DISCRETE AND CONTINUOUS
DYNAMICAL SYSTEMS SERIES B
Volume **17**, Number **6**, September **2012**

doi:10.3934/dcdsb.2012.17.xx

pp. **1–XX**

# OPTIMAL TREATED MOSQUITO BED NETS AND INSECTICIDES FOR ERADICATION OF MALARIA IN MISSIRA

Bassidy Dembele

Department of Mathematics and Computer Science, Grambling State University
Grambling, LA 71245, USA

Abdul-Aziz Yakubu

Department of Mathematics, Howard University
Washington, DC 20059, USA

Abstract. We extend the deterministic mathematical malaria model framework of Dembele *et al.* and use it to study the impact of protecting humans from mosquito bites and mass killing of mosquito vectors on malaria incidence in Missira, a village in Mali. As a case study, we fit our model to Missira malaria incidence data. Using the fitted model, we compute the optimal proportion of protected human population from infected mosquito bites and optimal proportion of killed moquitoes that would lead to the eradication of malaria in Missira.

1. **Introduction.** In 2008, an estimated $190 - 311$ million cases of malaria occurred worldwide. 90% of these cases occurred in Africa [7]. Though treatable and preventable, annually this mosquito-borne disease kills an estimated $708,000 - 1,003,000$; a large percentage of them children in Africa. In much of sub-Saharan Africa, parts of northern India, Indonesia, South America and elsewhere, malaria disease is endemic, but epidemics are uncommon [7].

Malaria parasite is transferred to a potential human victim when he or she is bitten by an infected female mosquito. *Plasmodium falciparum*, the deadliest of the four species of the parasite, is transmitted by the mosquito *Anopheles gambiae*. Plasmodium falciparum causes more than 90% of malaria death [7-9]. The intensity of malaria transmission depends on many factors (e.g. climate conditions, environmental and bio-geographical factors). Malaria is hard to control in endemic regions of Africa because transmission is efficient and transmission rates are so high that most people receive many infective bites per year [3]-[14], [17]-[25]. Killeen *et al.* used a kinetic model of mosquito foraging for aquatic habitats and vertebrate host to estimate the intensity of malaria transmission [16]. In [6], Carter *et al.* investigated the relationship between malaria incidence, anopheles mosquito population size and distance from breeding sites of the anopheles malaria vector in urban and peri-urban regions of Africa and the Indian ocean. Menach *et al.* proved that draining, fouling or filling standing water can be more effective at controlling malaria than applying larvicide [21]. In other words, killing a large number of mosquitoes or protecting a large number of people from mosquito bites can control the spread of malaria.

2000 *Mathematics Subject Classification.* Primary: 34C60; Secondary: 34C25, 34D23, 92D30.
*Key words and phrases.* Bed nets, Infecticide, Malaria, Mssira.

Malaria disease is endemic in Mali, and about 50, 000 Malians die from the disease every year. In Mali, malaria is responsible for 25% of deaths in children under the age of 5 [7]-[9]. In a clinical study, Allen and Cisse [1], used a sample of 76 children (ages 1 to 10 years) to determine the frequency of malaria in Missira, a village in Mali. For their study, Allen and Cisse [1] used Core $^{TM}$ Malaria pf rapid assay kit to test for the presence of plasmodium falciparum. The test kit has a sensitivity of 99% and a specificity of 96%. Allen and Cisse obtained that 22.37% of the children had plasmodium falciparum parasite in their blood (see Table 1).

**Table 1.** Clinical Malaria Data From Missira [1]

| **Malaria Status** | **Number of Children** | **Percentage** |
|---|---|---|
| Infected | 17 | 22.37% |
| Non-infected | 59 | 77.63% |
| Total | 76 | 100% |

In the present paper, we extend the deterministic mathematical malaria model framework of Dembele *et al.* and use it to study the impact of protecting humans from mosquito bites and mass killing of the mosquito vector on malaria incidence in Missira [8], [9]. Our mathematical model results collaborate the clinical results of Allen and Cisse in Table 1. A key observation in our modeling approach is the fact that the total population in the Missira study of Allen and Cisse is much smaller than the total population of Missira. Consequently, the total population of infected mosquitoes is essentially not affected by the clinical study of Allen and Cisse [1].

