

A model for endemic malaria with delay and variable population

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ABSTRACT

We derive and study an SEIS deterministic differential equation model for endemic malaria with variable human and mosquito populations. Mosquito deaths in earlier life stages and a delay, T , to cater for the time lapse between egg laying and adult mosquito eclosion are explicitly included. For $T=0$, oscillatory solutions are not possible. Conditions are derived for the existence, uniqueness and stability of the equilibria in the model. We show that the stability or instability of the positive vector equilibrium solution depends strongly on the size of the parameter T . We identify a threshold parameter R_0 , and show that the disease free equilibrium always exists and is locally and asymptotically stable when $R_0 < 1$. We show that the prevalence of malaria in endemic regions can be discussed simply by measuring the proportions of susceptible humans and mosquitoes at equilibrium.

Key words: Endemic equilibrium, threshold, delay.

RESUME

Nous avons dérivé et étudié un modèle déterminatif d'équation différentielle (SEIS) pour l'étude du paludisme endémique avec comme variables les populations humaine et de moustique. La mortalité des moustiques aux stades larvaires et le délai, T de développement entre la ponte des oeufs et la mue imaginale ont été explicitement considérés dans la modèle. A $T=0$, il n'y a pas de solution oscillatoire. Les conditions démontrons que la stabilité ou l'instabilité de la solution positive du vecteur à paramètre seuil, R_0 et démontré que l'équilibre des populations non infectées existe toujours et est localement et asymptotiquement stable quand $R_0 < 1$. Nous démontrons que la prévalence du paludisme en zone endémique peut être comprise simplement par l'estimation des proportions des humains et des moustiques susceptibles à l'équilibre.

Mots clés: *Equilibre endémique, seuil, délai*

Introduction

Malaria is a parasitic vector-borne disease endemic in many parts of the world. At present, at least 300 million people are affected world wide and there are between 1 - 1.5 million malaria related deaths annually Bradley (1996). 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 (Giles and Warrel (1993), WHO Reports (1980-1999), Elizabeth et al. (1992)). An excellent source of medical information on various diseases is found in Benenson (1990).

The literature on the biology of malaria is vast. See, for example, Ngwa and Shu (2000) for a good review and the references therein. 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. 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. With such diseases, the effects of changes in population size and

disease induced mortality are far from negligible can crucially influence disease dynamics. Here, we focus our attention on an endemic region and derive a deterministic mathematical model for the dynamics of malaria transmission in varying human and mosquito populations; factors which have hitherto been omitted in most mathematical models. Disease induced deaths are also explicitly included. Our model also incorporates mosquito deaths in life stages prior to the adult stage and a delay T to account for the time lapse between egg laying and adult mosquito eclosion. This delay could also be regarded as a parameter to capture seasonality. In fact, variations in climatic conditions have a profound effect on the life of mosquitoes, the development of the malaria parasite and consequently the incidence of malaria. The regular changes in mosquito population density caused by seasonality also explain interesting patterns in the overall dynamics of malaria. It is obvious that a rise in mosquito population can lead to a malaria epidemic in the human population, but less obvious when the prevalence of malaria amongst mosquitoes will occur Aron and May (1982). Hence, dependence of malaria transmission on the population density of mosquitoes results in epidemiological patterns that usually vary with the seasons. See, for example, Ross (1911) and Macdonald (1950, 1952, 1957).

The rest of the paper is organised as follows: In section 2 we present the essential assumptions, terminology and the derivation of our model. An analysis of the model in the absence of the disease is presented in Section 3 where we examine the existence, uniqueness and stability of the equilibrium solutions for the equations governing the total human and mosquito populations. In Section 4, we examine the entire model in the presence of the malaria disease. Then, we rescale the model and then proceed to examine the conditions for the existence, uniqueness and stability of endemic and disease free equilibrium solutions. We round up the paper with some concluding remarks in Section 5.

2 Derivation of the basic model

In this section we briefly derive the mathematical model studied in this paper. We define the following contact parameters.

- c_{hv} = the infectivity of an infectious 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 an infectious 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 basic unit of study is the parasite². However, since protozoans are micro-organisms with the ability to multiply rapidly and directly within the host's system, measuring the number of parasites within the host populations is usually difficult. Consequently, the infected host provides a most convenient unit of study. So we divide the human and mosquito populations into states (classes or compartments) representing disease status. The states contain susceptible, incubating and infectious individuals. At time t , there are S_h susceptible humans, E_h incubating humans, I_h infectious humans, S_v susceptible mosquitoes, E_v incubating mosquitoes and I_v infectious mosquitoes. The infected mosquito's period of infectiousness ends with its death. $N_h = S_h + E_h + I_h$ and $N_v = S_v + E_v + I_v$ are respectively the total human and vector populations at time t . The model assumes that all new-borns are susceptible in both populations (no vertical transmission) and a uniform birth rate. The per capita recruitment rates for humans and mosquitoes are $K_h > 0$ and $K_v > 0$ respectively, where K is assumed to be a monotonic decreasing and continuously differentiable function of the total population. The per capita natural removal rate for humans and mosquitoes are μ_h and μ_v respectively, where μ is a continuously differentiable and monotonic increasing function of the total population. Incubating individuals in both populations become infectious with rates $\nu_h > 0$ and $\nu_v > 0$. Infectious and infected human individuals recover with rate r_h to join the susceptible class or die from the disease at the additional rate $\gamma_h > 0$. Vector recruitment into the susceptible class is not instantaneous; i.e., mosquitoes that are recruited

²For malaria, it is possible to obtain qualitative measures not just for the prevalence (presence or absence of infection) but also for the intensity of infection (based, for example, on the number or proportion of infected red blood cells per sample). Further more, infection does not mean infectiousness since a patient may harbour many liver and blood stages of the parasite but no gametocytes. In other words, a more relevant quantitative measure of infectiousness is provided by the counts of gametocytes per unit blood sample Anderson and May (1979, 1991)

into the susceptible class at time t are from eggs laid by adult mosquitoes at the earlier time $t - T$, $T > 0$. This time interval can be significantly long when compared with the life span of the mosquito, and so is included in the model. Hence, the mosquito recruitment at time t is a function of the total mosquito population at the earlier time $t - T$.

