includes natural death in all bird compartments. The [TU] model also allows for vertical transmission of the virus from mosquito parent to offspring, while the [WCL] model does not. The two models differ in their assumptions on the disease transmission terms.

The [WCL] model was formulated in continuous time whereas the [TU] model was formulated in discrete time, with time steps of a week. The calculation for the basic reproduction number was made for the [WCL] but not for the [TU] model. We formulate discrete- and continuous-time versions for both models, and make the calculation of basic reproduction number for each formulation, so as to facilitate model comparison.

3. West Nile virus model of Wonham et al [WCL]

3.1. **Continuous time model.** We now give the continuous time WN virus model as formulated by Wonham et al. [WCL]. The authors include age structure for the female mosquito population by dividing this population into larvae and adults, with birth into the larval stage, and natural death in each stage. The adult stage is divided into susceptible, exposed (latent) and infectious compartments. For the one season model, they assume that WN virus can cause reservoir death, but natural birth and death of the reservoir population is ignored. This population is divided into susceptible, infectious, recovered and dead compartments. Cross-infection between mosquitoes and birds is modeled by mass action incidence normalized by the total population of birds. This arises since female mosquitoes only take a fixed number of blood meals per unit time, and follows a similar term used to model malaria; see, for example, [AM]. Since humans are dead-end hosts, they are not included in this model. In the common notation, the dynamics are given by the following ordinary differential equation system:

Vectors (V):

$$\begin{aligned}
\frac{dL_V}{dt} &= b_V(S_V + E_V + I_V) - m_V L_V - d_L L_V \\
\frac{dS_V}{dt} &= -\alpha_V \beta_R \frac{I_R}{N_R} S_V + m_V L_V - d_V S_V \\
\frac{dE_V}{dt} &= \alpha_V \beta_R \frac{I_R}{N_R} S_V - (\kappa_V + d_V) E_V \\
\frac{dI_V}{dt} &= \kappa_V E_V - d_V I_V
\end{aligned}$$

Reservoirs (R):

$$\begin{aligned}
\frac{dS_R}{dt} &= -\alpha_R \beta_R \frac{S_R}{N_R} I_V \\
\frac{dI_R}{dt} &= \alpha_R \beta_R \frac{S_R}{N_R} I_V - (\delta_R + \gamma_R) I_R \\
\frac{dR_R}{dt} &= \gamma_R I_R \\
\frac{dX_R}{dt} &= \delta_R I_R.
\end{aligned}$$

For the existence of a disease free equilibrium it is assumed that vector birth and death rates balance in the absence of disease. This is expressed by the following parameter constraint: $b_V = d_V\,(1 + d_L/m_V)$. The disease free equilibrium is:

$$(L_V, S_V, E_V, I_V, S_R, I_R, R_R, X_R) = \left(\frac{b_V}{m_V + d_L} A_V^*, A_V^*, 0, 0, N_R^*, 0, 0, 0 \right)$$

The infected variables are (E_V, I_V, I_R) and with $\mathcal{F}$, the rate of appearance of new infections, and $\mathcal{V}$, the rate of transfer between compartments,

$$\begin{bmatrix} E_V \\ I_V \\ I_R \end{bmatrix}_t = \mathcal{F} - \mathcal{V} = \begin{bmatrix} \alpha_V \beta_R \frac{I_R}{N_R} S_V \\ 0 \\ \alpha_R \beta_R \frac{S_R}{N_R} I_V \end{bmatrix} - \begin{bmatrix} (\kappa_V + d_V) E_V \\ d_V I_V - \kappa_V E_V \\ (\delta_R + \gamma_R) I_R \end{bmatrix}$$

The corresponding linearized matrices at the DFE are:

$$F = \begin{bmatrix} 0 & 0 & \alpha_V \beta_R \frac{A_V^*}{N_R^*} \\ 0 & 0 & 0 \\ 0 & \alpha_R \beta_R & 0 \end{bmatrix}, \qquad V = \begin{bmatrix} \kappa_V + d_V & 0 & 0 \\ -\kappa_V & d_V & 0 \\ 0 & 0 & \delta_R + \gamma_R \end{bmatrix}$$

Then

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\alpha_V \beta_R A_V^*}{(\delta_R+\gamma_R)N_R^*} \\ 0 & 0 & 0 \\ \frac{\alpha_R\beta_R\kappa_V}{(\kappa_V+d_V)d_V} & \frac{\alpha_R\beta_R}{d_V} & 0 \end{bmatrix}$$

so the spectral radius of FV^{-1} is

$$\mathcal{R}_0 = \sqrt{\frac{\alpha_V\alpha_R\beta_R^2\kappa_V}{d_V(d_V+\kappa_V)(\delta_R+\gamma_R)}\frac{A_V^*}{N_R^*}}$$

as found in [WCL]. It can be seen that $\mathcal{R}_0$ is the geometric mean of $\frac{\alpha_V\beta_R\kappa_V}{d_V(d_V+\kappa_V)}\frac{A_V^*}{N_R^*}$ and $\frac{\alpha_R\beta_R}{(\delta_R+\gamma_R)}$. The first term is the product of the infection rate to the vector at the DFE, the average time that a vector spends in the infective class and the probability that a vector entering the exposed class survives to become infective. The second term is the product of the infection rate to the reservoir and the average time that a bird spends in the infective class before dying or recovering.