The paper is organized as follows: In Section 2, we introduce the deterministic compartmental malaria model [2], [8], [9], [15]. We fit our mathematical model to the clinical data of Allen and Cisse (Table 1) in Section 3. In Section 4, we compute optimal proportions of protected people and optimal proportions of killed mosquitoes that would lead to the eradication of malaria in Missira. We summarize our results in Section 5.

2. **Malaria model.** As in Dembele *et al.* [8], [9], we use a non-autonomous system of a continuous-time compartmental malaria model to study malaria epidemic in Mali. In our model, human population $(N_h)$ is divided into three compartments, susceptible $(S_h)$, infected $(I_h)$, and recovered $(R_h)$ individuals. Mosquito population $(N_m)$ is divided into two compartments, susceptible $(S_m)$ and infected $(I_m)$ mosquitoes. Susceptible individuals are those who have no parasite in their bloodstream, but can get infected with the malaria parasite after receiving bites from infected mosquitoes. Infected individuals are those who have received bites from infected mosquitoes and are infected with the malaria disease. Initially, these individuals are not infectious because the parasite (plasmodium falciparum) has not reached its final stage of sexual development in their bloodstream and it is even not detectable by blood smear. After 12 days of infection, infected humans become infectious and can develop symptoms of malaria. However, with proper treatment, infected humans can recover completely from the disease. Recovered individuals are those who have received treatment by radical core using actimisine (ACT) and have recovered from malaria. Susceptible mosquitoes are those with no malaria parasites but can get infected with the malaria parasite after biting malaria infected humans. Infected mosquitoes have the malaria parasites as a result of taking blood meal from infected humans. Once infected by humans, it takes about 10 days for the parasite (plasmodium falciparum) to develop in the infected mosquito and become

infectious (sporozoites).

Following Dembele *et al.* [8], [9], we use the following system of equations to describe the spread of the malaria disease.

$$\left.\begin{array}{rcl}
\frac{dS_h}{dt} &=& \lambda_h N_h + \beta_h R_h - \mu_h(t) S_h - \frac{(1-c_h)(\alpha_{mh}(t) b_m I_m) S_h}{N_h}, \\
\frac{dI_h}{dt} &=& \frac{(1-c_h)(\alpha_{mh}(t) b_m I_m) S_h}{N_h} - (\mu_h(t) + \alpha_h) I_h, \\
\frac{dR_h}{dt} &=& \alpha_h I_h - (\beta_h + \mu_h(t)) R_h, \\
\frac{dS_m}{dt} &=& \lambda_m(t) N_m - \mu_m(t) S_m - \frac{(1-c_m)(\alpha_{hm}(t) b_m I_h) S_m}{N_h}, \\
\frac{dI_m}{dt} &=& \frac{(1-c_m)(\alpha_{hm}(t) b_m I_h) S_m}{N_h} - \mu_m(t) I_m, \\
\frac{dN_h}{dt} &=& (\lambda_h - \mu_h(t)) N_h, \\
\frac{dN_m}{dt} &=& (\lambda_m(t) - \mu_m(t)) N_m, \\
N_h &=& S_h + I_h + R_h, \\
N_m &=& S_m + I_m,
\end{array}\right\} \tag{1}$$

where the parameters are defined in Table 2**.**

**Table 2.** Model Parameters

| **Parameter** | **Definition** |
|---|---|
| $\alpha_{hm}$ | Human infectivity rate |
| $\alpha_{mh}$ | Mosquito infectivity rate |
| $b_m$ | Mosquito biting rate |
| $\lambda_h$ | Human birth rate |
| $\lambda_m$ | Mosquito birth rate |
| $\beta_h$ | Loss of immunity rate |
| $\alpha_h$ | Human recovery rate |
| $\mu_h$ | Human death rate |
| $\mu_m$ | Mosquito death rate |
| $c_h$ | Proportion of protected human |
| $c_m$ | Proportion of destroyed mosquito |

