A Mathematical Model for Endemic Malaria with Variable Human and Mosquito Populations

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Abstract—A deterministic differential equation model for endemic malaria involving variable human and mosquito populations is analysed. Conditions are derived for the existence of endemic and disease-free equilibria. A threshold parameter $\tilde{R}_0$ exists and the disease can persist if and only if $\tilde{R}_0$ exceeds 1. The disease-free equilibrium always exist and is globally stable when $\tilde{R}_0$ is below 1. Numerical simulations show that the endemic equilibrium, when it exists, is unique and is globally stable. © 2000 Elsevier Science Ltd. All rights reserved.

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1. INTRODUCTION

Malaria is a parasitic vector borne disease endemic in many parts of the world. At present, at least 300 million people are affected worldwide and there are between 1 and 1.5 million malaria related deaths annually [1]. The incidence of malaria in many urban centres of the world is increasing and almost all areas of high endemicity lie in developing countries where inadequate drainage provides large stagnant water reservoirs which are ideal breeding sites for disease vectors such as the Anopheles mosquito [2–7].

The malaria parasite is transmitted from person to person by a female Anopheles mosquito each time the infected insect takes a blood meal. The symptoms of malaria fever in an infected human include bouts of fever and anaemia.[1] On average, the incubation period is about 12 days in humans and about ten days in mosquitoes. The period can be longer depending on the strain of the parasite.

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[1]Whether or not the Plasmodium parasites cause their mosquito hosts any discomfort is not clear. It does seem that mosquitoes that are carrying the malaria parasite have a decreased life expectancy, higher mortality rates, low fertility rates, and higher man-biting rates than their noninfected counterparts [8].

The literature on the mathematical models for communicable diseases is vast. See, for example, the survey by Hethcote *et al.* [9]. In most of these models, the assumption of constant total populations is often made. Mathematical modelling of malaria has flourished since the days of Ross [10], who was the first to model the dynamics of malaria transmission and Macdonald [11-13] who expounded on Ross' work, introducing the theory of superinfection. Using data from the Garki project [14], many studies have been carried out on the epidemiology of malaria, and one of the most outstanding is the mathematical model proposed by Dietz *et al.* [15], which Nedelman [16] has analysed in detail. Further works on the subject include [17-19] and the review by Nedelman [20]. Today, we are faced with the need to predict the dynamics and transmission of indirectly transmitted diseases with greater accuracy and over longer periods of time, and more often with limited empirical data.

The dynamics of many communicable diseases have been extensively analysed under the assumption that the duration of immunity[2] is independent of exposure to infection [9,22,23]. However, the immunity to malaria appears to be sustained by continuing exposure [24,25]. Hence, the conventional definition of immunity as absolute refractoriness to infection may be too restrictive, as immunity may confer protection against severe illness without eliminating chronic, mild infections [26]. Hence, asymptomatic immune carriers may be infective. This phenomenon of incomplete immunity permitting transmission is known to exist for malaria and complicates disease control strategies as the reservoir of infection now includes symptomatic and asymptomatic infected individuals.

It is clear that the assumption of constant population size in epidemiological models, which is relatively valid when studying diseases of short duration with limited effects on mortality, may no longer be valid when dealing with endemic diseases such as malaria, which has a high mortality rate. In fact, the interaction between the mosquito and human populations necessarily introduces a high variability on the mosquito populations for which an assumption of constant population will not be valid. For the sake of mathematical tractability in the analyses of mathematical models for malaria transmission with variable population densities, information about infected mosquitoes is often traded for information about all the mosquitoes and infected people through the pseudoequilibrium hypothesis. In his articles, Nedelma [16,20] shows that in the malaria model of Dietz, Molineaux and Thomas [15], the inoculation rate depends on a pseudoequilibrium approximation to the differential equation describing the mosquito dynamics. This approximation can be quite restrictive if we consider the fact that transmission of the malaria parasite from person to person depends on the most part (aside from cases of blood transfusion) on the dynamics of the vector population. It is therefore necessary to study the dynamics and transmission of the malaria parasite when attention is paid to the dynamics of the mosquito populations.

Here we develop and analyse a model that incorporates compartments for the mosquito population. Following the ideas advanced by Aron [24-26], we introduce in our model a class of persons who are partially immune to the disease malaria, but who may be infectious. We assume density dependent death rates in both vector and human populations so that the total populations are varying with time through a modification of the logistic equation that includes disease related deaths; cf., [27]. The assumption of density dependent death rates in a population, first introduced by Verhulst [28], has been used, for example, by Nisbet and Gurney [29] in a study of predator-prey models, Shears [30] in a study on situations such as famine in developing countries, and Matessi [31] on the dynamics of a single population.

The rest of the paper is organised as follows. In Section 2, we briefly outline the derivation of the model and investigate the existence of steady states in Section 3. A linear stability analysis around the steady states is performed in Section 4, where we show that the disease-free equilibrium is globally stable. In Section 5, we present some numerical simulations and round up the paper with some concluding remarks in Section 6.

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[2]An excellent source of medical information on various diseases, showing the types of immunity, is found in [21].

2. THE BASIC MODEL

To motivate the derivation of our mathematical model, we define the following contact parameters.

$c_{hv}, \tilde{c}_{hv}$ = respective infectivity of an infectious nonimmune and a partially immune human. Defined as the probability that a bite by a susceptible mosquito on an infected human will transfer the infection to the mosquito.

c_{vh} = the infectivity of the mosquito. Defined as the probability that a bite by an infected mosquito on a susceptible human will transfer the infection to the human.

a_v = the man-biting rate of the mosquitoes. Defined as the average number of bites given to humans by each mosquito per unit time.

The human and vector populations are divided into classes or states containing susceptible, incubating, infectious, and immune individuals. At time t, there are S_h susceptible humans, E_h incubating humans, I_h infectious humans, R_h partially immune humans, S_v susceptible mosquitoes, E_v incubating mosquitoes, and I_v infectious mosquitoes. The mosquito population does not have an immune class, since their infective period ends with their death. $N_h = S_h + E_h + I_h + R_h$ and $N_v = S_v + E_v + I_v$ are, respectively, the total human and vector populations at time t. The model assumes all newborns are susceptible in both populations (no vertical transmission) and a uniform birth rate. The per capita birth rates for humans and mosquitoes are $\lambda_h > 0$ and $\lambda_v > 0$, respectively. Immune human individuals loose their immunity at a rate $\beta_h > 0$. Incubating individuals in both populations become infectious with rates $\nu_h > 0$ and $\nu_v > 0$. Infectious human individuals either recover with rate r_h (without any substantial gain in immunity) to join the susceptible class or with rate α_h into the immune compartment, where they remain for the period of their immunity before joining the susceptible class. All individuals in both human and mosquito populations experience per capita death rates of $f_h(N_h)$, $f_v(N_v)$, respectively, where for the moment, f_h and f_v are assumed to be strictly monotone increasing continuously differentiable functions of their arguments. Infected human individuals die from the disease at the additional rate $\gamma_h > 0$.

