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Malaria dynamics of transmission for individuals with multi-layered susceptibility

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The alarming prevalence of vector-borne diseases, such as malaria, has long been a global concern due to their ability to infect individuals across all social classes, thus leading to high morbidity and mortality rates. This study investigates the role of mosquito bites frequency in dynamics of transmission of malaria. Mainly, featuring the mathematical classification of susceptible individuals into high and low risk. The present study employs a time-dependent, social hierarchy-structured deterministic model to analyse the vulnerability of multi-layered classes to the transmission dynamics of malaria disease. This analysis takes into account the interaction between the human population and the mosquito vector population. Human infection statuses are divided into four categories: susceptible, infected, and recovered individuals, with further stratification of susceptible individuals based on their risk level. Concurrently, the total vector population is divided into susceptible and infected mosquitoes. The disease free equilibrium, basic reproduction number and endemic equilibrium were computed. The findings show that the higher the number susceptible humans subjected to high risk the higher number of infected human individuals.

Keywords: *Malaria; transmission; low-risk susceptible; high-risk susceptible; multi-layered classes.*

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1. Introduction

Malaria is a disease that is transmitted to human population by the bites of female anopheles mosquitoes. Moreover, it is one of the communicable disease regarded as a global threat [1]. Most of regions that are highly affected with malaria are Sub-Saharan Africa, Asia and America [2, 3]. The Sub-Saharan Africa is known with high morbidity and mortality rate, a region where *Plasmodium falciparum* is the main cause of a serious morbidity and high mortality rate [4, 5]. Therefore, it is inevitable to talk of malaria without mentioning Sub-Saharan Africa.

To eradicate malaria in Sub-saharan Africa, provision of insecticide mosquito treated nets (ITNs) which regarded as an effective way of preventing malaria has been a key [7]. However, providing of ITNs to more than 65% of Sub-Saharan African families has not shown to reduce morbidity and mortality, contrarily has increased [8]. Since the use of ITNs mainly focus on reducing the frequency of mosquito bites, mosquito contact rate, and increase mosquito death rate. The morbidity and mortality have shown to be high regardless of many families using ITNs. This implies that the study on the how frequently people are bitten by mosquito is less studied.

Consequently, this study focuses to investigate how the frequency of mosquito bites affects the dynamics of transmission of malaria incorporating behavioural responses, occupation and location of human individuals. In categorizing the susceptible human population into high-and low-risk individuals. Low risk individuals be susceptible people exposed to moderate mosquito frequency bites while high risk be those exposed to high frequency of mosquito bites due to their behaviour, location and occupation. Monthly data from Tanzania for September 2022 to December 2023 has been used for parameter estimation and fitting the model.

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2. Model formulation

In capturing the dynamics of transmission of malaria where the susceptible population categorises into multi-layered groups. The total human population ($N_H(t)$) is divided into four categories: susceptible ($H_S(t)$), infected ($H_I(t)$), and recovered ($H_R(t)$) with further stratification of Susceptible human individuals into high risk susceptible ($H_{Sh}(t)$) and low risk susceptibles ($H_{Sl}(t)$). More precisely, $H_S(t) = H_{Sh}(t) + H_{Sl}(t)$. Moreover, the total mosquito population($N_M(t)$) is divided into Susceptible mosquitoes ($M_S(t)$) and Infected mosquitoes ($M_I(t)$).

The high-risk vulnerable population grows at a rate of Δ, which is a fraction of the total recruitment in the susceptible population Λ_H, due to either births or immigrants. However, the remaining fraction $(1-\Delta)$ of Λ_H goes into low risk vulnerable. The high risk vulnerable humans also grow by the rate ρ of human immunity losing rate Φ, while $(1-\rho)$ of Φ becomes the low risk vulnerable humans. At the same time the low and high risk vulnerable population decrease by forces of infection Γ_1 and Γ_2 respectively. So that,

$$\Gamma_1 = p_{MH}\, b\, \frac{M_I(t)}{N_H(t)},$$
$$\Gamma_2 = p_{MH}\, b\, \zeta\, \frac{M_I(t)}{N_H(t)},$$

where p_{MH} is the rate of mosquito transmission to humans. While, b and ζ indicate the mosquito-human contact rate and the modification parameter for increasing mosquito bite frequency, respectively. Furthermore, both low and high risk vulnerable human populations are reduced by the natural mortality rate μ_H.

