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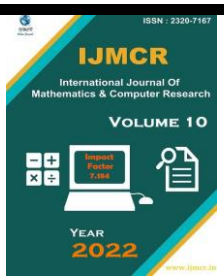

Mathematical Analysis of Seasonal Malaria Transmission in Swampy and Deltaic Regions.

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ABSTRACT

Climatic characteristics involving weather conditions affect malaria transmission in most deltaic regions of the world. Here, we propose a simple mosquito-human interaction model incorporating features of seasonal malaria pathogenesis. We obtain the basic reproduction number and show in our analysis some conditions for local and global stability of the solution, suggesting that intervention strategies should be targeted at reducing seasonal contacts of mosquitoes and humans. The model simulations compare well with malaria infection data.

KEYWORDS: Malaria, Deltaic, Seasonality, Sinusoidal, Meteorological, Transmission, Swampy.

1. INTRODUCTION AND GENERAL BACKGROUND

Malaria is one of the most important infectious disease that is very much common in tropical climatic regions and posing serious public health challenges in the poorest countries of the world. Thus it impacts on major socioeconomics decisions in the affected regions in particular and major world health policies in general. Malaria is caused by a plasmodium parasite associated with the female anopheles mosquito through its struggle to have a blood meal from humans. The disease could lead to a lot of complications including blockage of blood vessels in vital organs, enlargement of the spleen *[3]*, brain damage, kidney and liver failure *[4]*.

Mathematical models are very important tools in explicating the mode of transmission, which later direct and guide the proper implementation of intervention programmes. Malaria transmission could be seasonal or perennial. Knowing the duration, start and end of the malaria transmission season is important in terms of planning control strategies *[2]*. According to WHO's *2021 World malaria report*, there were an estimated 241 million malaria cases and 627 000 malaria deaths worldwide in *2020 [6]*. This trend follows from about 14 million additional cases with 69 thousands more deaths in 2020 compared to *2019* figure.

Despite the discovery of a malaria vaccine, people still fall sick after use and it is still only 30% effective against death. Hence, the recommendation of its use in combination with other therapeutic or preventive drugs *[7]*. There has been a rising debate in recent times as to whether or not global warming is associated with a rise in the number of malaria cases. According to WHO, malaria pathogenesis has the potential to significantly increase in response to climate change since temperature and rainfall have significant impact in the population dynamics of the mosquito vector *[24,25]*. Patterns of malaria transmission and disease vary markedly between regions and even within individual countries, which could be due to ecological differences and variations between malaria parasites and mosquito vectors *[8]*.

Malaria is endemic throughout Nigeria which accounted for *25* percent of all estimated malaria cases in the WHO African region in *2006* with transmission occurring all year round in the south where the Niger delta region is situated with more seasonality in the north *[9]*. Malaria accounts for nearly *110* million clinically diagnosed cases per year, *60* percent hospitalisations. An estimated *300,000* children die of malaria each year. It is also believed to contribute up to *11* percent maternal mortality, *25* percent infant mortality, and *30* percent under-five mortality. In addition to the direct health impact of malaria, there are also severe social and economic burdens on communities and the country as a whole in form of treatment costs, prevention, loss of work time, etc. *[12]*. In the 2020 World Malaria Report, Nigeria had the highest number of global malaria cases estimated as 27% of global malaria cases in *2019* and accounting for the highest number of deaths being 23% of global malaria deaths *[5]*. Understanding the pattern of

malaria transmission is an important step in regional malaria control. This led to the recommendation of seasonal malaria chemoprevention by the World Health Organisation in *2012 [10],* in which Nigeria, through its National Malaria Elimination Program strategy recommends seasonal malaria chemoprevention in nine states in the Sahel region where, malaria treatment is administered once a month for four months during the intense malaria transmission season*[11]*

The Niger Delta Region of Nigeria located at the southern part of the country covers a total of *70,000* km^2 with its riverine area consisting of coastal belt of swamps and vegetated tidal flats formed by a reticulate pattern of interconnected meandering creeks and distributaries of the River Niger. About *70* percent of Nigerian's crude oil and gas production is from the area *[22],* so a lot of oil prospecting companies operate in the region with foreign nationals including those from countries where malaria is said to have been eradicated. Therefore epidemiological researches including mathematical modelling aimed at eradicating or adequately controlling malaria in the region is more of a global interest than regional interest since it is possible for malaria to be exported alongside with oil and gas from the region. The disease is endemic in the region and it is the number one case of mortality and morbidity in the area *[13].*

The region experiences roughly about four months of dry season and eight months of rainy season. The dry season spreads from mid-November to the end of February and a two to three weeks break between July and August within the rainy season. The rainy season creates a favourable environment for mosquitoes to breed since they lay eggs on stagnant water that surrounds the swamps and creeks of the entire region. In order to have a good understanding of our problem and assess the intensity of malaria transmission in the region. The effect of seasonality and mosquito abundance in the pathogenesis of malaria has been emphasized by most mathematical models. Malaria transmission is both seasonal and heterogeneous, and mathematical models targeting appropriate intervention strategies that seek to predict the effects of possible intervention strategies should capture realistic seasonality of vector abundance [1].

A seasonal malaria model describing the Brazilian Amazon region was proposed in *[14].* The model included treatment of infected individuals while linking the latent period to the effect of environmental temperature influence. The model analysis shows temperature increase greatly affects the latent period, which drastically reduce the health care efficiency. Most existing models of malaria seasonality *[15, 16, 18, 19, 20]* use parameters of rainfall, temperature, and/or vegetation indices to describe suitable transmission patterns. However, the work of *[17]* proposes a statistical modelling framework that characterises seasonal patterns derived directly from monthly health facility data across various Islands in Madagascar. Thus modelling framework that accommodates location-specific seasonal characteristics will improve our understanding and planning of intervention strategies especially, with the aid of more available data.