The effective contact rates between the two populations, which may be defined as the average number of contacts per day that will lead to the infection of one party if the other party was infectious, depends on a number of factors: the man-biting rate[3] of the mosquitoes, the transmission probabilities between the species, and the number of individuals in both population. Briefly, if a_v is the man-biting rate of the mosquito, then there are $a_v N_v / N_h$ bites per human per time. Since there are S_h susceptible humans and the proportion of the total number of bites that are potentially infectious to humans is I_v / N_v, the number of potentially infectious bites given to susceptible humans is $a_v I_v S_h / N_h$ bites per time. However, only a fraction of these bites, namely c_{vh}, successfully infect humans. We thus have

$$\text{exposure rate of humans} = \left(\frac{c_{vh} a_v I_v}{N_h} \right) S_h. \tag{1}$$

Similarly, assuming that the reservoir of possible infections from humans includes the classes I_h and R_h, we have

$$\text{exposure rate of mosquitoes} = \left(\frac{c_{hv} a_v I_h}{N_h} \right) S_v + \left(\frac{\tilde{c}_{hv} a_v R_h}{N_h} \right) S_v. \tag{2}$$

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[3]We are assuming that the populations are confined in a particular geographic area, small enough so that each bite has an equal probability of being taken from any particular human. Since the transmission of the parasite is controlled by the man-biting habit of the mosquito, these assumptions are, in fact, a restricted form of homogeneous mixing based on the idea that the mosquitoes have a man-biting rate.

Now using standard mass action laws, we write the equations that describe the spread of the disease in the form

$$\frac{dS_h}{dt} = \lambda_h N_h + \beta_h R_h + r_h I_h - f_h(N_h) S_h - \left(\frac{c_{vh} a_v I_v}{N_h}\right) S_h,$$

$$\frac{dE_h}{dt} = \left(\frac{c_{vh} a_v I_v}{N_h}\right) S_h - (\nu_h + f_h(N_h)) E_h,$$

$$\frac{dI_h}{dt} = \nu_h E_h - (r_h + \alpha_h + \gamma_h + f_h(N_h)) I_h,$$

$$\frac{dR_h}{dt} = \alpha_h I_h - (\beta_h + f_h(N_h)) R_h, \tag{3}$$

$$\frac{dS_v}{dt} = \lambda_v N_v - f_v(N_v) S_v - \left(\frac{c_{hv} a_v I_h}{N_h}\right) S_v - \left(\frac{\tilde{c}_{hv} a_v R_h}{N_h}\right) S_v,$$

$$\frac{dE_v}{dt} = \left(\frac{c_{hv} a_v I_h}{N_h}\right) S_v + \left(\frac{\tilde{c}_{hv} a_v R_h}{N_h}\right) S_v - (\nu_v + f_v(N_v)) E_v,$$

$$\frac{dI_v}{dt} = \nu_v E_v - f_v(N_v) I_v,$$

and

$$\frac{dN_h}{dt} = \lambda_h N_h - f_h(N_h) N_h - \gamma_h I_h, \qquad \frac{dN_v}{dt} = \lambda_v N_v - f_v(N_v) N_v, \tag{4}$$

where all parameters in the model are assumed positive and the equations for $N_h = S_h + E_h + I_h + R_h$ and $N_v = S_v + E_v + I_v$ are obtained by adding up the relevant equations in (3). These equations are valid for $N_h > 0$ with $0 \leq S_h/N_h$, I_h/N_h, $R_h/N_h \leq 1$. We interpret those quantities involving division by N_h as zero whenever $N_h = 0$; cf., [32].

We remark here that the above formulation is different from that given in [33] where Esteva and Vargas develop an SIR model for dengue fever, a vector borne disease where the vector is the mosquito *Aedes aegypti*. There they model the rate of recruitment into the infectious human compartment with a term $\lambda_h S_h I_v/N_v$ which will require that λ_h have quasi-dimensional units[4] of per time. However, they go on to explain that this parameter is the product of the average biting rate of the mosquitoes, the proportion of bites that lead to infection, and ratio between the vector population size and the human population size. This gives a quasidimension of bites per human per time to λ_h. Hence, there appears to be a conflict in dimensions there. However, in classical epidemic models involving populations of the same species, contact rates are often assumed to be directly proportional to the total population as in [22], or constant as in [35], or some intermediate form as in [36]. Their approximation, therefore, seems reasonable.[5]

It can be shown, using standard techniques described in [37], that if initial conditions are specified for each of the state variables in system (3) at time $t = 0$ with $S_h(0) + E_h(0) + I_h(0) + R_h(0) = N_h(0)$, then there exists a unique solution satisfying these initial conditions for all $t \geq 0$ with $S_h(t) + E_h(t) + I_h(t) + R_h(t) = N_h(t)$ for all $t \geq 0$. It can also be verified that if $N_h(0) > 0$, then $N_h(t) > 0$ for all t, whereas if $N_h(0) = 0$, then $N_h(t) = 0$ for all t. Of course, similar arguments apply to the vector equations with corresponding expressions. Thus, system (3) is well posed from a mathematical and physical standpoint.

From (4), the equation describing the behaviour of the total vector population has at least two steady-state solutions: $N_v^* = 0$ and $N_v^* = f_v^{-1}(\lambda_v)$. Since, by hypothesis, f_v is a strictly monotonic increasing function of N_v, f_v^{-1} exists and is unique. A linearisation about $N_v^* = 0$ yields the linear approximation $\dot{N}_v = (\lambda_v - f_v(0)) N_v$, where the dot represents differentiation with respect to t. Hence, $f_v(0)$ is the linear death rate for the mosquito population and we assume that $\lambda_v \geq f_v(0) \geq 0$. This further implies that $f_v^{-1}(\lambda_v)$ exists and is nonnegative. Similar

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[4]See, for example, the book by Nåsell [34] for an example of quasi-dimensional units in Ross' model [10].

[5]Nedelman [16] studies the parameters in the mathematical model of Dietz, Molineaux and Thomas [15] and gives reasonable dimensions to parameters such as biting rates.

considerations on the total human population also shows that we require $\lambda_h \geq f_h(0) \geq 0$. An analysis of an SEIR model with generalised density dependent death rates may be found in [32]. Here, for positive constants μ_h, μ_v, μ_{2h}, and μ_{2v}, we consider the forms

$$f_v(N_v) = \mu_v + \mu_{2v}N_v, \qquad f_h(N_h) = \mu_h + \mu_{2h}N_h, \tag{5}$$

for f_v and f_h, so that in the absence of the disease, both populations are modelled by the logistic growth model with carrying capacities $(\lambda_h - \mu_h)/\mu_{2h}$ and $(\lambda_v - \mu_v)/\mu_{2v}$, for the human and vector populations, respectively. These carrying capacities exist and are nonnegative if $\lambda_h \geq \mu_h$ and $\lambda_v \geq \mu_v$.

It is easier to analyse the model in terms of proportions of susceptible, incubating, infectious, and immune individuals, so we make the change of variables

$$u = \frac{S_h}{N_h}, \qquad v = \frac{E_h}{N_h} \qquad w = \frac{I_h}{N_h}, \qquad R = \frac{R_h}{N_h}, \qquad x = \frac{S_v}{N_v}, \qquad y = \frac{E_v}{N_v}, \qquad z = \frac{I_v}{N_v}, \tag{6}$$

so that

$$u + v + w + R = 1 \Rightarrow v = 1 - u - w - R, \qquad x + y + z = 1 \Rightarrow y = 1 - x - y. \tag{7}$$