Moreover, infected human population increases by Γ_1 and Γ_2, reduced by μ_H and by the disease mortality rate represented by q. Additionally, the number of recovered human beings increase by λ and decrease by Φ and by natural death μ_H.

The number of vulnerable mosquitoes increases proportionally with the birth of adult mosquitoes Λ_M. At the same time the susceptible mosquito declines by mosquito natural death rate μ_M, and by force of infection Γ_3. So that,

$$\Gamma_3 = p_{HM}\, b\, \frac{H_I(t)}{N_H(t)},$$

where p_{HM} is the rate of transmission from human to mosquito. Figure 1 explains pictorially the dynamic transmission of malaria regarding compartments and corresponding with the assumptions applied.

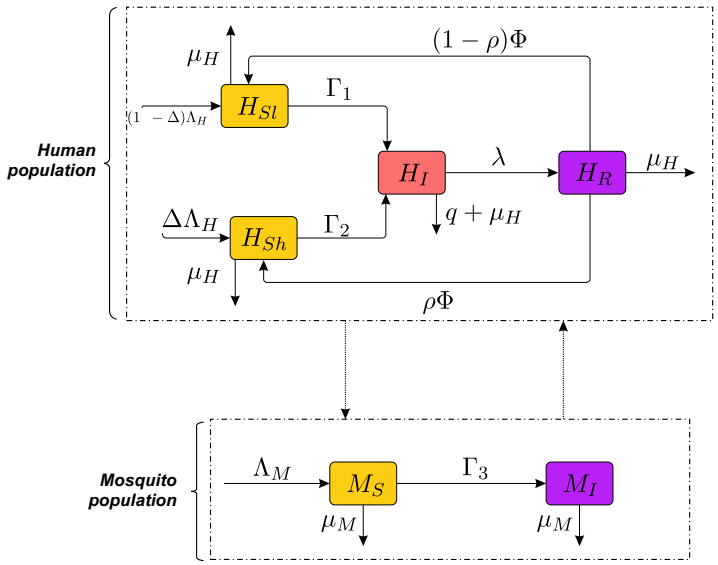

Fig. 1. Schematic depiction of dynamic transmission.

$$\begin{aligned}
\frac{dH_{Sl}(t)}{dt} &= (1-\Delta)\Lambda_H + (1-\rho)\Phi H_R(t) - p_{MH} b \frac{M_I(t)}{N_H(t)} H_{Sl}(t) - \mu_H H_{Sl}(t),\\
\frac{dH_{Sh}(t)}{dt} &= \Delta\Lambda_H + \rho\,\Phi H_R(t) - p_{MH} b\,\zeta \frac{M_I(t)}{N_H(t)} H_{Sh}(t) - \mu_H H_{Sh}(t),\\
\frac{dH_I(t)}{dt} &= p_{MH} b \frac{M_I(t)}{N_H(t)} H_{Sl}(t) + p_{MH} b\,\zeta \frac{M_I(t)}{N_H(t)} H_{Sh}(t) - (q+\mu_H+\lambda) H_I(t),\\
\frac{dH_R(t)}{dt} &= \lambda H_I(t) - (\mu_H+\Phi) H_R(t),\\
\frac{dM_S(t)}{dt} &= \Lambda_M - \mu_M\, M_S(t) - p_{HM} b \frac{H_I(t)}{N_H(t)} M_S(t),\\
\frac{dM_I(t)}{dt} &= p_{HM} b \frac{H_I(t)}{N_H(t)} M_S(t) - \mu_M M_I(t).
\end{aligned} \tag{1}$$

The model (1) of this study is the extension of different models in [3, 11]. Where every single parameter in the model is assumed to be positive. The model is developed in the consideration of the following assumptions:

(i) It is assumed that high-risk susceptible human are people subjected to high frequency of mosquito bites, contrarily the low risk susceptible human are subjected to low frequency mosquito bites.
(ii) The modification parameter for increased mosquito bites in high risk susceptible human ζ, is greater than one.
(iii) The model considers the short period of time that very a few number of people can change their behaviour, occupation or change their place of dwelling. Thus assuming that no person can move from low to high risk and vice-verse.

