

Impact of Hygiene on Malaria Transmission Dynamics: A Mathematical Model

Temidayo Oluwafemi* and Emmanuel Azuaba

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Abstract

Malaria continues to pose a major public health challenge, especially in developing countries, as 219 million cases of malaria were found in 89 countries. In this paper, a mathematical model using non-linear differential equations is formulated to describe the impact of hygiene on malaria transmission dynamics. The model is divided into seven compartments which includes five human compartments namely; unhygienic susceptible human population (S_u), hygienic susceptible human population (S_h), unhygienic infected human population (I_u), hygienic infected human population (I_h) and the recovered human population (R_h) while the mosquito population is subdivided into susceptible mosquitoes (S_v) and infected mosquitoes I_v . The positivity of the solution shows that a domain exists where the model is biologically meaningful and mathematically well-posed. The Disease-Free Equilibrium (DFE) point of the model is obtained. Then, the basic reproduction number is computed using the next generation method and established the condition for local stability of the disease-free equilibrium. Thereafter the global stability of the disease-free equilibrium was obtained by constructing the Lyapunov function of the model system. Also, sensitivity analysis of the model system was carried out to identify the influence of the parameters on the basic reproduction number. The result shows that the natural death rate of the mosquitoes is most sensitive to the basic reproduction number.

Keywords: mathematical model, malaria, hygiene, stability analysis, basic reproduction number, lyapunov function, sensitivity analysis

1. INTRODUCTION

Malaria is one of the infectious diseases with an adverse effect on the human population. Some of the malaria parasites live in humans and the remaining is transmitted between human host and mosquito vector by the infected female Anopheles mosquitoes. In rare cases, people may be infected via contaminated blood, or a fetus may become infected by its mother during pregnancy or after delivery. Two of the five parasites species – *Plasmodium falciparum* and *Plasmodium vivax* pose the greatest public health challenges [1] – [3]. According to the World Health Organization [4], 219 million cases of malaria were reported in 89 countries and the estimated death cases were 435,000 with the African region carrying a disproportionately high share of the global malaria burden. Malaria is the third leading cause of death

most especially for children under five years, after pneumonia and diarrheal diseases.

The transmission dynamics of malaria mainly happened in poor environmental conditions. These conditions include unsafe water supplies, poor personal hygiene, poor sanitary facilities, poor living standards, and unhygienic food. Poor personal hygiene may result in water-borne diseases [5]. Poor environmental sanitation (hygiene) and housing conditions might be significant risk factors for malaria burden [6]. Enebeli et al. (2019) concluded that poor access to water, sanitation, and hygiene practices of caregivers directly relates to the prevalence of malaria among their children [7]. A mathematical model of malaria dynamics was developed with naturally acquired transient immunity in the presence of protected travellers [3]. A non-autonomous model was also developed to assess the impact of different microclimate conditions on the transmission dynamics of malaria [8]. A mathematical model has been proposed for the transmission dynamics of malaria by incorporating change via education as a control strategy [9]. The human population follows the susceptible-protected-exposed-infectious-recovered (SPEIR) pattern and the mosquito population follows susceptible-exposed-infectious (SEI) patterns. An analytical study was carried out to investigate the local stability of the system while the basic reproduction number was obtained using

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the next-generation matrix method. The result shows that the disease-free equilibrium of the system is locally asymptotically stable if $R_0 < 1$. The impact of temperature in malaria disease transmission dynamics was mathematically studied [10]. The SEIR model was suitable for the human population and LSEI compartment model was suitable for mosquito population. It was observed that temperature affects the transmission dynamics of malaria significantly. The impact of drug-resistance in malaria transmission was also modelled [11]. Many mathematical models have been developed to study malaria dynamics but none has been discussed to study the impact of hygiene on malaria transmission dynamics as proposed in this work.

In this work, we propose a deterministic mathematical model for assessing the impact of hygiene on malaria transmission dynamics. The basic reproduction number is computed and the local and global stability of the disease-free equilibrium are established. Furthermore, the sensitivity analysis of the parameters is also evaluated.