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 rate³ of the mosquitoes, the transmission probabilities between the species and the number of individuals in both populations. The exposure rates to the infection have previously been derived; Ngwa and Shu (2000). These are:

$$\text{humans infected per unit time} = \left(\frac{c_{vh} a_v I_v}{N_h} \right) S_h, \tag{1}$$

$$\text{mosquitoes infected per unit time} = \left(\frac{c_{hv} a_v I_h}{N_h} \right) S_v. \tag{2}$$

We model the equation for the mosquito population as follows: Each mosquito goes through four life stages: egg \rightarrow larva \rightarrow pupa \rightarrow adult. Suppose that initially there are N_v adult mosquitoes each of which lays eggs at a rate $K_v(N_v)$. Suppose that $T_1 > 0$ is the time that elapses before the eggs hatch into larvae. Also suppose that these eggs have a natural death rate $\mu_{1v} > 0$. Then the expected number of eggs (total density of eggs), E_e , from time $t - T_1$ to time t is

$$E_e(t) = \int_{t-T_1}^t K_v(N_v(s)) N_v(s) e^{-\mu_{1v}(t-s)} ds, \tag{3}$$

where N_v represents the current mosquito population size, and $e^{-\mu_{1v}T_1}$ is the survival probability of the eggs during the time interval T_1 . On differentiating (3) and rearranging, we obtain the rate of production of larvae as

$$K_v(N_v(t - T_1)) N_v(t - T_1) e^{-\mu_{1v}T_1}.$$

Continuing in the same manner, we obtain the rate of entry into the adult mosquito compartment (the adult mosquito eclosion rate) as

$$K_v(N_v(t - T_1 - T_2 - T_3)) N_v(t - T_1 - T_2 - T_3) e^{-\mu_{1v}T_1 - \mu_{2v}T_2 - \mu_{3v}T_3},$$

where T_i and μ_{iv} , $i = 1, 2, 3$ are respectively the time spent in life stage i and the death rate constant for life stage i , excluding the adult life stage. We assume, for simplicity, that death in all previous life stages occurs at the uniform rate μ_{1v} for all i (i.e. $\mu_{1v} = \mu_{2v} = \mu_{3v}$) and that $T = T_1 + T_2 + T_3$. Then we may model the recruitment rate into the adult mosquito compartment as

$$\text{adult mosquito recruitment rate} = K_v(N_v(t - T)) N_v(t - T) e^{-\mu_{1v}T}. \tag{4}$$

³We 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 man-biting habit of the mosquito controls the transmission of the parasite, 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 temporal spread of the disease within the human and mosquito populations in the form:

$$\begin{aligned}
 \frac{dS_h}{dt} &= K_h(N_h)N_h + r_h I_h - \mu_h(N_h)S_h - \left(\frac{c_{vh}a_v I_v}{N_h}\right) S_h; \\
 \frac{dE_h}{dt} &= \left(\frac{c_{vh}a_v I_v}{N_h}\right) S_h - (\nu_h + \mu_h(N_h)) E_h; \\
 \frac{dI_h}{dt} &= \nu_h E_h - (r_h + \gamma_h + \mu_h(N_h)) I_h; \\
 \frac{dS_v}{dt} &= K_v(N_v(t-T))N_v(t-T)e^{-\mu_v T} - \mu_v(N_v(t))S_v - \left(\frac{c_{hv}a_v I_h}{N_h}\right) S_v \\
 \frac{dE_v}{dt} &= \left(\frac{c_{hv}a_v I_h}{N_h}\right) S_v - (\nu_v + \mu_v(N_v)) E_v; \\
 \frac{dI_v}{dt} &= \nu_v E_v - \mu_v(N_v)I_v;
 \end{aligned}
 \tag{5}$$

with appropriate initial conditions at time $t = 0$. Here,

$$\frac{dN_h}{dt} = K_h(N_h)N_h - \mu_h(N_h)N_h - \gamma_h I_h; \tag{6}$$

$$\frac{dN_v(t)}{dt} = K_v(N_v(t-T))N_v(t-T)e^{-\mu_v T} - \mu_v(N_v(t))N_v(t); \tag{7}$$

where all parameters in the model are assumed positive and the equations for $N_h = S_h + E_h + I_h$ and $N_v = S_v + E_v + I_v$ are obtained by adding up the relevant equations in (5). 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. Greenhalg (1997).

It can be shown using standard techniques described in Hale (1969) that if initial conditions are specified for each of the state variables at time $t = 0$ with $S_h(0) + E_h(0) + I_h(0) = N_h(0)$, then there exist a unique solution satisfying these initial conditions for all $t \geq 0$ with $S_h(t) + E_h(t) + I_h(t) = N_h(t)$ 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, for appropriate initial data, similar arguments apply to the vector's equations with corresponding expressions. Thus the system (5) is well posed from a mathematical and physical standpoint.

The analysis of a simplified version of system (5) for which $T = 0$, $K_h(N_h)N_h = K_h$, $K_v(N_v)N_v = K_v$, $\mu_h(N_h) = \mu_h$ and $\mu_v(N_v) = \mu_v$ where K_h , K_v , μ_h and μ_v are positive constants, was done by Bigina (1999) who established that under these conditions, the model (5) cannot exhibit oscillatory solutions and that the system possesses a unique *disease free* equilibrium, E_0 , which is locally and asymptotically stable when a unique threshold parameter $R_0 \leq 1$ and an *endemic* equilibrium, E_1 , which exists and is locally and asymptotically stable, for all $t > 0$, when $R_0 > 1$. R_0 was also explicitly calculated⁴, and in our notation may be written in the form

$$R_0 = \frac{\mu_h \nu_h \nu_v a_v^2 c_{hv} c_{vh} K_v}{\mu_v^2 K_h (\mu_v + \nu_v) (\mu_h + \nu_h) (\mu_h + r_h + \gamma_h)}. \tag{8}$$

In this paper, we study and analyse the model (5) under more general conditions. To do this we make the following assumptions:

⁴The quantity R_0 is dimensionless and determines an invasion criterion for the infection. We shall discuss its epidemiological importance and interpretation in Section 4.

