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# STABILITY ANALYSIS OF THE MODELS FOR MALARIA'S EFFECTS ON HUMANS BASED ON THE GENETIC STRUCTURE

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## ABSTRACT

Malaria, according to encyclopedia Britannica, is a relapsing infection caused by plasmodium, transmitted to humans through the bite of an anopheles mosquito. The composition of the genes in humans can either be homozygous (AA, SS) or heterozygous (AS), the homozygous are usually prone to the infection of malaria. The homozygous sickle cell genes (SS) encounter serious problems with blood shortage due to the sickle cell, this makes the malaria infection in them more complicated. The heterozygous sickle cell, however, develops a resistance to the infection through the immunity offered by the single sickle cell. This paper studies malaria's effects on the homozygous and heterozygous genes through the system of ordinary differential equations. The model was analyzed for stability, the reproduction number was obtained, and a simulation was performed using the reproduction number and some of the parameters to find out which of the parameters is most sensitive to the control of the spread of malaria. We found that contact rates and infection rates are highly sensitive parameters in malaria transmission. Therefore, minimizing mosquito-human contact is essential for disease control. Furthermore, our results showed that individuals with sickle cell trait have improved recovery rates, underscoring the protective benefits of this trait against malaria.

**Keywords**: Heterozygous, Homozygous, Sickle cell diseases, Plasmodium, Genotype

## INTRODUCTION

Malaria transmission occurs when an infected mosquito bites a person, the parasite that causes malaria is introduced into his bloodstream and continues to reproduce in cells away from the immune system. In the end, the parasite ruptures its cellular haven and unleashes chemicals that harm the tissue around it. The liver of the host first becomes infected, followed by the RBCs. To fully recover from malaria, one must not only combat the parasite but also repair any harm the parasite and the immune system's war on it have done (Henna, 2019).

Sickle cell disease results from a mutation in the gene that codes for β-globin, known as the haemoglobin (Hb)S mutant allele as opposed to the wild type, known as the HbA allele. In the homozygous situation (HbSS), this mutation results in sickle cell anemia, a serious disorder that is still fatal in places where access to modern medication is not possible. Although the mutation has been linked to several conditions or diseases like hematuria, splenic infarction, and exercise-related sudden death, it causes a much milder condition in carriers of the heterozygous condition (HbAS), also known as sickle cell trait (SCT) (Tsaras *et al.*, 2009). On the other hand, people who have the sickle cell trait (one sickle gene and one normal hemoglobin gene), commonly known as sickle cell disease, have a slight advantage when it comes to malaria. Because of this, sickle cell carriers are more prevalent in locations where malaria is common. Sickle cell trait was found to offer 60% protection from overall mortality. Like a common cold, sickle cell illness is not transmitted through contact. Either a person is born with it or not. If you are born with sickle cell disease, either of your parents (or one parent with sickle cell trait and the other with another haemoglobin characteristic) has the sickle cell trait. While a person with sickle cell trait cannot go on to have sickle cell illness, they remain capable of transmitting the gene to their offspring (Amber Yates, 2022).

In areas where malaria is prevalent, the body's defense mechanism is most active between 2 and 16 months of age, before the development of long-term immunity. The malaria parasite cycles between humans and female Anopheles mosquitoes. In humans, the parasite first multiplies in red blood cells and then in liver cells. Infected individuals transmit the parasite to mosquitoes, which spread the disease, but surprisingly, the infected mosquitoes do not suffer from the parasite, unlike their human hosts (Grosse *et al.*, 2011).

Ibrahim and Kogi (2020) analysed data from 2001 to 2005 to investigate the prevalence of malaria in a few hospitals in Zaria, Nigeria. Children were the most afflicted, and the results showed consistently high infection rates. The distribution of malaria cases was fairly uniform throughout the year, with no notable seasonal variations, and gender-based differences were not statistically significant. It was recommended that data collection must be improved by specifying the age of patients, priority should be given to youngsters in the malaria preventive measures, and demographic details should take care of occupation and socioeconomic status.