When humans are not protected from infected mosquito bites by either mosquito treated bed nets or insecticides and the mosquito birth rate, mosquito death rate and human birth rate are periodic, then $c_h = c_m = 0$ and System (1) reduces to the model of Dembele *et al.* [8], [9]. When $\lambda_m(t)$, $\mu_m(t)$ and $\mu_h(t)$ are $T-$*periodic* and $c_h = c_m = 0$, Dembele *et al.* obtained the basic reproduction number for System (1) as

$$\mathcal{R}_0 = \frac{\alpha\gamma(\frac{1}{T}) \displaystyle\int_0^T \frac{N_m(t)}{N_h(t)} dt}{(\lambda_h + \alpha_h)(\frac{1}{T}) \displaystyle\int_0^T \lambda_m(t) dt}, \tag{2}$$

where $N_h(t) \neq 0$ for all $t$ and $\gamma$ is a positive constant.

Following Dembele *et al.* [8], [9], we make the following change of variables.

$$s_h = \frac{S_h}{N_h},\ i_h = \frac{I_h}{N_h},\ r_h = \frac{R_h}{N_h},\ s_m = \frac{S_m}{N_m} \text{ and } i_m = \frac{I_m}{N_m}.$$

Consequently,

$$s_h + i_h + r_h = 1 \text{ and } s_m + i_m = 1.$$

That is,

$$r_h = 1 - s_h - i_h \text{ and } i_m = 1 - s_m.$$

In the new variables, System (1) reduces to the following system of three equations.

$$\left.\begin{array}{rcl} \frac{ds_h}{dt} &=& (\lambda_h + \beta_h)(1 - s_h) - \beta_h i_h - (1 - c_h)\gamma(t) s_h i_m, \\ \frac{di_h}{dt} &=& (1 - c_h)\gamma(t) s_h i_m - (\lambda_h + \alpha_h) i_h, \\ \frac{di_m}{dt} &=& (1 - c_m)(1 - i_m)\alpha i_h - \lambda_m i_m. \end{array}\right\} \tag{3}$$

**Lemma 2.1.** *In System (3), the compact set*

$$\Gamma = \{(s_h, i_h, i_m) \mid 0 \leq s_h, i_h, i_m \leq 1,\ 0 \leq s_h + i_h \leq 1 \text{ and } 0 \leq i_m \leq 1\}$$

*is positively invariant.*

*Proof.* If $s_h = 0$, then

$$\frac{ds_h}{dt} = \lambda_h + \beta_h(1 - i_h) \geq 0.$$

If $i_h = 0$, then

$$\frac{di_h}{dt} = \gamma(1 - c_h) s_h i_m \geq 0.$$

If $i_m = 0$, then

$$\frac{di_m}{dt} = \alpha(1 - c_m) i_h \geq 0.$$

Furthermore, if $s_h + i_h = 1$ then $\frac{ds_h}{dt} + \frac{di_h}{dt} = -\alpha_h i_h \leq 0$, and if $i_m = 1$ then $\frac{di_m}{dt} = -\lambda_m i_m \leq 0$. Therefore, no solution with initial condition in the compact region $\Gamma$ can leave $\Gamma$ at any time $t \geq 0$. □

Let

$$J(c_h, c_m) = \int_0^{t_f} (i_h + B c_h^2(t) + C c_m^2(t)) dt$$

be an objective functional to be minimized, and let

$$\begin{array}{rcl} U &=& \{(c_h(t), c_m(t)) \in [0,1] \times [0,1] \mid c_h, \\ && c_m \text{ are Lebesgue integrable functions and } t \in [0, t_f]\}, \end{array}$$

where $B$ and $C$ are constants. Our goal is to find an optimal proportion of protected humans and optimal proportion of killed mosquitoes that will minimize the human infected population. In the following result, we assert the existence of such an optimal control $c^* = (c_h^*, c_m^*) \in U$.