2. MATERIALS AND METHODS

2.1. Model Formulation

In this model, the total human population denoted by (N_H) is subdivided into unhygienic susceptible human population (S_u) , hygienic susceptible human population (S_h) , unhygienic infected human population (I_u) , hygienic infected human population (I_h) and the recovered human population (R) . The mosquito population denoted by (N_v) is subdivided into susceptible mosquitoes (S_v) and infected mosquitoes (I_v) . See the equations 2.1 and 2.2.

$$N_H = S_u + S_h + I_u + I_h + R. \tag{2.1}$$

$$N_v = S_v + I_v. \tag{2.2}$$

Let Λ_H be the recruitment rate of the human population. A fraction $(1 - \alpha)\Lambda_H$ enters unhygienic susceptible human class while the remaining fraction $(\alpha\Lambda_H)$ enters the hygienic susceptible human class. The unhygienic susceptible class is increased by the rate at which unhygienic human class lose

immunity after recovery given as ω_u , and reduced by the rate of progression to hygienic class (τ_1) , the force of infection for the unhygienic class (λ_u) and natural human death rate (μ_H) . The hygienic susceptible human compartment is increased by the τ_1 , the rate at which hygienic human loss immunity after recovery at ω_h , while the compartment is reduced by natural human death rate μ_H and the force of infection for the hygienic class $(1 - \zeta)\lambda_h$. The I_u is increased by λ_u and reduced by μ_H , rate of progression from I_u to I_h given as τ_2 . Malaria induced death for unhygienic human class and recovery for unhygienic human are denoted as δ_u and θ_u . The I_h is increased by $(1 - \zeta)\lambda_h$ and τ_2 then reduced by the recovery rate for a hygienic human class given as θ_h . Malaria induced death for hygienic human class is denoted as δ_h . The human recovery class (R) is increased by θ_h and θ_u then reduced by μ_H , ω_h , and ω_u . The S_v is increased by the mosquito recruitment rate given as Λ_v , reduced by the mosquitoes death rate μ_v , and force of infection for mosquito given as λ_v . Meanwhile, the I_v is increased by λ_v and μ_v .

Given the above description and definitions of variables and parameters in Table 1 and 2, the following are the model equations:

$$\frac{dS_u}{dt} = (1 - \alpha)\Lambda_H - (\tau_1 + \lambda_u + \mu_H)S_u + \omega_u R, \tag{2.3}$$

$$\frac{dS_h}{dt} = \alpha\Lambda_H + \omega_h R + \tau_1 S_u - ((1 - \zeta)\lambda_h + \mu_H)S_h, \tag{2.4}$$

$$\frac{dI_u}{dt} = \lambda_u S_u - (\tau_2 + \delta_u + \theta_u + \mu_H)I_u, \tag{2.5}$$

$$\frac{dI_h}{dt} = (1 - \zeta)\lambda_h S_h + \tau_2 I_u - (\delta_h + \theta_h + \mu_H)I_h, \tag{2.6}$$

$$\frac{dR}{dt} = \theta_u I_u + \theta_h I_h - (\omega_u + \omega_h + \mu_H)R, \tag{2.7}$$

$$\frac{dS_v}{dt} = \Lambda_v - \lambda_v S_v - \mu_v S_v, \tag{2.8}$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \tag{2.9}$$

“where”

$$\lambda_u = \frac{b_1 \beta_{vh} I_v}{N_H}, \lambda_h = \frac{b_2 \beta_{vh} I_v}{N_H}, b_1 > b_2, \lambda_v = \frac{b_3 \beta_{hv} (I_u + \rho I_h)}{N_H}, \delta_u > \delta_h, \theta_h > \theta_u. \tag{2.10}$$

