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An Epidemiological Model of Malaria at Techiman Municipality, Ghana

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Abstract

The study examined the prevalence of Malaria in the Techiman Municipality of Ghana. We used $SIRS$ model to analyze, model and predict the prevalence of Malaria disease in the municipality. The study is made up of two sections. An $SIRS$ model without the vital dynamics and an $SIRS$ with vital dynamics were used to explain the spread of the Malaria in the Techiman Municipality followed by the Fred Brauer simple treatment model ($SITR$) to determine whether the treatment of malaria in the municipality is beneficial. The model has two equilibrium states: the disease-free equilibrium and the endemic equilibrium states respectively. The stability condition of each equilibrium point is discussed. The basic reproductive number (R_0) of Malaria without the vital dynamics is estimated to be 0.7820 and the basic reproductive number (R_0) of Malaria with the vital dynamics is estimated to be 0.5787 in the Techiman Municipality. The basic reproductive numbers of the $SITR$ model and the modified malaria model were also estimated to be 0.8522 and 0.1252 respectively. Our work shows that the reproductive number (R_0) of Malaria infection at Techiman Municipality is less than 1($R_0 < 1$). Our work also shows that malaria treatment is beneficial in the municipality. According to the results of this study whenever the transmission rate coefficient in any of the models is increased, $R_0 > 1$, but when the transmission rate coefficient is reduced, $R_0 < 1$. We recommend that Malaria control measures should be intensified in the municipality so as to decrease the rate of transmission.

Keywords: SIRS model, Stability analysis, Equilibrium points, Mathematical model, Epidemiological model.

1. Introduction

Malaria is endemic throughout the tropics. Of the approximately 3.4 billion people worldwide who are exposed annually, 1.2 billion are at high risk; the World Health Organization (WHO) states that more than 207 million developed symptomatic malaria in 2012 [1]. Between 2000 and 2010, the number of reported annual malaria cases in 34 malaria-eliminating countries decreased by 85 percent from 1.5 million to $232{,}000$ cases [2]. Most of these are attributable to *P. falciparum*, but *P. vivax* and *P. knowlesi* can also cause severe disease. Malaria deaths peaked at 1.82 million in 2004 and fell to 1.24 million in 2010 ($714{,}000$ children < 5 years and $524{,}000$ individuals ≥ 5 years); over 80 percent of the deaths occur in sub-Saharan Africa [3]. The WHO's estimates of deaths from malaria ($627{,}000$ in 2012; uncertainty range $473{,}000$ to $789{,}000$) are approximately half the reliable estimates above.

Malaria is transmitted via the bite of a female *Anopheles* spp mosquito, which occurs mainly between dusk and dawn. Other comparatively rare mechanisms for transmission include congenitally acquired disease, blood transfusion, sharing of contaminated needles, and organ transplantation [4,5]. Malaria occurs throughout most of the tropical

regions of the world, with *P. falciparum* causing the largest burden of disease, followed by *P. vivax* [6]. *P. falciparum* predominates in Africa, New Guinea, and Hispaniola (Haiti and the Dominican Republic); *P. vivax* is more common in the Americas and the western Pacific. The prevalence of these two species is approximately equal in the Indian subcontinent, eastern Asia, and Oceania [7 – 9]. *P. malariae* is uncommon and is found in most endemic areas, especially in sub-Saharan Africa. *P. ovale*, even less common, is relatively unusual outside of Africa and, where it is found, comprises <1 percent of isolates. *P. knowlesi*, similar morphologically to *P. malariae*, has been identified by molecular methods in patients in Malaysia, the Philippines, Thailand, and Myanmar [10]; this species has not yet been proven to be transmitted from humans to mosquitoes (ie, a monkey reservoir may be required to infect mosquitoes).

In Ghana, there is compelling evidence from entomological studies, community prevalence studies and the 2011 Multiple Indicator Cluster Survey (MICS) that the burden of malaria is significantly lower in Accra, Kumasi and Tamale than in smaller communities located in the same ecological zone. Compared to children living in smaller communities of the same ecological zone, the prevalence of malaria parasitemia among children living in the largest cities of each zone was 73% to 86% lower in Accra, 79% to 85% lower in Kumasi, and 34% to 68% lower in Tamale [11]. Additionally, the prevalence of malaria infection was higher for children living in the poorest urban households compared to those living in combatively wealthier households [12].

