Modeling and Simulation of Spread and Effect of Malaria Epidemic

ALUKO,Olabisi Babatope *honaob@yahoo.com.com*
College of Technology,Department of Mathematics and Statistics
Osun State Polytechnic,
Iree, Osun State,Nigeria

BABATUNDE *hezecomp@yahoo.com*
College of Engineering,Science, and Technology,
Department of Information and Communication Technology
Osun State University
Osogbo,+234(35),Nigeria

Oluleye Hezekiah *babatundeho@uniosun.edu.ng*
College of Engineering,Science, and Technology,
Department of Information and Communication Technology
Osun State University
Osogbo,+234(35),Nigeria

ISIKILU Idayat Temilade *isikiluidaya@yahoo.com*
College of Technology,Department of Mathematics and Statistics
Osun State Polytechnic,
Iree, Osun State,Nigeria

OJO Bamidele *deleomoojo@yahoo.com*
Community High School,
Iroko, Ota
Ogun State, ,Nigeria

Abstract

The purpose of this paper is to consider malaria infection *(A)* and the control of malaria *(B)* as the two sets of soldiers engage in a war. The principal objectives are to see if it is possible with time to reduce and eradicate malaria in our environment taking reasonable precaution. The methodology approach is to model a mathematical equation using battling method approach to find the time*(t)* that control malaria in our environment will conquer the malaria infection i.e. when *A(t)=0*. The number of provided facilities *(n)* for the protection of malaria is also considered and varied. The result shows that as the number of malaria control increases the control time is decreasing

Keywords: Mathematical Modeling, Plasmodium Falciparum, Battling Methods & Merozoites.

1. INTRODUCTION

Despite considerable effort over the years to control malaria in our environment, many aspects of malaria and particularly the interactions between the parasite and its host which result in the disease are still poorly understood in our country.

A malaria infection starts with the bite of an infected female anopheles mosquito which introduces a few sporozoite stages that migrate to and infect liver cells. Asexual replication in the liver cell results in the release of thousands of merozoites that initiate the blood stage of the infection. This stage is responsible for the pathology associated with malaria (anaemia, fever, malase, anorexia est.). Merozoites infect red blood cells (RBCs) where they multiply to produce 8 – 32 new

merozoites (depending on the malaria species), which are released by lysis of the RBCs. The released merozoites can infect new RBCs, causing a rapid increase in parasites and infected cells. During this initial or acute phase of infection, different parasite strains reach different densities and cause varying degrees of anaemia and other pathology [5], [4], [11], [13], [12]. While most studies agree that specific immunity does not play a major role in this initial dynamics, there is considerable controversy over which factors drive the dynamics shortly after infection [2], [8], [9], [14], [15]. Gives two reasons why it is difficult to determine the contribution of these different factors to the dynamics of acute infection. The first problem is that there is relatively limited data on the dynamics of the parasite and the loss of RBCs following infection of humans with human malaria parasites such as parasite falciparum and parasite vivax. One approach to overcome this limitation is to use data from well characterized model system such as infections of mice with species such that parasite chabondi or parasite yoeli. The second problem is that the dynamic of infection may involve many interacting populations which fall into three groups. (i) The parasite and its resource i.e merozoites, uninfected and infected RBCs (ii) The inmate immune response i.e macrophages, dendritic cells, crytokines e.t.c and (iii) The adaptive immune response i.e B & T cells and antibodies.

[15] Bring mathematical models of the early stages of malaria infection into contact with experimental data on the time course of both the parasite density and the loss of RBCs following infection. [17] Mathematical modeling has long been applied to malaria control and is particularly relevant to day in light of rapid country progress in reaching high intervention coverage targets and given the intensifying global efforts to achieve the malaria control among the children. Mathematical modeling has been a valuable decision making tools for vaccination strategies against infections disease in particular for those covered by the Expand Program on Immunization (EPI). [1]Compared with other organisms that cause infectious disease parasite falciparum has a complex life cycle, expressing many different potential targets for vaccines and various candidate vaccine targeting different stages of the parasites are in clinical development. [3] The history of infective or partially effective control of malaria and failed vaccination attempts has led to the assumption that the efficacy of a malaria vaccine is unlikely to approach 100%, but since parasite falciparum is one of the most frequent causes of morbidity and mortality in areas where it is endemic, [16], [4], [7] even a partially protective vaccines may be highly cost-effective and a critically important public health tool. Mathematical models of both the natural history and epidemiology of malaria are needed to guide the process malaria control. Malaria models have several roles that transcend their obvious limitation in making precise predictions. A mathematical model was developed elsewhere [10] taking into account some biological features related to malaria disease such as partial acquired immunity, immunology memory and duration of sporogony. From the model equilibrium points were determined and the stability of these points was analyzed.