Here we propose a simple malaria model using seasonally induced or variable contact parameters to characterise the case of the Niger Delta using malaria infection data obtained from the region. In section *1* we present the introduction and the general background. We present the model formulation in section *2* and the analysis in section *3*. The numerical solution is given in section 4 and the paper is rounded up with discussion and conclusion in section 5.

2. THE PROPOSED MODEL

In this section we present a four compartment malaria model with the following model variables representing the human population, H and the mosquito population, M.

S_h	Susceptible human population
I_h	Infectious human population
S_m	Susceptible mosquito population
I_m	Infectious mosquito population

The nature of the data collected in the Niger Delta Region showing high infection in the rainy season and low infection in the dry season suggests either a seasonally varying contact rate or a variable birth rate or both. Seasonal variation in mosquito abundance in response to annual variation in temperature and rainfall can cause strong seasonal patterns of disease incidence in malaria epidemic regions*[21]*. The model explains the dynamics of both human and mosquito populations as they progress from susceptible noninfectious states to infectious states. Here we have assumed that all infected humans and mosquitos become infectious. Thus, malaria is transmitted when a susceptible human is bitten by an infected Anopheles mosquito or when a susceptible Anopheles mosquito bites an infectious human. We assume that susceptible humans and mosquitoes get infected at a rates, $\beta_m I_m \frac{S_h}{M}$ and $\beta_h I_h \frac{S_m}{N}$ respectively, where β_m and β_h are rate constants. The fractions $\frac{S_h}{M}$ and $\frac{S_m}{N}$ further explain the probability of contacts with susceptible humans and mosquitoes. A study carried out showed
that combinations of mean monthly temp range $(28 - 32^0C)$, maximum temperature $(24 - 28^0C)$,) and high rainfall provide suitable conditions for seasonal transmission *[20],* and since malaria shows strong seasonality we assume simple sinusoidal fluctuation in which Susceptible mosquitoes are recruited into the mosquito population through a seasonal birth rate $\lambda_m f(t)$, where

$$f(t) = c_0(1 + c_1 \cos 2\pi t), \qquad (1)$$

is a continuous, bounded, positive, periodic and nonzero function of time. The parameter, c_0
is the baseline biting rate and $0 < c_1 < 1$ measures the degree of seasonality. Mosquitoes die at a rate $\alpha_m I_m$ from the disease, and naturally at a rate $\mu_m M$ whereas, infectious humans recover at a rate $\gamma_h I_h$ into the susceptible class. We

assume a natural recruitment and mortality rates of $\lambda_h N$ and $\mu_h N$ for the human population while $\alpha_h I_h$ of them die as a result of the disease. The model consistent with the given assumptions is

$$\frac{dS_h}{dt} = \lambda_h N + \gamma_h I_h - \beta_m I_m \frac{S_h}{M} - \mu_h S_h \quad (2)$$
$$\frac{dI_h}{dt} = \beta_m I_m \frac{S_h}{M} - \alpha_h I_h - \gamma_h I_h - \mu_h I_h \quad (3)$$
$$\frac{dS_m}{dt} = \lambda_m f(t) M - \beta_h I_h \frac{S_m}{N} - \mu_m S_m \quad (4)$$
$$\frac{dI_m}{dt} = \beta_h I_h \frac{S_m}{N} - \alpha_m I_m - \mu_m I_m \quad (5)$$
$$\frac{dN}{dt} = \lambda_h N - \alpha_h I_h - \mu_h N \quad (6)$$
$$\frac{dM}{dt} = \lambda_m f(t) M - \alpha_m I_m - \mu_m M \quad (7)$$

Noting that *(6)* and *(7)* are obtained by adding $(2) - (3)$ and $(4) - (5)$ respectively, we impose at $t = 0$, $N = N_0$, $M = M_0$ as initial conditions for the human and mosquito populations, and working with population fractions, we rescale the system by writing

$$S = \frac{S_h}{N},\ I = \frac{I_h}{N},\ X = \frac{S_m}{M},\ Y = \frac{S_m}{M},\ \widehat{N} = \frac{N}{N_0},\ \widehat{M} = \frac{M}{M_0},\ \hat{t} = \frac{t}{t_0}, \quad (8)$$

where *S, I, X, Y*, $\widehat{N}$, $\widehat{M}$ and $\hat{t}$ are nondimensional variables. We note that for the susceptible human equation,

$$\frac{N}{t_0}\frac{dS}{d\hat{t}} = \frac{dS_h}{dt} - S\frac{dN}{dt}. \quad (9)$$

By expressing other state variables in this form including the human and mosquito populations and substituting these in $(2) - (7)$ we have,

$$\frac{dS}{d\hat{t}} = t_0\lambda_h(1 - S) - t_0\beta_m f(t)SY + t_0\gamma_h I_h + t_0\alpha_h SI, \quad (10)$$
$$\frac{dI}{d\hat{t}} = t_0\beta_m f(t)SI - t_0\lambda_h I - \alpha_h I - t_0\gamma_h I + t_0\alpha_h I^2, \quad (11)$$
$$\frac{dX}{d\hat{t}} = t_0\lambda_m f(t)(1 - X) - t_0\beta_h f(t)XI + t_0\alpha_m XY, \quad (12)$$
$$\frac{dY}{d\hat{t}} = t_0\beta_h f(t)XI - t_0\alpha_m Y - t_0\lambda_m f(t)Y + t_0\alpha_m Y^2, \quad (13)$$
$$\frac{dN}{d\hat{t}} = t_0\lambda_h N - t_0\alpha_h NI - t_0\mu_h N, \quad (14)$$
$$\frac{dN}{d\hat{t}} = t_0\lambda_m f(t)M - t_0\alpha_m YM - t_0\mu_m M. \quad (15)$$