**Theorem 2.2.** *In System (3), there exists $c^* = (c_h^*, c_m^*) \in U$ such that $J(c_h^*, c_m^*) = \min J(c_h, c_m)$. That is, there exists an optimal use of treated bed nets and insecticide that will minimize the infected human population.*

*Proof.* In System (3), all the coefficients are bounded. Hence, the set of controls and corresponding state variables is nonempty. The control set $U$ is convex and closed. Since the system is bilinear in $c_h$ and $c_m$, the right hand side of the system is bounded by a linear function in the state and control variables. Moreover, the integrand of the objective function is convex in $c_h$ and $c_m$. We then obtain that there exists an optimal pair, $c_h^*$ and $c_m^*$ that minimizes $J$. □

3. **Model fit to Missira data.** To measure the effects of mosquito bed nets usage and mosquito annihilation with insecticides on the clinical study, we fit our model to the Missira data in Table 1. Following Dembele *et al.*, we choose a specific positive and bounded mosquito infection rate $\gamma$ so that Model (3) fits the clinical data in Table 1, where estimates of the other model parameters are obtained from the real population data of Mali (see Table 3 and [8], [9]) and $c_h = c_m = 0$.

**Table 3.** Estimates of Model Parameters [9]

| **Model Parameter** | **Estimate (per day)** |
|---|---|
| $\alpha_{hm}$ | $1.75 \times 10^{-1}$ |
| $\alpha_{mh}$ | $1.75 \times 10^{-1}$ |
| $b_m$ | 1 *bite* |
| $\lambda_h$ | $10^{-4}$ |
| $\lambda_m$ | *Estimated* |
| $\beta_h$ | $3 \times 10^{-2}$ |
| $\alpha_h$ | $2.5 \times 10^{-1}$ |
| $\alpha$ | $1.75 \times 10^{-1}$ |
| $\mu_h$ | $9 \times 10^{-5}$ |
| $\mu_m$ | $3.3 \times 10^{-2}$ |

Following Dembele *et al.*, we choose

$$\gamma(t) = \frac{0.175t}{1+t^2} + 0.175$$

and

$$\frac{N_m}{N_h} = \frac{t}{1+t^2} + 10.$$

Using $\frac{dN_m}{dt} = (\lambda_m(t) - \mu_m)N_m$, we obtain that

$$\lambda_m(t) = \frac{1-t^2}{(1+t^2)\,(10t^2+t+10)} + \lambda_h - \mu_h + \mu_m.$$

In Mali, there are two main seasons, a wet season from April to September and a dry season from October to March. Due to exponential growth of mosquito population in the wet season, in general, malaria incidence in Mali is higher in the wet season than in the dry season. The mosquito growth is mainly affected by mosquito birth rate. As a result, the mosquito birth rate can be viewed as a one year periodic function. In the clinical studies of Allen and Cisse, data was collected in the wet season. Consequently, the mosquito birth rate, $\lambda_m(t)$, is a time dependent function but not a periodic function over the wet season, but all the other parameters are constant over the wet season (see Table 3).