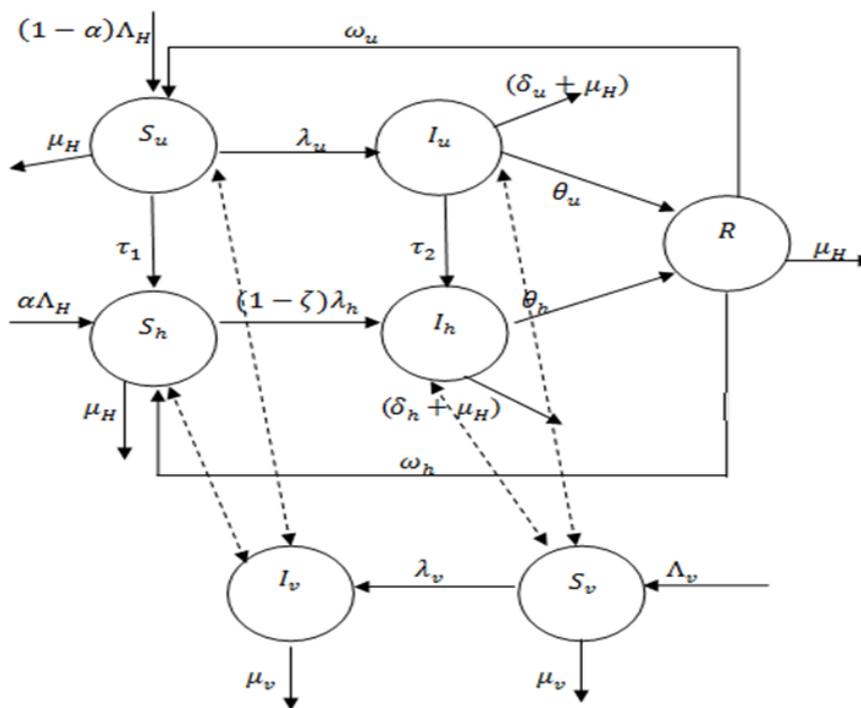


Figure 1. Model flow diagram.

2.2. Invariant Region

The invariant region can be obtained by the \$t > 0\$ following theorem.

Theorem 1

The solutions of the model are feasible for all if they enter the invariant region

$$\Omega = \Omega_H \times \Omega_v. \tag{2.11}$$

Proof:

Let

$$\Omega = (S_u, S_h, I_u, I_h, R, S_v, I_v) \in \mathbb{R}_+^7, \tag{2.12}$$

be any solution of the system with non-negative initial conditions. Hence, all feasible solution set of the human population of the malaria model enters the region

$$\Omega_H = \left\{ (S_u, S_h, I_u, I_h, R) \in \mathbb{R}_+^5 : S_u \geq 0, S_h \geq 0, I_u \geq 0, I_h \geq 0, R \geq 0, N_H \leq \frac{\Lambda_H}{\mu_H} \right\}. \tag{2.13}$$

Similarly, the feasible solution set of the vector population enter the region

$$\Omega_v = \left\{ (S_v, I_v) \in \mathbb{R}_+^2 : S_v \geq 0, I_v \geq 0, N_v \leq \frac{\Lambda_v}{\mu_v} \right\}. \tag{2.14}$$

Therefore, the region \$\Omega\$ is positively invariant

i.e. the solution remains positive for all initial values.

Thus, the model is biologically meaningful and mathematically well-posed in the domain \$\Omega\$.

2.3. Disease Free Equilibrium (DFE)

The DFE of the model equations can be found by setting the right hand of the model (2.3) – (2.9) to zero, i.e.

$$\frac{dS_u}{dt} = \frac{dS_h}{dt} = \frac{dI_u}{dt} = \frac{dI_h}{dt} = \frac{dR}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0.$$

which gives

$$(1 - \alpha)\Lambda_H - (\tau_1 + \lambda_u + \mu_H)S_u + \omega_u R = 0, \tag{2.3}$$

$$\alpha\Lambda_H + \omega_h R + \tau_1 S_u - ((1 - \zeta)\lambda_h + \mu_H)S_h = 0, \tag{2.4}$$

Table 1. Variables.

Symbols	Description
\$S_u\$	Unhygienic susceptible human
\$S_h\$	Hygienic susceptible human
\$I_u\$	Unhygienic infected human
\$I_h\$	Hygienic infected human
\$R\$	Recovered human
\$S_v\$	Susceptible mosquitoes
\$I_v\$	Infected mosquitoes

Table 2. Model Parameters.

Symbols	Description
Λ_H	Recruitment rate of human population
Λ_v	Recruitment rate of mosquitoes
τ_1	Progression from S_u to S_n
τ_2	Progression from I_u to I_n
δ_u	Disease—induced death for the unhygienic human class
δ_h	Disease—induced death for the hygienic human class
b_1	Biting rate of mosquito for unhygienic human class
b_2	Biting rate of mosquito for hygienic human class
β_{vh}	Transmission probability of infection from mosquito to human
β_{hv}	Transmission probability of infection from human to mosquitoes
λ_u	The force of infection for unhygienic human class
λ_h	The force of infection for hygienic human class
λ_v	Force of infection for mosquitoes
b_3	Biting rate of mosquitoes
ζ	Rate of reduction of infection for hygienic class
ρ	Modification parameter
θ_u	Rate of recovery for unhygienic human class
θ_h	Rate of recovery for hygienic human class
ω	Rate at which recovered human become susceptible
α	Hygienic rate
μ_H	Natural human death rate
μ_v	Natural death rate of mosquitoes
N_H	Total human population