Eridani (2011) explored the relationship between Sickle Haemoglobin (HbS) and malaria resistance. Early findings indicated that people with the sickle cell trait (HbS trait) were less vulnerable to malaria, and research has validated this protective effect. However, the presence of the HbS gene with alpha thalassemia appears to reduce this protection, which may explain why the HbS trait is less common among Mediterranean groups. Overall, malaria protection appears to be the product of several interacting elements, and proper understanding of these mechanisms could help develop better malaria preventive and treatment measures, as the illness continues to affect many locations throughout the world.

One copy of the gene alone, however, increases tolerance. Sickle-cell anaemia, a potentially fatal illness, results from having two versions. Long-standing research by immunologists has demonstrated that sickle-cell mice were more resistant to malaria than normal mice. The high prevalence of malaria in Africa may explain why many people there also have the sickle-cell trait. Researchers have long suspected that the genetic variation associated with sickle-cell trait provides some protection against malaria, making individuals with this trait more resilient to infection by the malaria parasite. But later it became apparent that the molecular machinery for clearing up the mess is already in place when the human or animal with the sickle-cell trait is born since they have to break down and detoxify misshaped red blood cells their entire lives.

This study aims to investigate the differential impact of malaria parasites on various genetic structures, specifically comparing the effects on homozygous genes (AA, SS) and heterozygous gene (AS), to elucidate the potential protective role of the sickle cell trait against malaria.

## MATERIALS AND METHODS

### Model Formulation

When modeling diseases using ordinary differential equations, assuming vertical transmission is vital, where recovered individuals become susceptible again, allowing for potential re-infection, as seen in malaria and other diseases (Akinwande, 2018). The population is categorized into main subgroups, including the susceptible homozygous ($S_1$), the infected homozygous ($I_1$), the susceptible heterozygous ($S_2$), the infected heterozygous ($I_2$), the susceptible homozygous ($S_3$), the infected homozygous ($I_3$), the recovered class (R), the carrier mosquito ($M_2$), and the non-carrier mosquito ($M_1$). People enter the susceptible classes through birth and re-infection from the recovery class and leave the class through infection and natural death. The infected classes are populated from the susceptible classes and leave the class through recovery, natural death, and death due to infection. It was assumed in this work that infants are not infected with malaria from birth and that the mosquitoes are non-plasmodium carriers from birth, becoming infected through contact with infected humans. The chances of re-infection are also considered in the model. The transmission dynamics of malaria, as it affects humans based on their genetic structure, is given by the schematic diagram and equations 1-8 below.

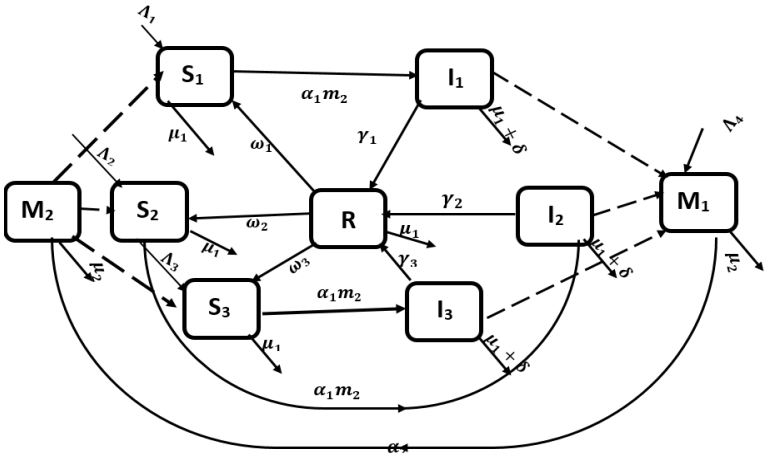


Figure 1: Schematic Diagram of the model

$$\frac{dS_1}{dt} = \Lambda_1 + \rho_1 R - \alpha_1 S_1 M_2 - \mu_1 S_1 \quad (1)$$