Assumption 1 For $N_h \in (0, \infty)$ we assume that K_h and μ_h satisfy the following conditions:

- (i) $K_h(N_h) > 0$ and $\mu_h(N_h) > 0$, where K_h and μ_h are monotonic functions of their arguments with K_h monotonically decreasing and μ_h monotonically increasing.
- (ii) $K_h(N_h) > 0$ and $\mu_h(N_h) > 0$ are continuously differentiable with $K'_h(N_h) < \mu'_h(N_h)$, $\forall N_h$.
- (iii) $K_h(0+) > \mu_h(0+)$ and $K_h(\infty) < \mu_h(\infty)$.

It is easy to verify that Assumption 1(iii) ensures that when $\gamma = 0$ Equation (6) has a nonzero equilibrium solution⁵. Assumptions 1(i) and 1(ii) ensure that such an equilibrium solution is globally and asymptotically stable to small perturbations. A similar set of assumptions can be derived for the equation governing the total mosquito population.

3 The basic model in the absence of the disease

In this section we analyse the basic model in the absence of the disease. Since the only dynamic coupling between the two populations (mosquitoes and humans), at least in the context of our model, arises as a result of the presence of the disease, malaria, it is necessary that, to be able to quantify the relative abundance of the disease within the population, we must analyse the behaviour of the total populations in the absence of the disease. This first analysis must be detailed enough to be able to serve as a tool to ascertain the effect and consequences of the disease in the population. To this effect, we need suitable forms for the functions K_h , K_v , μ_h and μ_v . An analysis of an SEIR model with generalised density dependent death rates may be found in Greenhalgh (1997). Here for positive parameters λ_{1h} , λ_{2h} , λ_v , μ_{1h} , μ_{2h} , μ_{4v} and m , we select the following forms for K and μ :

$$\left. \begin{aligned} K_h(N_h) &= \frac{\lambda_{1h}}{N_h} + \lambda_{2h}, & \mu_h(N_h) &= \mu_{1h} + \mu_{2h}N_h \\ K_v(N_v(t-T)) &= \lambda_v e^{-mN_v(t-T)}, & \mu_v(N_v) &= \mu_{4v} \text{ a constant.} \end{aligned} \right\} \quad (9)$$

Here, λ_{1h} is a rate representing a constant human migration term, λ_{2h} and λ_v are respectively the linear birth rates and μ_v is the vector's linear death rate, m measures how fast K_v decreases. If we apply the principle that requires that we interpret the quantities involving division by the total population as zero whenever the total population is zero, then Assumption 1 (iii) implies that $\lambda_{2h} > \mu_{1h}$ and $\lambda_v > \mu_{4v}$. With the forms chosen in (9), Assumptions 1 (i) & (ii) are automatically satisfied and in this case, the dynamics of the total mosquito and human populations in the absence of the disease are modelled by the decoupled equations

$$\frac{dN_h}{dt} = \left(\frac{\lambda_{1h}}{N_h} + \lambda_{2h} \right) N_h - (\mu_{1h} + \mu_{2h}N_h)N_h; \quad (10)$$

$$\frac{dN_v(t)}{dt} = \lambda_v e^{-mN_v(t-T)} N_v(t-T) e^{-\mu_{1v}T} - \mu_{4v}N_v(t). \quad (11)$$

⁵This is clear since the conditions specified in the Assumption imply that the curves of K_h and μ_h will intersect once, and only once, for some $N_h^* \in (0, \infty)$. N_h^* is the unique positive equilibrium solution in the absence of the disease.

We put forth the following definition.

Definition 2 *In the context of this paper, we call a solution $N_h(t)$ or $N_v(t)$, $t \in [0, \infty)$ of (10) or (11) realistic if the solution is non-negative for all time.*

We proceed to show that the equations (10) and (11) that describe the dynamics of the total human and vector populations do indeed have realistic solutions when the foregoing conditions are met.

3.1 Analysis of the equation governing the total human population

In this subsection we show that under certain conditions, equation (10) has a globally stable steady state solution which may be identified as the environmental carrying capacity. To establish this result, we shall restrict ourselves to immigrations into our region of interest. That is λ_{1h} is always positive. It is immediate that if $\lambda_{1h} \leq 0$, then we must require that the linear birth rate should outweigh the linear death rate at all times.

Theorem 3 *For given positive parameters λ_{1h} , λ_{2h} , μ_{1h} and μ_{2h} , equation (10) has realistic equilibrium solutions $N_h = 0$ and $N_h = N_h^* > 0$. Moreover, the unique positive equilibrium solution N_h^* always exists and is globally stable when $\lambda_{1h} > 0$ and $\lambda_{2h} > \mu_{1h}$.*

Proof: The equilibrium solutions are easily obtained by equating the right hand side of (10) to zero and solving for N_h . This gives the positive equilibrium solution as

$$N_h^* = \frac{(\lambda_{2h} - \mu_{1h}) + \sqrt{(\lambda_{2h} - \mu_{1h})^2 + 4\lambda_{1h}\mu_{2h}}}{2\mu_{2h}} \tag{12}$$

Now, by using standard techniques⁶, (10) can be solved explicitly to obtain the solution

$$N_h(t) = \frac{\kappa [(\lambda_{2h} - \mu_{1h}) + M_1] - [(\lambda_{2h} - \mu_{1h}) - M_1] \exp(-M_1 t)}{2\mu_{2h} [\kappa - \exp(-M_1 t)]}, \tag{13}$$

where

$$M_1 = \sqrt{(\lambda_{2h} - \mu_{1h})^2 + 4\lambda_{1h}\mu_{2h}}$$

and κ is a constant that can be determined from the initial condition at time $t = 0$. It is now a trivial matter to see from (13) that $N_h(t) \rightarrow N_h^*$ (given in (12)) as $t \rightarrow \infty \forall N_h(0) \in (0, \infty)$.

■

How the solution (13) approaches N_h^* depends on whether $N_h(0)$ is bigger or smaller than N_h^* . Hence, if $N_h(0) < N_h^*$ ($N_h(0) > N_h^*$), then for the appropriate parameter regime as indicated in Theorem 3, $N_h(t)$ grows (decays) monotonically to N_h^* for all time $t > 0$. The behaviour of the solution of Equation (10) is characteristic of a class of models with a density dependent regulatory mechanism that compensates for the effect of overcrowding.

⁶Observe that (10) is an ordinary differential equation of Riccati type with evident solution N_h^* given by (12).