$$\lambda_u S_u - (\tau_2 + \delta_u + \theta_u + \mu_H) I_u = 0, \quad (2.5) \quad \text{At DFE, } I_u = I_h = I_v = 0,$$

$$(1 - \zeta) \lambda_h S_h + \tau_2 I_u - (\delta_h + \theta_h + \mu_H) I_h = 0, \quad (2.6) \quad \text{So we have,}$$

$$\theta_u I_u + \theta_h I_h - (\omega_u + \omega_h + \mu_H) R = 0, \quad (2.7) \quad (1 - \alpha) \Lambda_H - (\tau_1 + \mu_H) S_u = 0,$$

$$\Lambda_v - \lambda_v S_v - \mu_v S_v = 0, \quad (2.8) \quad \alpha \Lambda_H + \tau_1 S_u - \mu_H S_h = 0,$$

$$\lambda_v S_v - \mu_v I_v = 0 \quad (2.9) \quad \Lambda_v - \mu_v S_v = 0.$$

After computing simultaneously, we have

$$S_u = \frac{(1 - \alpha)\Lambda_H}{(\tau_1 + \mu_H)},$$

$$S_h = \frac{\Lambda_H(\tau_1 + \alpha\mu_H)}{\mu_H(\tau_1 + \mu_H)},$$

$$S_v = \frac{\Lambda_v}{\mu_v}.$$

Therefore, the DFE point of the model is given by

$$E_0 = (S_u^0, S_h^0, I_u^0, I_h^0, R^0, S_v^0, I_v^0) = \left(\frac{(1-\alpha)\Lambda_H}{(\tau_1+\mu_H)}, \frac{\Lambda_H(\tau_1+\alpha\mu_H)}{\mu_H(\tau_1+\mu_H)}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0 \right). \tag{2.15}$$

2.4. Basic Reproduction Number (R_0)

The R_0 is defined as the number of secondary malaria infections produced by one infected individual in a completely susceptible community. The next-generation method [12] will be employed to compute R_0 . The $F(x)$ is the rate of new infection appearance while $V(x)$ is the rate of transfer of individuals into compartments. Therefore,

$$F = \begin{pmatrix} 0 & 0 & \frac{b_1 \beta_{vh}(1-\alpha)\mu_H}{(\tau_1 + \mu_H)} \\ 0 & 0 & \frac{(1-\zeta)b_2 \beta_{vh}(\alpha\mu_H + \tau_1)}{(\tau_1 + \mu_H)} \\ \frac{b_3 \beta_{hv}\Lambda_v\mu_H}{\Lambda_H\mu_v} & \frac{\rho b_3 \beta_{hv}\Lambda_v\mu_H}{\Lambda_H\mu_v} & 0 \end{pmatrix}, \tag{2.16}$$

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\tau_2 & k_2 & 0 \\ 0 & 0 & \mu_v \end{pmatrix}, \tag{2.17}$$

whereas

$$k_1 = (\tau_2 + \delta_u + \theta_u + \mu_H), k_2 = (\delta_h + \theta_h + \mu_H),$$

$$k_3 = (\omega + \mu_H). \tag{2.18}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 & 0 \\ \frac{\tau_2}{k_1 k_2} & \frac{1}{k_2} & 0 \\ 0 & 0 & \frac{1}{\mu_v} \end{pmatrix},$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{b_1 \beta_{vh}(1-\alpha)\mu_H}{(\tau_1 + \mu_H)\mu_v} \\ 0 & 0 & \frac{(1-\zeta)b_2 \beta_{vh}(\alpha\mu_H + \tau_1)}{(\tau_1 + \mu_H)\mu_v} \\ \frac{b_3 \beta_{hv}\Lambda_v\mu_H}{\Lambda_H\mu_v k_1} + \frac{\tau_2 \rho b_3 \beta_{hv}\Lambda_v\mu_H}{\Lambda_H\mu_v k_1 k_2} & \frac{\rho b_3 \beta_{hv}\Lambda_v\mu_H}{\Lambda_H\mu_v k_2} & 0 \end{pmatrix}. \tag{2.19}$$