3. Model analysis

3.1. Disease free equilibrium

The disease free equilibrium (DFE) of the model (1), that computed with consideration of the absence of infection, taking that $H_I = H_R = M_I = 0$ into model (1) denoted by

$$\mathscr{E}_0 = \left(\frac{\Lambda_H(1-\Delta)}{\mu_H}, \frac{\Lambda_H \Delta}{\mu_H}, 0, 0, \frac{\Lambda_M}{\mu_M}, 0 \right). \tag{2}$$

3.2. Basic reproduction number

In order to determine the basic reproduction number $\mathscr{R}_0$, we use the procedure outlined in [12]. It is composed of the non-negative matrix $\mathscr{F}$ (of the new infection term) and the matrix $\mathscr{V}$ (of the transition term) associated with the model (1), and then determining the spectral radius of $\mathscr{F}\mathscr{V}^{-1}$. In order to achieve this, we classify the infection statuses of the model and deconstruct the right-hand side into $F - V$. Let, F and V;

$$F = \begin{pmatrix} p_{MH} b \frac{M_I(t)}{N_H(t)} H_{Sl}(t) + p_{MH} \zeta b \frac{M_I(t)}{N_H(t)} H_{Sh}(t) \\ p_{HM} b \frac{H_I(t)}{N_H(t)} M_S(t) \end{pmatrix}, \quad V = \begin{pmatrix} (q+\mu_H+\lambda) H_I(t) \\ \mu_M M_I(t) \end{pmatrix}.$$

Thus,

$$\mathscr{F} = \begin{pmatrix} 0 & p_{MH}\Delta b\zeta - \frac{(\Lambda_H\Delta - \Lambda_H) p_{MH} b}{\Lambda_H} \\ \frac{\Lambda_M p_{HM} \mu_H b}{\Lambda_H \mu_M} & 0 \end{pmatrix}, \quad \mathscr{V} = \begin{pmatrix} g_1 & 0 \\ 0 & \mu_M \end{pmatrix}.$$

Therefore,

$$\mathscr{F}\mathscr{V}^{-1} = \begin{pmatrix} 0 & \frac{\Delta b p_{MH}\zeta - (\Delta - 1) b p_{MH}}{\mu_M} \\ \frac{\Lambda_M b \mu_H p_{HM}}{\Lambda_H g_1 \mu_M} & 0 \end{pmatrix}$$

Thus, by the next generation operator technique, the basic reproduction number is given by

$$\mathscr{R}_0 = \frac{b}{\mu_M}\sqrt{\frac{\Delta\Lambda_M\,\mu_H\,p_{HM}\,p_{MH}\zeta + (1-\Delta)\Lambda_M\,\mu_H\,p_{HM}p_{MH}}{\Lambda_H g_1}} \tag{3}$$

Theorem 1 (Ref. [13]). *If $\mathscr{R}_0 < 1$, then the disease free equilibrium is locally asymptotically stable and unstable otherwise.*

Using model (1) malaria can be eradicated from the population with both low- and high-risk susceptible human individuals when the basic reproduction number $\mathscr{R}_0$ is reduced to less than one. This is depicted in Theorem 1.