The current study therefore aims at analyzing, modeling and predicting the spread of Malaria disease in the Techiman Municipality of Ghana. The district was chosen for the study because it is one of the districts where the disease is considered to be endemic [13].

2. Mathematical Model

The model is a modification of Kermack and Mechandrick model [14]. In the standard $SIRS$ model, the population is divided into three states, susceptibles S, infectives I, and removed or immune R. The first group are the individuals who are capable of becoming infected with a particular disease, the second group consists of individuals who are infected with a disease and R represents those who have recovered from disease or malaria infection.

The population of individuals in the compartments S, I and R, at time t, is denoted by $S(t)$, $I(t)$ and $R(t)$ respectively. β is the transmission rate coefficient, γ is the removed rate coefficient and τ is the loss of immunity coefficient.

The diagram in **Figure1** is the schematic diagram of SIRS model without vital dynamics.

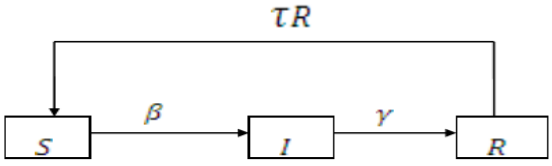


Figure 1: Schematic diagram of $SIRS$ model without vital dynamics

2.1 *The Model Assumptions*

The disease spread in a closed environment; that is no emigration or immigration, and neither birth nor death in the population, so that the total population remains a constant X for all t, that is

$S(t) + I(t) + R(t) = X$. If we let $S = S(t)/X$, $I = I(t)/X$ and $R = R(t)/X$ where R, I and R are the susceptible, infected and recovery fractions in the population.

1. The infected individuals do not gain permanent immunity and become susceptible again after they are recovered from the infection.
2. Age, sex, social status and race do not affect the probability of being infected
3. The members of the population mix homogeneously (have the same interactions with one another to the same degree).

The model equations are:

$$\frac{dS}{dt} = \tau R - \beta SI \quad (1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I \quad (2)$$

$$\frac{dR}{dt} = \gamma I - \tau R \quad (3)$$

Where β is the rate of transmission coefficient, γ is the removed rate and τ is the immunity loss coefficient respectively.

We then substitute $R = X - I - S$ into equation 1-3 and obtain

$$\frac{dS}{dt} = \tau(X - I - S) - \beta SI \quad (4)$$

$$\frac{dI}{dt} = \beta SI - \gamma I \quad (5)$$

$$\frac{dR}{dt} = \gamma I - \tau(X - I - S) \quad (6)$$

The equation for R is then decoupled from the first two equations of the system, so we need to consider the system.

$$\frac{dS}{dt} = \tau(X - I - S) - \beta SI$$

and

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Table 1: Model Parameters

Parameter	Description	Value
β	Transmission rate	1.9335
γ	Recovery rate	1.6757
μ	Natural death rate	1.6142
α	Disease-induced death rate	0.0512
τ	Immunity loss rate	0.0300
η	Rate of removal from the treated class	0.4065
b	birth rate	12.156

3. Model Analysis

Equilibrium Points

We now evaluate the equilibrium points or steady states of the ODE 4, 5 and 6. The points to be found are the disease-free (where $I = 0$), and endemic (Where $I \neq 0$). Setting the right hand side of the equations in the system 4, 5 and 6 to zero and then solve for the values S, I and R, we

obtain

$$(S^*, I^*, R^*) = \left[\frac{\gamma}{\beta}, \frac{\tau}{\beta}\left(\frac{\beta X - \gamma}{\tau + \gamma}\right), \frac{\gamma}{\beta}\left(\frac{\beta X - \gamma}{\tau + \gamma}\right)\right] \quad (7)$$

3.1 The Disease-Free Equilibrium and its analysis

The disease-free equilibrium is the situation where there is no infection. That is where $I = 0$. Setting only $\frac{dS}{dt} = 0$, the disease-free equilibrium is $(S^*, I^*) = (X, 0) = (1, 0)$