3.2. **Discrete time version of Wonham et al. model – version 1.** To uniquely write down the difference equations from the original continuous time model, the ordering of events needs to be specified. Thus, we consider the following set of assumptions.

(1) Birth, infection and transfer between compartments occur at the beginning of the time step.
(2) Natural and disease-induced mortality occur at the end of the time step.

Note that a different ordering of events is considered in the Appendix.

Under assumptions (1) and (2), the difference equation system takes the form:

$$\begin{aligned}
L_V(t+1) &= (1-\hat{d}_L)\hat{b}_V(S_V(t)+E_V(t)+I_V(t)) + (1-\hat{d}_L)(1-\hat{m}_V)L_V(t)\\
S_V(t+1) &= (1-\hat{d}_V)S_V(t)(1-\hat{\alpha}_V)^{\hat{\beta}_R I_R(t)/N_R(t)} + (1-\hat{d}_V)\hat{m}_V L_V(t)\\
E_V(t+1) &= (1-\hat{d}_V)S_V(t)\left(1-(1-\hat{\alpha}_V)^{\hat{\beta}_R I_R(t)/N_R(t)}\right) + (1-\hat{d}_V)(1-\hat{\kappa}_V)E_V(t)\\
I_V(t+1) &= (1-\hat{d}_V)\hat{\kappa}_V E_V(t) + (1-\hat{d}_V)I_V(t)\\
S_R(t+1) &= S_R(t)(1-\hat{\alpha}_R)^{\hat{\beta}_R I_V(t)/N_R(t)}\\
I_R(t+1) &= (1-\hat{\delta}_R)S_R(t)\left(1-(1-\hat{\alpha}_R)^{\hat{\beta}_R I_V(t)/N_R(t)}\right) + (1-\hat{\delta}_R)(1-\hat{\gamma}_R)I_R(t)\\
R_R(t+1) &= \hat{\gamma}_R I_R(t)\\
X_R(t+1) &= X_R(t) + \hat{\delta}_R\left(S_R(1-(1-\hat{\alpha}_R)^{\hat{\beta}_R I_V(t)/N_R(t)}) + (1-\hat{\gamma}_R)I_R(t)\right)
\end{aligned}$$

By way of example, we derive in detail the equation for $S_V(t+1)$. First, the expected number of bites made by a susceptible mosquito in a unit time interval is $\hat{\beta}_R$, and the expected number of times the mosquito bites an infected bird is $\hat{\beta}_R I_R(t)/N_R(t)$. The probability of a mosquito avoiding the infection arising from a single bite on an infected bird is $1-\hat{\alpha}_V$ and hence the probability of a susceptible

mosquito avoiding infection in a given time step is $(1-\hat{\alpha}_V)^{\hat{\beta}_R I_R(t)/N_R(t)}$. The number of susceptible mosquitoes remaining susceptible in a given time step, $S_V(t)(1-\hat{\alpha}_V)^{\hat{\beta}_R I_R(t)/N_R(t)}$, is augmented by the number of larvae maturing $\hat{m}_V L_V(t)$, and then is diminished by natural mortality which is avoided with probability $1-\hat{d}_V$. Using a similar approach, it is possible to derive the other equations above.

Here, the parameter constraint for existence of a disease free equilibrium is:

$$\hat{b}_V(1-\hat{d}_V) = \hat{d}_V\left(1+\frac{\hat{d}_L}{\hat{m}_V(1-\hat{d}_L)}\right)$$

The disease free equilibrium is:

$$(L_V, S_V, E_V, I_V, S_R, I_R, R_R, X_R) = \left(\frac{(1-\hat{d}_L)\hat{b}_V}{(1-\hat{d}_L)\hat{m}_V+\hat{d}_L}A_V^*, A_V^*, 0, 0, N_R^*, 0, 0, 0\right)$$

The infected variables are E_V, I_V and I_R. Linearizing the equations for these variables about the DFE and writing the resulting matrix as $F+T$, where F includes only new infections and the column sums of T are less than one, gives the following nonnegative matrices:

$$F = \begin{bmatrix} 0 & 0 & -(1-\hat{d}_V)\hat{\beta}_R\ln(1-\hat{\alpha}_V)\dfrac{A_V^*}{N_R^*} \\ 0 & 0 & 0 \\ 0 & -(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R) & 0 \end{bmatrix}$$

$$T = \begin{bmatrix} (1-\hat{d}_V)(1-\hat{\kappa}_V) & 0 & 0 \\ (1-\hat{d}_V)\hat{\kappa}_V & 1-\hat{d}_V & 0 \\ 0 & 0 & (1-\hat{\delta}_R)(1-\hat{\gamma}_R) \end{bmatrix}$$

Then the matrix $F(I-T)^{-1}$ is given as

$$\begin{bmatrix} 0 & 0 & \dfrac{-(1-\hat{d}_V)\hat{\beta}_R\ln(1-\hat{\alpha}_V)A_V^*}{(\hat{\delta}_R+\hat{\gamma}_R(1-\hat{\delta}_R))N_R^*} \\ 0 & 0 & 0 \\ \dfrac{-(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R)(1-\hat{d}_V)\hat{\kappa}_V}{\hat{d}_V(\hat{d}_V+\hat{\kappa}_V(1-\hat{d}_V))} & \dfrac{-(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R)}{\hat{d}_V} & 0 \end{bmatrix}$$

and the spectral radius of $F(I-T)^{-1}$ is

$$\mathcal{R}_0 = \sqrt{\frac{(1-\hat{d}_V)^2(1-\hat{\delta}_R)\hat{\beta}_R^2\hat{\kappa}_V\ln(1-\hat{\alpha}_V)\ln(1-\hat{\alpha}_R)}{\hat{d}_V(\hat{d}_V+\hat{\kappa}_V(1-\hat{d}_V))(\hat{\delta}_R+\hat{\gamma_R}(1-\hat{\delta}_R))}\frac{A_V^*}{N_R^*}}$$