Due to the significance of seasonality in the recruitment of mosquitoes into the mosquito population, we rescale time with the recruitment rate of mosquito. Thus, by defining the following dimensionless parameters

$$t_0 = \frac{1}{\lambda_m},\ a = \frac{\lambda_h}{\lambda_m},\ b = \frac{\beta_m}{\lambda_m},\ d = \frac{\gamma_h}{\lambda_m},\ e = \frac{\alpha_h}{\lambda_m},\ q = \frac{\beta_h}{\lambda_m},\ g = \frac{\alpha_m}{\lambda_m}, h = \frac{\mu_h}{\lambda_m}, r = \frac{\mu_m}{\lambda_m}, \quad (16)$$

and substituting these into $(10) - (15)$, we obtain the following nondimensional system;

$$\frac{dS}{d\hat{t}} = a(1 - S) + dI - bf(t)SY + eSI, \quad (17)$$
$$\frac{dI}{d\hat{t}} = bf(t)SY - (a + d + e)I + eI^2, \quad (18)$$
$$\frac{dX}{d\hat{t}} = f(t)(1 - X) - qXI + gXY, \quad (19)$$
$$\frac{dY}{d\hat{t}} = qXI - f(t)Y - gY + gY^2, \quad (20)$$
$$\frac{dN}{d\hat{t}} = (a - h)N - eNI, \quad (21)$$
$$\frac{dM}{d\hat{t}} = (f(t) - r)M - gYM, \quad (22)$$

subject to the initial conditions,

$$N(0) = 1, M(0) = 1,\ S(0) = S_0,\ I(0) = 1 - S_0, X(0) = X_0,\ Y(0) = 1 - X_0. \quad (23)$$

3. MODEL ANALYSIS

3.1 Determining the Disease Threshold (R_0)

The disease threshold, in other words known as the basic reproduction number is the number of secondary infection that may possibly be generated by introducing one infectious agent into an infection free environment and in this case, an infectious human or mosquito. We consider the vector equation

$$\ddot{\iota}' = (\theta_a - \theta_b)\pi_c,$$

describing the emergence of new infections and their systemic transition among compartments wherein $\theta_a\pi_c$ represents emergence of new infection, $\theta_a\pi_c$ the infection distribution vector and π_c is the reservoir of infection vector.

$$\theta_a = \begin{bmatrix} 0 & bf(t) \\ q & 0 \end{bmatrix},\quad \theta_b = \begin{bmatrix} (a + d + e) & 0 \\ 0 & (g + f(t)) \end{bmatrix},\quad \pi_c = \begin{bmatrix} I \\ Y \end{bmatrix}$$

The largest eigenvalue of $G_0 = \theta_a\theta_b^{-1}$ is the basic reproduction number.

$$G_0 = \frac{1}{(a+d+e)(g+f(t))}\begin{bmatrix} (g + f(t)) & 0 \\ 0 & (a + d + e) \end{bmatrix},$$

$$= \frac{1}{(a+d+e)(g+f(t))}\begin{bmatrix} 0 & bf(t)(g + f(t)) \\ q(a + d + e) & 0 \end{bmatrix}$$

The highest eigenvalue of G_0 in terms of σ^* gives:

$$\sigma^{*2} = R_0 = \frac{qbf(t)}{(g+f(t))(a+d+e)} \quad (24)$$

3.2 Positivity, Existence and Uniqueness of Solution

The model is described in the domain

$$\Omega \in \mathbb{R}^6 = \{S,\ I, X, Y, N, M : S \geq 0, I \geq 0, X \geq 0, Y \geq 0, N > 0, M > 0, S + I = 1,\ X + Y = 1\} \quad (25)$$

Suppose $at\ t = 0$ all variables are non-negative, it implies that $S(0) + I(0) = 1$ and $X(0) + Y(0) = 1$. If $S = 0$, and all other variables are in Ω, then, $\frac{dS}{dt} \geq 0$, this is also the case for variables in (18) - (20). If $N = 0$ and $M = 0$, then $\frac{dN}{dt} = 0$ and $\frac{dM}{dt} = 0$. But if $N > 0$ and $M > 0$, assuming $a > h$, and $f(t) > r$, then with suitable initial conditions, $\frac{dN}{dt} > 0$ and $\frac{dM}{dt} > 0$ for all values of $t > 0$. We note that the right-hand side of *(17)* - *(22)* is continuous with continuous partial derivatives. Thus, solutions exist and are unique.

The model has mathematically and biologically relevant solutions in the domain $\Omega\ \forall\ t \in [0, \infty)$

3.3 Steady State Solution

The equilibrium point is $E_0 = (S, I, X, Y) = (1,0,1,0)$. At the disease free state, there are neither infected mosquitoes nor infected humans, wherein $I = 0$ and $Y = 0$. Substituting these into the right hand side of (17), and (19) gives $S = 1$, and $X = 1$. We note that the populations of humans and mosquitoes are gradually increasing provided $> h$, and $f(t) > r$ with the population of mosquitoes expected to exhibit some oscillatory behaviour due to the choice of the function, *f(t)* describing seasonal mosquito birth rate.