$$\frac{dI_1}{dt} = \alpha_1 S_1 M_2 - \gamma_1 I_1 - (\mu_1 + \delta) I_1 \quad (2)$$

$$\frac{dS_2}{dt} = \Lambda_2 + \rho_2 R - \alpha_1 S_2 M_2 - \mu_1 S_2 \quad (3)$$

$$\frac{dI_2}{dt} = \alpha_1 S_2 M_2 - \gamma_2 I_2 - (\mu_1 + \delta) I_2 \quad (4)$$

$$\frac{dS_3}{dt} = \Lambda_3 + \rho_3 R - \alpha_1 S_3 M_2 - \mu_1 S_3 \quad (5)$$

$$\frac{dI_3}{dt} = \alpha_1 S_3 M_2 - \gamma_3 I_3 - (\mu_1 + \delta) I_3 \quad (6)$$

$$\frac{dR}{dt} = \gamma_1 I_1 + \gamma_2 I_2 + \gamma_3 I_3 - (\rho_1 + \rho_2 + \rho_3 + \mu_1) R \quad (7)$$

$$\frac{dM_1}{dt} = \Lambda_4 - \alpha_2 M_1 (I_1 + I_2 + I_3) - \mu_2 M_1 \quad (8)$$

$$\frac{dM_2}{dt} = \alpha_2 M_1 (I_1 + I_2 + I_3) - \mu_2 M_2 \quad (9)$$

**Table 1: Definition of variables and parameter**

| Variables | Description | Parameters | Description |
|---|---|---|---|
| $S_1$ | Susceptible AA individual | $\gamma_1$ | Recovery rate in AA |
| $S_2$ | Susceptible AS individual | $\gamma_2$ | Recovery rate in AS |
| $S_3$ | Susceptible SS individual | $\gamma_3$ | Recovery rate in SS |
| $I_1$ | Infected AA individual | $\rho_1$ | Reinfection rate in AA |
| $I_2$ | Infected AS individual | $\rho_2$ | Reinfection rate in AS |
| $I_3$ | Infected SS individual | $\rho_3$ | Reinfection rate in SS |
| $R$ | Number of recovered humans | $\delta$ | Death due to infection |
| $M_1$ | Number of non-plasmodium carrier mosquitoes | $\mu_1$ | Typical death rate in humans |
| $M_2$ | Number of plasmodium carrier mosquitoes | $\mu_2$ | Typical and induced death rates in mosquitoes |
| $\Lambda_1$ | Typical birth rate in humans (AA) | $\Lambda_2$ | Typical birth rate in humans (AS) |
| $\Lambda_3$ | Typical birth rate in humans (SS) | $\Lambda_4$ | Typical birth rate in mosquitoes. |

**Equilibrium State Existence in the Model**

At equilibrium,

$$\frac{dS_1}{dt}=\frac{dI_1}{dt}=\frac{dS_2}{dt}=\frac{dI_2}{dt}=\frac{dS_3}{dt}=\frac{dI_3}{dt}=\frac{dR}{dt}=\frac{dM_1}{dt}=\frac{dM_2}{dt}=0 \quad (10)$$

Consider arbitrary equilibrium points

$$(S_1,S_2,S_3,I_1,I_2,I_3,R,M_1,M_2\quad)= (S_1^0,S_2^0,S_3^0,I_1^0,I_2^0,I_3^0,R,M_1^0,M_2^0\quad) \quad (11)$$

