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Mathematical analysis of an age-structured model for malaria transmission dynamics

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ABSTRACT

A new deterministic model for assessing the role of age-structure on the transmission dynamics of malaria in a community is designed. Rigorous qualitative analysis of the model reveals that it undergoes the phenomenon of backward bifurcation, where the stable disease-free equilibrium of the model co-exists with a stable endemic equilibrium when the associated reproduction number (denoted by $\mathcal{R}_0$) is less than unity. It is shown that the backward bifurcation phenomenon is caused by the malaria-induced mortality in humans. A special case of the model is shown to have a unique endemic equilibrium whenever the associated reproduction threshold exceeds unity. Further analyses reveal that adding age-structure to a basic model for malaria transmission in a community does not alter the qualitative dynamics of the basic model, with respect to the existence and asymptotic stability of the associated equilibria and the backward bifurcation property of the model. Numerical simulations of the model show that the cumulative number of new cases of infection and malaria-induced mortality increase with increasing average lifespan and birth rate of mosquitoes.

1. Introduction

Malaria, caused by the protozoan plasmodium parasite, is transmitted to humans by female anopheles mosquitoes (after taking a blood meal from the human host). Although malaria has been endemic in many parts of the world for hundreds of thousands of years [21], the disease continues to inflict major public health burden in affected areas, notably the tropical and subtropical areas in Africa, Asia and South America [52]. Malaria, which is endemic in over 100 countries (affecting over one-third of the world's population) [52], accounted for 216 million cases and 655,000 deaths in 2010 [52,53]. Furthermore, malaria inflicts significant mortality among children under the age of five [9].

There is currently no effective and safe vaccine for use against malaria in humans (although concerted global efforts are underway to develop such a vaccine [2,11,12,20,27,33,35,36,38,43,44,47,48,51]). Consequently, malaria control is based on the use of preventive measures (such as mosquito-reduction strategies and personal protection against mosquito bite) and the use of anti-malaria drugs (see, for instance, [16,18,40,41,52,55]).

Numerous mathematical models have been designed and used to gain insight into malaria transmission dynamics in a community, dating back to the classical malaria models of Ross [42] and Macdonald [32]. Although these classical models have, over the decades, been extended to incorporate various aspects related to malaria transmission dynamics and control, such as repeated exposure [37], the use of preventive and therapeutic strategies [55], effect of climate change [31] etc., not much work has been done in modeling the effect of age-structure on malaria spread and control. This is particularly important considering the fact that malaria mortality is age-dependent (with children under the age of five bearing the most burden [52]). The aim of this study is to design, and rigorously analyse, a new age-structured ordinary differential equation (ODE) model for the transmission dynamics of malaria in a community. The model to be designed represents an extension of other age-structured ODE models in the literature, particularly those in [1,39]. The model is formulated in Section 2, and qualitatively analysed in Section 3. An equivalent model, with no age-structure, is considered in Section 4. Numerical simulations are reported in Section 5.

2. Model formulation

The new age-structured malaria model is designed by splitting the total human population at time t, denoted by $N_H(t)$, into the mutually-exclusive sub-populations of susceptible juveniles ($S_{HJ}(t)$), susceptible adults ($S_{HA}(t)$), exposed juveniles ($E_{HJ}(t)$), exposed adults ($E_{HA}(t)$), symptomatic juveniles ($I_{HJ}(t)$), symptomatic adults ($I_{HA}(t)$), recovered juveniles ($R_{HJ}(t)$) and recovered adults ($R_{HA}(t)$), so that

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$$N_H(t) = S_{HJ}(t) + S_{HA}(t) + E_{HJ}(t) + E_{HA}(t) + I_{HJ}(t) + I_{HE}(t) + R_{HJ}(t) + R_{HA}(t).$$

It should be emphasized that individuals in the exposed (E_{HJ} and E_{HA}) classes are infected (i.e., they are in the early stage of infection, but show no clinical symptoms of malaria).

The total mosquito population at time t, denoted by $N_V(t)$, is sub-divided into the mutually-exclusive compartments of susceptible ($S_V(t)$) and infected ($I_V(t)$) mosquitoes, so that

$$N_V(t) = S_V(t) + I_V(t).$$

The population of susceptible juveniles is generated by the birth (or immigration) of juveniles (at a rate Π_J). Although vertical transmission of malaria can occur (see [15] and some of the references therein), it is assumed that all children are born susceptible (i.e., it is assumed that vertical transmission does not occur). This population is increased by loss of infection-acquired immunity by recovered juveniles [28,37] (at a *per capita* rate ψ_{HJ}). It is decreased by infection, following effective contacts with infected mosquitoes, at a rate λ_{HJ}, given by

$$\lambda_{HJ} = \frac{b_1(N_V, N_H)\beta_{HJ} I_V}{N_V}. \tag{2.1}$$

In (2.1), $b_1(N_V, N_H)$ is the *per capita* biting rate of mosquitoes on susceptible humans (juveniles and adults) *per* unit time, and β_{HJ} is the probability of infection of susceptible juveniles *per* bite by an infected mosquito. It is further decreased by maturation to adulthood (at a rate ξ; this rate is assumed, for mathematical convenience, to be same for all the epidemiological classes for humans) and natural death (at a rate μ_H; it is assumed that natural death occurs in all human epidemiological classes at this rate). Thus,

$$\frac{dS_{HJ}}{dt} = \Pi_J + \psi_J R_{HJ} - \lambda_{HJ} S_{HJ} - (\xi + \mu_H) S_{HJ}. \tag{2.2}$$