We also arbitrarily scale time t with the quantity $1/\mu_v$ by setting $\tau = \mu_v t$, the total populations by their respective carrying capacities by setting $N_h = ((\lambda_h - \mu_h)/\mu_{2h})N_h^*$, $N_v = ((\lambda_v - \mu_v)/\mu_{2v})N_v^*$. Hence, we introduce the following dimensionless parameters after dropping the asterisks:

$$\tau = \mu_v t, \qquad \lambda = \frac{\lambda_h}{\mu_v}, \qquad \beta = \frac{\beta_h}{\mu_v}, \qquad \gamma = \frac{\gamma_h}{\mu_v}, \qquad \nu = \frac{\nu_h}{\mu_v}, \qquad r = \frac{r_h}{\mu_v},$$

$$\alpha = \frac{\alpha_h}{\mu_v}, \qquad \epsilon = \frac{\mu_h}{\mu_v}, \qquad \xi(N_h, N_v) = \frac{c_{vh} a_v \mu_{2h}(\lambda_v - \mu_v)N_v}{\mu_{2v}\mu_v(\lambda_h - \mu_h)N_h}, \tag{8}$$

$$a = \frac{\lambda_v}{\mu_v}, \qquad b = \frac{c_{hv} a_v}{\mu_v}, \qquad c = \frac{\tilde{c}_{hv} a_v}{\mu_v}, \qquad e = \frac{\nu_v}{\mu_v},$$

where aside from the parameter, r, we have used Greek letters for parameters relating to human population. System (3) becomes

$$\frac{du}{d\tau} = \lambda(1 - u) + \beta R + rw + \gamma wu - \xi uz,$$

$$\frac{dw}{d\tau} = \nu(1 - u - R) + \gamma w^2 - (r + \alpha + \gamma + \lambda + \nu)w,$$

$$\frac{dR}{d\tau} = \alpha w + \gamma wR - (\beta + \lambda)R, \tag{9}$$

$$\frac{dx}{d\tau} = a(1 - x) - bxw - cxR,$$

$$\frac{dz}{d\tau} = e(1 - x) - (a + e)z,$$

where we have used (7) to eliminate v and y from the system and have written ξ in a form most suitable to emphasize the density dependence in the contact rates. The equations for the total populations, which in fact, determine the behaviour of ξ, now take form

$$\frac{dN_h}{d\tau} = (\lambda - \epsilon)(1 - N_h)N_h - \gamma N_h w, \qquad \frac{dN_v}{d\tau} = (a - 1)(1 - N_v)N_v. \tag{10}$$

We assume that $\lambda - \epsilon - \gamma > 0$, which clearly guarantees the existence of a nonnegative steady-state solution for $N_h(t)$ as $t \to \infty$. Now, the model in terms of proportions, (9), is defined in the subset $\Omega \times [0, \infty)$ of $\mathbf{R}_+^6$, where

$$\Omega = \{u, w, R, x, z : 0 \leq u, w, R, x, z \leq 1, \, 0 \leq u + w + R \leq 1, \, 0 \leq x + z \leq 1\} \tag{11}$$

and the original quantities can be recovered from the proportions through (6)–(8).

3. EXISTENCE OF STEADY-STATE SOLUTIONS

In this section, we present some results concerning the existence of equilibrium or constant solution for the model formulated above. To do this, we shall make use of a threshold parameter, which we shall denote by $\tilde{R}_0$.

PROPOSITION 3.1. *The model formulated in terms of proportions has at least one equilibrium solution $E : (u, w, R, x, z) = (u^*, w^*, R^*, x^*, z^*)$ with u^*, w^*, R^*, x^*, z^* all nonnegative, whose existence and properties are determined by the threshold parameter $\tilde{R}_0$, where*

$$\tilde{R}_0 = \frac{\xi e \nu (\alpha c + b(\beta + \lambda))}{a(a+e)(\beta+\lambda)(\lambda+\nu)(\alpha+r+\gamma+\lambda)}. \tag{12}$$

PROOF. Let $(u^*, w^*, R^*, x^*, z^*)$ be a constant solution of model (9). We easily express u^*, w^*, x^*, and z^* in terms of R^* in the form

$$
\begin{aligned}
w^*(R^*) &= \frac{(\beta+\lambda)R^*}{\alpha+\gamma R^*}, \\
u^*(R^*) &= 1 - R^* + \frac{(\beta+\lambda)R^* \left(\gamma(\beta+\lambda)R^* - M(\alpha+\gamma R^*)\right)}{(\alpha+\gamma R^*)^2 \nu}, \\
x^*(R^*) &= \frac{a\left(\alpha+\gamma R^*\right)}{a\alpha + (\alpha c + b(\beta+\lambda) + a\gamma)R^* + c\gamma R^{*2}}, \\
z^*(R^*) &= \left(\frac{e}{a+e}\right) \frac{(\alpha c + b(\beta+\lambda) + c\gamma R^*)R^*}{a\alpha + (\alpha c + b(\beta+\lambda) + a\gamma)R^* + c\gamma R^{*2}}.
\end{aligned} \tag{13}
$$

Substituting these in the first of (9) and equating to zero yields a sixth-order polynomial in R^* of the form

$$R^* \left(A_5 R^{*5} + A_4 R^{*4} + A_3 R^{*3} + A_2 R^{*2} + A_1 R^* + A_0 \right) = 0, \tag{14}$$

where

$$
\begin{aligned}
A_5 ={}& acD\tilde{R}_0\gamma^4\nu^2\xi, \\
A_4 ={}& \gamma^3\nu \left(Ac(\beta+\lambda)(-B + a\beta(\beta+\lambda) + a\alpha\nu) + AD\left(c + a\tilde{R}_0\right)\xi \right. \\
& \left. + cD\tilde{R}_0\left(B - a(\beta+\lambda)^2 + a(2\alpha - \gamma)\nu\right)\xi\right), \\
A_3 ={}& \gamma^2 \left(A(\beta+\lambda)(A(-B + a\beta(\beta+\lambda) + a\alpha\nu) \right. \\
& + \nu(-(B(c\alpha + a\gamma)) + a(\beta+\lambda)(a\beta\gamma - c\alpha\lambda) + a\alpha(c\alpha + a\gamma)\nu)) \\
& + D\left(A^2 + A\tilde{R}_0\left(B - a(\beta+\lambda)^2\right) + A\left(2c\alpha + a\left(2\tilde{R}_0\alpha + \gamma - \tilde{R}_0\gamma\right)\right)\nu \right. \\
& \left.\left. + c\tilde{R}_0\alpha\nu\left(2B - a(\beta+\lambda)^2 + a(\alpha - 3\gamma)\nu\right)\right)\xi\right), \\
A_2 ={}& \alpha\gamma \left(A(\beta+\lambda)(-(A(B + a\lambda(\beta+\lambda) - a\alpha\nu)) \right. \\
& + a\gamma\nu\left(-2B + a\left(\beta^2 - \lambda^2 + 2\alpha\nu\right)\right)) + D\left(2A^2 + A\tilde{R}_0\left(2B - a(\beta+\lambda)^2\right)\right. \\
& \left.\left. + A\left(\left(c + a\tilde{R}_0\right)\alpha - 3a\left(-1 + \tilde{R}_0\right)\gamma\right)\nu + c\tilde{R}_0\alpha\nu(B - 3a\gamma\nu)\right)\xi\right), \\
A_1 ={}& \alpha^2 \left(-(aA\gamma(\beta+\lambda)\nu(B + a\lambda(\beta+\lambda) - a\alpha\nu)) \right. \\
& \left. + D\left(A^2 + AB\tilde{R}_0 - 3aA\left(-1 + \tilde{R}_0\right)\gamma\nu - ac\tilde{R}_0\alpha\gamma\nu^2\right)\xi\right), \\
A_0 ={}& aAD\alpha^3\nu\left(1 - \tilde{R}_0\right)\xi,
\end{aligned}
$$

and

$$
\begin{aligned}
A &= \nu(\alpha c + b(\beta+\lambda)), \qquad B = a(\alpha\nu + M(\beta+\lambda)), \\
D &= \frac{a}{\xi}(\beta+\lambda)(\nu(\alpha+\gamma+r) + \lambda M), \\
M &= \alpha + r + \gamma + \nu + \lambda.
\end{aligned} \tag{15}
$$