3.3. **Comparison of discrete with continuous time version.** The discrete time version in Section 3.2 has a natural correspondence with the continuous time version considered earlier in Section 3.1. In particular, due to the assumed order of events for the discrete system in Section 3.2, the events in step (1) are conditioned upon surviving mortality in the previous step (2). Hence the parameters are conditioned upon surviving natural ($\hat{d}_V$ or $\hat{d}_L$) or disease-induced ($\hat{\delta}_R$) mortality. This means that δ_R is replaced by $\hat{\delta}_R$, d_V is replaced by $\hat{d}_V$, γ_R is replaced by $\hat{\gamma}_R(1-\hat{\delta}_R)$, b_V is replaced by $\hat{b}_V(1-\hat{d}_L)$, κ_V is replaced by $\hat{\kappa}_V(1-\hat{d}_V)$, and m_V is replaced by $\hat{m}_V(1-\hat{d}_L)$. The infection rate $\alpha_V\beta_R$ is replaced by $-(1-\hat{d}_V)\hat{\beta}_R\ln(1-\hat{\alpha}_V)$, and infection rate $\alpha_R\beta_R$ is replaced by $-(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R)$. With these substitutions the above basic reproduction number is identical to the previous continuous time version (Section 3.1), and $\mathcal{R}_0$ can again be interpreted as a geometric mean.

4. West Nile Virus model of Thomas and Urena [TU]

4.1. **Discrete time model.** We now give the discrete time model, as formulated by Thomas and Urena [TU], with the time step of one week. In the model, the authors include compartments for susceptible, exposed and infectious mosquitoes; susceptible, infectious and recovered birds; and susceptible, infectious and recovered humans. They assume that a proportion of mosquito births is infected, and so goes into the exposed class (vertical WN virus transmission). Birds are assumed to die naturally (not from the virus), human death is omitted, and mass action incidence is assumed. The biting rate parameter, which is defined as 'the probability that one mosquito bites one bird' in a given week is constrained to lie between zero and one, unlike the biting parameter of Section 3.2, $\hat{\beta}_R$, which is defined as the number of bites made per susceptible mosquito per time step. Furthermore, there are no separate transmission parameters $\hat{\alpha}_V$ and $\hat{\alpha}_R$ in the original model [TU]. The probability of virus transmission between mosquitoes and birds is implicitly assumed to be $\hat{\alpha}_V = \hat{\alpha}_R = 1$. To distinguish the biting rate parameters in this model from the model of Section 3.2, we use $\tilde{\beta}_R$ and $\tilde{\beta}_H$ for the [TU] model in this section. The order of events is not explicitly stated in [TU]. Control by insecticide spraying every other week is through the introduction of a time dependent function $c(t)$ (death due to spraying), namely

$$c(t) = \begin{cases} 0, & t \text{ even} \\ \text{constant } c \in (0,1), & t \text{ odd} \end{cases}$$

We keep this spraying function in our model formulation, so as to facilitate comparison with the model in [TU], although the control level is taken to be equal to zero in the analysis that follows.

In the common notation, the dynamics are given by the difference equation system for vectors, reservoirs and humans:

Vectors (V):

$$\begin{aligned} S_V(t+1) &= (1-\tilde{\beta}_R)^{I_R(t)}(1-\hat{d}_V+\hat{b}_V-c(t))S_V(t) \\ &\quad +(1-\hat{\rho}_V)\hat{b}_V(E_V(t)+I_V(t)) \\ E_V(t+1) &= (1-(1-\tilde{\beta}_R)^{I_R(t)})(1-\hat{d}_V+\hat{b}_V-c(t))S_V(t) \\ &\quad +(1-\hat{\kappa}_V)(1-\hat{d}_V-c(t))E_V(t)+\hat{\rho}_V\hat{b}_V(E_V(t)+I_V(t)) \\ I_V(t+1) &= (1-\hat{d}_V-c(t))(I_V(t)+\hat{\kappa}_V E_V(t)) \end{aligned}$$

Reservoirs (R):

$$\begin{aligned} S_R(t+1) &= \hat{b}_R(I_R(t)+R_R(t)) + (1-\tilde{\beta}_R)^{I_V(t)}(1-\hat{d}_R+\hat{b}_R)S_R(t) \\ I_R(t+1) &= (1-(1-\tilde{\beta}_R)^{I_V(t)})(1-\hat{d}_R+\hat{b}_R)S_R(t) + (1-\hat{\gamma}_R)(1-\hat{d}_R)I_R(t) \\ R_R(t+1) &= (1-\hat{d}_R)R_R(t) + \hat{\gamma}_R(1-\hat{d}_R)I_R(t) \end{aligned}$$

Humans (H):

$$\begin{aligned} S_H(t+1) &= \hat{b}_H N_H(t) + (1-\tilde{\beta}_H)^{I_V(t)}S_H(t) \\ I_H(t+1) &= (1-(1-\tilde{\beta}_H)^{I_V(t)})S_H(t) + (1-\hat{\gamma}_H)I_H(t) \\ R_H(t+1) &= R_H(t) + \hat{\gamma}_H I_H(t) \end{aligned}$$

Note that we have corrected a bracket in their $I_V(t+1)$ equation, and have also omitted a term $(1-\hat{\kappa}_V)\hat{b}_V E_V(t)$ on the right side of the equation for $E_V(t+1)$ as given in [TU, equation (3)] as this does not seem biologically reasonable, since vertically transmitted disease is accounted for by the term containing ρ_V. The total number of humans is given by $N_H = S_H + I_H + R_H$.