3.3. Endemic equilibrium

Endemic equilibrium (EE) denoted by $\mathscr{E}_1 = (H_{Sl}^{**}, H_{Sh}^{**}, H_I^{**}, H_R^{**}, M_S^{**}, M_I^{**})$ is the equilibrium point of the model (1) which considers the presence of infection in the population. Following the approach adopted in [6, 13, 14] we consider the human mortality rate due to disease equal to zero $q \approx 0$, so that $N_H = \frac{\Lambda_H}{\mu_H}$ and the forces of infection be

$$\Gamma_1^{**} = \frac{p_{MH}\,b\,M_I^{**}\mu_H}{\Lambda_H}, \quad \Gamma_2^{**} = \frac{p_{MH}\,b\,\zeta\,M_I^{**}\mu_H}{\Lambda_H}, \quad \Gamma_3^{**} = \frac{p_{HM}\,b\,H_I^{**}\mu_H}{\Lambda_H}$$

therefore we have model (1) turn into

$$\begin{aligned}
(1-\Delta)\Lambda_H + (1-\rho)\Phi H_R^{**} - \Gamma_1^{**}H_{Sl}^{**} - \mu_H H_{Sl}^{**} &= 0,\\
\Delta\Lambda_H + \rho\,\Phi H_R^{**} - \Gamma_2^{**}H_{Sh}^{**} - \mu_H H_{Sh}^{**} &= 0,\\
\Gamma_1^{**}H_{Sl}^{**} + \Gamma_2^{**}H_{Sh}^{**} - \mu_H H_E^{**} - g_1 H_I^{**} &= 0,\\
\lambda\,H_I^{**} - g_2 H_R^{**} &= 0,\\
\Lambda_M - \mu_M M_S^{*} - \Gamma_3^{**}M_S^{**} &= 0,\\
\Gamma_3^{**}M_S^{**} - \mu_M M_I^{**} &= 0,
\end{aligned} \tag{4}$$

thus solving the system (4) we get,

$$\begin{aligned}
H_{Sl}^{**} &= \frac{\Gamma_2^{**}\Lambda_H\Phi\lambda(\Delta-\rho)+g_1g_2\Lambda_H\mu_{H(1-\Delta)}+\Gamma_2^{**}g_1g_2\Lambda_H(1-\Delta)}{K},\\
H_{Sh}^{**} &= \frac{\Delta\Gamma_1^{**}\Lambda_H(g_1g_2-\Phi\lambda)+\Delta\Lambda_H g_1g_2\mu_H+\Gamma_1^{**}\Lambda_H\Phi\lambda\rho}{K},\\
H_I^{**} &= \frac{\Gamma_1^{**}\Gamma_2^{**}\Lambda_H g_2+(\Delta\Gamma_2^{**}g_2+g_2(1-\Delta)\Gamma_1^{**})\Lambda_H\mu_H}{K},\\
H_R^{**} &= \frac{\Gamma_1\Gamma_2^{**}\Lambda_H\lambda+(\Delta\Gamma_2^{**}\lambda_1+\lambda(1-\Delta)\Gamma_1^{**})\Lambda_H\mu_H}{K},\\
M_S^{**} &= \frac{\Lambda_M}{\Gamma_3^{**}+\mu_M},\\
M_I^{**} &= \frac{\Gamma_3^{**}\Lambda_M}{\Gamma_3^{**}\mu_M+\mu_M^2}.
\end{aligned}$$

Since

$$K = \Gamma_1^{**}\Gamma_2(g_1g_2-\Phi\lambda) + g_1g_2\mu_H^2 + \Gamma_1^{**}\lambda\Phi\mu_H\rho + \Gamma_1^{**}g_1g_2\mu_H(1-\zeta) + \Gamma_2^{**}\mu_H(g_1g_2-\lambda\Phi),$$

$$g_1 = q+\mu_H+\lambda, \quad g_2 = \mu_H+\Phi,$$

$$\Gamma_1^{**} = \frac{\Gamma_3^{**}\Lambda_M b\mu_H p_{MH}}{\big(\Gamma_3^{**}\mu_M+\mu_M^2\big)\Lambda_H}, \quad \Gamma_2^{**} = \frac{\Gamma_3^{**}\Lambda_M b\mu_H p_{MH}\zeta}{\big(\Gamma_3^{**}\mu_M+\mu_M^2\big)\Lambda_H}.$$