3.2 Analysis of the equation governing the total mosquito population

In this subsection we show that under certain conditions, the equation (11) can exhibit oscillatory solutions. The basic idea being that these oscillations in the population density of the mosquitoes will in turn induce oscillations in the prevalence of the disease, malaria, in the community. This is significant since malaria is transmitted from person to person by mosquitoes and the whole transmission cycle is driven by the mosquito's man-biting rate.

On examining equation (11), we note that the presence of the delay parameter, T , in the model assumes that the past history of the mosquito population, prior to time $t = 0$ say, is known. That is, we must specify an initial condition on some interval, at least of length T , on $(-\infty, 0]$. To proceed, we set $N_v(t) = y_0(t)$, $-T \leq t \leq 0$, for some positive, continuous and bounded function y_0 , regardless of whether or not $y_0(t)$ satisfies (11). We then seek a continuous extension of $y_0(t)$ into the future, to the function $N_v(t)$ that satisfies (11) $\forall t > 0$. We note that $N'_v(0)$ is interpreted as a right hand derivative at 0. We can therefore formulate a solution procedure for the equation (11) on the intervals $[(k - 1)T, kT]$, $k = 1, 2, 3, \dots$ as follows: for a given function $y_0(t)$, $t \in [-T, 0]$, set

$$N_v(t) = y_k(t), \quad t \in [(k - 1)T, kT], \quad k = 1, 2, 3, \dots$$

Then (11) can be solved in a hierarchical manner through the equations:

$$\frac{dy_k(t)}{dt} + \mu_{Av}y_k(t) = \lambda_v e^{-m y_{k-1}(t-T)} y_{k-1}(t-T) e^{-\mu_{Av}T}, \quad t \in [(k - 1)T, kT], \quad (14)$$

$$y_k((k - 1)T) = y_{k-1}((k - 1)T), \quad k = 1, 2, \dots, \quad (15)$$

$y_0(t)$ given.

Now, proceeding in time intervals of length T , (14) can be applied as often as desired and a solution, up to any required time t , for the delay differential equation can be constructed. Note that to ensure continuity, in addition to the condition (15), we require that N_v have right derivatives at $t = 0, t = T, t = 2T, t = 3T \dots$.

A few steps of the solution procedure outline above shows that the integrals quickly become cumbersome and one cannot draw any general conclusion concerning the behaviour of the resulting solution. Hence, to get an insight into the nature of the solution, we perform a linear stability analysis about the steady state solution. We easily verify that if N_v^* is the positive equilibrium solution of (11), its value is

$$N_v^* = \frac{1}{m} \ln \left(\frac{\lambda_v}{\mu_{Av} e^{\mu_{Av}T}} \right). \quad (16)$$

Since we are primarily interested in determining whether or not there exists a parameter regime for which the parameter T can induce oscillations⁷ in the solution of equation (11), we perform a linear stability analysis in the neighbourhood of N_v^* . The following results show that there exists a parameter regime for which the steady state N_v^* will be stable to small perturbations and one for which it is not. We shall need the following definition, cf. Cooke et al. (1999).

⁷It is a simple matter to prove that a single autonomous non-delay differential equation of the form $y' = f(y)$, $y(0) = y_0$ cannot exhibit periodic solutions. See, for example, Murray (1989).

Definition 4 Let σ and p^* , with $\sigma \in (\frac{\pi}{2}, \pi)$, be the solution of the equations

$$\sin \sigma = -\cos \sigma \left(\frac{\mu_{1v}}{\mu_{4v}} \sigma \cos \sigma + p^* \sin \sigma \right), \quad \frac{\sin \sigma - \sigma \cos \sigma}{\sigma - \sin \sigma \cos \sigma} = \frac{2\sigma \mu_{1v} \cos \sigma}{\mu_{4v} \sin \sigma} + p^*. \quad (17)$$

For $\frac{\lambda_v}{\mu_{4v}} > p^* + 1$, define T^* and T^{**} by

$$T^* = \frac{\bar{x}_1}{\mu_{4v}}, \quad T^{**} = \frac{\bar{x}_2}{\mu_{4v}}, \quad (18)$$

where \bar{x}_1 and $\bar{x}_2 > \bar{x}_1$ are the two positive solutions of the equations

$$\bar{x} = \frac{-\sigma}{\tan \sigma}, \quad \frac{\sigma}{\sin \sigma} = \bar{x} \left(\ln \left(\frac{\lambda_v}{\mu_{4v} e} \right) - \frac{\mu_{1v}}{\mu_{4v}} \bar{x} \right). \quad (19)$$

Theorem 5 Let N_v^* be the unique positive steady state of equation (11). Then for positive parameters λ_v , μ_{1v} , and μ_{4v} , we have

- (i) If $\frac{\lambda_v}{\mu_{4v}} < e^{p^*+1}$, then the unique positive equilibrium N_v^* of equation (11) is locally and asymptotically stable independent of T .
- (ii) If $\frac{\lambda_v}{\mu_{4v}} > e^{p^*+1}$, then there exists $0 < T^* < T^{**}$ such that as T increases from zero, N_v^* loses stability at $T = T^*$. As T further increases from T^* , stability is regained at $T = T^{**}$.

Proof: The linear stability of the equilibrium N_v^* of (11) is studied by linearising about the steady state N_v^* . That is, we write $N_v(t) = N_v^* + n(t)$ where $|n(t)| \ll 1$, substitute in (11) and expand the nonlinear terms in a Taylor series about N_v^* retaining only first order terms in $n(t)$. This gives the equation

$$\frac{dn(t)}{dt} = -Mn(t - T) - \mu_{4v}n(t); \quad (20)$$

$$MT = \mu_{4v}T \left(p - \frac{\mu_{1v}}{\mu_{4v}}(\mu_{4v}T) \right), \quad p = \ln \left(\frac{\lambda_v}{\mu_{4v}e} \right). \quad (21)$$

