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Age- and bite-structured models for vector-borne diseases

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ABSTRACT

The biology and behaviour of biting insects is a vitally important aspect in the spread of vector-borne diseases. This paper aims to determine, through the use of mathematical models, what effect incorporating vector senescence and realistic feeding patterns has on disease. A novel model is developed to enable the effects of age- and bite-structure to be examined in detail. This original PDE framework extends previous age-structured models into a further dimension to give a new insight into the role of vector biting and its interaction with vector mortality and spread of disease. Through the PDE model, the roles of the vector death and bite rates are examined in a way which is impossible under the traditional ODE formulation. It is demonstrated that incorporating more realistic functions for vector biting and mortality in a model may give rise to different dynamics than those seen under a more simple ODE formulation. The numerical results indicate that the efficacy of control methods that increase vector mortality may not be as great as predicted under a standard host–vector model, whereas other controls including treatment of humans may be more effective than previously thought.

1. Introduction

The role of biting insects is of the utmost importance in the transmission dynamics of vector-borne diseases; without them many diseases simply could not spread. Vector biology such as longevity and biting rate has long been known to determine not only the persistence of such diseases but also to affect the size and speed of epidemics and the equilibrium prevalence of endemics. Indeed, in the early mathematical models of malaria, Ross indicates that vector death rate and bite rate are important with both featuring in his threshold theorem for malaria (Ross, 1916).

The Ross–Macdonald ordinary differential equation (ODE) model (Macdonald, 1957; Ross, 1911) and its many variations dominate the literature in vector-borne disease modelling. However, key assumptions regarding insect behaviour and biology are often disregarded or overlooked. Taking a basic model of vector-borne disease, one can use a mechanistic approach driven by observation of the biology of transmission and introduce more of the inherent complexity. It is important that this is introduced in such a way that the direct effects of the new elements can be ascertained. Here, the biology and corresponding behaviour of the vector is scrutinised.

It will be assumed that the basic underlying vector-borne disease model takes the form:

$$\text{Hosts}\quad\begin{cases}\dfrac{dS_H}{dt} &= b_H N_H - d_H S_H + \gamma_H I_H - \lambda_H S_H\\ \dfrac{dE_H}{dt} &= -d_H E_H - \sigma_H E_H + \lambda_H S_H\\ \dfrac{dI_H}{dt} &= -(d_H + D_H) I_H + \sigma_H E_H - \gamma_H I_H\end{cases}$$

$$\text{Vectors}\quad\begin{cases}\dfrac{dS_V}{dt} &= b_V N_V - \lambda_V S_V - d_V S_V\\ \dfrac{dE_V}{dt} &= \lambda_V S_V - \sigma_V E_V - d_V E_V\\ \dfrac{dI_V}{dt} &= \sigma_V E_V - d_V I_V\end{cases} \tag{1.1}$$

where $\lambda_i = \alpha p_i I_j/(N_H + m)$ is the force of infection of species j on species i ($j \neq i$). This term is a standard "criss-cross" transmission term associated with purely disassortative mixing. It arises through a vector biting at a rate α, picking a single host from all other hosts (N_H) and other animals (m) and the probability of transmission from infected host/vector to susceptible vector/host being successful (p_V/p_H). Other parameter notation is given in Table 1.

This susceptible-exposed-infected (SEI) host–vector model has recovery (at a rate γ_H) for hosts, but not vectors and additionally

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Table 1
Parameters for the SEI Ross–Macdonald model (1.1).

Parameters and variables	Description
b_H	Per capita host birth rate
d_H	Host death rate
λ_H	Force of infection upon host
γ_H	Host recovery rate
σ_H	Inverse of host latent period
D_H	Disease-induced host death rate
p_H	Probability of host becoming infected from a single infected bite
b_V	Per capita vector birth rate
d_V	Vector death rate
λ_V	Force of infection upon vector
σ_V	Inverse of vector latent period
α	Average bite rate
p_V	Probability of vector becoming infected from a single bite on an infected host
m	Number of other animals available for blood feeding (assuming no feeding preference between hosts) or number of other animals scaled by the vector's relative preference of these animals over the primary hosts (see Rock et al., in press for more details on vector preference).

Table 2
New parameters for the age and bite-structured model (other parameters remain the same as the standard ODE model (1.1).)