3.4 Local Stability Analysis of the Disease Free Equilibrium (E_0)

Lemma 3.1: The disease free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: We assume that $0 < c_1 < 1$ so that *f(t)* is positive including all other parameters of system $(17) - (22)$ and the linearization of the system about the disease free state yields the following characteristic equation with eigenvalue, λ.

$$(\lambda + a)(\lambda + f(t))\{\lambda^2 + (a + b + e + f(t) + g)\lambda + (f(t) + g)(a + b + e) - gbf(t)\} = 0 \qquad (26)$$

If $R_0 < 1$, then using *(24) in (26)*, we observe that all the coefficients of the quadratic polynomial are positive and nonzero, so by the Decartes' rule of signs there are no positive real eigenvalues, This means there are two negative eigenvalues or a pair of complex conjugate roots with negative real parts. Thus the disease free equilibrium is locally asymptotically stable if $R_0 < 1$. We note that if $R_0 > 1$, then there will be one sign change and by using the Descartes' rule of sign we conclude that the disease free state is unstable for $R_0 > 1$.

3.5 Global Stability Analysis of the Disease Free Equilibrium (E_0)

Lemma 3.2: The disease free equilibrium is globally asymptotically stable in Ω if $R_0 < 1$.

Proof: We consider the function, $\Psi: \{(S, I, X, Y) \in \Omega : S, X > 0\} \to \mathbb{R}$, where

$$\Psi = \frac{1}{f}(1 - S) + \frac{1}{f}I + (1 - X) + Y. \qquad (27)$$

We note that $\Psi \geq 0$ and is continuously differentiable on the interior of Ω. We show that the disease free state is a global minimum of Ψ on Ω when $R_0 < 1$. The derivative of Ψ computed along solutions of the system is

$$\frac{d\Psi}{dt} = \frac{-a}{f}(1 - S) - \frac{d}{f}I + bSY - \frac{e}{f}SI + bSY - \frac{1}{f}(a + d + e)I + \frac{e}{f}I^2 - f(1 - X) - qXI - gXY + qXI - (f + g)Y + gY^2,$$

and after some simplifications gives,

$$\frac{d\Psi}{dt} = -2\left\{\left[\left(\frac{a+d}{q}\right) - f\right](1 - S) + qS(1 - S) + (f - b)(1 - X) + \frac{e}{f}SI + gXY + bI(1 - X)\right\} \qquad (28)$$

We observe from *(28)* that $\frac{d\Psi}{dt} \leq 0$ whenever $b < f < \frac{a+d}{q}$. For $(I, Y) = (0, 0)$, $\frac{d\Psi}{dt} \leq 0$ and (I, Y) is the positively invariant subset in the interior of Ω and by LaSalle's invariant principle *[26]*, $(I, Y) \to (0, 0)$, while $(S, X) \to (1, 1)$ on the boundary of Ω. We show that

$R_0 = \frac{qbf}{(g+f)(a+d+e)} = \frac{qbf}{g(a+d+e)+f(a+d+e)} = \frac{qbf}{g(a+d+e)+f(a+d)+fe} = \frac{b}{\left(\frac{a+d}{q}\right)+\theta}$, where $\theta = \frac{g(a+d+e)+fe}{qf} > 0$ due to the positivity condition of the model parameters. Since $b < \frac{a+d}{q}$, it follows that $R_0 < 1$. Thus the disease free state is globally asymptotically stable if it is less than unity.

4. NUMERICAL SOLUTION

The numerical solution is obtained by using MATLAB's ODE45, variable order Runge-Kutta method with relative and absolute tolerance of 10^{-9}. The dimensionless parameters used for the simulations are defined in *(16)* with numerical values $a = 0.0047$, $b = 0.0592$, $d = 0.01063$, $e = 0.000765$, $q = 0.921$, $g = 0.00052$, $h = 0.00446$, $r = 0.0385$, $c_0 = 0.0635$, $c_1 = 0.132$, $\sigma = 0.088$, with initial conditions *S = 1, I = 0, X = 0.8, Y = 0.2, N = 1, M = 1*. The original parameter values are estimates obtained from *[20]* with little adjustments through our model fitting to malaria infection data. In order to compare the model with data we write $S = 1 - I$ and $X = 1 - Y$. Substituting these into system $(17) - (22)$, we have a reduced system involving infectious humans and mosquitoes as follows:

$$\frac{dI}{d\hat{t}} = bf(t)(1 - I)Y - (a + d + e)I + eI^2, \qquad (29)$$

(18)

$$\frac{dY}{d\hat{t}} = q(1 - Y)I - f(t)Y - g(1 - Y)Y, \qquad (30)$$

We seek the numerical solution of these together with *(21)* and *(22)*.