The nonlinearities in the infection terms differ from those assumed in Section 3.2. For example, the nonlinearity in the fraction of susceptible mosquitoes avoiding infection, $(1-\tilde{\beta}_R)^{I_R(t)}$, differs from the nonlinearity for the discrete model of Section 3.2, $(1-\hat{\alpha}_V)^{\hat{\beta}_R I_R(t)/N_R(t)}$, unless $1-\tilde{\beta}_R = (1-\hat{\alpha}_V)^{\hat{\beta}_R/N_R(t)}$, or equivalently $\hat{\beta}_R \ln(1-\hat{\alpha}_V)/N_R = \ln(1-\tilde{\beta}_R)$. A similar argument applied to the bird population shows that the nonlinear incidence terms are equal only when $\hat{\beta}_R \ln(1-\hat{\alpha}_R)/N_R = \ln(1-\tilde{\beta}_R)$.

The model assumptions that lead to the differences in nonlinear transmission terms can be summarized as follows. If a single infected mosquito were introduced into a population of birds, the probability that any given susceptible bird avoids infection during the weekly time-step is $1-\tilde{\beta}_R$, according to [TU], and is $(1-\hat{\alpha}_V)^{\hat{\beta}_R/N_R(t)}$, according to [WCL]. The first assumes that the probability of avoiding infection is independent of the bird population size, while the second assumes that the probability of avoiding infection is an increasing function of bird population size.

Constraints for the existence of a DFE are $\hat{d}_V = \hat{b}_V, \hat{d}_R = \hat{b}_R, \hat{b}_H = 0$ and $c(t) = 0$, and the DFE has $S_V = N_V^*, S_R = N_R^*, S_H = N_H^*$ and all other state variables zero.

The model of Thomas and Urena [TU] includes vertical transmission of disease through infected vector births (the parameter ρ_V). This must also be included in $\mathcal{F}$ giving rise to a term in the matrix F. With $c(t) = 0$ and infected variables E_V, I_V, I_R, I_H gives $F(I-T)^{-1}$ as a 4-by-4 matrix, where the I_H variable plays no role in the calculation because humans are dead-end hosts and so do not impact infection of other species (see Appendix for matrices F and T). This matrix is reducible, with two of its eigenvalues being zero, thus its spectral radius is given as the largest eigenvalue of the reduced matrix

$$\begin{bmatrix} \hat{\rho}_V & \dfrac{-\ln(1-\tilde{\beta}_R)N_V^*}{d_R+\hat{\gamma}_R(1-\hat{d}_R)} \\ \dfrac{-\ln(1-\tilde{\beta}_R)\hat{\kappa}_V(1-\hat{d}_V)N_R^*}{\hat{d}_V(\hat{d}_V+\hat{\kappa}_V(1-\hat{d}_V))} & 0 \end{bmatrix}$$

For $\hat{\rho}_V > 0$, some births are infected, then $\mathcal{R}_0$ is is equal to

$$\frac{1}{2}\left(\hat{\rho}_V + \sqrt{\hat{\rho}_V^2 + 4\frac{(1-\hat{d}_V)\hat{\kappa}_V \ln^2(1-\tilde{\beta}_R)N_V^* N_R^*}{\hat{d}_V(\hat{d}_V + \hat{\kappa}_V(1-\hat{d}_V))(\hat{d}_R + \hat{\gamma}_R(1-\hat{d}_R))}}\right).$$

If there are no infected births, then $\mathcal{R}_0$ is reduced and is given explicitly as a square root, namely:

$$\mathcal{R}_0 \quad = \quad \sqrt{\frac{(1-\hat{d}_V)\hat{\kappa}_V \ln^2(1-\tilde{\beta}_R)N_V^* N_R^*}{\hat{d}_V(\hat{d}_V + \hat{\kappa}_V(1-\hat{d}_V))(\hat{d}_R + \hat{\gamma}_R(1-\hat{d}_R))}}$$

Although the $\mathcal{R}_0$ for $\rho_V = 0$ is superficially different from the $\mathcal{R}_0$ for the discrete-time version of the Wonham et al. model in Section 3.2, the two can be closely connected if it is assumed that the value of $\tilde{\beta}_R$ can be related to the parameters $\hat{\beta}_R$, $\hat{\alpha}_V$, $\hat{\alpha}_R$, and N_R as discussed earlier. Namely, $\hat{\beta}_R \ln(1-\hat{\alpha}_V)/N_R^* = \ln(1-\tilde{\beta}_R)$ and $\hat{\beta}_R \ln(1-\hat{\alpha}_R)/N_R^* = \ln(1-\tilde{\beta}_R)$. Once these assumptions are made, the only difference between the $\mathcal{R}_0$ terms is in the precise way that the mortality terms appear in the formula. As demonstrated for the [WCL] model in the Appendix, the manner in which the mortality terms appear in $\mathcal{R}_0$ depends upon the precise ordering of events within one time step of the discrete time model. We believe that this is the reason for the discrepancy between the two $\mathcal{R}_0$ terms (for the [TU] model with $\rho_V = 0$ of this section and for the discrete [WCL] model of Section 3.2), but have not pursued analysis of this further. This is because, when formulating their model, Thomas and Urena did not precisely specify the ordering of events within a time step.