The system equations 1 – 8 transform into

$$\Lambda_1+\rho_1R-\alpha_1S_1M_2-\mu_1S_1=0 \quad (12)$$
$$\alpha_1S_1M_2-(\gamma_1+\mu_1+\delta)I_1=0 \quad (13)$$
$$\Lambda_2+\rho_2R-\alpha_1S_2M_2-\mu_1S_2=0 \quad (14)$$
$$\alpha_1S_2M_2-(\gamma_2+\mu_1+\delta)I_2=0 \quad (15)$$
$$\Lambda_3+\rho_3R-\alpha_1S_3M_2-\mu_1S_3=0 \quad (16)$$
$$\alpha_1S_3M_2-(\gamma_3+\mu_1+\delta)I_3=0 \quad (17)$$
$$\gamma_1I_1+\gamma_2I_2+\gamma_3I_3-(\rho_1+\rho_2+\rho_3+\mu_1)R=0 \quad (18)$$
$$\Lambda_4-\alpha_2M_1(I_1+I_2+I_3)-\mu_2M_1=0 \quad (19)$$
$$\alpha_2M_1(I_1+I_2+I_3)-\mu_2M_2=0 \quad (20)$$

Let,

$$k_1=\gamma_1+\mu_1+\delta;k_2=\gamma_2+\mu_2+\delta;k_3=\gamma_2+\mu_2+\delta;k_4=\rho_1+\rho_2+\rho_3+\mu_1 \quad (21)$$
$$\Lambda_1+\rho_1R-\alpha_1S_1M_2-\mu_1S_1=0 \quad (22)$$
$$\alpha_1S_1M_2-k_1I_1=0 \quad (23)$$
$$\Lambda_2+\rho_2R-\alpha_1S_2M_2-\mu_1S_2=0 \quad (24)$$
$$\alpha_1S_2M_2-k_2I_2=0 \quad (25)$$
$$\Lambda_3+\rho_3R-\alpha_1S_3M_2-\mu_1S_3=0 \quad (26)$$
$$\alpha_1S_3M_2-k_3I_3=0 \quad (27)$$
$$\gamma_1I_1+\gamma_2I_2+\gamma_3I_3-k_4R=0 \quad (28)$$
$$\Lambda_4-\alpha_2M_1(I_1+I_2+I_3)-\mu_2M_1=0 \quad (29)$$
$$\alpha_2M_1(I_1+I_2+I_3)-\mu_2M_2=0 \quad (30)$$

At Disease Free State the infected classes are assumed to be zero $I_1=I_2=I_3=0$

Substituting this in (28) and (30) gives,

$$M_2=0,R=0; \quad (31)$$

Substituting (31) into (22), (24,) and (26) gives

$$S_1=\frac{\Lambda_1}{\mu_1};S_2=\frac{\Lambda_2}{\mu_1};S_3=\frac{\Lambda_3}{\mu_1} \quad (32)$$

Thus, the DFE($E^0$) exists at the points

$$\in_0=(S_1,S_2,S_3,I_1,I_2,I_3,M_1,M_2\quad)= \left(\frac{\Lambda_1}{\mu_1},\frac{\Lambda_2}{\mu_1},\frac{\Lambda_3}{\mu_1},0,0,0,\frac{\Lambda_4}{\mu_2},0\right) \quad (33)$$

**Computation of Basic Reproduction Number ($R_0$)**

Following the approach of (Somma *et al.*, 2017), the basic reproduction number of the model was calculated using next-generation matrix operations, building on the work of (Diekmann *et al.*, 2000) and improvements made by (Van den Driessche & Watmough, 2002). The effective basic reproduction number is determined by the largest eigenvalue or spectral radius of ($FV^{-1}$). Specifically, the basic reproduction number is given by the largest eigenvalue or spectral radius of $FV^{-1}$.

$$FV^{-1}=\left\{\left[\frac{\partial F_i(E^0)}{\partial x_i}\right]\left[\frac{\partial V_i}{\partial x_i}\right]^{-1}\right\} \quad (34)$$

Where $F_i$ is the rate of appearance of new infection in compartment $i$, $V_i$ is the transfer of infections from one compartment $i$ to another and $E^0$is the Disease-Free Equilibrium.