Clearly, $R^* = 0$ is a solution. Notice that A_5 is positive while the sign of A_0 coincides with that of $(1 - \tilde{R}_0)$, so that if $\tilde{R}_0 > 1$, $A_0 < 0$, in which case, we have at least one sign change in the sequence of coefficients $\{A_5, \ldots, A_0\}$. Hence, by Descartes Rule of signs, there exists at least one positive real root for (14) aside from the root $R^* = 0$, whenever $\tilde{R}_0 > 1$. ∎

REMARK. From an epidemiologically realistic point of view, all we require is that there be a solution $R^* \in [0, 1]$ satisfying (14). When such a solution exists, we call it realistic and values for the other steady states are given by (13). When $R^* = 0$, the steady state postulated by Proposition 3.1 is the solution $E_0 : (u, w, R, x, z) = (1, 0, 0, 1, 0)$, called the disease-free equilibrium DFE. We thus have the following result.

PROPOSITION 3.2. *If $N_v = 0$ or any of ν, e, or ξ is zero, then the only realistic solution R^* of (14) is the solution $R^* = 0$ and the model formulated in terms of proportions has only the disease-free equilibrium $E_0 : (u, w, R, x, z) = (1, 0, 0, 1, 0)$ as a constant solution.*

PROOF. If $N_v = 0$, then from (8), $\xi = 0$ and the first of (9) shows that the only possible nonnegative constant solution for the system is the solution E_0. The rest of the proposition follows immediately upon substitution of these values of ξ, ν, or e in the coefficients of (14). ∎

Proposition 3.2 gives us some conditions under which the parameter $\tilde{R}_0$ can vanish. Since the parameter ξ is a grouping of many variables including the total vector and host populations, the condition $\xi = 0$ can be interpreted in a variety of ways. For example, $\xi = 0$ could mean that the transmission probability is zero or that the total vector population is zero, $\xi \to 0$ as $N_v \to 0$. The form of polynomial (14) clearly shows that the DFE always exist. When $\tilde{R}_0 > 1$, a second equilibrium different from the DFE is established, as is seen in the following result.

PROPOSITION 3.3. *Let $\tilde{R}_0 > 1$, then the condition*

$$\alpha\lambda - \beta\gamma \geq 0 \tag{16}$$

is a sufficient condition to guarantee the existence of at least one value $R^* \in (0, 1)$ that solves (14). That is, when $\tilde{R}_0 > 1$, the model formulated in terms of proportions has at least one realistic equilibrium solution different from the disease-free equilibrium, called the endemic equilibrium. Moreover, when $\gamma = 0$, this endemic equilibrium, $E_{\gamma=0}$, is unique and is expressible in terms of $\tilde{R}_0$.

PROOF. Consider the function $g : \mathbf{R} \to \mathbf{R}$ defined by

$$g(R^*) = A_5 R^{*5} + A_4 R^{*4} + A_3 R^{*3} + A_2 R^{*2} + A_1 R^* + A_0,$$

where the coefficients A_i, $i = 0, \ldots, 5$ are those of (14). We easily verify that

$$g(0) = A_0 = \frac{ea\nu A^2 \alpha^3}{a + e} \left(\frac{1 - \tilde{R}_0}{\tilde{R}_0} \right).$$

Hence, $g(0) < 0$, when $\tilde{R}_0 > 1$. Some algebraic manipulation shows that

$$\begin{aligned}
g(1) = (\beta + \lambda) &\Big(aM\tilde{R}_0 (\alpha + \gamma) \lambda (\beta + \lambda) (c(\alpha + \gamma) + b(\beta + \lambda)) (M(\alpha + \gamma) - \gamma(\beta + \lambda)) \\
&+ (M(\alpha + \gamma) - \gamma(\beta + \lambda)) ((-\beta\gamma + \alpha\lambda)(c\alpha + b(\beta + \lambda))(c(\alpha + \gamma) + b(\beta + \lambda)) \\
&+ a(\alpha + \gamma) \Big(c\beta \Big(\tilde{R}_0 \alpha (r + \alpha) + \Big(r\tilde{R}_0 + \Big(-1 + 2\tilde{R}_0 \Big) \alpha \Big) \gamma + \tilde{R}_0 \gamma^2 \Big) \\
&+ c\Big(\alpha \Big(\alpha + \tilde{R}_0 (r + \alpha) \Big) + \tilde{R}_0 (r + 2\alpha) \gamma + \tilde{R}_0 \gamma^2 \Big) \lambda + b(\beta + \lambda) \Big(\tilde{R}_0 (r + \alpha) \beta \\
&+ \Big(-1 + \tilde{R}_0 \Big) \beta\gamma + \Big(\alpha + \tilde{R}_0 (r + \alpha + \gamma) \Big) \lambda \Big) \Big) \nu \\
&+ (\alpha + \gamma)^2 (r + \alpha + \gamma)(c\alpha + b(\beta + \lambda))((a + c)(\alpha + \gamma) + b(\beta + \lambda))\nu^2 \Big).
\end{aligned}$$

It is now a trivial matter to see that when condition (16) holds, $g(1) \geq 0$ when $\tilde{R}_0 > 1$. The existence of the root $R^* \in (0,1)$ follows from the intermediate value theorem. Now, when $\gamma = 0$, (14) reduces to a first-degree polynomial in R^* and using the parameter groupings in (15), we easily establish the equilibrium solutions

$$u^* = \frac{A+B}{A+B\tilde{R}_0}, \qquad w^* = \frac{a\nu(\beta+\lambda)\left(\tilde{R}_0 - 1\right)}{A+B\tilde{R}_0},$$

$$R^* = \frac{\nu\alpha a\left(\tilde{R}_0 - 1\right)}{A+B\tilde{R}_0}, \qquad x^* = \frac{A+B\tilde{R}_0}{(A+B)\tilde{R}_0}, \tag{17}$$

$$z^* = \frac{D\left(\tilde{R}_0 - 1\right)}{A+B},$$

which are clearly realistic only when $\tilde{R}_0 > 1$ with $\tilde{R}_0 = 1$ giving the DFE.   ∎

From the remark above, the constant solutions postulated by Proposition 3.1 are realistic if and only if each of them lies in the compact interval $[0,1]$. Simple calculations show that $R^* = 1$ is not realistic because u^* would then be negative. So we shall assume that $0 \leq R^* < 1$. Similarly, $w^* = 1$ is not realistic and so we require $0 \leq w^* < 1$. Using these in (13), we have

$$0 \leq w^* < 1 \ \Rightarrow\ 0 \leq \frac{(\beta+\lambda)R^*}{\alpha+\gamma R^*} < 1 \ \Rightarrow\ 0 \leq R^* \leq \frac{\alpha}{\beta+\lambda-\gamma} < 1. \tag{18}$$