Inserting the forces of infection Γ_1^{**} and Γ_2^{**} into the force of infection Γ_3^{**}, we obtain the quadratic equation

$$X_1(\Gamma_3^{**})^2 + X_2\Gamma_3^{**} + X_3 = 0. \tag{5}$$

The values of X_1, X_2 and X_3 can be expressed as follows:

$$\begin{aligned}
X_1 = \big(&\Lambda_M^2 b^2 p_{MH}^2\zeta(g_1g_2-\Phi\lambda) + \Lambda_H\Lambda_M b\mu_H p_{MH}\zeta(g_1g_2-\rho\Phi\lambda) + \Lambda_H\Lambda_M b\mu_M p_{MH}(g_1g_2-(1-\rho)\Phi\lambda)\\
&+\Lambda_H\Lambda_M b g_1g_2\mu_M p_{MH} + \Lambda_H^2 g_1g_2\mu_M^2\big) > 0,
\end{aligned}$$

$$X_2 = \big(\Delta\Lambda_H\Lambda_M b^2 g_2\mu_H\mu_M p_{HM}p_{MH}(\zeta-1) + \Lambda_M^2 b^3 g_2\mu_H p_{HM}p_{MH}^2\zeta + \Lambda_H\Lambda_M\Phi b\lambda_1\mu_M^2 p_{MH}\rho\zeta$$
$$+\Lambda_H\Lambda_M b^2 g_2\mu_H\mu_M p_{HM}p_{MH} - \Lambda_H\Lambda_M b g_1 g_2\mu_M^2 p_{MH}(\zeta+1) + \Lambda_H\Lambda_M\Phi b\lambda_1\mu_M^2 p_{MH}(1-\rho)$$
$$-2\,\Lambda_H^2 g_1 g_2\mu_M^3\big),$$
$$X_3 = \Lambda_H^2 g_1 g_2\mu_M^4(1-\mathscr{R}_0^2).$$

It is clear, that X is positive since the expressions $g_1g_2 - \Phi\lambda$, $g_1g_2 - \rho\Phi\lambda$, and $g_1g_2 - (1-\phi)\Phi\lambda$ are always positive. However, the value of X_3 is positive if $\mathscr{R}_0$ is less than 1 and negative if $\mathscr{R}_0$ exceeds 1. The occurrence of backward bifurcation is determined by the value of X_3 [17]. Hence, the following results can be obtained.

Theorem 2. *The model (1) gives:*

(i) Existence of EE when $\mathscr{R}_0 > 1$, implying that $X_3 < 0$;
(ii) Existence of EE when $X_2 < 0$, and $X_3 = 0$ or $X_2^2 - 4X_1X_3 = 0$;
(iii) Existence of backward bifurcation when $X_3 > 0$ for $\mathscr{R}_0 < 1$, $X_2 < 0$ and $X_2^2 - 4X_1X_3 > 0$;
(iv) No existence of EE otherwise.

In (i) and (ii), the existence of EE is shown when $R_0 > 1$ and $R = 1$, respectively. In addition, (iii) shows the probability of existence of backward bifurcation. In demonstrating whether the backward bifurcation exists in the model we calculate the critical value denoted by $\mathscr{R}_0^c$ by $X_2^2 - 4X_1X_3 = 0$, such that

$$\mathscr{R}_0^c = \sqrt{1 - \frac{X_2}{4X_1\Lambda_H^2 g_1 g_2\mu_M^4}}. \tag{6}$$

Thus, it is depicted that the backward bifurcation can occur when $\mathscr{R}_0^c < \mathscr{R}_0 < 1$.

Theorem 3 (Refs. [6, 13]). *If $\mathscr{R}_0 > 1$, then endemic equilibrium is globally asymptotically stable and unstable otherwise.*

Theorem 3 demonstrates also the model (1) that malaria exists to the population when basic reproduction number $\mathscr{R}_0$ is greater than one.

4. Model fitting

In order to validate the model to the real collected data, model fitting was performed to help obtain the parameter values that fit to the data. In this scenario we used least square method to fit the model, which implemented using python programming language by *scipy.optimize.curve_fit* tool. This fitting process is depicted in Figure 2 and Table 1 shows the parameter values obtained from fitting the model to real malaria confirmed cases from Tanzania ministry of health for September 2022 to December 2023. Using the data in Table 1 obtained by fitting the model to the real data, we computer the basic reproduction number $\mathscr{R}_0$ of the model (1). Thus, the malaria basic reproduction number $\mathscr{R}_0$ for Tanzania from September 2022 to December 2023 is 3.32516. Referring Theorem 3, value of $\mathscr{R}_0 = 3.32516$ indicates that endemic of malaria existed in Tanzania from September 2022 to December 2023.