We analyze the stability of the disease-free equilibrium by considering the linearized system of the system of equations 4 and 5 about the equilibrium point by taking the Jacobian matrix.

$$J = \begin{pmatrix} \beta I - \gamma & \beta S - \gamma \\ \beta I & \beta S - \tau \end{pmatrix}$$

At the critical point $(S^*, 0) = (X, 0)$ the Jacobian matrix is given by

$$J(X, 0) = \begin{pmatrix} -\gamma & -\beta X - \gamma \\ 0 & \beta X - \tau \end{pmatrix}$$

$$|J-\lambda| = \begin{vmatrix} -\gamma-\lambda & -\beta X-\gamma \\ 0 & \beta X-\tau-\lambda \end{vmatrix}$$

$$\lambda_1 = -\gamma \text{ and } \lambda_2 = \beta X-\tau$$

For $Det\ (J-\lambda)$ to be asymptotically stable, both eigenvalues must be negative. For $Det\ (J-\lambda) = 0$, it is clear that $\lambda_1 = -\gamma$ is negative and therefore if $\lambda_2 = \beta X-\tau < 0$, then both eigenvalues are negative and $R_0 < 1$. Hence the disease-free equilibrium is asymptotically stable, if $\lambda_2 = \beta X-\tau > 0$, then $Det(J-\lambda)$ is unstable.

3.2 The Basic Reproductive Number of Malaria infection without the vital dynamics

The basic reproductive number(R_0) is the average number of infectives produced when one infective individual is introduced into a completely susceptible population. [15, 16]

We determine the basic reproductive number of the model as follows:

$$\frac{ds}{dt} = \tau(X-S-I)-\beta SI$$

$$\frac{dI}{dt} = \beta SI-\gamma I$$

From the infected compartment,

$$F = \frac{\delta F}{\delta I} = \beta S, \quad V = \frac{\delta V}{\delta I} = \gamma,$$

$$V^{-1} = \frac{1}{\gamma} \qquad FV^{-1} = \frac{\beta S}{\gamma}$$

We consider initial condition $S = S_0$.

This implies

$$FV^{-1} = \frac{\beta S_0}{\gamma}$$

but $FV^{-1} = R_0$, therefore

$$R_0 = \frac{\beta S_0}{\gamma} \qquad (8)$$

When $R_0 > 1$ there is an endemic and the solution is unstable, and epidemic occurs. When $R_0 < 1$ there is no epidemic and the disease dies out. When $R_0 = 1$, the disease become endemic, meaning the disease remains in the population at a constant rate, as one infected individual transmits the disease to one susceptible [17].

3.3 The Endemic Equilibrium and its analysis

We now consider the case where $R_0 > 1$ where the system has an endemic infection. The $(S^*, 0)$ is unstable. From equations 7 and 8, we obtain the endemic equilibrium point as

$$(S^*, I^*) = \left[\frac{\gamma}{\beta}, \frac{\tau}{\beta}\left(\frac{\beta X - \gamma}{\tau + \gamma}\right)\right]$$

The equilibrium point is endemic with non-zero infected in the steady state $(I^* > 0)$ when the condition $\beta X > \gamma$ is satisfied.

The Jacobian matrix of the fixed point derived from the equations 7 and 8 is given by

$$J = \begin{bmatrix} -\tau - \beta I & -\tau - \beta S \\ \beta I & \beta S - \gamma \end{bmatrix} \quad (9)$$

When $S = \frac{\gamma}{\beta}$ and $I = \frac{\tau}{\beta}\left(\frac{\beta X - \gamma}{\tau + \gamma}\right)$ is substituted into the Jacobian matrix (4) we get

$$J = \begin{bmatrix} -\tau\left(\frac{\tau + \beta X}{\tau + \gamma}\right) & -(\tau + \gamma) \\ \tau\left(\frac{\beta X - \gamma}{\tau + \gamma}\right) & 0 \end{bmatrix}$$

The eigenvalues of the model is given by

$$\lambda_1 \lambda_2 = \frac{1}{2}\left[-\tau\left(\frac{\tau + \beta X}{\tau + \gamma}\right) \pm \sqrt{\tau^2\left(\frac{\tau + \beta X}{\tau + \gamma}\right)^2 - 4\tau(\beta X - \gamma)}\right] \quad (10)$$

The real parts of which are always negative for an endemic steady state, since $\beta X > \gamma$. Hence the fixed point is asymptotically stable [18].