Observe that the last inequality in (18) is strictly less than 1. Otherwise, we have $w^* = 1$, which is not allowed. It is then a simple matter to demonstrate that when the parameters are chosen so that (16) and (18) are satisfied, then u^* as defined by (13) lies in the compact interval $[0,1]$. The corresponding steady-state values for the total vector and human populations are computed from (10). The steady-state solution for which the total vector and human populations are both zero is not realistic in the sense that we have nothing to prove (both populations are extinct!). One easily sees from (10) that when $\lambda < \epsilon$ (respectively, $a < 1$), the total human (respectively, vector) population tends to zero as $t \to \infty$. Here, we consider only the cases $\lambda > \epsilon$ (respectively, $a > 1$), from which it is easy to see that, in the absence of the disease, there is exponential growth of both populations near $N_h = 0$ (respectively, $N_v = 0$) with saturation near $N_h = 1$ (respectively, $N_v = 1$). In the presence of the disease, when $\gamma = 0$, the endemic equilibria for both the vector and total populations are $(N_h, N_v) = (1,1)$. As disease related deaths set in, a new equilibrium for the total human population, whose size is determined by the relative magnitude of γ, is established. This equilibrium is obtained by substituting the steady-state value w^* into the appropriate equation from (10). This gives

$$N_h^* = 1 - \frac{\gamma}{\lambda-\epsilon}w^*. \tag{19}$$

Observe that if the disease related death rate is severe enough (i.e., γ is large enough), this equilibrium can cease to exist. Using w^* from (13), we have

$$0 \leq N_h^* \leq 1 \ \Rightarrow\ 0 \leq R^* \leq \frac{(\lambda-\epsilon)\alpha}{\gamma(\beta+\epsilon)}.$$

Hence, the endemic equilibrium R^* when it exists must be a root of polynomial (14) and, in addition, should satisfy

$$0 \leq R^* < \min\left\{1, \frac{(\lambda-\epsilon)\alpha}{\gamma(\beta+\epsilon)}, \frac{\alpha}{\beta+\lambda-\gamma}\right\}. \tag{20}$$

This minimum will exist whenever $\lambda \geq \epsilon$ since $\beta+\lambda > \gamma$.

There are two distinct ways of considering a disease as being brought under control in a population of varying size. The stricter way is to demand that the total number of infectives, i.e., the reservoir of infection, here I_h, I_v (and possibly R_h) $\to 0$ with increasing time, while a weaker demand will be that the proportions w, z (and possibly R) tend to zero with increasing time; cf., [38,39]. Thus, we shall seek conditions for the stability of the endemic proportional state $(u^*, w^*, R^*, x^*, z^*)$ with $w^* > 0$, $R^* > 0$, and $z^* > 0$ and for the stability of the DFE $(u^*, w^*, R^*, x^*, z^*) = (1, 0, 0, 1, 0)$. We will see in Section 4 that the stability of these equilibria depend critically on the parameter $\tilde{R}_0$, defined by (12), and on condition (16). The parameter $\tilde{R}_0$ is the *basic reproduction ratio*. It is usually defined as the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual during its entire period of infectiousness, and mathematically as the dominant eigenvalue of a positive linear operator [40].

In our formulation, ξ depends on the transmission probabilities, the man-biting rate of the mosquitoes, the carrying capacities of the environment for both species, as well as on the ratio between the vector and human populations. Hence, the behaviour of the proportions depend strongly on the behaviour of the total vector and human populations. These total populations are observable, and can be measured in a given population, making our representation meaningful. Here $\tilde{R}_0$ increases with increasing vector population.

4. STABILITY OF THE EQUILIBRIA

The local stability of the equilibrium solutions can be examined by linearising system (9) about the equilibrium solution $(u^*, w^*, R^*, x^*, z^*)$. This gives the Jacobian matrix

$$
J_E = \begin{pmatrix}
\gamma w^* - \lambda - \xi z^* & r + \gamma u^* & \beta & 0 & -\xi u^* \\
-\nu & 2\gamma w^* - M & -\nu & 0 & 0 \\
0 & \alpha + \gamma R^* & \gamma w^* - \beta - \lambda & 0 & 0 \\
0 & -bx^* & -cx^* & -\dfrac{a}{x^*} & 0 \\
0 & 0 & 0 & -e & -(a+e)
\end{pmatrix}, \qquad (21)
$$

where we have used the fact that $a(1 - x^*) - bx^*w^* - cx^*R^* = 0$ to replace $a + bw^* + cR^*$ by a/x^*. The eigenvalues of J_E are the solutions of the fifth-order polynomial equation

$$
\zeta^5 + a_1\zeta^4 + a_2\zeta^3 + a_3\zeta^2 + a_4\zeta + a_5 = 0, \qquad (22)
$$

where

$$
a_1 = a + e + \frac{a}{x^*(R^*)} + B_1 + B_2 + B_3,
$$

$$
\begin{aligned}
a_2 = {}& \frac{a}{x^*(R^*)}(a+e) + \left(a + e + \frac{a}{x^*(R^*)}\right)(B_1 + B_2 + B_3) \\
& + B_1(B_2 + B_3) + B_2 B_3 + \nu(B_4 + B_5),
\end{aligned}
$$

$$
\begin{aligned}
a_3 = {}& \frac{a}{x^*(R^*)}(a+e)(B_1 + B_2 + B_3) \\
& + \left(a + e + \frac{a}{x^*(R^*)}\right)(B_1(B_2 + B_3) + B_2 B_3 + \nu(B_4 + B_5)) \\
& + B_1(B_2 B_3 + \nu B_4) + \nu(B_5 B_3 + \beta B_4),
\end{aligned}
$$

$$
\begin{aligned}
a_4 = {}& \frac{a}{x^*(R^*)}(a+e)(B_1(B_2 + B_3) + B_2 B_3 + \nu(B_4 + B_5)) \\
& + \left(a + e + \frac{a}{x^*(R^*)}\right)(B_1(B_2 B_3 + \nu B_4) + \nu(B_5 B_3 + \beta B_4)) \\
& - \xi e \nu u^*(R^*) x^*(R^*) b,
\end{aligned}
$$

$$a_5 = \frac{a}{x^*(R^*)}(a+e)(B_1(B_2B_3 + \nu B_4) + \nu B_5 B_3 + \beta B_4)$$
$$- \xi e \nu u^*(R^*) x^*(R^*)(cB_4 + bB_3),$$

with B_i, $i = 1, \ldots, 5$ functions of the steady-state solution R^* given by (13). Here,

$$\begin{aligned}
B_1 &= \lambda + \xi z^*(R^*) - \gamma w^*(R^*), & B_2 &= M - 2\gamma w^*(R^*), \\
B_3 &= \beta + \lambda - \gamma w^*(R^*), & B_4 &= \alpha + \gamma R^*, \\
B_5 &= r + \gamma u^*(R^*).
\end{aligned} \qquad (23)$$

Now, from a stability point of view, all we wish to know is whether there exists a value ζ, that is a solution to (22) with $\Re e(\zeta) > 0$. If such a ζ exist, then the equilibrium solution is locally unstable to small perturbations, otherwise, it is locally and asymptotically stable.

Using the expressions in (13), we have

$$\begin{aligned}
B_1 &= \lambda + \xi z^*(R^*) - \gamma w^*(R^*) = \xi z^*(R^*) + \frac{\lambda\alpha - \gamma\beta R^*}{\alpha + \gamma R^*} > 0, \\
B_2 &= M - 2\gamma w^*(R^*) = (\alpha + \nu + r) + \frac{\alpha\lambda - \gamma\beta R^*}{\alpha + \gamma R^*} + \gamma(1 - w^*(R^*)) > 0, \\
B_3 &= \beta + \lambda - \gamma w^*(R^*) = (\beta + \lambda)\left(1 - \frac{\gamma R^*}{\alpha + \gamma R^*}\right) > 0,
\end{aligned}$$

since $\lambda\alpha - \gamma\beta \geq 0$ from (16) and $0 \leq w^*, R^*, x^* < 1$. Hence, all groupings in the coefficients of (22) are positive. The coefficients $a_1, \ldots, a_3$ are always positive; a_4 and a_5 could be negative. Given equations (16) and (18) and the expression $\tilde{R}_0$ give by (12), we can show that whenever $\tilde{R}_0 > 1$, a_4 and a_5 are both positive. Hence, there are no positive real roots for polynomial (22). The special case $\gamma = 0$ is illuminating. We have the following results.