Then Model (3) becomes the following deterministic non-autonomous system of equations.

$$\left.\begin{array}{rcl} \frac{ds_h}{dt} &=& (\lambda_h+\beta_h)(1-s_h) - \beta_h i_h - 0.175(1-c_h)\left(\frac{t}{1+t^2}+1\right)s_h i_m, \\ \frac{di_h}{dt} &=& 0.175(1-c_h)\left(\frac{t}{1+t^2}+1\right)s_h i_m - (\lambda_h+\alpha_h)i_h, \\ \frac{di_m}{dt} &=& (1-c_m)(1-i_m)\alpha i_h - \left(\frac{1-t^2}{(1+t^2)(10t^2+t+10)} + \lambda_h - \mu_h + \mu_m\right) i_m. \end{array}\right\} \quad (4)$$

Using Equation (2), the model parameter estimates of Table 3 and $\min_{0\le t\le T=1}\{\gamma(t)\} = 0.175$ together with our choice of $\lambda_m(t)$, we obtain that $\mathcal{R}_0$ for Missira is greater

than 1.49. That is, Malaria is endemic in Missira. Furthermore, using the initial population size,

$$(s_h(0), i_h(0), i_m(0)) = (0.8, 0.1, 0.1),$$

we compute the percentage of malaria infections when $c_h = c_m = 0$. The percentage of malaria cases we obtained from Model (4) is 22.37% (see Figure 1), about that same as that of the clinical study of Allen and Cisse in Table 1 [1].

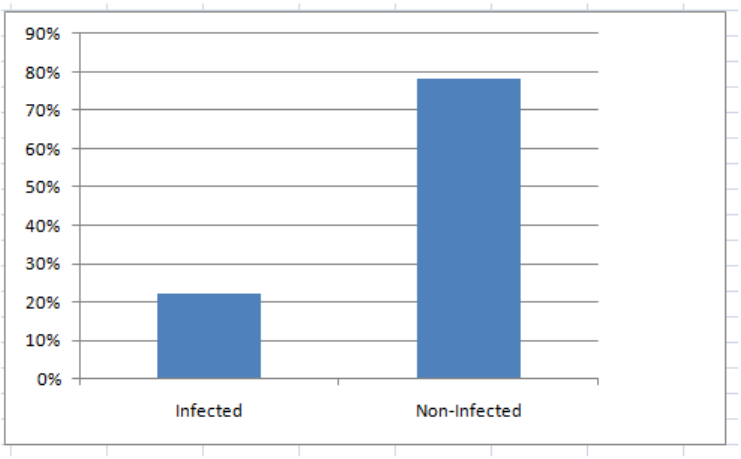


FIGURE 1. Mathematical model results on malaria incidence in Missira when humans are not protected and mosquitoes are not killed.

4. **Malaria in Missira: Treated mosquito bed nets and insecticides.** The incidence of malaria disease has decreased in about 41% of the population of Mali that regularly sleep in chemically treated mosquito bed nets. However, malaria disease remains endemic in Mali. Because the chemically treated mosquito bed nets are mostly used at night during bedtime, mosquitoes in Mali have opportunities to bite people before bedtime. Consequently, the use of treated mosquito bed nets is not enough for complete eradication of the malaria disease in Mali. Next, we use Model (4) to study the impact of adopting each of the following three policies on the incidence of malaria in Missira.

- Protecting humans from mosquito bites (e.g. sleeping in chemically treated mosquito bed nets).
- Killing mosquitoes (e.g. using insecticides to kill mosquitoes).
- Protecting humans from mosquito bites and killing mosquitoes.

4.1. **Protection Of humans from mosquito bites.** In this section, we compute optimal proportion of human population that must be protected from mosquito bites in order to contain the spread of the malaria disease, where mosquitoes are not killed. For this computation, we fix all the parameters in System (4) at their

current values in Table 3 and let $c_m = 0$. Using the initial condition $(0.8, 0.1, 0.1)$ in System (4), we obtain an optimal value of $c_h^* = 0.9$. That is, when at least 90% of the population of Missira is protected from mosquito bites and mosquitoes are not killed, then the proportion of malaria infected individuals is zero (see Table 4). However, when $c_m = 0$ and $c_h \leq 0.80$, then at least 1% of the human population is infected with malaria (see Table 4). That is, malaria remains endemic in Missira when not more than 80% of humans are protected from mosquito bites and mosquitoes are not being killed.