The population of susceptible adults is generated by the maturation of susceptible juveniles (at the rate ξ) and by the loss of infection-acquired immunity by recovered adults (at a rate ψ_A). It is decreased by infection at a rate λ_{HA}, given by

$$\lambda_{HA} = \frac{b_1(N_V, N_H)\beta_{HA} I_V}{N_V}, \tag{2.3}$$

where β_{HA} is the probability of infection of susceptible adults *per* bite by an infected mosquito. This population is further decreased by natural death. Hence,

$$\frac{dS_{HA}}{dt} = \xi S_{HJ} + \psi_A R_{HA} - \lambda_{HA} S_{HA} - \mu_H S_{HA}. \tag{2.4}$$

The population of exposed juveniles is generated, following the infection of susceptible juveniles, at the rate λ_{HJ}. It is decreased by the development of clinical symptoms of malaria (at a rate σ_{HJ}), maturation to adulthood (at the rate ξ) and natural death, so that

$$\frac{dE_{HJ}}{dt} = \lambda_{HJ} S_{HJ} - (\sigma_{HJ} + \xi + \mu_H) E_{HJ}. \tag{2.5}$$

Similarly, the population of exposed adults is generated by the maturation of exposed juveniles (at the rate ξ) and by the infection of susceptible adults (at the rate λ_{HA}). It is diminished by the development of malaria symptoms (at a rate σ_{HA}) and natural death. Hence,

$$\frac{dE_{HA}}{dt} = \xi E_{HJ} + \lambda_{HA} S_{HA} - (\sigma_{HA} + \mu_H) E_{HA}. \tag{2.6}$$

The population of symptomatic juveniles is generated when exposed juveniles develop clinical symptoms of malaria (at the rate σ_{HJ}). It is decreased by maturation (at the rate ξ), recovery (at a rate γ_J), natural death and disease-induced death (at a rate δ_{HJ}). Hence,

$$\frac{dI_{HJ}}{dt} = \sigma_{HJ} E_{HJ} - (\xi + \gamma_J + \mu_H + \delta_{HJ}) I_{HJ}. \tag{2.7}$$

Similarly, the population of symptomatic adults is generated at the rates σ_{HA} and ξ, and reduced by recovery (at a rate γ_A), natural death and disease-induced death (at a rate δ_{HA}), so that,

$$\frac{dI_{HA}}{dt} = \sigma_{HA} E_{HA} + \xi I_{HJ} - (\gamma_A + \mu_H + \delta_{HA}) I_{HA}. \tag{2.8}$$

The population of recovered juveniles is generated at the rate γ_J, and decreased by the loss of infection-acquired immunity (at the rate ψ_J), maturation (at the rate ξ) and natural death. Thus,

$$\frac{dR_{HJ}}{dt} = \gamma_J I_{HJ} - (\psi_J + \xi + \mu_H) R_{HJ}. \tag{2.9}$$

The population of recovered adults is increased by the recovery of symptomatic adults (at the rate γ_A) and the maturation of recovered juveniles (at the rate ξ). It is decreased by the loss of infection-acquired immunity (at the rate ψ_A) and natural death. Thus,

$$\frac{dR_{HA}}{dt} = \gamma_A I_{HA} + \xi R_{HJ} - (\psi_A + \mu_H) R_{HA}. \tag{2.10}$$

The population of susceptible mosquitoes is generated by the birth of adult mosquitoes (at a *per capita* rate Π_V). It is reduced by infection, following effective contacts with infected humans, at a rate λ_V, where

$$\lambda_V = \frac{b_2(N_V, N_H)\beta_V[\eta(E_{HJ} + E_{HA}) + I_{HJ} + I_{HA}]}{N_H}. \tag{2.11}$$

In (2.11), $b_2(N_V, N_H)$ is *per capita* biting rate of susceptible mosquitoes on infected humans, β_V is the probability of infection of a susceptible mosquito *per* bite on an infected human and $0 \leqslant \eta < 1$ is a modification parameter accounting for the assumption that exposed humans are less infectious than symptomatic humans. This population is further decreased by natural death (at a rate μ_V). Hence,

$$\frac{dS_V}{dt} = \Pi_V - \lambda_V S_V - \mu_V S_V. \tag{2.12}$$

The population of infected mosquitoes is generated by the infection of susceptible mosquitoes (at the rate λ_V) and decreased by natural death (at the rate μ_V). Thus,

$$\frac{dI_V}{dt} = \lambda_V S_V - \mu_V I_V. \tag{2.13}$$

It is assumed that mosquitoes do not suffer additional disease-induced death [39].

An important requirement for a mosquito-borne disease model, such as the model given by equations {(2.1),...,(2.13)}, is that the total number of bites made by mosquitoes must balance the total number of bites received by the human hosts (see, for instance, [3,17,37]). This constraint is implemented as follows. First of all, mosquitoes bite both susceptible and infected humans. Hence, it is assumed that the average number of mosquito bites received by humans depends on the total sizes of the populations of mosquitoes and humans in the community. Furthermore, it is assumed that the human hosts are always sufficient in abundance so that it is reasonable to consider the biting rate $b_2(N_V, N_H) = b_2$, a constant. Thus, in order for the total number of bites made by mosquitoes to balance the total number of bites received by the human hosts, the following conservation law must hold:

$$b_2 N_V = b_1(N_V, N_H) N_H, \tag{2.14}$$

so that,