Table 1. Parameter values of the model (1).

Parameter	Value (day^{-1})	Reference
b	0.065	[15]
ζ	1.4	Assumed
p_{HM}	0.002	Fitted
p_{MH}	0.01104	Fitted
q	0.000078904	Estimated
ρ	0.8	Assumed
Φ	0.3099	Fitted
μ_M	0.001	Fitted
μ_H	0.000004	Estimated
λ	0.08	Fitted
Δ	0.8	Assumed
Λ_H	200	Assumed
Λ_M	400	Assumed

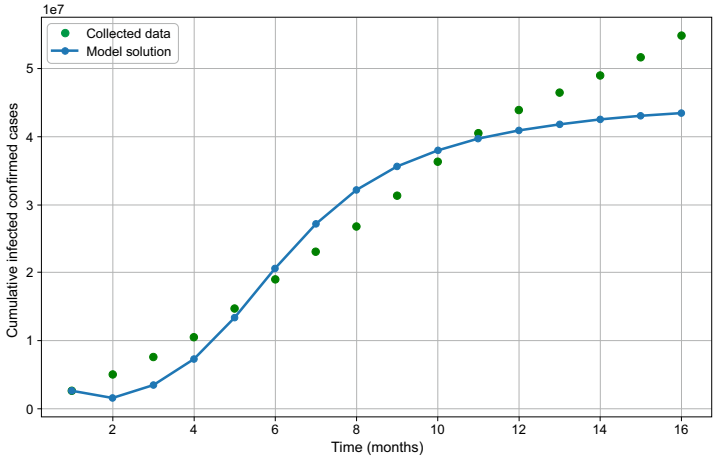

Fig. 2. The Model fitting to Tanzania's monthly malaria cases Sep 2022 – Dec 2023.

5. Numerical simulations

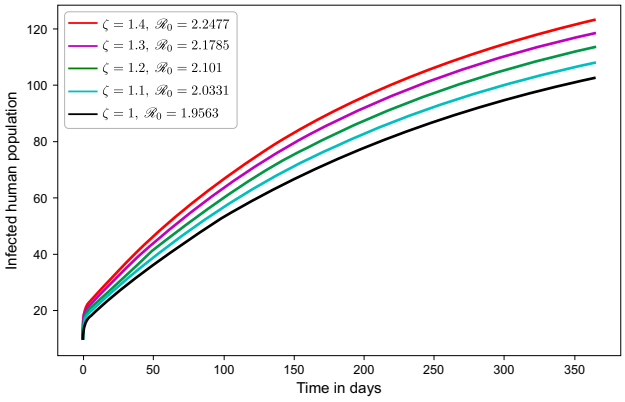

Fig. 3. Infected humans.

In this section we use Python 3.0 in both solving the model and numerical simulations, setting human population initial conditions as: $N_H(0) = 6549$, $H_{Sl}(0) = 2000$, $H_I(0) = 10$, $H_R(0) = 0$ and $H_{Sh}(0) = 4539$. We also set mosquito population initial conditions as $N_M(0) = 400000$, $M_I(0) = 20000$ and $M_S(0) = 38000$. Furthermore, the values of parameters as in Table 1 used for simulation with varying of the parameter ζ from 1 to 1.4. Figure 3 depicts that increase of modification parameter for increase in mosquito frequency bites in high-risk susceptible humans ζ, causes the increase in infected human individuals. Therefore, these findings depicts that in order to successfully control malaria from the population, the high-risk susceptible human individuals should be given a great attention compares to low-risk individuals. For instance, in addition to providing mosquito insecticide-treated nets, a control strategy should include repellent lotion for high-risk individuals, especially during outdoor activities. This will probably help to reduce the morbidity and mortality from the entire population. However, focusing