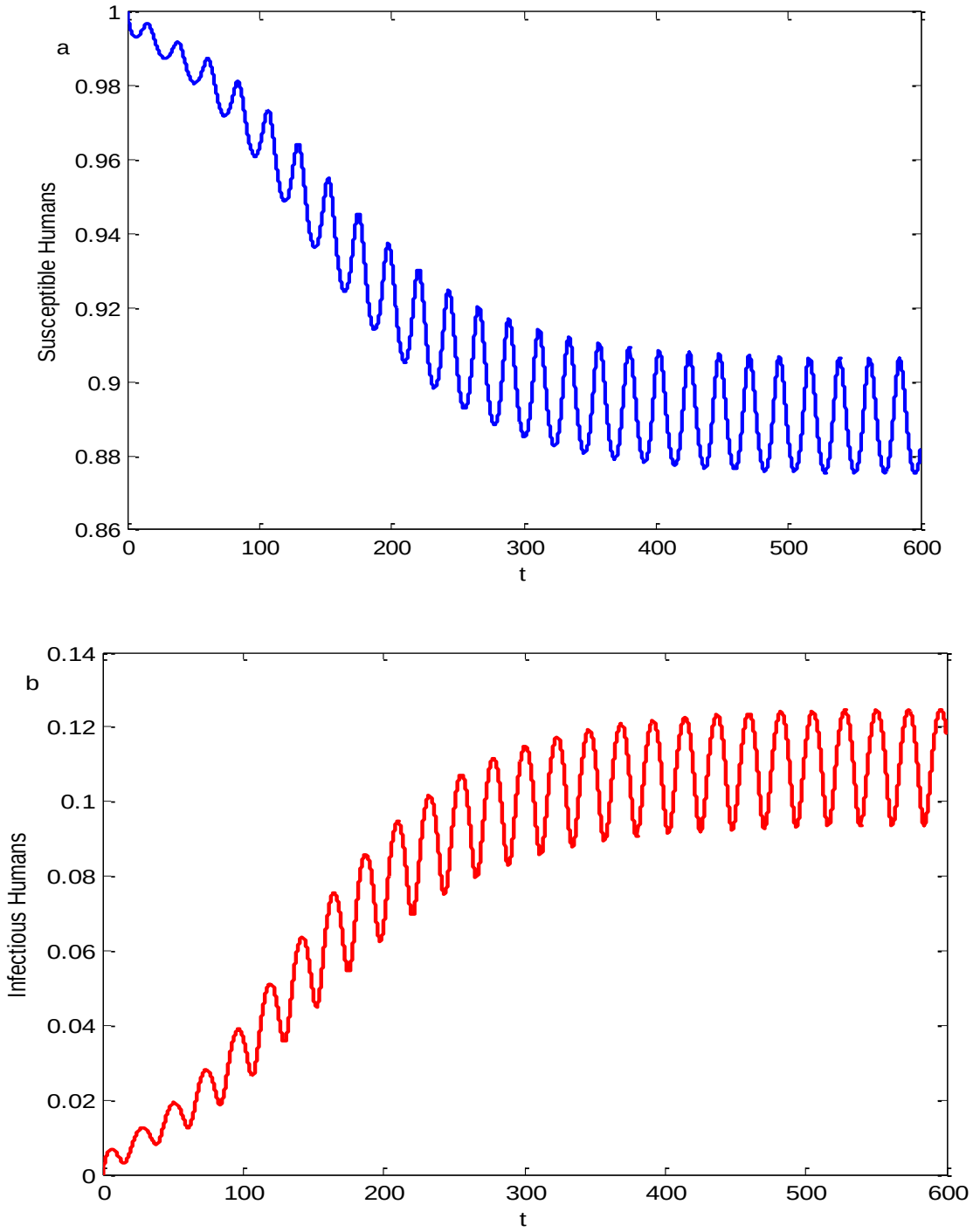

Fig.1. Results showing the endemic state of Susceptible and Infectious humans where t = 1, represents approximately 30 days in real time. The initial conditions used are S = 1, I = 0, X = 0.8, Y = 0.2, N = 1, M = 1. and the parameter values are $\mathbf{a = 0.0047,\ b = 0.0592,\ d = 0.01063,\ e = 0.000765,\ q = 0.921,\ g = 0.00052,\ h = 0.00446,\ r = 0.0385,\ c_0 = 0.0635,\ c_1 = 0.132,\ \sigma = 0.088.}$