Hence, linear stability or instability of N_v^* depends on whether or not $n(t)$ as defined by (20) grows unbounded as $t \rightarrow \infty$. To determine this, we seek solutions of (20) of the form

$$n(t) = n_0 e^{\xi t} \quad (22)$$

where n_0 is a proportionality constant that can be determined from the initial conditions and ξ is an eigenvalue that measures the temporal growth (or decay) of the solution as time evolves. Substitute the form (22) into (20) to obtain the dispersion relation

$$\xi T = -MT e^{-\xi T} - \mu_{4v}T. \quad (23)$$

Notice that $\xi = 0$ is not a solution of (23) and that (21) defines a parabola in the $(\mu_{4v}T, MT)$ -plane. If ξ is real and $MT > 0$, then (23) assures us that $\xi < 0$ and that $n(t) \rightarrow 0$ as $t \rightarrow \infty$. That is, N_v^* is linearly stable $\forall T > 0$. Also, if ξ is real, $MT < 0$ and $MT e^{-\xi T} < \mu_{4v}T$, then $\xi < 0$ and N_v^* is linearly stable to small perturbations for all T . However, all we wish to know is whether there are solutions of (23) with $\Re(\xi) > 0$ which will thus imply instability

with growing oscillations as t increases. To examine this, set $\xi = \mu + i\omega$ in (23) and equate real and imaginary parts to have the two equations

$$\mu T = -MTe^{-\mu T} \cos(\omega T) - \mu_{4v}T, \quad \omega T = MTe^{-\mu T} \sin(\omega T). \quad (24)$$

Observe that $|\xi|^2 \rightarrow \infty$ as $\mu \rightarrow -\infty$. Therefore, there exists a real number μ_0 which bounds $\Re(\xi)$ from above. We also observe that if $\omega = 0$ and $MT > 0$, then ξ is real and (23) shows that $\xi < 0$ for all T . We also observe that if $\omega \neq 0$ is a solution of (24), so is $-\omega$. Hence, without loss of generality, we assume that $\omega > 0$ and determine conditions on the parameter T that will guarantee the existence of solutions of (23) with $\Re(\xi) > 0$ in anticipation of limit cycle solutions.

The limiting case where $\mu = 0$ gives

$$MT = \frac{\omega T}{\sin(\omega T)}, \quad \mu_{4v}T = \frac{-\omega T}{\tan(\omega T)}. \quad (25)$$

We note that neither ωT nor $\sin(\omega T)$ is zero at a root of (24). We easily verify, by examining (24), that when $MT > 0$, $\omega T \in (\pi/2, \pi)$ since $\omega > 0$. In this case, (25) gives that part of the solution of (23) for which ξ is purely imaginary with $\mu_{4v}T > 0$ in the $(\mu_{4v}T, MT)$ -plane. Substituting (25) with $\sigma = \omega T$ and $p = p^*$ in (21) gives the first equation of (17). This gives the condition for the curve (21) and that defined by (23) when ξ is purely imaginary to intersect at $p = p^*$ which is the critical value of p in the $(\mu_{4v}T, MT)$ -plane. These two curves must be tangential at $p = p^*$ for the unique solution p^* . Differentiating (21) and the parametric equations of (25) with respect to $\mu_{4v}T$ in order to impose the condition of tangency leads to the second equation of (17) with $\sigma = \omega T$ and $p = p^*$.

It is now a trivial matter to see that if p , as given in (21) is such that $p < p^*$ (that is, $\lambda_v/\mu_{4v} = e^{p^*+1}$) then the intersection of the curves described in the last paragraph is not possible and Theorem 5(i) is proved. Also, if $p > p^*$ then the two curves will intersect at points where $\mu_{4v}T = \mu_{4v}T^*$ and $\mu_{4v}T = \mu_{4v}T^{**}$. These points are given in (18) and (19) and are obtained from (21) and (25), with $\sigma = \omega T$, $MT = \sigma/\sin \sigma$ and $\bar{x} = \mu_{4v}T$. As T passes through T^* , stability is lost and as T passes through T^{**} , stability is regained. This proves (ii) of the theorem. ■

Corollary 6 Assume $m > 0$, $\lambda_v > \mu_{4v}$, $\mu_{1v} = 0$ in (11) with positive initial data. Then

- (i) if $\lambda_v/\mu_{4v} \leq e^2$, then the unique positive equilibrium N_v^* , is locally and asymptotically stable for all $T > 0$.
- (ii) if $\lambda_v/\mu_{4v} > e^2$, then there exists $T_c > 0$ with

$$T_c = \frac{\pi - \cos^{-1}(\mu_{4v}/M)}{\sqrt{M^2 - \mu_{4v}^2}}$$

such that the unique positive equilibrium is stable when $T < T_c$ and unstable when $T > T_c$.

- (iii) For $T > T_c$ and $\lambda_v/\mu_{4v} > e^2$, N_v^* is linearly unstable to small perturbations and the instability is by growing oscillations with the period of oscillation approximately given by $2\pi/\sqrt{M^2 - \mu_{4v}^2}$

Proof: We take each item in turn

(i) Set $\mu_{1v} = 0$ in Theorem 5. M in (21) reduces to

$$MT = p\mu_{4v}T, \quad p = \ln\left(\frac{\lambda_v}{\mu_{4v}e}\right)$$

and (21) thus reduces to a straight line in the $(\mu_{4v}T, MT)$ -plane and the result then follows from the prove of Theorem 5.

(ii) All we need is to verify that T_c is as given. Now, consider the first of (24) and $|\xi|^2$ at $\mu = 0$ to have the solution

$$\mu = \mu_c = 0, \quad \omega_c T_c = \pi - \cos^{-1}\left(\frac{\mu_{4v}}{M}\right), \quad \omega_c = \sqrt{M^2 - \mu_{4v}^2}. \quad (26)$$

Clearly, $M^2 > \mu_{4v}^2$ since by hypothesis $\lambda_v/\mu_{4v} > e^2$. The result then follows.