**Table 4.** Malaria Iincidence When Humans Are Protected And Mosquitoes Are Not Killed

| **Protected Humans** | **Infected Humans** | **Non-Infected Humans** |
|---|---|---|
| 10% | 20% | 80% |
| 20% | 18% | 82% |
| 30% | 16% | 84% |
| 40% | 14% | 86% |
| 50% | 11% | 89% |
| 60% | 7% | 93% |
| 70% | 3% | 97% |
| 80% | 1% | 99% |
| 90% | 0% | 100% |

4.2. **Mass mosquito killing.** Now, we compute the optimal proportion of mosquito population that must be destroyed in order to contain the spread of the malaria disease, where human population is not protected from mosquito bites. For this computation, we fix all the parameters in System (4) at their current values in Table 3 and let $c_h = 0$. Using the initial condition $(0.8, 0.1, 0.1)$ in System (4), we obtain an optimal value of $c_m^* = 0.9$. That is, when 90% of the mosquito population in Missira is killed and individuals are not protected from mosquito bites, then the proportion of malaria infected individuals is zero. However, when $c_h = 0$ and $c_m \leq 0.8$ then the proportion of malaria infected individuals in the population is at least 2% (see Table 5). That is, malaria remains endemic in Missira when not more than 80% of the mosquito population is killed and humans are not protected from mosquito bites.

**Table 5.** Malaria Incidence When Humans Are Not Protected But Mosquitoes Are Killed

| **Killed Mosquitoes** | **Infected Humans** | **Non-Infected Humans** |
|---|---|---|
| 10% | 21% | 79% |
| 20% | 20% | 80% |
| 30% | 19% | 81% |
| 40% | 17% | 83% |
| 50% | 15% | 85% |
| 60% | 11% | 89% |
| 70% | 7% | 93% |
| 80% | 2% | 98% |
| 90% | 0% | 100% |

4.3. **Combined policy: Protecting humans and killing mosquitoes.** Now, we consider the case where both policies are adopted simultaneously. That is, we compute optimal proportion of the population that must protected from mosquito bites and optimal proportion of mosquito population that must be destroyed. For this computation, we fix all the parameters in System (4) at their current values in Table 3, where $c_h \neq 0$ and $c_m \neq 0$. In this case of combined policy, we obtain that several combinations of $c_h^*$ and $c_m^*$ values lead to a complete eradication of the malaria disease in the human population. For example, we obtain non-unique optimal values of $(c_h^*, c_m^*) = (0.50, 0.70)$ and $(c_h^*, c_m^*) = (0.60, 0.60)$. That is, when 50% (respectively, 60%) of the human population is protected from mosquito bites and 70% (respectively, 60%) of the mosquito population is killed, then the proportion of malaria infected individuals in the population is zero (see Table 6). However, when $(c_h, c_m) = (0.50, 0.30)$ or $(c_h, c_m) = (0.40, 0.40)$, then about 14% of the population is infected with malaria. That is, malaria remains endemic in Missira when 50% (respectively, 40%) of humans are protected from mosquito bites and 30% (respectively, 40%) of the mosquitoes population are killed.

**Table 6.** Malaria Incidence When Humans Are Protected And Mosquitoes Are Killed

| **Protected Humans** | **Killed Mosquitoes** | **Infected Humans** |
|---|---|---|
| 50% | 70% | 0% |
| 60% | 60% | 0% |
| 40% | 80% | 0% |
| 70% | 50% | 0% |
| 80% | 20% | 0% |

5. **Conclusion.** We used a simple deterministic malaria model to study the potential combined effects of protecting individuals from mosquito bites and killing mosquitoes on the incidence of malaria disease in Missira, a village in Mali. The conclusion of the model results are the following.