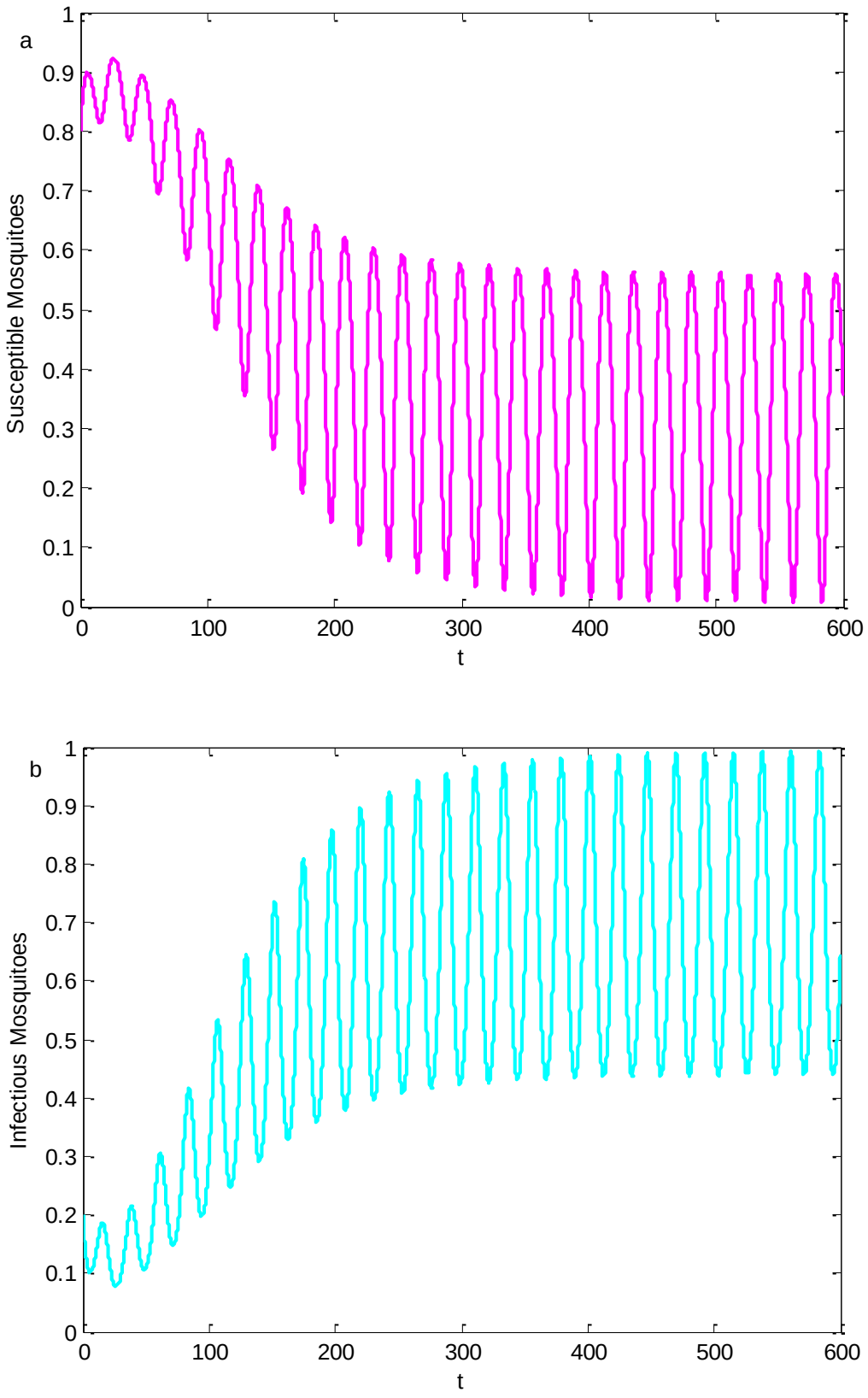

Fig.2. Results showing the Susceptible and Infectious mosquitoes. The initial conditions used are S = 1, I = 0, X = 0.8, Y = 0.2, N = 1, M = 1, and the parameter values are given above.

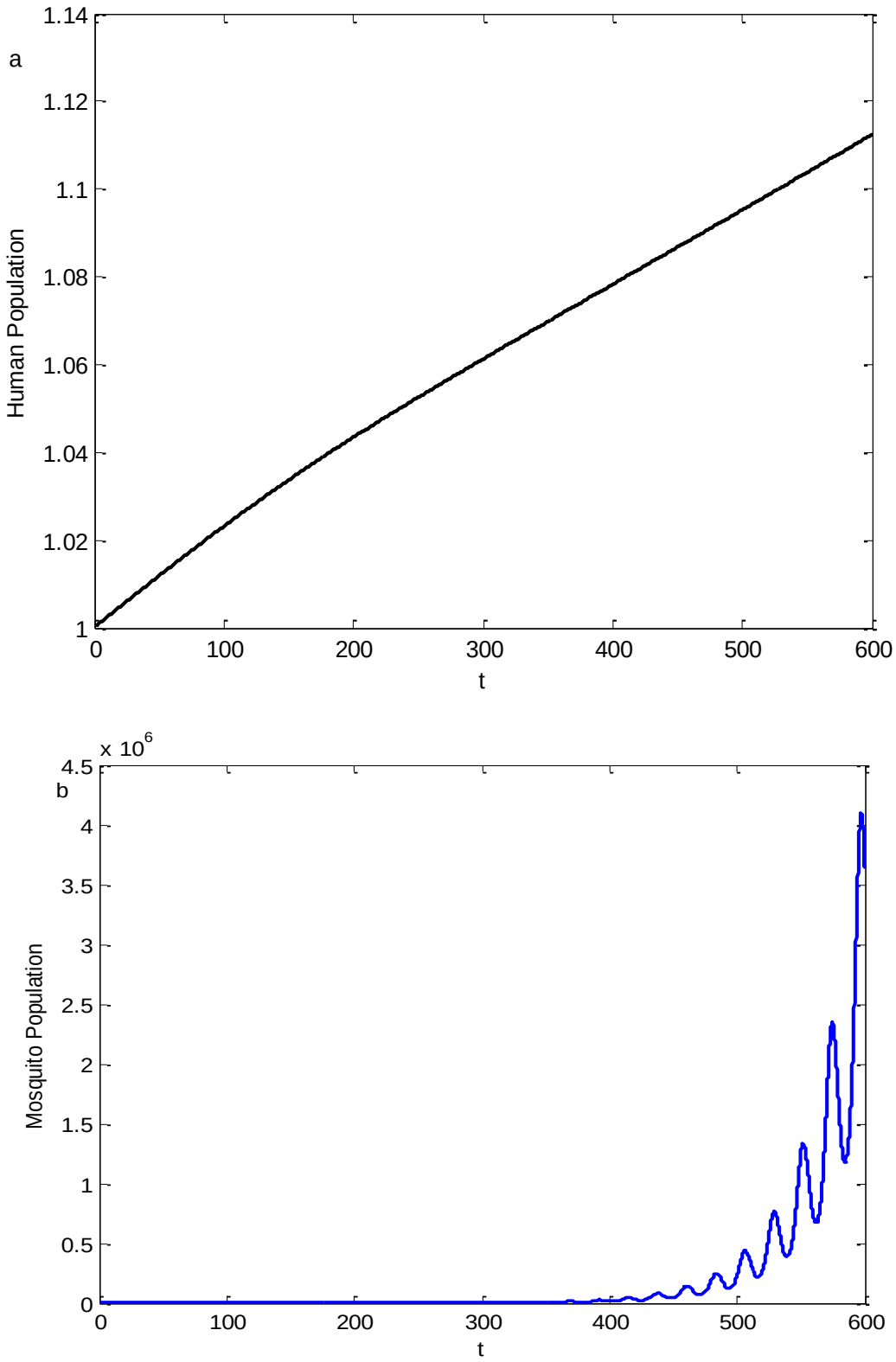

Fig.3. Results showing the Human and Mosquito populations where. The initial conditions used are S = 1, I = 0, X = 0.8, Y = 0.2, N = 1, M = 1, and the parameter values are given above.