(iii) The solution $\xi = \mu + i\omega$ with largest real part when $T = T_c$ is $\mu = \mu_c = 0$, $\omega = \omega_c = \sqrt{M^2 - \mu_{4v}^2}$. If we write $T = T_c + \epsilon$ for $|\epsilon| \ll 1$, we expect μ and ω to also differ from μ_c and ω_c by some small quantities. Let these be $\mu = \theta$, $\omega = \omega_c + \eta$, $0 < |\theta|, |\eta| \ll 1$, where θ and η are to be determined. Now substitute these into (24), expand the non-linear terms in a Taylor Series about μ_c, ω_c and T_c , retain only first order terms in the small quantities to have the linear approximations

$$(1 + \mu_{4v}T_c)\theta - \omega_c T_c \eta = \omega_c^2 \epsilon, \quad \omega_c T_c \theta + (1 + \mu_{4v}T_c)\eta = -\mu_{4v}\omega_c \epsilon. \quad (27)$$

Solving these simultaneously gives the first order approximations

$$\theta = \frac{\omega_c^2}{(1 + \mu_{4v}T_c)^2 + \omega_c^2 T_c^2} \epsilon, \quad \eta = -\frac{\omega_c[\mu_{4v}(1 + \mu_{4v}T_c) + \omega_c^2 T_c]}{(1 + \mu_{4v}T_c)^2 + \omega_c^2 T_c^2} \epsilon \quad (28)$$

where ω_c and T_c are given in (26). Thus near the bifurcation point $T = T_c$, we have the linear approximation

$$N_v(t) \approx N_v^* + \Re\{n_0 \exp(\theta + i(\omega_c + \eta)t)\},$$

where θ and η are given in (28). This shows that when $\epsilon > 0$, instability is by growing oscillations with the initial period given by

$$\frac{2\pi}{\sqrt{M^2 - \mu_{4v}^2} + \eta} \approx \frac{2\pi}{\sqrt{M^2 - \mu_{4v}^2}}, \quad \text{to } O(1) \text{ for small } \epsilon. \quad (29)$$

Hence, for $T < T_c$ ($T > T_c$) we will observe damped (growing) oscillations with the initial period of oscillation given by (29). ■

The above analysis shows that there exists a realistic parameter regime for which oscillatory solutions of equation (11) are possible; a situation which is not possible when there is no delay in the model (that is $T = 0$). Hence, including delay in the model increases complexity and allows for a wider range of possibilities. Our results also demonstrate that μ_{1v} plays a significant role in the dynamics of the mosquito population. For example, we have shown that when $\mu_{1v} > 0$, an instability window wherein $T^* < T < T^{**}$ exist so that for $T < T^*$ and/or $T > T^{**}$ the unique positive equilibrium solution N_v^* is locally and asymptotically stable to small perturbations and unstable otherwise.

4 The basic model in the presence of the disease

In this section, we examine and analyse the basic model derived in Section 2. It is easier to analyse the model in terms of proportions of susceptible, incubating and infectious individuals; so we make the change of variables:

$$u = \frac{S_h}{N_h}, v = \frac{E_h}{N_h}, w = \frac{I_h}{N_h}, x = \frac{S_v}{N_v}, y = \frac{E_v}{N_v}, z = \frac{I_v}{N_v}, \tag{30}$$

so that

$$u + v + w = 1 \Rightarrow v = 1 - u - w, \quad x + y + z = 1 \Rightarrow y = 1 - x - z. \tag{31}$$

We also arbitrarily scale time t with the quantity $1/\mu_{4v}$ by setting $\tau = \mu_{4v}t$, and scale the total populations with their respective positive disease free equilibrium states by setting $N_h = N_h^*N$, $N_v = N_v^*\bar{N}$, where N_h^* and N_v^* are given by (12) and (16) respectively. Hence, we introduce the following dimensionless parameters:

$$\begin{aligned} \tau &= \mu_{4v}t, \quad \delta = \frac{\lambda_{1h}}{\mu_{4v}}, \quad \lambda = \frac{\lambda_{2h}}{\mu_{4v}N_h^*}, \quad \gamma = \frac{\gamma_h}{\mu_{4v}}, \quad \nu = \frac{\nu_h}{\mu_{4v}}, \\ \alpha &= 1 - \frac{\lambda_{2h} - \mu_{1h}}{\mu_{2h}N_h^*}, \quad \varepsilon = \frac{c_{vh}a_vN_v^*}{\mu_{4v}N_h^*}, \quad \rho = \frac{\mu_{2h}N_h^*}{\mu_{4v}}, \quad \beta = \frac{r_h}{\mu_{4v}}, \\ b &= \frac{c_{hv}a_v}{\mu_{4v}}, \quad c = \frac{\nu_v}{\mu_{4v}}, \quad d = mN_v^*. \end{aligned} \tag{32}$$

The system (5) becomes:

$$\begin{aligned} \frac{du}{d\tau} &= Q(N)(1 - u) + (\beta + \gamma u)w - R(N, \bar{N})uz \\ \frac{dw}{d\tau} &= \nu(1 - u) + (\gamma w - S(N))w \\ \frac{dx}{d\tau} &= a(\bar{N}; T)(1 - x) - bxw \\ \frac{dz}{d\tau} &= c(1 - x) - (a(\bar{N}; T) + c)z \end{aligned} \tag{33}$$

where

$$\begin{aligned} a(\bar{N}; T) &= e^{d(1-\bar{N}(\tau-T))} \frac{\bar{N}(\tau-T)}{\bar{N}(\tau)}, \quad Q(N) = \lambda + \frac{\delta}{N} \\ R(N, \bar{N}) &= \varepsilon \frac{\bar{N}}{N}, \quad S(N) = \nu + \gamma + \beta + Q(N). \end{aligned}$$

We have also used (31) to eliminate v and y from the system and have written $Q(N)$, $R(N, \bar{N})$, $S(N)$, and $a(\bar{N}; T)$ as such to emphasize the density dependence in the contact and transition rates. For notational simplicity, we simply write Q , R , S , and a to represent these density dependent quantities. We note that our representation is meaningful as the total populations are observables that can be measured in a given situation. The equations for the total populations, which in fact determine the behaviour of $Q(N)$, $R(N, \bar{N})$, $S(N)$ and $a(\bar{N}; T)$, now take the form

$$\frac{dN}{d\tau} = \rho(1 - N)(N + \alpha) - \gamma Nw, \quad \frac{d\bar{N}}{d\tau} = e^{d(1-\bar{N}(\tau-T))} \bar{N}(\tau-T) - \bar{N}(\tau). \tag{34}$$

Now, the model in terms of proportions, (33), is defined in the subset $\Omega \times [0, \infty)$ of \mathbf{R}_+^5 where

$$\Omega = \{u, w, x, z : 0 \leq u, w, x, z \leq 1, 0 \leq u + w \leq 1, 0 \leq x + z \leq 1\} \quad (35)$$

and the original quantities can be recovered from the proportions through (30), (31) and (32).

4.1 Existence of steady state solutions

In this subsection we present some results concerning the existence of equilibrium or constant solutions for the model formulated above. To do this we shall make use of a threshold parameter, which we shall denote by R_0 . We give the following modification to Definition 2.