- If humans are not protected from mosquito bites and mosquitoes are not killed, then about 22.37% of the population of Missira remains infected with malaria disease. This model result agrees with the clinical results of Allen and Cisse in Table 1.
- If at least 90% of the population of Missira is protected from mosquito bites and mosquitoes are not killed, then malaria disease would be eradicated from Missira. In the absence of mosquito annihilation, malaria remains endemic in Missira when not more than 80% of the population is protected from mosquito bites.
- If at least 90% of the mosquito population is killed and humans are not protected from mosquito bites, then malaria disease would be eradicated from Missira. In the absence of human protection, malaria remains endemic in Missira when not more than 80% of the mosquito population is killed.
- Several combinations of proportion of protected humans and proportion of killed mosquitoes lead to malaria eradication. For example, protecting 50% (respectively, 60%) of humans and killing 70% (respectively, 60%) of the mosquito population would eradicate malaria from Missira.

Currently, about 41% of the people in Mali regularly use chemically treated mosquito bed nets to protect themselves from mosquito bites. By the above model results, the best policy is the simultaneous adoption of both interventions, protecting humans and killing mosquitoes. However, several optimal combinations of the two interventions lead to malaria eradication. For example, by increasing the percentage of treated bed net usage from 41% to 60% (respectively, 50%) via educational campaign and using insecticides to kill 60% (respectively, 70%) of the mosquito population, the deadly malaria disease can be eradicated from Missira. These conclusions of our model results should be tested clinically. If it turns out that the dual strategy of protecting humans from infected mosquito bites and mass killing of mosquitoes is the best policy, then the cost of the interventions could be used to determine the best combination for adoption. In some geographical locations, the cost of using chemically treated mosquito bed nets could be less than that of using insecticides to kill mosquitoes.

To obtain our model results, we first chose the infection function $\gamma(t)$. The function is allowed to vary over time and it fits well the clinical data of Allen and Cisse. Estimating the "range" of functions that "fit" the observed clinical data and determining how our model results depend on these functions are interesting questions for future studies.

In their clinical study, Allen and Cisse used a sample of 76 children with ages ranging from 1-10 years old to determine the frequency of malaria incidence. Malaria is responsible for 25% of deaths in children of Mali. Using age-structured deterministic and stochastic malaria models to study cumulative and age-specific incidence of malaria could be useful for future malaria investigations.

**Acknowledgments.** This research has been partially supported by the National Marine Fisheries Service, Northeast Fisheries Science Center (Woods Hole, MA 02543), Department of Homeland Security, DIMACS and CCICADA of Rutgers University, Mathematical Biosciences Institute of the Ohio State University and National Science Foundation under grants DMS 0931642 and 0832782.

## REFERENCES

[1] A. D. Allen and M. Cisse, *Determination of the frequency and correlation between glucose 6-phosphate dehydrogenase deficiency and sickle cell anemia (HbS) in a west African village (Mali), a malaria endemic region*, Technical Report, Department of Biology (Howard Hughes Program), Howard University, 2010.

[2] N. T. J. Bailey, "The Mathematical Theory of Epidemics," Hafner Publishing Co., New York, 1957.

[3] A. Bekessey, L. Molineaux and J. Storey, *Estimation of incidence and recovery rates of Plasmodium falciparum parasitaemia from longitudinal data*, Bull. World Health Organ, **54** (1976), 685–693.

[4] P. Carnevale, J. Mouchet, M. Coosemans, J. Julvez, S. Manguin, R. D. Lenoble and S. Sircoulou, "Biodiversit du Paludisme dans le Monde," John Libbey Eurotext, Paris, 2004.

[5] D. Coulibaly, D. Diallo, M. Thera, A. Dicko, A. Guindo, A. Kone, Y. Cissoko, S. Coulibaly, A. Djimde, K. Lyke, O. Doumbo and C. Plowe, *Impact of preseason treatment on incidence of falciparum malaria and parasite density at a site for testing malaria vaccines in Bandiagara, Mali*, Am. J. Trop. Med. Hyg., **67** (2002), 604–610.

[6] R. Carter, K. N. Mendis and D. Roberts, *Spatial targeting of interventions against malaria*, Bulletin of the World Health Organization, **78** (2000), 1401–1411.