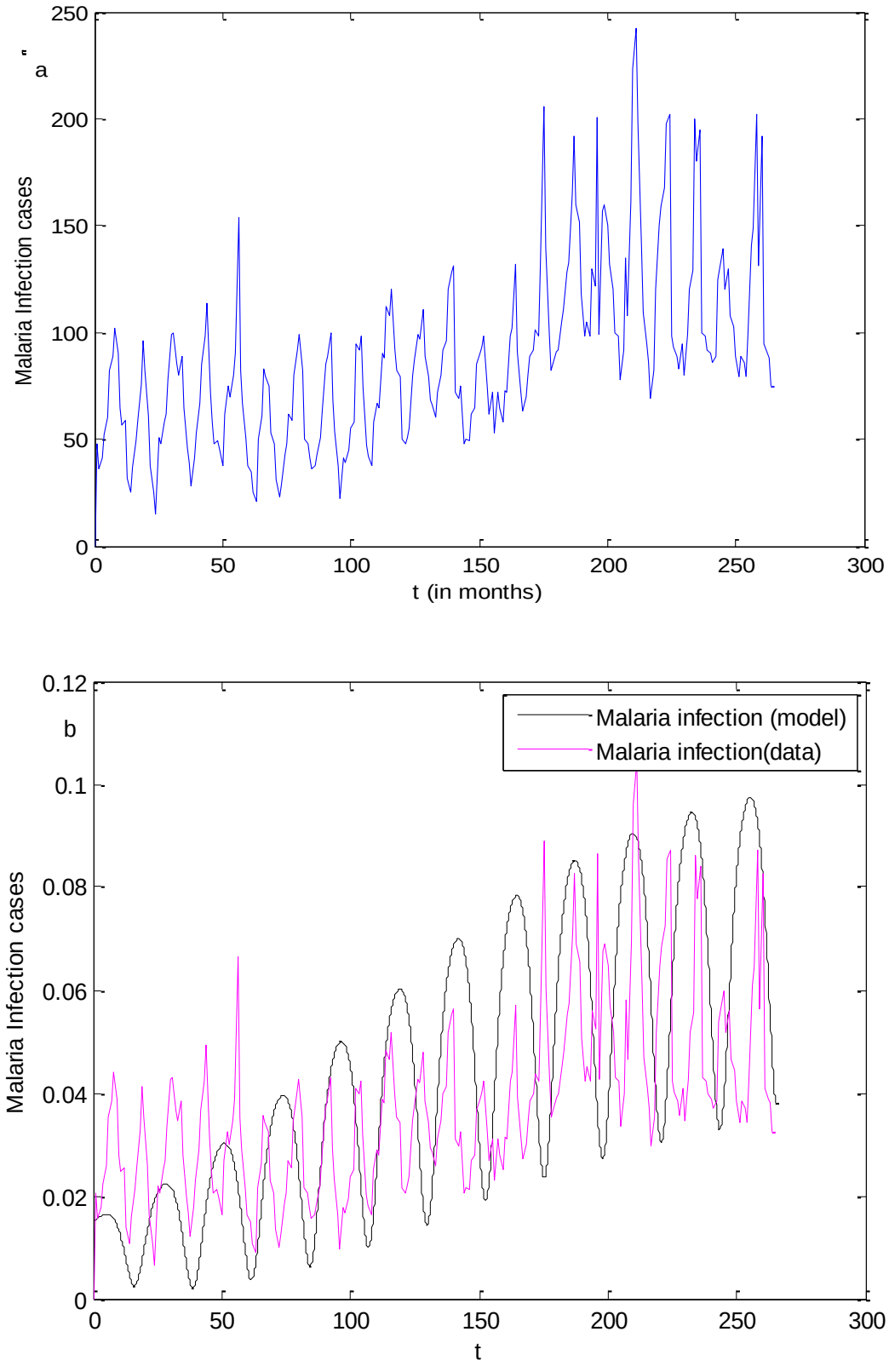

Fig.4. Results showing malaria infection cases from 2000 to 2021 obtained from the University of Port Harcourt Teaching Hospital, where t = 1, represents approximately 30 days in real time. Fig.4a is a line plot of the data while Fig.4b is model fitting of the line plot. The initial conditions used are I = 0.015, Y = 0.03, N = 1, M = 1. and the parameter values are the same as those in Fig.1 except that .we have used the value $c_1 = 0.232$.