PROPOSITION 4.1. *The disease-free equilibrium is locally and asymptotically stable when $\tilde{R}_0 \leq 1$. Moreover, when $\gamma = 0$ and $\tilde{R}_0 > 1$, the unique endemic equilibrium $E_{\gamma=0}$, given by Proposition 3.3 is also locally and asymptotically stable.*

PROOF. The local stability of the DFE is determined by considering the value $R^* = 0$ in (22). Since the coefficients a_1, a_2, and a_3 are nonnegative, it suffices to show that a_4 and a_5 are also positive when $\tilde{R}_0 \leq 1$ and $R^* = 0$. This can easily be seen by making ξ, say, the subject of equation (12) and substituting the resulting value of ξ together with $R^* = 0$ in a_4 and a_5. When $\gamma = 0$, the nonzero positive value for R^*, which exists for $\tilde{R}_0 > 1$ is given by (17). Again, we easily verify that for this value of R^*, a_4 and a_5 are positive when $\tilde{R}_0 > 1$. The positivity of all the coefficients imply that there are no positive real roots for polynomial (22) satisfying the required conditions. The necessary and sufficient condition for local asymptotic stability then follows from the Routh-Hurwitz conditions applied to polynomial (22). The straightforward but rather lengthy calculations are omitted. ∎

The next result concerns the global stability of the disease-free equilibrium. We use the direct method of Liapunov; [37,41].

PROPOSITION 4.2. *The DFE is globally and asymptotically stable if $\tilde{R}_0 \leq 1$.*

PROOF. Consider the function $\ell : \Omega \times [0, \infty) \to \Re$ defined by

$$\ell = \frac{\lambda}{\lambda + \nu}(R + w) + \frac{\nu}{\nu + \lambda}(1 - u) + \frac{a}{a + e}z + \frac{e}{a + e}(1 - x). \qquad (24)$$

Clearly $\ell > 0 \, \forall (u, w, R, x, z) \in \Omega \setminus \{E_0\}$ where Ω is the region defined in (11) and $\{E_0\}$ is the

singleton $\{(1,0,0,1,0)\} \in \Omega$. Some calculations show that

$$
\begin{aligned}
\frac{d\ell}{d\tau} =& \left(\frac{eb}{a+e} - (r+\lambda)\right) w + \left(\frac{ec}{a+e} - (\beta+\lambda)\right) R \\
&+ \left(\frac{\xi\nu}{\lambda+\nu} - a\right) z - \gamma Ru - \frac{\gamma\lambda}{\lambda+\nu} wv - \left(\frac{\xi\nu}{\lambda+\nu} + \frac{ec}{a+e}\right) Rz \\
&- \left(\frac{\xi\nu}{\lambda+\nu} + \frac{eb}{a+e}\right) wz - \frac{\xi\nu}{\lambda+\nu} vz - \left(\frac{eb}{a+e} w + \frac{ec}{a+e} R\right) y,
\end{aligned}
\tag{25}
$$

from which we deduce that the orbital derivative of ℓ in $\Omega \setminus \{E_0\}$ is nonpositive whenever

$$
b \le \frac{(r+\lambda)(a+e)}{e}, \qquad c \le \frac{(\beta+\lambda)(a+e)}{e}, \qquad \xi \le \frac{a(\lambda+\nu)}{\nu}.
\tag{26}
$$

We easily verify that inequalities (26) put together imply that $\tilde{R}_0 \le 1$. ℓ is a Liapunov function for the system. The Liapunov-LaSalle theorem [36] assures us that all paths in $\Omega \setminus \{E_0\}$ approach the largest positively invariant subset $\tilde{\Omega} \subset \Omega$, wherein

$$
\frac{d\ell}{d\tau} = 0.
$$

$\tilde{\Omega}$ is the set $\{(w = 0, R = 0, z = 0)\}$. Hence, $(w, R, z) \to (0, 0, 0)$ as $\tau \to \infty$. Now on the u- and x-axes, we have the equations $\dot{u} = \lambda(1 - u)$ and $\dot{x} = a(1 - x)$, where the dot represents differentiation with respect to τ. These imply that $(u(\tau), x(\tau)) \to (1, 1)$ as $\tau \to \infty$. Hence, E_0 is the only omega limit point of system (9) on the boundary of Ω, and therefore, cannot be the omega limit point of any other orbit starting in the interior of Ω. ∎

Propositions 4.1 and 4.2 state that there are two possible realistic equilibrium points: one where the disease has died out proportionally and the other, if $\tilde{R}_0 > 1$, where there is a unique endemic equilibrium. $\tilde{R}_0$ is a unique threshold parameter which determines the behaviour of the system. Assuming that the stability result for the endemic equilibrium is also global, then assuming that initially there is at least one infectious mosquito (or human), then if $\tilde{R}_0 \le 1$, we expect the disease to die out proportionally, whereas if $\tilde{R}_0 > 1$, then we expect the disease to tend proportionally to the unique endemic equilibrium, thereby establishing itself in the community.

5. SIMULATIONS

In the last section, we analysed the model formulated in terms of proportions. We showed that the endemic equilibrium, when it exists, is locally and asymptotically stable. In this section, we demonstrate numerically that the model formulated in terms of proportions does indeed possess a globally and asymptotically stable endemic equilibrium solution if $\tilde{R}_0 > 1$ whenever the initial conditions are prescribed in the region Ω defined by (11). To achieve this, a program was written in Fortran to integrate equations (9),(10) and the output was comprehensively verified using a detailed output from a number of runs.

To explore the behaviour of the system and to demonstrate the global stability of the endemic equilibrium, we select parameters bearing in mind the nondimensionalisation (8). For a given set of parameters, the value of ξ depends on the values of the corresponding nonzero steady-state values of N_v^* and N_h^*. We note from (10) that when $\gamma = 0$, $(N_h^*, N_v^*) = (1, 1)$. We selected the value $\lambda = 0.00184$ which, in dimensional terms, corresponds to approximately a per capita human birth rate of 28 births per thousand per year, $\beta = 0.35$, which in dimensional terms corresponds to a period of immunity of approximately 68.5 days. As β is increased, the period of immunity is reduced and as $\beta \to \infty$, the model becomes the standard SEIS model in epidemiology. We report results for $\gamma = 0.001$ which, in dimensional terms, corresponds to a disease-induced death rate of approximately 1.52%. We set $\nu = 2.0$, which corresponds to an intrinsic incubation period of

12 days. We assume that a smaller proportion of humans recover from infection without gaining immunity and set $r = 0.2$, $\alpha = 0.3$. These correspond to an infectious period of between 80 and 120 days for individuals in each category. We assume that the mosquito population has a net positive growth rate and take $a = 1.002$. Following estimates given in [17,18,20], we take $b = 10.0$. This gives the transmission rate from human to vector to be about 0.4167. Though we assume that immune human individuals may still be infectious, we assume that the infectiousness of members from this class is small compared to that of members in the infectious compartment and arbitrarily set $c = b/10$. We take $e = 2.4$ which corresponds to an extrinsic incubation period of about ten days. We have assumed a mosquito life span of about 24 days. The values of μ_{2h} and μ_{2v}, c_{vh} etc., are lumped into the parameter ξ which also contains the ratio of the vector and mosquito populations at the equilibrium.