Definition 7 *We shall call a solution of the problem formulated in terms of proportions realistic if it lies in the compact interval $[0,1]$.*

Proposition 8 *The model formulated in terms of proportions has at least one equilibrium solution $E : (u, w, x, z) = (u^*, w^*, x^*, z^*)$ with u^*, w^*, x^*, z^* all nonnegative, whose existence and properties are determined by the threshold parameter R_0 where*

$$R_0 = \frac{R\nu bc}{a(a+c)(Q+\nu)(Q+\beta+\gamma)}. \quad (36)$$

Moreover when $R_0 > 1$ and $\gamma = 0$, there is a unique non-trivial realistic equilibrium solution $E_{\gamma=0}$ that is expressible in terms of R_0 . When $R_0 \leq 1$, the only realistic equilibrium solution is the solution $E_0 : (u, w, x, z) = (1, 0, 1, 0)$ which is called the disease free equilibrium .

Proof: Let (u^*, w^*, x^*, z^*) be a constant solution of the model (33). We easily express u^* , x^* and z^* in terms of w^* in the form

$$\begin{aligned} u^*(w^*) &= \frac{\nu + (\gamma w^* - S)w^*}{\nu}, \\ x^*(w^*) &= \frac{a}{a + bw^*}, \\ z^*(w^*) &= \frac{bcw^*}{(a+c)(a+bw^*)}. \end{aligned} \quad (37)$$

Substituting these in the first equation of (33) and equating to zero yields a fourth order polynomial in w^* of the form

$$w^*(A_3w^{*3} + A_2w^{*2} + A_1w^* + A_0) = 0; \quad (38)$$

where

$$\begin{aligned} A_3 &= \gamma^2 a B_1^2 B_2 B_3 R_0, \\ A_2 &= \gamma a B_1 (\gamma - B_2 B_3 R_0 (B_1 (R B_2 c + B_3 \nu) + R c)), \\ A_1 &= a B_1 B_2 B_3 R_0 (B_1 (\nu (\beta + \gamma) + Q S) + S R c) - \gamma a B_1 (\nu B_3 + R B_2 c), \\ A_0 &= a \nu c B_1 B_2 B_3 (1 - R_0), \end{aligned}$$

and

$$B_1 = (a + c), \quad B_2 = \frac{Q\nu}{cR}, \quad B_3 = \frac{Q + \beta + \gamma}{\nu}.$$

Clearly $w^* = 0$ is a solution. Notice that A_3 is positive while the sign of A_0 coincides with that of $(1 - R_0)$ so that if $R_0 > 1$, $A_0 < 0$ in which case we have at least one sign change in the sequence of coefficients $\{A_3, A_2, A_1, A_0\}$. Hence, by Descartes' rule of signs, there exists at least one positive real root for (38) aside from the root $w^* = 0$ whenever $R_0 > 1$. When $w^* = 0$, we get the disease free equilibrium point $E_0 = (1, 0, 1, 0)$. When $\gamma = 0$, (38) reduces to a first order polynomial from which w^* is easily calculated. In this case we have the endemic equilibrium solution $E_{\gamma=0} = (u^*, w^*, x^*, z^*)$ with $0 < u^*, w^*, x^*, z^* < 1$ given by

$$u^* = \frac{1}{R_0 x^*}, \quad w^* = \frac{a\nu(R_0 - 1)}{\nu b + aSR_0}, \quad x^* = \frac{b\nu + aSR_0}{R_0(b\nu + aS)}, \quad z^* = \frac{\nu bc(R_0 - 1)}{B_1 R_0(b\nu + aS)}. \quad (39)$$

$E_{\gamma=0}$ is clearly unique when $R_0 > 1$. When $R_0 \leq 1$ the only realistic solution is the disease free equilibrium solution. ■

Remark: Observe that when $\gamma = 0$, the quantity $R_0 - 1$ appears only in the expressions for the proportions of infectives. This is the reason why (39) is called an endemic equilibrium. Also observe that the relation $u^* x^* = 1/R_0$ gives a measurable index which indicates that we can discuss the prevalence of the disease in the population simply by measuring the proportions of susceptible humans and mosquitoes in the population (at equilibrium). The next result indicates that there exist a parameter regime such that the polynomial (38) does indeed have a realistic solution aside from the disease free equilibrium⁸ when $\gamma \neq 0$.

Proposition 9 *Let $R_0 > 1$ and $Q + \nu - \gamma \geq 0$, then there exists at least one value $w^* \in (0, 1)$ that solves (38). That is, when $R_0 > 1$, the model formulated in terms of proportions has at least one realistic equilibrium solution different from the disease free equilibrium, called the endemic equilibrium.*

Proof: Consider the function $g : \mathbf{R} \rightarrow \mathbf{R}$ defined by

$$g(w^*) = A_3 w^{*3} + A_2 w^{*2} + A_1 w^* + A_0$$

where the coefficients $A_i, i = 0, 1, 2, 3$ are those of (38). We easily verify that $g(0) = A_0$ and see that $g(0) < 0$ when $R_0 > 1$. Some algebraic manipulation then shows that

$$g(1) = (Q + \beta)(B_1(a + b)(Q + \nu - \gamma) + Rbc)$$

It is now a trivial matter to see that when the condition $Q + \nu - \gamma \geq 0$ holds, $g(1) \geq 0$. The existence of the root $w^* \in (0, 1)$ then follows from the intermediate value theorem. ■

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 (ie, the reservoir of infection) here $I_h, I_v \rightarrow 0$ with increasing time, while a weaker demand is that the proportions w, z , tend to zero with increasing time; cf. Busenberg and van den Driessche (1990). Thus we shall seek conditions for the stability of the endemic

⁸Results earlier established; Ngwa and Shu (2000), can be used to show that when any of \bar{N}, R, ν, b or c is zero, then the only realistic solution of (38) is the solution $w^* = 0$.

proportional state (u^*, w^*, x^*, z^*) with $w^* > 0$ and $z^* > 0$ and for the stability of the DFE $(u^*, w^*, x^*, z^*) = (1, 0, 1, 0)$. We will see in Subsection 4.2 that the stability of these equilibria depend critically on the parameter R_0 , defined by (36). The parameter 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; Diekmann et al. (1990).