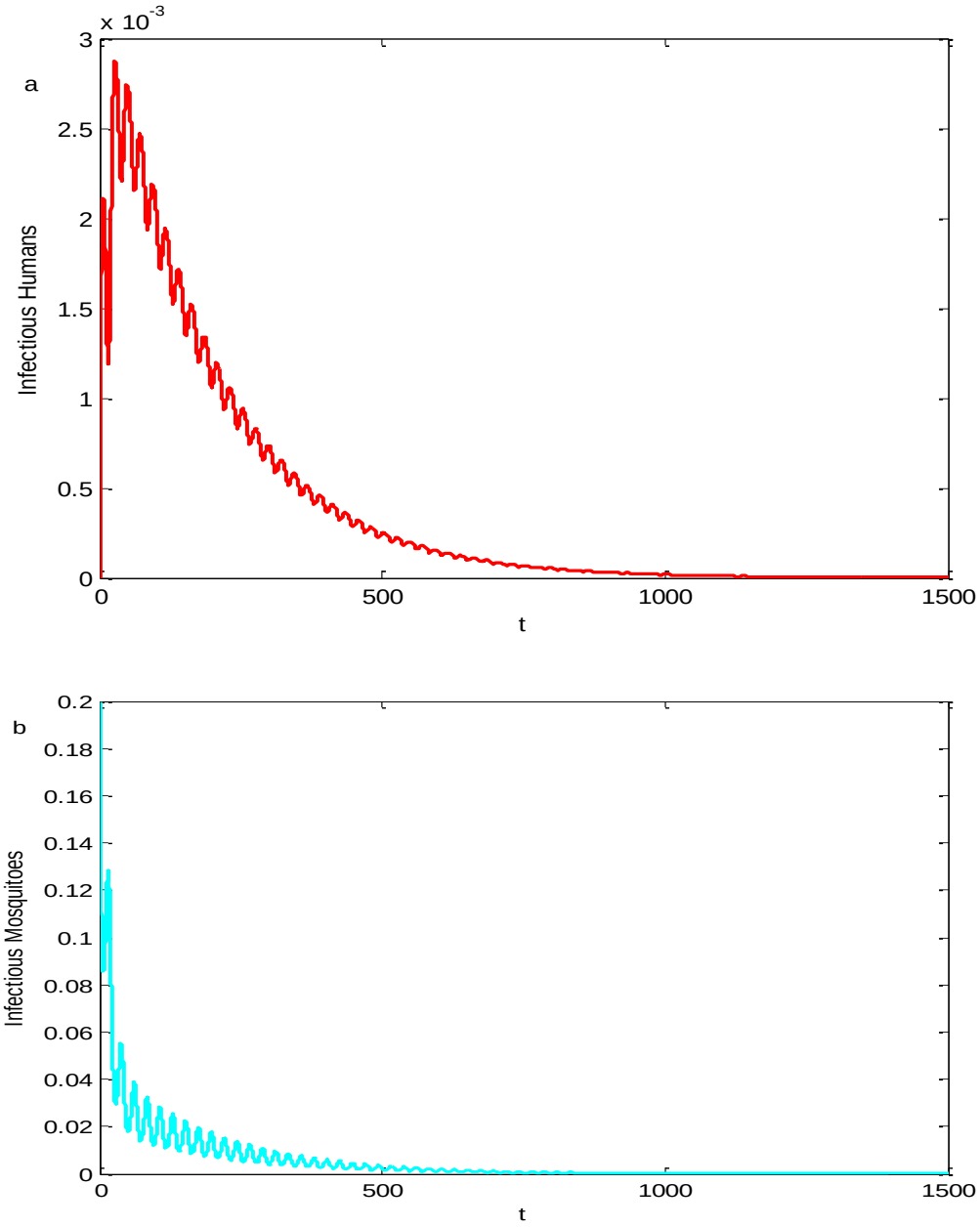

Fig.5. Results showing Infectious humans and mosquito fractions when $R_0 < 1$. The initial conditions used are S = 1, I = 0, X = 0.8, Y = 0.2, N = 1, M = 1, and the parameter values are the same as those in Fig.1 except that .we have used the value $b = 0.0192$ and $q = 0.521$.

5 DISCUSSION AND CONCLUSION

5.1 Discussion

In this model, we describe malaria transmission in a simple form accommodating the dynamics of location-specific seasonal characteristics, which we believe will create an enabling environment for various stakeholders to improve their understanding and planning of intervention strategies especially, with the aid of more available data. Due to temperature and rainfall induced seasonal patterns of disease incidence in malaria epidemic regions *[21]*, we assume a seasonal birth rate of mosquitoes, $\lambda_m f(t)$, where $f(t) = c_0(1 + c_1 \cos 2\pi t)$. The numerical simulations show oscillatory behaviour in the mosquito population and its compartments as shown in figures *2a,b* and *3a*. There are fluctuations in disease transmission in terms of infectivity and susceptibility as shown in figures *1a* and *1b*.

In figure *2a*, the number of susceptible mosquitoes decreases with increasing amplitude as more mosquitoes get infected in figure *2b* describing a situation of endemic malaria as the number of infected humans increases. This is an expected behaviour in as much as the threshold disease indicator, R_0, is greater than unity. However, following the definitions of the parameters in *(1)*, $f \in (c_0, 2c_0)$ and if R_0 is less than unity due to reduction in the baseline biting rate, and or, the infection rates of mosquitoes and humans, the disease goes into extinction as time progresses with a characteristic of continuously reduced seasonal pattern with decaying amplitude as shown in *figure 5*. The populations of humans

and mosquitoes are gradually increasing in figures *3a* and *3b* with that of malaria showing some oscillatory behaviour due to our assumption of a simple seasonal birth rate. We note that even with different initial conditions of the populations, the system converges to a certain equilibrium level oscillating around the average endemic state.

We investigate some data on the number of malaria infection cases for *22* years obtained from the University of Port Harcourt Teaching Hospital in Rivers State in the Niger Delta region of Nigeria. A plot of the data as shown in figure *3* appears to suggest some seasonal pattern of infection. We compare our model with a line plot of the data points in figure 4 where the characteristic feature of seasonality is well represented. The results suggest that the level of seasonality in infection may likely follow the rainfall pattern. However, in order to demonstrate a reasonable degree of objectivity we will be careful not to make any general statement about the data. Malaria transmission also depends on climatic conditions which may affect the abundance and survival of mosquitoes, such as rainfall pattern, temperature and humidity. In many places, transmission is seasonal with the peak during and just after the rainy season [23]. The intricacies of monthly or annual variation of malaria transmission may be better explicated with meteorological data like average monthly and annual rainfall and temperature. Thus, the availability of such data may assist in arriving at better results.

5.2 Conclusion

In this work, we presented a mathematical model on malaria transmission dynamics driven by seasonal forcing integrating simple features of host-vector-parasite interactions. The model focuses on the contributions of seasonality due to effect of rainfall, temperature and other metrological features of the environment in the transmission of malaria. This will create a better understanding and provide a clear policy direction to biologists and public health groups to adopt better regional specific strategies in disease control. We remark that despite the inherent oscillatory behaviour of the solution due to the strength of seasonality, the disease free equilibrium is locally and globally asymptotically stable if the disease control index, R_0 is less than unity. From the simulations, a reduction in the infection rate of humans and mosquitos is very important in the control of the disease despite the level of seasonality as suggested by R_0. Thus intervention strategies should be targeted at reducing seasonal contacts of mosquitoes and humans.

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