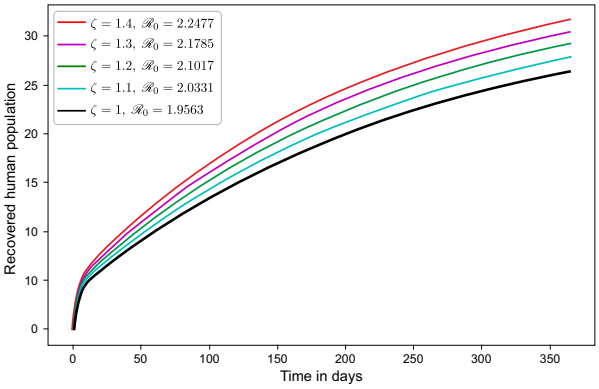

Fig. 4. Recovered humans.

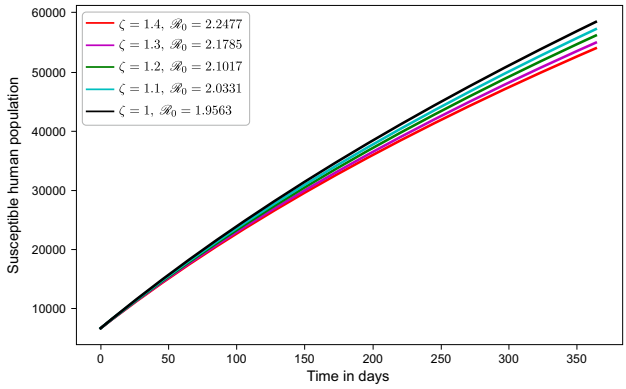

Fig. 5. Susceptible humans.

only to high risk-human individuals will not eradicate the disease from the entire population. Figures 4 and 5 also depict the recovered and susceptible human individuals as the parameter ζ varies.

6. Conclusion

Malaria is a disease that has a great detrimental effect to the human development. Consequently, the non-linear differential equation time-dependent model has developed to capture the human population with both high and low-risk susceptible humans due to human behaviours, occupation and location. We successfully computed the basic reproduction number $\mathscr{R}_0$, disease free equilibrium and endemic equilibrium. The disease free equilibrium is locally asymptotically stable when $\mathscr{R}_0 < 1$. Endemic exists to the population when $\mathscr{R}_0 > 1$. Moreover, it has depicted graphically that when $\mathscr{R}_0 > 1$, the greater the exposure of susceptible individuals to infection, the higher the spread of infection throughout the population precisely at long run. This suggests that to successfully control malaria in the population a great attention should be give to highly susceptible, probably avoid great morbidity and mortality in the entire human population.

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Динаміка передачі малярії в осіб з багаторівневою сприйнятливістю

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Тривожна поширеність трансмісивних захворювань, таких як малярія, вже давно викликає глобальне занепокоєння через їх здатність заражати людей у всіх соціальних класах, що призводить до високих рівнів захворюваності та смертності. Це дослідження вивчає роль частоти укусів комарів у динаміці передачі малярії, загалом, з математичною класифікацію вразливих осіб на групи високого та низького ризику. У цьому дослідженні використовується залежна від часу, структурована соціальною ієрархією детермінована модель для аналізу вразливості багаторівневих класів до динаміки передачі малярії. Цей аналіз враховує взаємодію між людською популяцією та популяцією комарів–переносників. Інфекційні статуси людей поділяються на чотири категорії: сприйнятливі, інфіковані та одужавші особи з подальшою стратифікацією сприйнятливих осіб на основі рівня ризику. Одночасно загальна популяція переносників поділяється на сприйнятливих та інфікованих комарів. Було розраховано рівновагу без захворювання, базове репродуктивне число та ендемічну рівновагу. Отримані дані показують, що чим більша кількість сприйнятливих людей піддається високому ризику, тим більша кількість інфікованих людей.

Ключові слова: *малярія; передавання; сприйнятливі особи з низьким ризиком; сприйнятливі особи з високим ризиком; багаторівневі класи.*