4.2. **Continuous time version of** [TU] **model.** Assuming a small time step, we replace $t+1$ in the original [TU] model (modified as noted in Section 4.1) with $t+\Delta t$, where $\Delta t \to 0$. We replace probabilities by corresponding rates, so that $\hat{a} = a\Delta t$, where a is $b_V, d_V, \kappa_V, b_R, d_R, \gamma_R$, respectively, and set $\tilde{\beta}_R = \beta_R \Delta t$. Then we expand the functions with respect to Δt using a Taylor series and neglect all higher order terms (such as $(\Delta t)^2, (\Delta t)^3$). The resulting differential equation system, obtained as the limit with $\Delta t \to 0$, reads:

$$\frac{dS_V}{dt} = -\beta_R S_V I_R - d_V S_V + b_V S_V + b_V(1-\rho_V)(E_V + I_V)$$
$$\frac{dE_V}{dt} = \beta_R S_V I_R - d_V E_V - \kappa_V E_V + \rho_V b_V (E_V + I_V)$$
$$\frac{dI_V}{dt} = -d_V I_V + \kappa_V E_V$$
$$\frac{dS_R}{dt} = b_R N_R - d_R S_R - \beta_R I_V S_R$$
$$\frac{dI_R}{dt} = \beta_R I_V S_R - (d_R + \gamma_R) I_R$$
$$\frac{dR_R}{dt} = \gamma_R I_R - d_R R_R$$
$$\frac{dS_H}{dt} = -\beta_H I_V S_H + b_H N_H$$
$$\frac{dI_H}{dt} = \beta_H I_V S_H - \gamma_H I_H$$
$$\frac{dR_H}{dt} = \gamma_H I_H$$

The disease free equilibrium, with parameter constraints $b_V = d_V, b_R = d_R$ and $b_H = 0$, is:

$$(S_V, E_V, I_V, S_R, I_R, R_R, S_H, I_H, R_H) = (N_V^*, 0, 0, N_R^*, 0, 0, N_H^*, 0, 0)$$

The infected variables are E_V, I_V, I_R and I_H but I_H can be excluded from the $\mathcal{R}_0$ calculations by an argument similar to that used in the original [TU] model in Section 4.1. Then in variables E_V, I_V, I_R the matrices at the DFE are:

$$F = \begin{bmatrix} \rho_V b_V & \rho_V b_V & \beta_R N_V^* \\ 0 & 0 & 0 \\ 0 & \beta_R N_R^* & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} d_V + \kappa_V & 0 & 0 \\ -\kappa_V & d_V & 0 \\ 0 & 0 & d_R + \gamma_R \end{bmatrix}$$

Consequently,

$$FV^{-1} = \begin{bmatrix} \rho_V & \rho_V & \dfrac{\beta_R N_V^*}{(d_R + \gamma_R)} \\ 0 & 0 & 0 \\ \dfrac{\beta_R \kappa_V N_R^*}{d_V(d_V + \kappa_V)} & \dfrac{\beta_R N_R^*}{d_V} & 0 \end{bmatrix}$$

so the spectral radius of FV^{-1} is, for $\rho_V = 0$,

$$\mathcal{R}_0 = \sqrt{\frac{\beta_R^2 \kappa_V N_V^* N_R^*}{d_V(d_V + \kappa_V)(d_R + \gamma_R)}}$$

For $\rho_V > 0$,

$$\mathcal{R}_0 = \frac{1}{2}\left(\rho_V + \sqrt{\rho_V^2 + 4\frac{\beta_R^2 \kappa_V N_V^* N_R^*}{d_V(d_V+\kappa_V)(d_R+\gamma_R)}}\right)$$

With the ratio of A_V^* and N_R^* replaced by the product $N_V^* N_R^*$ and δ_R replaced by d_R, the expression for $\mathcal{R}_0$ in the continuous [WCL] model in Section 3.1 is analogous to the above expression with $\rho_V = 0$, once we recall that, for the [TU] model, $\alpha_V = \alpha_R = 1$. As discussed below in Section 5, the main difference is due to the different assumptions made about the disease transmission terms. An identification between the $\mathcal{R}_0$ expressions for this continuous model and the previous discrete model (Section 4.1) can be made as for the [WCL] models of Section 3. If β_R is replaced by $-\ln(1-\tilde{\beta}_R)$, and κ_V and γ_R are conditioned on surviving natural death, then the $\mathcal{R}_0$ of Section 4.1 is obtained.

5. Discussion and Concluding remarks

As demonstrated in Section 3, analysis of the change in $\mathcal{R}_0$ with model parameters for the Wonham et al. [WCL] model predicts that a reduction in mosquito density can be used to control WN virus outbreaks. This prediction is shared by the model of Thomas and Urena for WN virus [TU]. However, [WCL] also predicts that a reduction in bird density will actually exacerbate, rather than control, a WN virus outbreak. By way of contrast, this second result is *not* predicted by the model of Thomas and Urena [TU]. In fact, for the [TU] model, a reduction in bird density will control, rather than exacerbate, a WN virus outbreak. Clearly, both results cannot be simultaneously correct from a biological perspective.