$$f_i=\begin{pmatrix}\alpha_1S_1^0M_2^0\\ \alpha_1S_2^0M_2^0\\ \alpha_1S_3^0M_2^0\\ \alpha_2M_1^0(I_1^0+I_2^0+I_3^0)\end{pmatrix};v_i=\begin{bmatrix}k_1&0&0&0\\0&k_2&0&0\\0&0&k_3&0\\0&0&0&\mu_2\end{bmatrix} \quad (35)$$

$$FV^{-1}=\begin{bmatrix}0&0&0&\frac{S_1\alpha_1}{\mu_2}\\0&0&0&\frac{S_2\alpha_1}{\mu_2}\\0&0&0&\frac{S_3\alpha_1}{\mu_2}\\\frac{M_1\alpha_2}{k_1}&\frac{M_1\alpha_2}{k_2}&\frac{M_1\alpha_2}{k_3}&0\end{bmatrix} \quad (36)$$

The characteristics polynomial of (36), gives

$$|FV^{-1}-\lambda I|=0 \quad (37)$$

$$|FV^{-1}-\lambda|=\begin{vmatrix}-\lambda&0&0&\frac{S_1\alpha_1}{\mu_2}\\0&-\lambda&0&\frac{S_2\alpha_1}{\mu_2}\\0&0&-\lambda&\frac{S_3\alpha_1}{\mu_2}\\\frac{M_1\alpha_2}{k_1}&\frac{M_1\alpha_2}{k_2}&\frac{M_1\alpha_2}{k_3}&-\lambda\end{vmatrix}= \begin{vmatrix}-\lambda&0&0&\frac{\Lambda_1\alpha_1}{\mu_1\mu_2}\\0&-\lambda&0&\frac{\Lambda_2\alpha_1}{\mu_1\mu_2}\\0&0&-\lambda&\frac{\Lambda_3\alpha_1}{\mu_1\mu_2}\\\frac{\Lambda_4\alpha_2}{\mu_2k_1}&\frac{\Lambda_4\alpha_2}{\mu_2k_2}&\frac{\Lambda_4\alpha_2}{\mu_2k_3}&-\lambda\end{vmatrix}=0 \quad (38)$$

$$-\lambda\left[-\lambda(\lambda^2-\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2})\right]-0+\frac{\alpha_1\Lambda_1}{k_1\mu_1\mu_2}(0)=0 \quad (39)$$

$$\lambda^2-\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2}=0 \quad (40)$$

$$\lambda=\pm\sqrt{\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2}} \quad (41)$$

$$\lambda_1=0,\lambda_2=-\sqrt{\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2}},\lambda_3=+\sqrt{\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2}} \quad (42)$$

$\lambda_3$is the spectral radius of $\rho(FV^{-1})$

$$R_0=\sqrt{\frac{\alpha_1\alpha_2\Lambda_3\Lambda_4}{k_3\mu_1\mu_2^2}} \quad (43)$$

**Stability analysis of Disease-Free equilibrium(DFE)**

$$\begin{pmatrix}-\alpha_1M_2-\mu_1&0&0&0&0&0&\rho_1&0&-\alpha_1S_1\\\alpha_1M_2&-k_1&0&0&0&0&0&0&\alpha_1S_1\\0&0&-(\alpha_1M_2+\mu_1)&0&0&0&\rho_2&0&-\alpha_1S_2\\0&0&\alpha_1M_2&-k_2&0&0&0&0&\alpha_1S_2\\0&0&0&0&-(\alpha_1M_2+\mu_1)&0&\rho_3&0&-\alpha_1S_3\\0&0&0&0&\alpha_1M_2&k_3&0&0&\alpha_1S_3\\0&\gamma_1&0&\gamma_2&0&\gamma_3&k_4&0&0\\0&\alpha_2M_1&0&\alpha_2M_1&0&\alpha_2M_1&0&\alpha_2(I_1+I_2+I_3)-\mu_2&0\\0&\alpha_2M_1&0&\alpha_2M_1&0&\alpha_2M_1&0&\alpha_2(I_1+I_2+I_3)&\mu_2\end{pmatrix} \quad 44$$