The R_0 is the largest eigenvalue or spectral radius of FV^{-1} . Hence,

$$R_0 = \sqrt{\frac{b_3 \beta_{vh} \beta_{hv} \Lambda_v \mu_H (b_1 \mu_H (1-\alpha) (k_2 + \tau_2 \rho) + b_2 k_1 \rho (\alpha \mu_H + \tau_1) (1-\zeta))}{\Lambda_H \mu_v^2 k_1 k_2 (\tau_1 + \mu_H)}}. \tag{2.20}$$

Theorem 2

The DFE E_0 for model system is locally asymptotically stable if $R_0 < 1$ and unstable otherwise.

Proof:

At DFE, the Jacobian matrix is given by

$$J = \begin{bmatrix} -(\tau_1 + \mu_H) & 0 & 0 & 0 & \omega & 0 & 0 \\ \tau_1 & -\mu_H & 0 & 0 & \omega & 0 & 0 \\ 0 & 0 & -k_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_v & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_v \end{bmatrix}$$

Table 3. Indices of Sensitivity.

Symbols	Sensitivity Index
Λ_H	-1
Λ_v	1
τ_1	-0.00013
τ_2	-0.000041
δ_u	-0.000041
δ_h	-0.29
b_1	0.00022
b_2	1
β_{vh}	1
β_{hv}	1
b_3	1
ζ	-0.087
ρ	1
θ_u	-0.000015
θ_h	-0.71
α	-0.00011
μ_H	1
μ_v	-2

$$|J - \lambda I| = \begin{vmatrix} -(\tau_1 + \mu_H) - \lambda & 0 & 0 & 0 & \omega & 0 & 0 \\ \tau_1 & -\mu_H - \lambda & 0 & 0 & \omega & 0 & 0 \\ 0 & 0 & -k_1 - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_2 - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_3 - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_v - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_v - \lambda \end{vmatrix}$$

The eigenvalues are:

$$\lambda_1 = -(\tau_1 + \mu_H), \lambda_2 = -\mu_H, \lambda_3 = -k_1, \lambda_4 = -k_2, \lambda_5 = -k_3, \lambda_6 = \lambda_7 = -\mu_v.$$

It is observed that all the eigenvalues are negative, this implies $R_0 < 1$ that at the DFE point is locally asymptotically stable, this means that malaria infection can be eliminated from the population.

2.5. Global stability of the Disease Free Equilibrium (DFE)

Theorem 3

The DFE of the model system is globally asymptotically stable if $R_0 \leq 1$.

Proof:

Consider the following Lyapunov function:

$$V(t) = b_3\beta_{nv}\Lambda_v\mu_H(k_2 + \tau_2\rho)I_u + b_3\rho\Lambda_v\mu_H\beta_{nv}k_1I_h + \Lambda_Hk_1k_2\mu_vI_v, \tag{2.21}$$

Differentiating yield

$$\frac{dV}{dt} = b_3\beta_{nv}\Lambda_v\mu_H(k_2 + \tau_2\rho)(\lambda_u S_u - k_1 I_u) + b_3\rho\Lambda_v\mu_H\beta_{nv}k_1((1 - \zeta)\lambda_h S_h + \tau_2 I_u - k_2 I_h) + \Lambda_Hk_1k_2\mu_v(\lambda_v S_v - \mu_v I_v), \tag{2.22}$$

Table 4. Parameter values of model.

Symbols	Values	Source
Λ_H	100	[13]
Λ_v	1000	[14]
τ_1	0.25	(Assumed)
τ_2	0.5	(Assumed)
δ_u	0.13	(Assumed)
δ_h	0.06	(Assumed)
b_1	0.17	(Assumed)
b_2	0.1	(Assumed)
β_{vh}	0.03	[2]
β_{nv}	0.09	[2]
b_3	0.12	[15]
ζ	0.08	(Assumed)
ρ	0.5	(Assumed)
θ_u	0.05	(Assumed)
θ_h	0.15	(Assumed)
ω	0.7902	[14]
α	0.46	(Assumed)
μ_H	0.00004	[13]
μ_v	0.0000569	[14]