4. SIRS Model of Malaria with Vital Dynamics

We consider the $SIRS$ model with exponential birth, natural death, disease-induced death rates, and standard incidence.

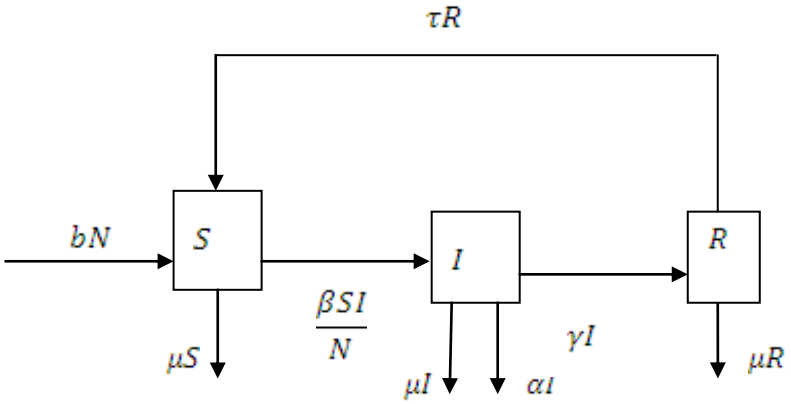


Figure 2: The schematic diagram of $SIRS$ model with vital dynamics

$$\frac{dS}{dt} = bN - \mu S - \frac{\beta SI}{N} + \tau R \quad (11)$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - (\alpha + \mu + \gamma)I \quad (12)$$

$$\frac{dR}{dt} = \gamma I - (\mu + \tau)R \quad (13)$$

Where $b =$ birth rate, $\mu =$ natural death rate, $\alpha =$ disease-induced death rate coefficient, $\gamma =$recovery rate coefficient, $\tau =$ immunity loss rate coefficient. The total population is given by $N(t) = S(t) + I(t) + R(t)$.

We non-dimentionalize the systems 11, 12 and 13 as follows

Let

$$m = \frac{S}{N}, \quad n = \frac{1}{N} \text{ and } \quad w = \frac{R}{N}$$

The system of equations 11, 12 and 13 is then modified to

$$\frac{dm}{dt} = b - bm - \beta mn + \tau w + \alpha mn \quad (14)$$

$$\frac{dn}{dt} = \beta mn - (b + \alpha + \gamma)n + \alpha n^2 \quad (15)$$

$$\frac{dw}{dt} = \gamma n - (b + \tau)w + \alpha nw \quad (16)$$

The system of equations 14, 15, and 16 above is equivalent to the system equations:

$$\frac{dn}{dt} = \beta mn - (b + \alpha + \gamma)n + \alpha n^2$$

$$\frac{dw}{dt} = \gamma n - (b + \tau)w + \alpha nw$$

Since $m + n + w = 1.$ The system $D = \{n, w: n \geq 0, w \geq 0, n + w < 1\}$ is invariant for the system of equations 14 and 15.

From the system of equations 14 and 15, the modified reproductive number is given by

$$R_1 = \frac{\beta}{b + \mu + \gamma} \quad (17)$$

The system of equations 14 and 15 has only the disease-free equilibrium $E_0(0, 0)$, which is globally asymptotically stable in D, if $R \leq 1.$ This disease-free equilibrium becomes unstable, and there appears a

positive equilibrium $E^*(n^*, w^*)$, which is globally asymptotically stable in D if $R_1 > 1$. When the disease-free equilibrium is globally asymptotically stable, the infective fraction goes to zero and the disease dies out.

Also from the system 4.1, the basic reproductive number of the model is given by

$$R_0 = \frac{\beta}{\alpha + \mu + \gamma} \tag{18}$$

where β is the transmission coefficient, $\frac{1}{\alpha+\mu+\gamma}$ is the infectious period and R_0 is the average number of secondary infections produced by one infected individual during the mean course of infection in a fully susceptible population[19].