A resolution of this quandary arises from an analysis of how model assumptions shape the formula for $\mathcal{R}_0$. The two models make different assumptions about mosquito feeding efficiencies, leading to different disease transmission terms.

The [WCL] model follows [AM] in using modified mass action terms that assume efficient mosquito searching even when host densities are low ($\alpha_R\beta_R\frac{S_R}{N_R}I_V$ for mosquitoes to birds and $\alpha_V\beta_R\frac{I_R}{N_R}S_V$ for birds to mosquitoes). This assumption corresponds to a disease transmission rate that depends only on the proportion of birds susceptible or infected, and is independent of the actual density of birds. Thus a constant disease transmission rate is assumed over a range of bird densities (Figure 1).

The [TU] model uses simple mass action terms that assume the encounter rate between mosquitoes and hosts is proportional to host density (in the continuous formulation, these are $\beta_R I_V S_R$ for mosquitoes to birds and $\beta_R S_V I_R$ for birds to mosquitoes). This assumption corresponds to a transmission rate that increases linearly with bird density (Figure 1). As noted earlier, the [TU] model does not explicitly state the order of events within in each time step. A further investigation of the [TU] model might reformulate the specification of the order of events within each time step, although we do not pursue this here.

Since WN-vector mosquitoes in North America typically exhibit a 3-day feeding cycle, it may be reasonable to imagine a saturating functional response of transmission rate to bird density, of which the [WCL] and [TU] models each represent a part (Figure 1). Incorporating such dynamics into a single model would require a different transmission term [MBH].

Although both model assumptions have a sound theoretical basis, they yield starkly different predictions as to the effect of bird control (as calculated from $\mathcal{R}_0$) on WN virus. When bird densities are low, the [WCL] model predicts that the remaining birds receive more bites and become local hot spots for disease transmission with each bird having a high probability of becoming infected and passing on the virus. The [TU] model, in contrast, predicts that the disease will die out in regions of low bird density. Thus, the $\mathcal{R}_0$ of [TU] predicts that bird control would be effective in controlling WN, whereas the $\mathcal{R}_0$ of [WCL] predicts that it would be counterproductive.

Because the $\mathcal{R}_0$ involves linearization about the equilibrium $S_R = N_R^*$, the model yielding correct $\mathcal{R}_0$ is the one whose functional response of transmission rate to bird density is valid for typical bird densities N_R^*. In other words, if reduction in bird density from N_R^* means no reduction in the overall biting rate, but simply that the remaining birds are bitten more frequently (i.e., N_R^* is in the 'flat' region of Figure 1), then the $\mathcal{R}_0$ of [WCL] is pertains. If, by way of contrast, reduction in bird density from N_R^* means a concomitant reduction in the overall biting rate (i.e., N_R^* is in the 'linear' region of Figure 1) then the [TU] model pertains.

This simple example illustrates our primary purpose. That is, to demonstrate clearly how slightly different, but seemingly reasonable assumptions, going into a model formulation for WN virus, can yield very different biological conclusions on the basis of analysis of $\mathcal{R}_0$. Indeed, as we have shown in Section 3 and the Appendix, a change as simple as moving from continuous to discrete time formulation can yield several plausible discrete-time models, each with a qualitatively different $\mathcal{R}_0$. As such, this paper is designed as a cautionary note which underscores the importance of model formulation and between-model comparison prior to inferring the efficacy of disease management methods. Section 2 and the subsequent explicit calculations address our secondary purpose, namely to set out clearly the calculations for the basic reproduction number for a continuous or discrete time model, with particular emphasis on models for WN virus.

The two models that we have taken from the literature, specifically the continuous time model of Wonham et al [WCL] and the discrete time model of Thomas and Urena [TU], plus the additional continuous time models by Lord and Day [LD2] and by Bowman et al [B] are, to our knowledge, the only mathematical models for West Nile virus currently in the literature. We are considering these, a model of St Louis Encephalitis virus by Lord and Day [LD2] and other studies on Encephalitis [LD1], [KSM], [TG], [TGM] for further comparisons.

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7. Appendix

7.1. **Discrete time version of Wonham et al. model – version 2 (different assumptions).** To see how the ordering of events can affect the model structure, and eventually $\mathcal{R}_0$, we consider a different set of assumptions from that in Section 3.2. The new assumptions are as follows:

(1) Disease-induced and natural mortality and birth occur at the beginning of the time step.

(2) Infection and transfer occur at the end of the time step.

The corresponding model in discrete time is formulated as:

$$\begin{aligned}
L_V(t+1) &= (1-\hat{m}_V)[\hat{b}_V(S_V(t)+E_V(t)+I_V(t))+(1-\hat{d}_L)L_V(t)]\\
S_V(t+1) &= (1-\hat{d}_V)S_V(t)(1-\hat{\alpha}_V)^{\frac{\hat{\beta}_R(1-\hat{\delta}_R)I_R(t)}{N_R(t)-\hat{\delta}_R I_R(t)}}\\
&\quad + \hat{m}_V[(1-\hat{d}_L)L_V(t)+\hat{b}_V(S_V(t)+E_V(t)+I_V(t))]\\
E_V(t+1) &= (1-\hat{d}_V)S_V(t)\left(1-(1-\hat{\alpha}_V)^{\frac{\hat{\beta}_R(1-\hat{\delta}_R)I_R(t)}{N_R(t)-\hat{\delta}_R I_R(t)}}\right)\\
&\quad + (1-\hat{d}_V)(1-\hat{\kappa}_V)E_V(t)
\end{aligned}$$