At DFE, it was found that

$$\begin{aligned} \frac{dV}{dt} &\leq \left(\frac{b_3 \beta_{vh} \beta_{hv} \Lambda_v \mu_H (b_1 \mu_H (1-\alpha)(k_2 + \tau_2 \rho) + b_2 k_1 \rho (\alpha \mu_H + \tau_1)(1-\zeta))}{(\tau_1 + \mu_H)} - \Lambda_H k_1 k_2 \mu_v^2 \right) I_v, \\ \frac{dV}{dt} &\leq \left(\frac{b_3 \beta_{vh} \beta_{hv} \Lambda_v \mu_H (b_1 \mu_H (1-\alpha)(k_2 + \tau_2 \rho) + b_2 k_1 \rho (\alpha \mu_H + \tau_1)(1-\zeta))}{\Lambda_H k_1 k_2 \mu_v^2 (\tau_1 + \mu_H)} - 1 \right) I_v, \\ \frac{dV}{dt} &\leq (R_0^2 - 1) I_v, \end{aligned} \quad (2.23)$$

From the equation above, $\frac{dV}{dt} \leq 0$, if $R_0 \leq 1$. (2.24)

Hence, the DFE is globally asymptotically stable.

2.6. Sensitivity Analysis

In this section, sensitivity analysis is carried out to identify the parameters that have a great influence on the R_0 . The sensitivity index of R_0 to a given parameter P is given by the relation

$$\Pi_P^{R_0} = \frac{\partial R_0}{\partial P} \frac{P}{R_0}, \quad (2.25)$$

Table 3 shows the sensitivity indices of the basic reproduction number to the parameters. The parameters with positive indices indicate that the basic reproduction number increases as their values increase. While the parameters with negative sensitivity indices indicate an increase in these parameters will result in the decline of the basic reproduction number and vice-versa.

3. RESULT AND DISCUSSIONS

First, this system of model is biologically meaningful and mathematically well-posed in the given domain Ω . The R_0 of the model is computed using the next-generation method. The existence of the disease-free equilibrium of the system is established and the condition for the local stability of the disease-free equilibrium and global stability of the disease-free equilibrium follows using the Lyapunov function. The DFE is locally asymptotically stable if $R_0 < 1$ and globally asymptotically stable if $R_0 \leq 1$. Sensitivity analysis of the model equation is carried out as illustrated in Table 3. From the table, it shows that the natural death rate of mosquitoes (μ_v) is most sensitive to the Basic Reproduction Number.

4. CONCLUSIONS

In this work, the mathematical model to assess the impact of hygiene on malaria transmission dynamics was proposed and analyzed. The model is divided into the human population and vector (mosquito) population, the human population is further subdivided into the susceptible unhygienic human population, susceptible hygienic human population, infected unhygienic human population, infected hygienic human population and recovered human, while the vector population is subdivided into the susceptible vector and infected vector. We proved that the model equation is biologically meaningful and mathematically well-posed. The disease-free equilibrium (DFE) is established and it was observed that DFE is locally asymptotically stable if $R_0 < 1$ while globally asymptotically stable if $R_0 \leq 1$ using Lyapunov function. Sensitivity analysis of the model parameters is carried out and it shows that the natural death rate of mosquitoes is most sensitive to the Basic reproduction Number.

This implies that individuals must continue to engage in activities that promote both personal hygiene and environmental hygiene so as reduce the growth of mosquito hence curbing the spread of malaria also government and other Non-Governmental Organizations (NGOs) must continue to intensify campaigns on hygienic practices at individual and community levels. Future studies can be carried out on this model such as: establishing and proving the existence of the unique endemic equilibrium point, analyzing the stability (local and global stability) of the endemic equilibrium point, and solving the model equations using any analytic method available.

AUTHOR INFORMATION

Corresponding Author

Temidayo Oluwafemi — General Studies Department, Newgate College of Health Technology, Minna - 920211 (Nigeria);
Email: dayofemi985@gmail.com

Author

Emmanuel Azuaba — Department of Mathematics, Bingham University Karu, Nassarawa - 962101 (Nigeria) ;