Parameters and variables	Description	Note
t	Chronological time	
τ	Time since last bite (TSLB)	
a	Age of vector	Since biting maturity
$\alpha(\tau)$	Per capita bite rate	$\alpha(\tau)=\beta r(\tau)$
β	Maximum per capita bite rate	Constant
$r(\tau)$	"Desire to bite" probability that a vector will take a blood-meal if it finds a host	
δ	Kronecker delta	$\delta(x)=\begin{cases}1 & \text{if } x=0\\ 0 & \text{otherwise}\end{cases}$

disease-induced mortality (D_H) for hosts. Next a more complex model is derived from (1.1), however the following methodology could be applied to almost any ODE vector model.

2. Methodology

2.1. Age structure (vector senescence)

The *age* at which a vector becomes infectious affects the number of secondary infections that can result from this one individual. If infection occurs near the start of the vector's life, it will inflict a higher number of bites (Styer et al., 2007; Bailey, 1982). This notion is that on average the vector which is infected at a low age will spend longer infectious than its counterpart which was infected nearer to the end of its life; more bites occur (on average) whilst it is infected and consequently it spreads disease more to the host population. The relationship between vector survivorship and its important effects on both vectorial capacity and the basic reproductive ratio was first discussed by Macdonald in the 1950s (Macdonald, 1956, 1952, 1961), however it was not until much later that different type of distributions for vector mortality were used rather than simply altering the fixed daily survivorship.

Traditional ODE models such as the Ross–Macdonald model, make use of the simple Markovian formulation by assuming that the (instantaneous) death rate is constant regardless of age; this leads to exponentially distributed life expectancies. In some cases this may be a reasonable and/or justifiable assumption, however more recent work on vectors such as the mosquito (Styer et al., 2007; Bellan, 2010) and tsetse (Hargrove et al., 2011) indicate that not modelling realistic death rates may lead to inaccuracies when estimating the transmission and prevalence of vector-borne disease. This certainly warrants further investigation and is cited as one of the most overlooked aspects of vector-borne disease modelling; Styer et al. (2007) and Bellan (2010) emphasise the importance of vector senescence as part of the modelling procedure.

Others have also attempted to resolve this neglected insight into vector-borne disease modelling by means of *Lumped-Age Classes* whereby the vector population is partitioned into classes in which parameters (in particular the death rate) are assumed to be constant (Hancock and Godfray, 2007). This method is commonly found in single population age-structured models; instead of modelling ageing by some rate of loss and gain between classes, the technique utilises a delay differential equation (DDE) framework where individuals effectively spend fixed times in each stage. DDEs are general more complex to work with than ODEs, particularly during numerical simulation.

A natural way to introduce age structure within the vector population is via a partial differential equation (PDE) type model in a similar manner to creating an age structure in single species disease models (described by various authors Keeling and Rohani, 2008; Murray, 2002; Britton, 2003), whereby a more realistic death rate which is a function of age is chosen.

Imposing a PDE-type age structure on the SEI host–vector model necessitates:

- Dependence of both chronological time and age for vectors (but not for hosts, although hosts could be treated similarly):

$$S_H(t), E_H(t), I_H(t), S_V(a,t), E_V(a,t), I_V(a,t)$$

- Forced births for vectors (births must occur at age zero, $a=0$):

$$b_V\delta(a)$$

- Age dependent deaths for vectors:

$$-d_V(a)$$

- Inclusion of the ageing process for vectors:

$$-\frac{\partial N_V}{\partial a}$$

- A new infection term within the host population (the infection term for the vector population remains unchanged and it is assumed that infectiousness does not vary with age hence the probability of transmission is independent of age):

$$\lambda_H = \alpha p_H \frac{1}{(N_H+m)}\int_0^\infty I_V(u,t)\,\mathrm{d}u$$

2.2. Bite structure (vector feeding behaviour)

Age structure in vector-borne disease models is not in itself new, although vector-borne age-structured models predominantly focus on age in the host population rather than the vector (Geisse et al., 2012; Hethcote and Thieme, 1985). However, not only is vector ageing important but the feeding patterns of the vector also play a vital role in disease transmission. After a vector has reached biting maturity it will start to "desire" a blood-meal; as time passes the vector becomes more and more likely to feed given the opportunity.