In our formulation, some of our parameters are not constant, but rather depend on a variety of factors such as the size of the total populations in the respective 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 R_0 will increase with increasing vector population.

4.2 Stability of the Equilibria

In this subsection we examine the local stability of the steady states derived above.

Theorem 10 *If $R_0 \leq 1$, then the disease free equilibrium, E_0 , is locally and asymptotically stable while the equilibrium $E_{\gamma=0}$ is also locally and asymptotically stable when $R_0 > 1$.*

Proof: The local stability of the equilibrium solutions can be examined by linearising system (33) about the equilibrium solution (u^*, w^*, x^*, z^*) . This gives the Jacobian matrix

$$J_E = \begin{pmatrix} -A & B & 0 & -Ru^*(w^*) \\ -\nu & -C & 0 & 0 \\ 0 & -bx^*(w^*) & -D & 0 \\ 0 & 0 & -c & -B_1 \end{pmatrix} \tag{40}$$

where

$$\begin{aligned} A &= Q + Rz^*(w^*) - \gamma w^*, & B &= \beta + \gamma u^*(w^*), \\ B_1 &= a + c, & D &= a + bw^*, & C &= S - 2\gamma w^*. \end{aligned}$$

Observe hat the quatities A, B, C, D and B_1 so defined are all positive since $Q + \nu - \gamma \geq 0$ and $0 \leq w^* < 1$. The eigenvalues of J_E are the solutions of the fourth order polynomial equation

$$\zeta^4 + a_1\zeta^3 + a_2\zeta^2 + a_3\zeta + a_4 = 0; \tag{41}$$

where,

$$\begin{aligned} a_1 &= A + C + D + B_1 \\ a_2 &= D(A + C + B_1) + B_1(A + C) + AC + B\nu \\ a_3 &= D(B\nu + C(A + B_1)) + B_1(B\nu + AC) \\ a_4 &\neq DB_1(B\nu + AC) - R\nu bcu^*(w^*)x^*(w^*). \end{aligned}$$

Now, from a stability point of view, all we wish to know is whether there exist a value ζ , that is a solution to (41) with $\Re(\zeta) > 0$. If such a ζ exist, then the equilibrium solution

is locally unstable to small perturbations, otherwise it is locally and asymptotically stable. Observe that a_1 , a_2 and a_3 are all positive since the parameter groupings are positive. However, a_4 may or may not be positive. We easily establish using (36) and the expression for a_4 that in the case where $w^* = 0$, that is where we have only the disease free equilibrium, $a_4 = \nu c R B_1 B_2 B_3 (1 - R_0)$ and when $w^* \in (0, 1)$ with $\gamma = 0$, $a_4 = \nu c R B_1 B_2 B_3 (R_0 - 1)$ where $\nu B_3 = Q + \beta$. It is then a trivial matter to deduce from Descartes' rule of signs that when $R_0 \leq 1$, the disease free equilibrium is locally and asymptotically stable and when $R_0 > 1$ the endemic equilibrium is locally and asymptotically stable. The local and asymptotic stability of the endemic equilibrium is concluded by applying the Ruth Hurwitz conditions on the coefficients of the polynomial (41). The straightforward but tedious calculations are omitted. ■

Notice that the second equation of (34) is a scaled version of (11) and now contains only the parameters d and T . In this case, M in (21) becomes $d - 1$. The analysis of the equation (11) then carries over and we observe that the oscillations in the mosquito population will induce oscillations in the prevalence of the disease in the community. Equation (29) shows that the initial period of oscillation in the system may be written in dimensionless terms as $2\pi/\sqrt{d(d-2)}$ and the critical bifurcation parameter is $T_c = \pi - \cos^{-1}(1/M)/\sqrt{d(d-2)}$.

5 Concluding remarks

The results presented above show that there are two possible realistic equilibrium points: One where the disease has died out proportionally and the other, if $R_0 > 1$, where there is a unique endemic equilibrium. R_0 is a unique threshold parameter which determines the behaviour of the system. Assuming that the stability results are global, then assuming that initially there is at least one infectious mosquito (or human), then if $R_0 \leq 1$, we expect the disease to die out proportionally, whereas if $R_0 > 1$, then we expect the disease to tend proportionally to the unique endemic equilibrium, thereby establishing itself in the community.

In this paper we have developed an SEIS 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 pseudo-equilibrium hypothesis is often made. 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 have demonstrated that it is possible to use a single equation to study the population dynamics of an organism whose dynamics are known to be oscillatory, provided we introduce a delay in the model. Our study of the delay parameter shows that such a delay can indeed induce oscillatory phenomena in the dynamics of the mosquito population and that the death rate of the mosquitoes at earlier life stages plays a significant role in the dynamics of the mosquito population.

We reformulated the differential equation model in terms of proportions of susceptible, incubating and infectious individuals in both vector and human populations. 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 R_0 and the disease can persist

if and only if R_0 exceeds one. The disease free equilibrium always exists and is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$. We showed that the endemic equilibrium is locally and asymptotically stable when it exists.

Due to the oscillatory nature of the dynamics, our model provides a plausible framework for studying the control strategies for the containment of malaria. For example, continuous application of control measures, (such as the use of insecticides or impregnated bed nets) when the mosquito population is at its maximum amplitude may result to a substantial decrease in this population and in extreme cases may lead to extinction. On the other hand, instantaneous application of a control measure may not be effective because the initial application will normally lead to a depression in the prevalence of the infection in the population and the number of cases will start to rise again, once the mosquito population recovers, because of its oscillatory nature. We have thus demonstrated that it is important to pay sufficient attention to the dynamics of the mosquito population, especially taking into account the time lapse between egg laying adult mosquito eclosion. Given that the malaria parasite has displayed its ability to build up resistance to anti-malarial drugs, and that there is presently no vaccine for the disease, mosquito based control strategies remain one of the few methods available, despite current debate about its long term effectiveness.

The steady state relation when $\gamma = 0$, namely $u^*x^* = 1/R_0$ is of much significance. It indicates that in an endemic region, we could discuss malaria prevalence simply by measuring the proportions of susceptible humans and mosquitoes in the population. We have not presented analytic global stability results for the equilibria in the model. This and other aspect of the model such as numerical simulations, the modification of the recovery rates to include the use of anti malarial drugs are aspects under investigation.

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Received: 02/04/2001

Accepted: 30/05/2001