In a subsequent study [10] the previously developed model was used to assess the effects of global warning and local socioeconomic condition on malaria transmission. These effects were assessing analyzing the equilibrium points calculated at different but fixed values of the parameters of the models. Regarding malaria transmission, it was observed that the effects of global warning posed a major challenge and the effects variation in local socioeconomic conditions are much stronger than the effect of the increasing global temperatures.

In this study, we bring mathematical modeling of malaria infection in our environment and its control addressing the challenges of the model to provide useful prediction on malaria control using detail data from World Heath Organization of malaria cases, malaria recorded due to infection and cured due to malaria control shown in Table1. Find out that in the nearest future the number of malaria cases in our environment will be dropped drastically.

2. MODEL

In this model, we shall use the following ,based on our knowledge of malaria Infection:

1. Malaria is caused through parasite i.e. Plasmodium.

2. The vector of malaria is female Anopheles Mosquito

The plasmodium has four species as regards to the region

1. Plasmodium Malaria A
2. Plasmodium Ovale
3. Plasmodium Vivax
4. Plasmodium Falciparum

If we consider malaria infections *(A)* and control of malaria *(B)* as two different set of soldiers engaging in a war. The reason is that no human being would be ready to die irrespective of their ages. But malaria infections as well known, it is a common disease that anybody can contact. So, for the fact that we are more prone to malaria infection in our region, we say that the control of malaria and malaria infections can engage in a battle war in which one would fight another one to finish.

In a battle war, there would be some ammunition which each army engage in the war would use to fight or defend themselves. Malaria in its own side use any of the above four stated plasmodium to fight the control of malaria in our environment. In this sense, we represent malaria Infection by *A* while its ammunition is represented by 'ψ'. At time *(t),* we have A *(t)*

Also, control of malaria in our environment represented by *B* which has some ammunitions (management of malaria) to fight malaria infection. The ammunitions according to our research are:

1. The use of treated mosquito net
2. The use of ointment to rub body
3. Proper environmental sanitation
4. Proper drainage
5. Bush clearing
6. Use of mosquito insecticide
7. Use of chemicals on any stagnant water
8. Use of defensive drug / anti malaria medicine.

We regards all the above listed ammunitions as 'n' while this n can be varied according to the knowledge of individual on malaria. So, the killing power of malaria control on malaria infection is represented by 'θ'. At time 't' we have B (t).

Generally we all know that malaria infection is not an infectious disease likewise it can not be transmitted from one generation to another generation. Then, the control of malaria has more (n) weapons to fight malaria with one weapon.

3.0 Governing Equation

List of symbols

A – Malaria infection

B – Human malaria control

ψ - Malaria weapon

θ - Human weapon

n – No of malaria control

x – Malaria cases confirmed

y – Malaria death

z – Malaria cured

Combining all the factors approximately, we obtained model defining malaria infections over time as:

$$\frac{dA}{dt} = -n\theta B, \qquad n\geq 1 \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \text{(i)}$$

While a model defining control of malaria over time is

$$\frac{dB}{dt} = -\psi A \qquad \ldots\ldots\ldots\ldots\ldots \text{(ii)}$$

Equation (i) and (ii) are first order linear O.D.E. and can be solved.

$$\psi A^2 = n\theta B^2 + (\psi A_0^{\ 2} - n\theta B_0^{\ 2}) \qquad \ldots\ldots\ldots\ldots\ldots \text{(iii)}$$

Recall that: $A(0) = A_0$, $B(0) = B_0$

When will malaria control in human being conquer malaria infection totally?

$$\frac{d^2A}{dt^2} = -n\theta \frac{dB}{dt} \qquad \ldots\ldots\ldots\ldots\ldots \text{(iv)}$$

$$\frac{d^2B}{dt^2} = -\psi \frac{dA}{dt} \qquad \ldots\ldots\ldots\ldots\ldots \text{(v)}$$

Substitute for $\frac{dA}{dt}$ and $\frac{dB}{dt}$ into (iv) and (v) we have

$$\frac{d^2A}{dt^2} = n\psi\theta A \qquad A(0) = A_0 \qquad \ldots\ldots\ldots\ldots\ldots \text{(vi)}$$

$$\frac{d^2B}{dt^2} = n\psi\theta B \qquad B(0) = B_0 \qquad \ldots\ldots\ldots\ldots\ldots \text{(vii)}$$

Solving the second order O.D.E (vi) and (vii) we obtained

$$A(t) = \frac{1}{2}\left(\frac{n\theta B_0}{\sqrt{n\theta\psi}} + A_0\right)e^{\sqrt{n\theta\psi}\ t} + \frac{1}{2}\left(A_0 - \frac{n\theta B_0}{\sqrt{n\theta\psi}}\right)e^{-\sqrt{n\theta\psi}\ t} \qquad \ldots\ldots\ldots\ldots\ldots$$