At time $t = 0$, we have the following initial conditions in the proportions: $u(0) = 0.85$, $w(0) = 0.05$, $R(0) = 0.04$, $x(0) = 0.999999$, $z(0) = 0.000001$. The program was run for different sets of initial conditions and the qualitative form of the final finite amplitude steady-state solutions where the same. The simplest type of initial data which considered the whole population to be essentially susceptible and introduced one infectious mosquito (or human) at time $t = 0$, was also tried and tested. The results where qualitatively the same as those for the other cases. However, the general form of the solutions is determined by the way in which the quantity $\xi(N_h, N_v) \rightarrow \xi(N^*, 1)$ and on the relative growth rates of the two populations. The general behavior of the model is shown in Figures 1–4, where we plot the proportions relative to the total populations with time. These figures clearly show that the endemic steady-state solution is unique and is globally and asymptotically stable.

Figures 1 and 2, respectively, show the initial and long-term behaviour of the proportions, in the human population, plotted on the same graph with the total population to show the relative sizes of the proportions. While Figure 3 shows the behaviour of the system for smaller values
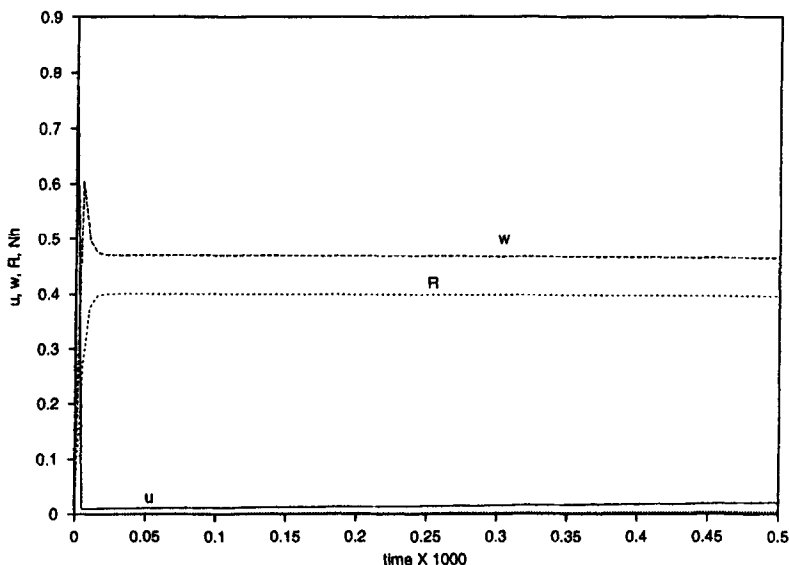

Figure 1. Initial behaviour of the proportions u, w, R, and the total human population N_h with time. The parameters are $\gamma = 0.001$, $\xi = 4.066$, $\alpha = 0.3$, $r = 0.2$ $\beta = 0.35$, $\lambda = 0.00184$, $\nu = 2.0$, $b = 10$, $c = 1.0$, $\lambda - \epsilon = 0.005$, $a = 1.002$, $e = 2.4$. For these, $\bar{R}_0 = 61.722$. As time increases from zero, the proportion of susceptible drops while those of infectious and removed classes increases. The proportion w peaks and drops again capturing the typical behaviour of infectious individuals during an epidemic.
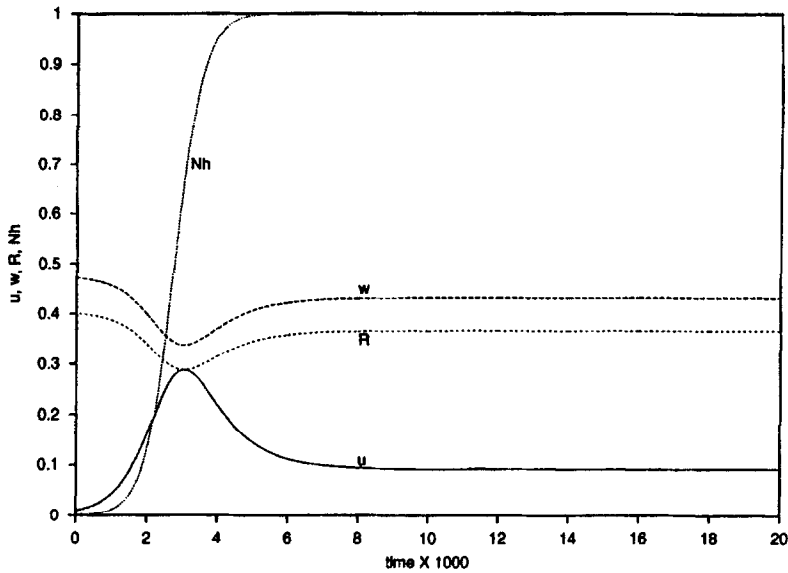
Figure 2. Long-term behaviour of the solutions shown in Figure 1. As more and more individuals lose their immunity and join the susceptible, the proportion of susceptible rises, after the initial drop, and then equilibrates at a final finite amplitude steady state. There is a depression in the reservoir of infections as the proportion of susceptible peaks to its maximum.
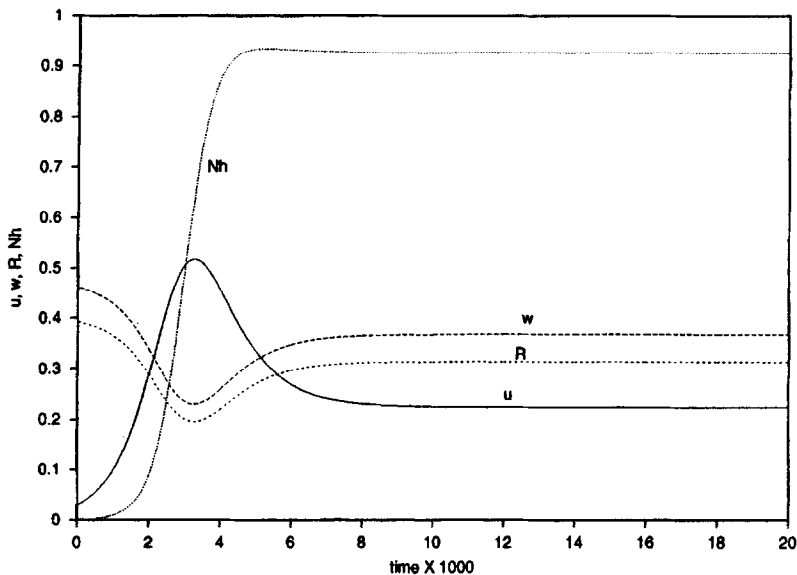

Figure 3. Diagram showing the long-term behaviour of the proportions in the human populations. $\xi = 1.3554$ and all other parameters are as in Figure 1. $\tilde{R}_0 = 20.57$. Observe that the proportions in the reservoir of infections are smaller in this case than in those of Figures 1 and 2. A large $\tilde{R}_0$, as is the case in Figures 1 and 2, implies that more individuals end up being infected. Because of disease related deaths, the total human population does not reach its carrying capacity, here normalised to one.

of ξ, Figure 4 shows the long-term behaviour of the proportions in the mosquito population. Here z is very high, showing that we are indeed in an endemic regime.