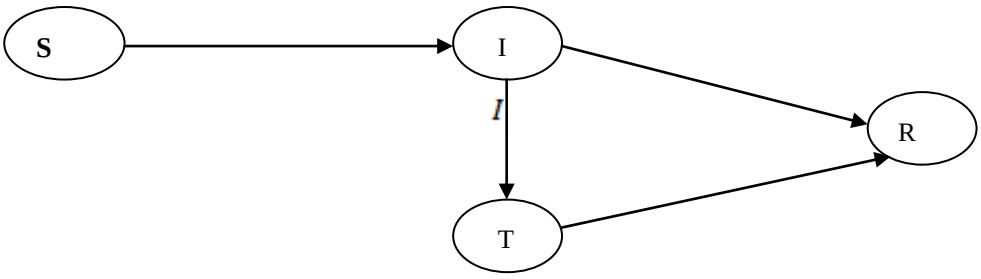
5. $SITR$ Model Of Malaria

We model the $SITR$ model by supposing that a fraction γ per unit time of infectives is selected for treatment, and that treatment reduces infectivity by a fraction τ. Suppose also that the rate of removal from the treated class is η and T is the treatment class.

We assume that:

1. the treatment moves infectives to a class T with infectivity decreased by a factor τ and with a recovery rate η.
2. treatment continues as long as an individual remains infective.
3. treatment is beneficial, if $\eta > \tau\alpha$.

Figure 3: The schematic diagram of $SITR$ model



The diagram above is the schematic diagram of $SITR$ model

$$\frac{dS}{dt} = -\beta S(I + \tau T) \tag{19}$$

$$\frac{dI}{dt} = \beta S(I + \tau T) - (\alpha + \gamma)I \tag{20}$$

$$\frac{dT}{dt} = \gamma I - \eta T \tag{21}$$

where $\tau, \gamma, \eta, \alpha,$ and β have their usual meanings.

The basic reproductive number of $SITR$ can be found by assuming that an infective in a totally susceptible population causes βX new infections in unit time, and the mean time spent in the infective class is $\frac{1}{(\alpha+\gamma)}$. In addition to this, a fraction $\frac{\gamma}{(\alpha+\gamma)}$ of infectives are treated. While in the treatment stage the number of new infections caused in unit time is $\tau\beta X$, and the mean time in treatment class is $1/\eta$. The reproductive number is therefore given by

$$R_0 = \frac{\beta X}{\alpha+\beta} + \frac{\gamma}{\alpha+\eta} \times \frac{\tau\beta X}{\eta}$$

$$R_0 = \frac{\beta X}{\alpha+\gamma}\left[1 + \frac{\tau\gamma}{\eta}\right] \qquad (22)$$

R_0 is a decreasing function of γ if $\eta > \tau\alpha$ [20].

6. Sensitivity analysis

We investigate the nature of the model by conducting the sensitivity analysis

6.1. *Sensitivity analysis of R_o of Malaria without the vital dynamics*

a. If β and γ values are increased and S_0 maintain the same, that is
$\beta = 3,\ \gamma = 2$ and $S_0 = 0.67738,\ R_0 = 1.02 > 1$

b. If a. If β and γ values are reduced and S_0 maintain the same, that is
$\beta = 0.5257,\ \gamma = 0.6354$ and $S_0 = 0.67738,\ R_0 = 0.5604 < 1$

6.2. *Sensitivity analysis of R_o of Malaria with the vital dynamics*

a. If β value is increased and α, μ and γ maintain the same, that is
$\beta = 4,\ \gamma = 1.6757,\ \alpha = 0.0512,$ and $\mu = 1.6142,\ R_0 = 1.197 > 1$

b. If β value is reduced and α, μ and γ maintain the same, that is
$\beta = 1,\ \gamma = 1.6757,\ \alpha = 0.0512,$ and $\mu = 1.6142,\ R_0 < 1$

6.4. Sensitivity analysis of the disease-free equilibrium

a. If β value is increased and all the other parameters maintain the same, that is
$\beta = 4,\ \gamma = 1.6757,\ \tau = 0.0300$ and **X= 0.0596,** $\lambda_1 = -1.6757$ and $\lambda_2 = 0.2084$
The eigenvalues are distinct. It is a saddle and the origin is unstable

b. If β value is reduced and all other parameters maintain the same, that is
$\beta = 0.0335,\ \gamma = 1.6757,\ \tau = 0.0300$ and **X= 0.0596,** $\lambda_1 = -1.6757$ and $\lambda_2 = -0.0288,$

the eigenvalues are both negative which is a nodal sink and the origin is asymptotically stable.