Using the characteristic equation $\left|J_{\varepsilon_0}-\lambda I\right|=0$

$$\begin{pmatrix} -\alpha_1 M_2 - \mu_1 - \lambda & 0 & 0 & 0 & 0 & 0 & \rho_1 & 0 & -\alpha_1 S_1 \\ \alpha_1 M_2 & -k_1 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_1 S_1 \\ 0 & 0 & -(\alpha_1 M_2 + \mu_1) - \lambda & 0 & 0 & 0 & \rho_2 & 0 & -\alpha_1 S_2 \\ 0 & 0 & \alpha_1 M_2 & -k_2 - \lambda & 0 & 0 & 0 & 0 & \alpha_1 S_2 \\ 0 & 0 & 0 & 0 & -(\alpha_1 M_2 + \mu_1) - \lambda & 0 & \rho_3 & 0 & -\alpha_1 S_3 \\ 0 & 0 & 0 & 0 & \alpha_1 M_2 & k_3 - \lambda & 0 & 0 & \alpha_1 S_3 \\ 0 & \gamma_1 & 0 & \gamma_2 & 0 & \gamma_3 & k_4 - \lambda & 0 & 0 \\ 0 & \alpha_2 M_1 & 0 & \alpha_2 M_1 & 0 & \alpha_2 M_1 & 0 & \alpha_2(I_1 + I_2 + I_3) - \mu_2 - \lambda & 0 \\ 0 & \alpha_2 M_1 & 0 & \alpha_2 M_1 & 0 & \alpha_2 M_1 & 0 & \alpha_2(I_1 + I_2 + I_3) & \mu_2 - \lambda \end{pmatrix} = 0 \quad (45)$$

At DFE $\left(S_1 = \frac{\Lambda_1}{\mu_1}, S_2 = \frac{\Lambda_2}{\mu_1}, S_3 = \frac{\Lambda_3}{\mu_1}, M_1 = \frac{\Lambda_4}{\mu_2},\right)$

$$\begin{pmatrix} -\mu_1 - \lambda & 0 & 0 & 0 & 0 & 0 & \rho_1 & 0 & \frac{-\alpha_1 \Lambda_1}{\mu_1} \\ 0 & -k_1 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\alpha_1 \Lambda_1}{\mu_1} \\ 0 & 0 & -\mu_1 - \lambda & 0 & 0 & 0 & \rho_2 & 0 & \frac{-\alpha_1 \Lambda_2}{\mu_1} \\ 0 & 0 & 0 & -k_2 - \lambda & 0 & 0 & 0 & 0 & \frac{\alpha_1 \Lambda_2}{\mu_1} \\ 0 & 0 & 0 & 0 & -\mu_1 - \lambda & 0 & \rho_3 & 0 & \frac{-\alpha_1 \Lambda_3}{\mu_1} \\ 0 & 0 & 0 & 0 & 0 & k_3 - \lambda & 0 & 0 & \frac{\alpha_1 \Lambda_3}{\mu_1} \\ 0 & \gamma_1 & 0 & \gamma_2 & 0 & \gamma_3 & k_4 - \lambda & 0 & 0 \\ 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & -\mu_2 - \lambda & 0 \\ 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & 0 & \mu_2 - \lambda \end{pmatrix} = 0 \quad (46)$$