(viii)

Similarly

$$B(t) = \frac{1}{2}\left(\frac{\psi A_0}{\sqrt{n\psi\theta}} - B_0\right)e^{-\sqrt{n\psi\theta}\ t} + \frac{1}{2}\left(B_0 - \frac{\psi A_0}{\sqrt{n\psi\theta}}\right)e^{-\sqrt{n\psi\theta}\ t} \qquad \ldots\ldots\ldots\ldots\ldots \text{(ix)}$$

Note: Human malaria control will conquer malaria infection totally when A (t) =0

$$t = -\frac{1}{2\sqrt{n\psi\theta}}\log_e\left(\frac{\frac{n\theta B_0}{\sqrt{n\psi\theta}} + A_0}{A_0 - \frac{n\theta B_0}{\sqrt{n\psi\theta}}}\right) \qquad \ldots\ldots\ldots\ldots\ldots \text{(x)}$$

5. Validity of the Model / Example

Table 1:- Malaria cases recorded in Nigeria.

Source: - World Heath Organization (WHO)

Year	Malaria Cases Confirmed (x)	Malaria Death (y)	Malaria Cured (z)
2001	2,253,519	4,317	2,249,202
2002	2,605,381	4,092	2,601,289
2003	2,608,479	5,343	2,603,136
2004	3,310,229	6,032	3,304,197
2005	3,532,108	6,494	3,525,614
2006	3,982,372	6,586	3,975,786
2007	2,969,950	10,289	2,975,786
2008	2,834,174	8,677	2,825,497
	$\sum x$ **= 24,096,212**	$\sum y =$ **51,830**	$\sum z =$ **24,044,382**

$$A_0 = B_0 = \frac{\sum_{i=1}^{8} x}{t} = 3{,}012{,}027$$

$$\psi = \frac{\sum_{i=1}^{8} y}{\sum_{i=1}^{8} x} = 0.00215$$

$$\theta = \frac{\sum_{i=1}^{8} z}{\sum_{i=1}^{8} x} = 0.9978$$

Case 1

If n = 5, $A_0 = B_0$=3,012,027, ψ = 0.00215, θ = 0.9978

$$t = -4.8263 \log_e \left(\frac{148{,}060{,}315}{-142{,}036{,}254} \right)$$

$$= 4.8263 \log_e (1.0424)$$

= 0.20042year

$\approx$ 2months, 12days and 4hours

Case 2.

If n = 8, $A_0 = B_0$ = 3,012,027, ψ = 0.00215, θ = 0.9978

$$t = -3.8124 \log_e \left(\frac{186{,}338{,}022.6}{-180{,}313{,}968.6} \right)$$