6.5. Sensitivity analysis of the endemic equilibrium state

a. If β value is increased and all other parameters maintain the same, that is

$\beta = 4$, $\gamma = 1.6757$, $\tau = 0.0300$ and **X= 0.0596,** $\lambda_1 = 0.21$ and $\lambda_2 = -0.41$

The eigenvalues are distinct. It is a saddle and the origin is unstable

b. If β value is reduced and all other parameters maintain the same, that is

$\beta = 0.0335$, $\gamma = 1.6757$, $\tau = 0.0300$ and **X= 0.0596,** $\lambda_1 = 0.22$ and $\lambda_2 = -0.22$

The eigenvalues are distinct. It is a saddle and the origin is unstable

7. Discussion of Results

We used $SIRS$ model to predict the spread of Malaria in the Techiman Municipality of Ghana. We discussed the existence and stability of the disease-free and endemic equlibria of the model and performed sensitivity analysis of the parameters. We also considered Fred Brauer simple treatment model ($SITR$) in the research.

Based on the data obtained from the municipality, we estimated the basic reproductive number of Malaria model without vital dynamics to be $R_0 = 0.7820$ in the Techiman Municipality. This indicates that the disease is not endemic in the municipality. The basic reproductive number of Malaria treatment model $(STIR)$, the basic reproductive number of malaria with vital dynamics and the modified reproductive number of Malaria were also estimated to be $R_0 = 0.8522$, $R_0 = 0.5787$ and $R_0 = 0.1252$ respectively. These reproductive numbers further confirm that Malaria infection in Techiman Municipality has not yet reach its endemic state since they are all less than 1.

According to the parameter values in the treatment model, we concluded that Malaria treatment in Techiman Municipality was beneficial since $\eta > \tau\alpha$.

In the stability analysis of the equilibrium points, the disease-free equilibrium is unstable. This is because the transmission rate of Malaria at Techiman Municipality is greater than the removed rate. Also the eigenvalues of the disease-free equilibrium point in the model are $\lambda_1 = 1.6757$ and $\lambda_2 = -1.2797$ which is a saddle point and therefore unstable. The endemic equilibrium point of the model was found to be asymptotically stable. The eigenvalues obtained at the endemic equilibrium point in the model are $\lambda_1 = -0.0146$ and $\lambda_2 = -0.0103.$ This is a nodal sink and therefore asymptotically stable.

In the sensitivity analysis of the reproductive numbers of Malaria model without vital dynamics, Malaria model with vital dynamics and the Malaria treatment model, whenever the value of the transmission rate coefficient (β) is increased, $R_0 > 1$. But whenever the value of β is reduced, $R_0 < 1$. This implies that if Malaria is not combated in Techiman Municipality, epidemic may occur.

In the stability analysis of the of the disease-free equilibrium, when the value of the transmission rate coefficient (β) is increased, the equilibrium point becomes unstable, but when it is reduced, it becomes asymptotically stable. This indicates that if the disease is unchecked epidemic may occur. The endemic equilibrium point is unstable, whether the value transmission rate is increased or decreased. this shows that it will be difficult to eradicated the disease if it becomes epidemic in the municipality.

8. Conclusion

Our work shows that the reproductive number of Malaria infection in the Techiman Municipality is less than 1($R_0 < 1$) and therefore the belief that Malaria is endemic at Techiman Municipality as posited by the GNA (2005) cannot be justified. We recommend that Malaria control measures should be intensified in the municipality so as to decrease the rate of transmission of malaria in order not to affect the development of human resource in Ghana.

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