Clearly four eigenvalues are clearly negative, these are

$$\left(-\mu_1 - \lambda\right)^2 = 0; \left(-\mu_2 - \lambda\right)^2 = 0 \quad (47)$$

$$\begin{pmatrix} -k_1 - \lambda & 0 & 0 & 0 & \frac{\alpha_1 \Lambda_1}{\mu_1} \\ 0 & -k_2 - \lambda & 0 & 0 & \frac{\alpha_1 \Lambda_2}{\mu_1} \\ 0 & 0 & k_3 - \lambda & 0 & \frac{\alpha_1 \Lambda_3}{\mu_1} \\ \gamma_1 & \gamma_2 & \gamma_3 & k_4 - \lambda & 0 \\ \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & -\mu_2 - \lambda \end{pmatrix} = 0 \quad (48)$$

$$-k_1 - \lambda \begin{vmatrix} -k_2 - \lambda & 0 & 0 & \frac{\alpha_1 \Lambda_2}{\mu_1} \\ 0 & -k_3 - \lambda & 0 & \frac{\alpha_1 \Lambda_3}{\mu_1} \\ \gamma_2 & \gamma_3 & -k_4 - \lambda & 0 \\ \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & -\mu_2 - \lambda \end{vmatrix} - \frac{\alpha_1 \Lambda_1}{\mu_1} \begin{vmatrix} 0 & -k_2 - \lambda & 0 & 0 \\ 0 & 0 & k_3 - \lambda & 0 \\ \gamma_2 & \gamma_3 & 0 & -k_4 - \lambda \\ \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 \end{vmatrix} = 0 \quad (49)$$

$$(-k_1 - \lambda)(-k_2 - \lambda) \begin{vmatrix} -k_3 - \lambda & 0 & \frac{\alpha_1 \Lambda_3}{\mu_1} \\ \gamma_3 & -k_4 - \lambda & 0 \\ \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 & -\mu_2 - \lambda \end{vmatrix} - \frac{\alpha_1 \Lambda_1}{\mu_1}(0) \begin{vmatrix} 0 & k_3 - \lambda & 0 \\ \gamma_3 & 0 & -k_4 - \lambda \\ \frac{\alpha_2 \Lambda_4}{\mu_2} & \frac{\alpha_2 \Lambda_4}{\mu_2} & 0 \end{vmatrix} = 0 \quad (50)$$

$$(-k_1 - \lambda)(-k_2 - \lambda)(-k_3 - \lambda)(-k_4 - \lambda)(-\mu_2 - \lambda) + (-k_1 - \lambda)(-k_2 - \lambda)(-k_4 - \lambda)\left(\frac{\alpha_1 \Lambda_3}{\mu_1}\right)\left(-\frac{\alpha_2 \Lambda_4}{\mu_2}\right) = 0 \quad (51)$$

$$(-k_1 - \lambda)(-k_2 - \lambda)(-k_3 - \lambda)(k_4\mu_2 + k_4\lambda + \mu_2\lambda + \lambda^2) + (-k_1 - \lambda)(-k_2 - \lambda)\left(\frac{\alpha_1 \Lambda_3 \alpha_2 \Lambda_4 k_4}{\mu_1 \mu_2}\right) + \left(\frac{\alpha_1 \Lambda_3 \alpha_2 \Lambda_4 \lambda}{\mu_1 \mu_2}\right) \quad (52)$$

$$(-k_1 - \lambda)(-k_2 - \lambda)\left[(-k_4 k_3 \mu_2 - k_4 k_3 \lambda - \mu_2 k_3 \lambda - k_3 \lambda^2 - k_4 \mu_2 \lambda - k_4 \lambda^2 - \mu_2 \lambda^2 - \lambda^3) + \left(\frac{\alpha_1 \Lambda_3 \alpha_2 \Lambda_4 k_4}{\mu_1 \mu_2}\right) + \left(\frac{\alpha_1 \Lambda_3 \alpha_2 \Lambda_4 \lambda}{\mu_1 \mu_2}\right)\right] \quad (53)$$

$$(\lambda^2 + a\lambda + b)(\lambda^3 + c\lambda^2 + d\lambda + e) = 0 \quad (54)$$