$$\begin{aligned}
I_V(t+1) &= \hat{\kappa}_V(1-\hat{d}_V)E_V(t)+(1-\hat{d}_V)I_V(t)\\
S_R(t+1) &= S_R(t)(1-\hat{\alpha}_R)^{\frac{\hat{\beta}_R(1-\hat{\delta}_R)I_V(t)}{N_R(t)-\hat{\delta}_R I_R(t)}}\\
I_R(t+1) &= (1-\hat{\gamma}_R)\left(S_R(t)\left(1-(1-\hat{\alpha}_R)^{\frac{\hat{\beta}_R(1-\hat{\delta}_R)I_V(t)}{N_R(t)-\hat{\delta}_R I_R(t)}}\right)+(1-\hat{\delta}_R)I_R(t)\right)\\
R_R(t+1) &= \hat{\gamma}_R\left(S_R(t)\left(1-(1-\hat{\alpha}_R)^{\frac{\hat{\beta}_R(1-\hat{\delta}_R)I_V(t)}{N_R(t)-\hat{\delta}_R I_R(t)}}\right)+(1-\hat{\delta}_R)I_R(t)\right)\\
X_R(t+1) &= X_R(t)+\hat{\delta}_R I_R(t)
\end{aligned}$$

Now, the parameter constraint for existence of a disease-free equilibrium is:

$$\hat{b}_V = \hat{d}_V\left(1+\frac{\hat{d}_L}{\hat{m}_V(1-\hat{d}_L)}\right)$$

The disease free equilibrium is:

$$(L_V,S_V,E_V,I_V,S_R,I_R,R_R,X_R) = \left(\frac{(1-\hat{d}_L)(1-\hat{m}_V)\hat{b}_V}{(1-\hat{d}_L)\hat{m}_V+\hat{d}_L}A_V^*,A_V^*,0,0,N_R^*,0,0,0\right)$$

The infected variables are E_V, I_V and I_R and

$$F = \begin{bmatrix} 0 & 0 & -(1-\hat{d}_V)(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_V)\dfrac{A_V^*}{N_R^*}\\ 0 & 0 & 0\\ 0 & -(1-\hat{d}_V)(1-\hat{\gamma}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R) & 0 \end{bmatrix}$$

$$T = \begin{bmatrix} (1-\hat{d}_V)(1-\hat{\kappa}_V) & 0 & 0\\ (1-\hat{d}_V)\hat{\kappa}_V & 1-\hat{d}_V & 0\\ 0 & 0 & (1-\hat{\delta}_R)(1-\hat{\gamma}_R) \end{bmatrix}$$

The matrix $F(I-T)^{-1}$ is given as

$$\begin{bmatrix} 0 & 0 & \dfrac{\hat{p}_V(1-\hat{\delta}_R)\hat{\beta}_R \ln(1-\hat{\alpha}_V)A_V^*}{(\hat{\delta}_R+\hat{\gamma}_R(1-\hat{\delta}_R))N_R^*} \\ 0 & 0 & 0 \\ \dfrac{-(\hat{p}_V)^2(1-\hat{\gamma}_R)\hat{\beta}_R \ln(1-\hat{\alpha}_R)\hat{\kappa}_V}{\hat{d}_V(\hat{d}_V+\hat{\kappa}_V(1-\hat{d}_V))} & \dfrac{-(\hat{p}_V)(1-\hat{\gamma}_R)\hat{\beta}_R \ln(1-\hat{\alpha}_R)}{\hat{d}_V} & 0 \end{bmatrix}$$

where here $\hat{p}_V$ is written for $(1-\hat{d}_V)$. Thus the spectral radius of $F(I-T)^{-1}$ is

$$\mathcal{R}_0 = \sqrt{\frac{(1-\hat{d}_V)^3(1-\hat{\gamma}_R)(1-\hat{\delta}_R)\hat{\beta}_R^2\hat{\kappa}_V \ln(1-\hat{\alpha}_V)\ln(1-\hat{\alpha}_R)}{\hat{d}_V(\hat{d}_V+\hat{\kappa}_V(1-\hat{d}_V))(\hat{\delta}_R+\hat{\gamma}_R(1-\hat{\delta}_R))}\frac{A_V^*}{N_R^*}}$$

Note that this $\mathcal{R}_0$ is different than the one calculated for the previous discrete time model in Section 3.2. However, as with the previous model, this $\mathcal{R}_0$ can be made identical to the earlier Wonham et al. [WCL] $\mathcal{R}_0$, once the parameters are conditioned upon surviving natural ($\hat{d}_V$ or $\hat{d}_L$) or disease-induced ($\hat{\delta}_R$) mortality, and the infection rates are modified appropriately. For these assumptions, this means that δ_R is replaced by $\hat{\delta}_R$, d_V is replaced by $\hat{d}_V$, γ_R is replaced by $\hat{\gamma}_R(1-\hat{\delta}_R)$, b_V is replaced by $\hat{b}_V(1-\hat{d}_L)$, κ_V is replaced by $\hat{\kappa}_V(1-\hat{d}_V)$, and m_V is replaced by $\hat{m}_V(1-\hat{d}_L)$ as in Section 3.2. The infection rate $\alpha_V\beta_R$ is replaced by $-(1-\hat{d}_V)(1-\hat{\delta}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_V)$, and infection rate $\alpha_R\beta_R$ is replaced by $-(1-\hat{\delta}_R)(1-\hat{\gamma}_R)\hat{\beta}_R\ln(1-\hat{\alpha}_R)$. Note that, due to the different set of assumptions regarding the ordering of events, these infection rates are expressed by different parameters than in the previous discrete time model (Section 3.2): a new factor of $1-\hat{\delta}_R$ appears in the $\alpha_V\beta_R$ term, and a new factor of $1-\hat{\gamma}_R$ appears in the $\alpha_R\beta_R$ term.