[7] Centers for Disease Control and Prevention, *Malaria*. Availabe from: http://www.cdc.gov/malaria.

[8] B. Dembele, A. Friedman and A.-A. Yakubu, *Malaria model with periodic mosquito birth and death rates*, J. Biol. Dyn., **3** (2009), 430–445.

[9] B. Dembele, A. Friedman and A.-A. Yakubu, *Mathematical model for optimal use of sulfadoxine-pyrimethamine as a temporary malaria vaccine*, Bulletin of Mathematical Biology, **72** (2010), 914–930.
[10] K. Dietz, *Mathematical models for malaria in different ecological, zones*, Presented to the 7*th* International Biometric Conference, Hannover, August 1970, 1621.
[11] K. Dietz, *Mathematical models for transmission and control of malaria*, in "Principles and Practice of Malariology" (eds. W. H. Wernsdorfer and I. McGregor), Churchill Livingstone, London, (1988), 1091–1113.
[12] A. N. Gideon, and W. S. Shu, *A Mathematical Model for Endemic Malaria with Variable Human and Mosquito populations*, United Nations Educational Scientific and Cultural Organization and International Atomic Energy Agency, IC, (1999), 127. Available from: http://www.ictp.trieste.it/puboff.
[13] N. J. Govella, F. O. Okumu and F. Killeen. Gerry, *Insecticide-treated nets can reduce malaria transmission by mosquitoes which feed outdoors*, Am. J. Trop. Med. Hyg., **82** (2010), 415–419.
[14] N. C. Grassly and C. Fraser, *Seasonal infectious disease epidemiology*, Proc. R. Soc. B, **273** (2006), 2541–2550.
[15] W. O. Kermack and A. G. McKendrick, *Contributions to the mathematical theory of epidemics (part I)*, Proc. R. Soc. A, **115** (1927), 700–721.
[16] G. F. Killeen, A. Seyoum and B. G. J. Knols, *Rationalizing historical successes of malaria control in Africa in terms of mosquito resource availability management*, Am. J. Trop. Med. Hyg., **71** (2004), 87–93.
[17] A. J. Lokta, *Contributions to the analysis of malaria epidemiology*, Am. J. Hyg., **3** (1923), 11–21.
[18] G. Macdonald, *The analysis of infection rates in diseases in which supperinfection occurs*, Trop. Dis. Bull., **47** (1950), 907–915.
[19] E. Martini, "Berechnungen und Beobachtungen zur Epidemiologie und Bekam pfung der Malaria," Gente, Hamburg, 1921.
[20] F. E. McKenzie and H. W. Bossert, *An integrated model of Plasmodium falciparum dynamics*, J. Theor. Biol., **232** (2005), 411–426.
[21] A. L. Menach, E. F. McKenzie, A. Flahault and D. L. Smith, *The unexpected importance of mosquito oviposition behaviour for malaria: Non-productive larval habitats can be sources for malaria transmission*, Malaria Journal, (2005), 4–23.
[22] National Institute of Allergy and Infectious Diseases, *Malaria*, Publication No. 02-7139, 2002.
[23] T. J. Norman and M. A. Baley, "The Biomathematics of Malaria," Oxford University Press, London, 1982.
[24] R. Ross, "The Prevention of Malaria," $2^{nd}$ edition, With Addendum on the Theory of Happenings, Murray, London, 1911.
[25] N. Sagoba, S. Doumbia, P. Vounatsou, I. Baber, M. Keita, M. Maiga, S. Toure, G. Dolo, T. Smith and J. M. C. Ribeiro, *Monitoring of larval habitats and mosquito densities in the Sudan savanna of Mali: Implications of malaria vector control*, Am. J. Trop. Med. Hyg., **77** (2007), 82–88.

Received May 2011; revised July 2011.

*E-mail address*: dem_77@hotmail.com
*E-mail address*: ayakubu@howard.edu