REFERENCES

- [1] E. Azuaba, J. M. Orverem, Y. M. Kura and U. J. Dahiru. (2020). “Mathematical Approach for Malaria Disease in the Presence of Drug Therapy and Treatment”. *International Journal of Mathematics, and Its Applications*. **8** (1): 77–88.
- [2] S. Olaniyi, K. O. Okosun and S. O. Adesanya. (2018). “Global Stability and Optimal Control Analysis of Malaria Dynamics in the Presence of Human Travelers”. *The Open Infectious Diseases Journal*. **10**: 166–186. [10.2174/1874279301810010166](https://doi.org/10.2174/1874279301810010166).
- [3] S. Olaniyi, K. O. Okosun, S. O. Adesanya and R. S. Lebelo. (2020). “Modeling Malaria Dynamics with Partial Immunity and protected travelers: optimal control and cost-effectiveness analysis”. *Journal of Biological Dynamics*. **14** (1): 90–115. [10.1080/17513758.2020.1722265](https://doi.org/10.1080/17513758.2020.1722265).
- [4] F. Kogan. (2020). “Remote Sensing for Malaria (Monitoring and Predicting Malaria from Operational Satellites)”. Springer, Cham. [10.1007/978-3-030-46020-4](https://doi.org/10.1007/978-3-030-46020-4).
- [5] G. O Mauti. E. M. Mauti and K. D. Kowanga. (2015). “Evaluation of Malaria Spread in Relation to Poor Environmental Conditions at Kibaha District (Tanzania)”. *Journal of Scientific & Innovative Research*. **4** (5) :203–206.
- [6] T. Nkuo-Akenji, N. N. Ntonifor, M. B. Ndikum, E. L. Abongwa, A. Nkweschu, D. N. Anong, M. Songmbe, M. G. Boyo, K. N. Ndamukong, and V. P. K. Titanji. (2006). “Environmental factors affecting malaria parasite prevalence in rural Bolifamba, South West Cameroon”. *African Journal of Health Sciences*. **13** (1-2): 40–46.
- [7] U. U. Enebeli, A. N. Amadi, O. K. Iro, E. T. Oparaocha, E. A. Nwoke, S. N. O. Ibe, N. N. Oti, U. M. Chukwuocha, C. R. Nwufo, C. O. Amadi, and I. Esomonu. (2019). “Assessment of Water, Sanitation and Hygiene Practices and the Occurrence of Childhood Malaria in Abia State, Nigeria”. *Researchjournal’s Journal of Public Health*. **5** (6): 1–15.
- [8] D. Okuonghae, and A. Nwankwo. (2019). “Mathematical Assessment of the Impact of Different Microclimate Conditions on Malaria Transmission Dynamics”. *Mathematical Biosciences and Engineering*. **16** (3): 1414–1444.
- [9] A. N. Goni, and S. Musa (2018). “Modeling the Effect of Education-Based Intervention in the control of Malaria”. *Science World Journal*. **13** (4).
- [10] G. Bhujju, G. R. Phaijoo, and D. B. Gurung. (2018). “Mathematical Study on Impact of Temperature in Malaria Disease Transmission Dynamics”. *Advances in Computer Sciences*. **1** (2).
- [11] O. K. Okosun, and O. D. Makinde. (2011). “Modeling the Impact of Drug Resistance in Malaria Transmission and Its Optimal Control Analysis”. *International Journal of Physical Sciences*. **6**: 6479-6487. [10.5897/IJPS10.542](https://doi.org/10.5897/IJPS10.542).
- [12] O. Diekmann, J. A. Hesterbeek, and M. G. Roberts. (2010). “Construction of Next-Generation Matrices for Compartmental Models in Epidemics”. *Journal of the Royal Society of Biology, Interface*. **7** (47): 875-885. [10.1098/rsif.2009.0386](https://doi.org/10.1098/rsif.2009.0386).
- [13] M. O. Oluwatayo. (2019). “Mathematical Model of the Coinfection Dynamics of Malaria-Toxoplasmosis in the Tropics”. *Biometrical Letters*. **56** (2): 139-163. [10.2478/bile-2019-0013](https://doi.org/10.2478/bile-2019-0013).
- [14] E. A. Bakare and C. R. Nwozo. (2017). “Bifurcation and Sensitivity Analysis of Malaria-Schistosomiasis Coinfection Model”. *International Journal of Applied Computational Mathematics*. [10.1007/s40819-017-0394-5](https://doi.org/10.1007/s40819-017-0394-5).
- [15] S. Olaniyi, and O. S. Obabiyi. (2013). “Mathematical Model for Malaria Transmission Dynamics in Human and Mosquito Populations with Nonlinear Forces of Infection”. *International Journal of Pure and Applied Mathematics*. **88** (1): 125-56.