7.2. **Matrices F and T for** [TU] **model.** The 4-by-4 matrices F and T used to compute $\mathcal{R}_0$ in Section 4.1 are given explicitly as:

$$F = \begin{bmatrix} \hat{\rho}_V\hat{b}_V & \hat{\rho}_V\hat{b}_V & -\ln(1-\tilde{\beta}_R)N_V^* & 0 \\ 0 & 0 & 0 & 0 \\ 0 & -\ln(1-\tilde{\beta}_R)N_R^* & 0 & 0 \\ 0 & -\ln(1-\tilde{\beta}_H)N_H^* & 0 & 0 \end{bmatrix}$$

$$T = \begin{bmatrix} (1-\hat{\kappa}_V)(1-\hat{d}_V) & 0 & 0 & 0 \\ \hat{\kappa}_V(1-\hat{d}_V) & 1-\hat{d}_V & 0 & 0 \\ 0 & 0 & (1-\hat{\gamma}_R)(1-\hat{d}_R) & 0 \\ 0 & 0 & 0 & (1-\hat{\gamma}_H) \end{bmatrix}$$

References

[AM] R. M. Anderson and R.M. May, *Infectious Diseases of Humans*, Oxford University, Oxford, 1991.

[B] C. Bowman, A. B. Gumel, P. van den Driessche, J. Wu, H. Zhu, *A mathematical model for assessing control strategies against West Nile virus*, Bull. Math. Biology, to appear.

[C] H. Caswell, *Matrix Population Models*, Sinauer, 2001.

[DH] O. Diekmann and J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*, Wiley, New York, 1999.

[KSM] B. H. Kay, A. J. Saul and A. McCullagh, *A mathematical model for the rural amplification of Murray Valley Encephalitis virus in Southern Australia*, Am. J. Epidemiology 125(4),(1987), 690–705.

[LS] C-K. Li and H. Schneider, *Application of Perron-Frobenius theory to population dynamics*, Mathematical Biology, 44, (2002), 450–462.

[LD1] C. C. Lord and J. F. Day, *Simulation studies of St. Louis Encephalitis virus in South Florida*, Vector borne and zootonic diseases, 1(4),(2001), 299–315.

[LD2] C. C. Lord and J. F. Day, *Simulation studies of St. Louis Encephalitis virus and West Nile viruses: the impact of bird mortality*, Vector borne and zootonic diseases, 1(4),(2001), 317–329.

[MBH] H. McCallum, N. Barlow and J. Hone, *How should pathogen transmission be modelled?* Tr. Ecol. Evol., 16(6), (2001), 295-300.

[TG] P. K. Tapaswi and A. K. Ghosh, *Dynamics of Japanese Encephalitis - a study in mathematical epidemiology*, IMA Journal of Mathematics Applied in Medicine and Biology 16(10), (1999), 1–27.

[TGM] P. K. Tapaswi, A. K. Ghosh and B. B. Mukhopadhyay, *Transmission of Japanese Encephalitis in a 3-population model*, Ecological Modelling 83 (1995), 295–309.

[TU] D. M. Thomas and B. Urena, *A model describing the Evolution of West Nile-like Encephalitis in New York City*, Math. Comput. Modelling 34 (2001), 771–781.

[VW] P. van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosc. 180 (2002), 29–48.

[WCL] M. J. Wonham, T. de Camino-Beck and M. A. Lewis, *An epidemiological model for West-Nile virus: invasion analysis and control application*, Proc. R. Soc. Lond. B, (2004), 501–507.

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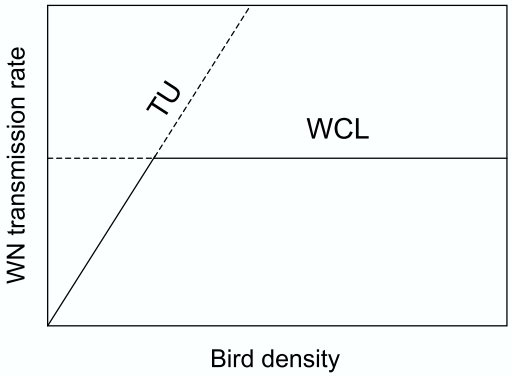

FIGURE 1. Two different assumptions about mosquito feeding efficiency lead to different WN transmission dynamics. The [WCL] model assumes the transmission rate is invariant across bird densities (horizontal line), whereas the [TU] model assumes transmission scales with bird density (diagonal line). When $S_R = N_R = N_R^*$, the per mosquito transmission rate to hosts is $\alpha_R\beta_R$ in the [WCL] model, whereas the per mosquito transmission rate to hosts is $\beta_R N_R^*$ in the [TU] model. Biologically, transmission rates likely exhibit a response that is a combination of the two (solid lines), depending on the equilibrium density of hosts N_R^*.