$$= 3.8124 \log_e (1.0334)$$

= 0.12525year

≈ 1month, 15days and 2hours

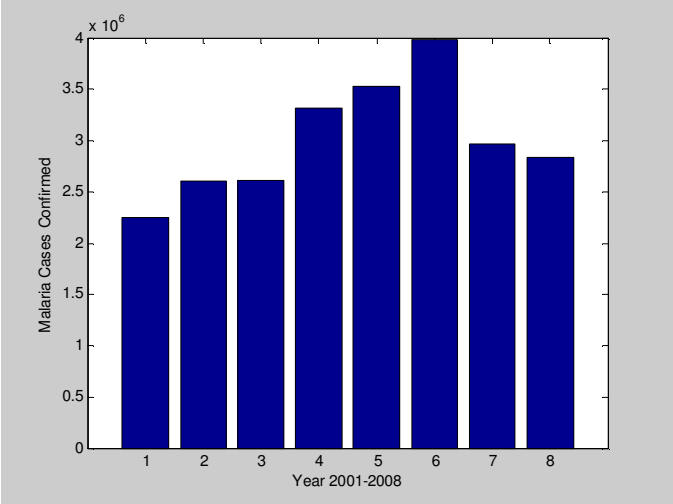

FIGURE1: Malaria Cases Confirmed

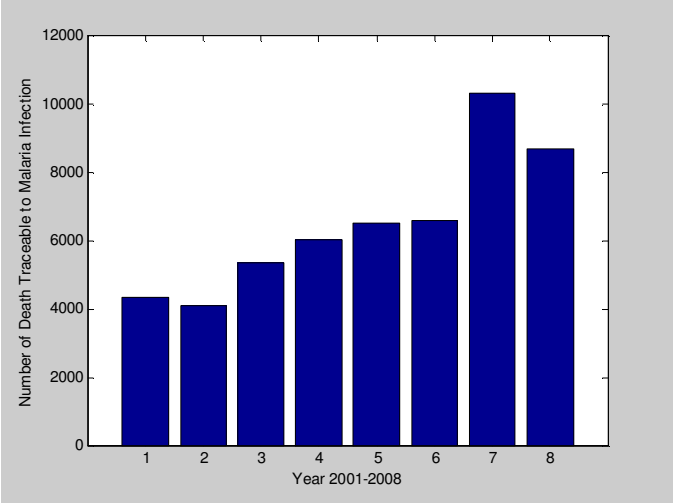

FIGURE 2: No of Death by Malaria

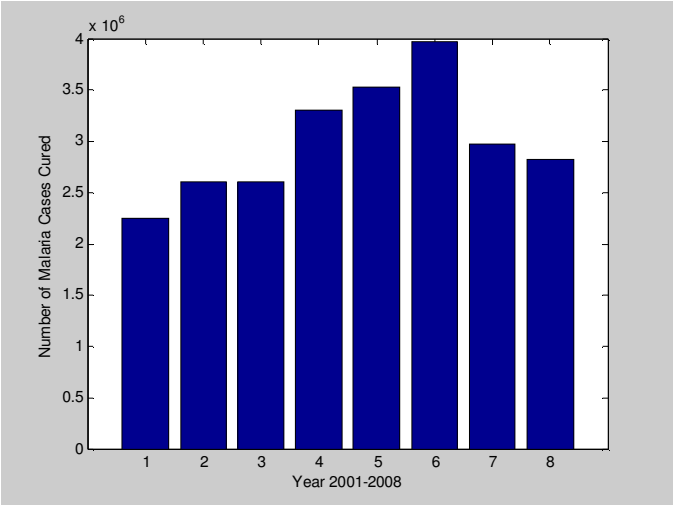

FIGURE 3: No of Malaria Infection Cured

REFERENCE

[1] Anderson, R.M. et al (1989). Non-linear phenomena in host-parasite interactions. Parasitology 99 (Suppl.), S59-S79

[2] Anderson, R.M., May, R.M. (1991). Infections Disease of Humans: Dynamics and control. Oxford, United Kingdom, Oxford University Press.

[3] Ballou, W.R. et al (2004). Update on the Clinical Development of Candidate Malaria Vaccines. Am J Trop Med Hyg 71 (2 suppl): pp239 – 247.

[4] Breman, J.G.; Egan, A.; Keusch, G.T. (2001). The intolerable burden of malaria: a new look at the numbers – Am J. trop med Hyg 64 (suppl): iv-vii.

[5] Field, J.W.; Niven, J.C. (1937). A note on prognosis in relation to parasites counts in acute subtertian malaria. Trans. R. Soc. Trop. Med. Hyg. 6, 569-574.

[6] Field, J.W. (1949). Blood examination and prognosis in acute falciparum malaria. Trans. R. Soc. Trop. Med. Hyg. 43, 33-48

[7] Greenwood, B. et al (2005). Malaria. Lancet: 1487- 1498

[8] Hellriegel, B. (1992). Modeling the immune response to malaria with ecological concepts: short- term behavior against long - term equilibrium. Proc. R. Soc. B 250, 249-256.

[9] Hetzel, C.; Anderson, R.M. (1996). The within – host cellular dynamics of blood stage malaria – theoretical and experimental studies. Parasitology 113, 25-38.

[10] Hyun, M Yang, (2001). A mathematical model for malaria transmission relating global warming and local socioeconomic conditions. Rev. saude public vol. 35 no 3 sao panlo June 2001.

[11] Kitchen, S.F. (1949a). Falciparum malaria in malariology (ed. M.F. Boyd), pp. 995-1016. London, UK: Saunders.

[12] Mackinnon, M.J.; Read, A.F. (2004). Virulence in Malaria: an evolutionary viewpoint. Phil Trans. R. Soc. B 359, 965 – 986.

[13] Molineaux, L. (2001). Plasmodium falciparum parasitaemia described by a new mathematical model. Parsitology 122, 379-391.

[14 Mc Queen, P.G.; Mckenzie, F.E. (2004). Age structured red blood cell susceptibility and the dynamics of malaria infections. Proc. Natl Acad. Sci USA 101, 9161 - 9166.

[15] Rustom Antia, et al. (2008). The dynamics of acute malaria infections .1. Effect of the parasites red blood cell preference. Proc R. Soc B 2008 275, 1449-1458.

[16] Snow, R.W. et al. (2005). The global distribution of clinical episodes of malaria. Nature 434:214-217

[17] Thomas Smith. et al (2006) Mathematical modeling of the impact of malaria vaccines on the clinical epidemiology and natural entory of plasmodrum falciparum malaria: Overview am. J Trop. Med. Hyg, 75 (suppl 2), 2006 pp 1-10