Since the threshold parameter $\tilde{R}_0$ determines whether or not the disease will establish itself permanently in the population, we show how this parameter varies with the reservoir of infections by plotting $w + R$ against $\tilde{R}_0$ in Figure 5. Our result coincides with the results obtained for
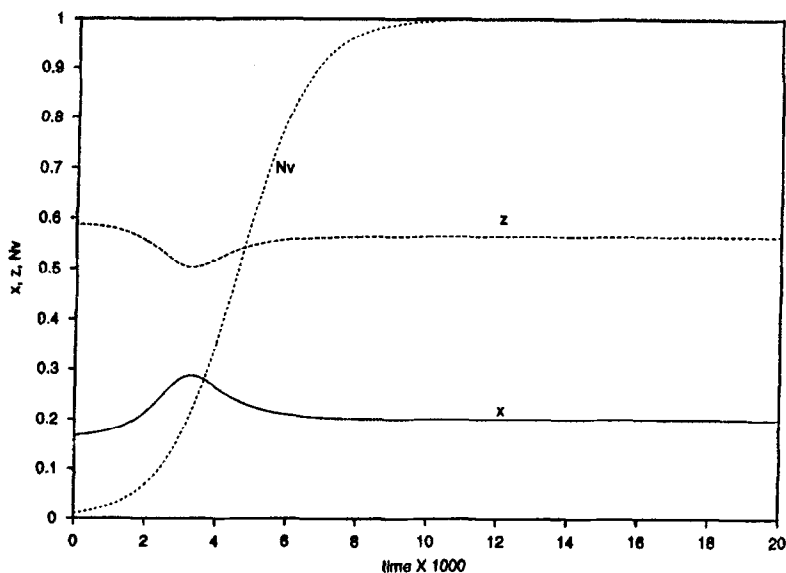
Figure 4. Diagram showing the long-term proportions in the vector population. The parameters are as in Figure 1. The initial conditions are explained in the text. With this parameter regime, more than 50% of the mosquitoes are infectious indicating the endemicity and prevalence of infection.
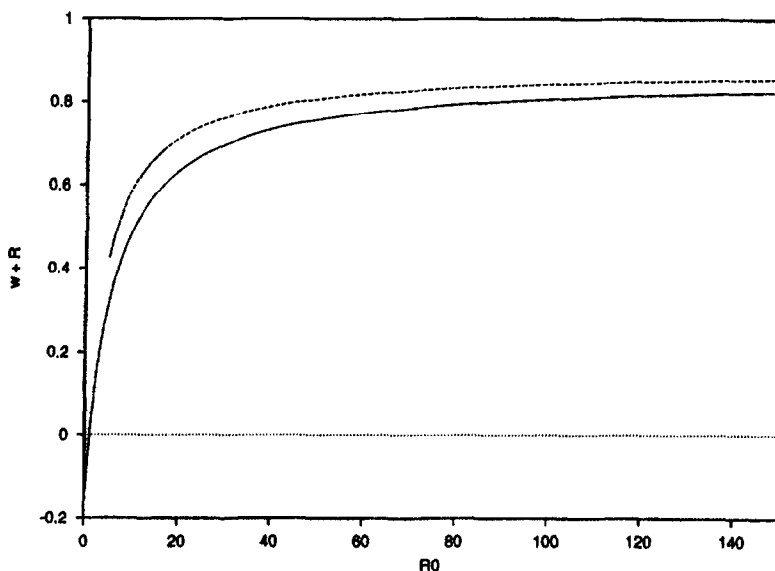

Figure 5. Diagram showing the behaviour of the reservoir of infection in the human population, $w + R$, as a function of the basic reproduction ratio $\tilde{R}_0$. In this figure, the dashed line is the computation from the numerical simulations while the solid curve is the theoretical prediction as defined by the steady-state solutions (13) when $\gamma = 0$. Parameters: $\lambda = 0.00184$, $\beta = 0.35$, $\gamma = 0$, $\xi = 1.355428$, $\nu = 2.0$, $r = 0.2$, $\alpha = 0.3$, $a = 1.002$, $b = 10.0$, $c = 1.0$, $e = 2.4$, $\lambda - \epsilon = 0.005$, as explained in the text.

epidemiological models. See, for example, [42,43]. Observe that though the proportions of humans in the reservoir of infections $w + R$ increases as $\tilde{R}_0$ increases, it also saturates for larger values of $\tilde{R}_0$. One can clearly see that the prevalence level (proportions in the reservoir of infections) varies with the basic reproduction ratio, only in a narrow range of values of the basic reproduction ratio, namely, when $\tilde{R}_0$ is not much above 20. Then small changes in $\tilde{R}_0$ lead to large changes in

the reservoir of infection. If $\tilde{R}_0$ is below one, then the prevalence of the disease is obviously always zero. In our model, if $\tilde{R}_0$ is above 50, then even large changes in $\tilde{R}_0$, which are often effected by large-scale spraying with insecticides to reduce mosquito populations, will be associated with very small changes in prevalence rates. This behaviour has far-reaching consequences in an attempt to control endemic malaria for it says that if the initial contact rate is far above its critical value one, then even large reductions in mosquito populations will show very little effect. This is especially so when these mosquitoes are given a chance to recover.

6. CONCLUDING REMARKS

In this paper, we have developed an SEIRS model for the dynamics and transmission of malaria which can be used to study other vector/indirectly transmitted diseases. We started off by briefly reviewing available literature on previous work in this area. Though mathematical models in malaria in particular and vector borne diseases in general are well established, the unrealistic assumption of a constant population size or the pseudoequilibrium hypothesis is often made. Following ideas advanced by Aron [24–26], we thus developed an SEIR-type model where attention was paid to the dynamics of the vector population and the class of immune human individuals; this class was assumed infectious. Though our primary objective had been to study malaria transmission, our model has applications to other infectious diseases of humans such as dengue fever, yellow fever, and sleeping sickness.

We reformulated the differential equation model in terms of proportions of susceptible, incubating, infectious, and removed (or partially immune) individuals in both vector and human populations. We showed that our model has a unique solution. We next examined the existence of equilibrium solutions to this model and gave conditions that are sufficient for the existence of realistic equilibria. The results of our model fit into the pattern of previously analysed models. There is a threshold parameter $\tilde{R}_0$ and the disease can persist if and only if $\tilde{R}_0$ exceeds one. The disease-free equilibrium always exists and is locally stable if $\tilde{R}_0 < 1$ and unstable if $\tilde{R}_0 > 1$. We showed that the disease-free equilibrium is globally stable when $\tilde{R}_0 \leq 1$. For $\tilde{R}_0 > 1$, our analysis showed that there exists an endemic equilibrium which is locally and asymptotically stable. Numerical simulations confirmed this result.

The behaviour of the proportion of infectious individuals in the human population, which we have called the reservoir of infection, with respect to the basic reproduction ratio, agrees with previously established results in the literature. See, for example, [42–44]. Our model shows that as long as the net growth rate of the mosquito population is positive, abruptly reducing the density of the mosquito population will not have a significant effect on the prevalence of malaria in endemic regions where the basic reproduction ratio $\tilde{R}_0$ is large. As each abrupt reduction may only result in a depression in the disease prevalence and the number of cases will start rising once the mosquito population recovers. Our model, therefore, provides a framework for studying control strategies for the containment of malaria.

We have not presented analytic global stability results for the endemic equilibrium. This and other aspects of the model such as Aron's [24] theory of acquired immunity by boosted exposure, the modification of the recovery rates to include the use of antimalarial drugs are aspects under investigation.

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