Where

$$a = k_1 + k_2; b = k_1 k_2; c = k_3 k_4 \mu_2; d = k_3 \mu_2 - k_3 k_4 + k_4 \mu_2 - \frac{\alpha_1 \alpha_2 \Lambda_3 \Lambda_4}{\mu_1 \mu_2}; e = k_3 k_4 \mu_2 - \frac{\alpha_1 \alpha_2 \Lambda_3 \Lambda_4 k_4}{\mu_1 \mu_2} \quad (55)$$

$$\lambda^5 + (a + c)\lambda^4 + (b + ac + d)\lambda^3 + (bc + ad + e)\lambda^2 + (bd + ae)\lambda + be = 0 \quad (56)$$

$$k_1 k_2 k_3 k_4 \mu_2 - \frac{\alpha_1 \alpha_2 \Lambda_3 \Lambda_4 k_1 k_2 k_4}{\mu_1 \mu_2} \quad (57)$$

Divide both sides by $k_1 k_2 k_3 k_4 \mu_2$, we have

$$1 - \frac{\alpha_1 \alpha_2 \Lambda_3 \Lambda_4 k_4}{\mu_1 \mu_2^2} \quad (58)$$

$$1 - (R_0)^2 \quad (59)$$

Hence, $(R_0)$ is globally asymptotically stable at DFE.

## RESULTS AND DISCUSSION

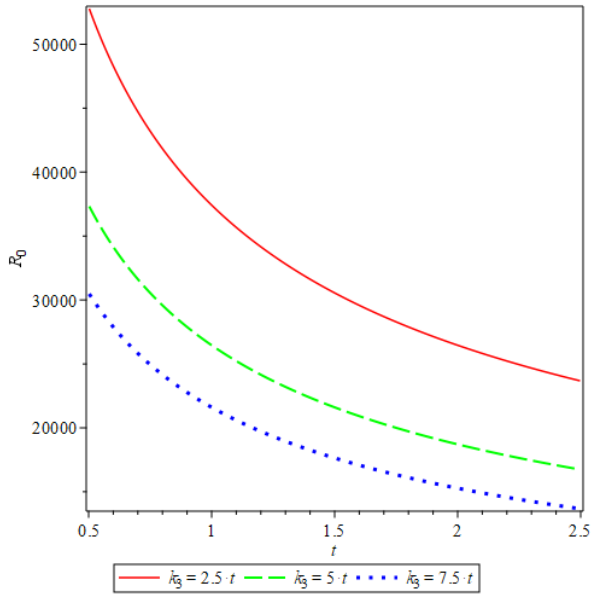


Figure 2: Graph of Ro against time varying $k_3$

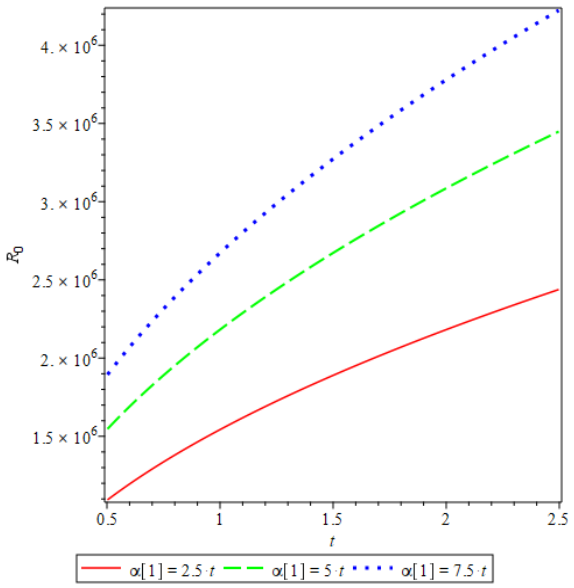


Figure 3: Graph of $R_0$ against time varying $\alpha_1$

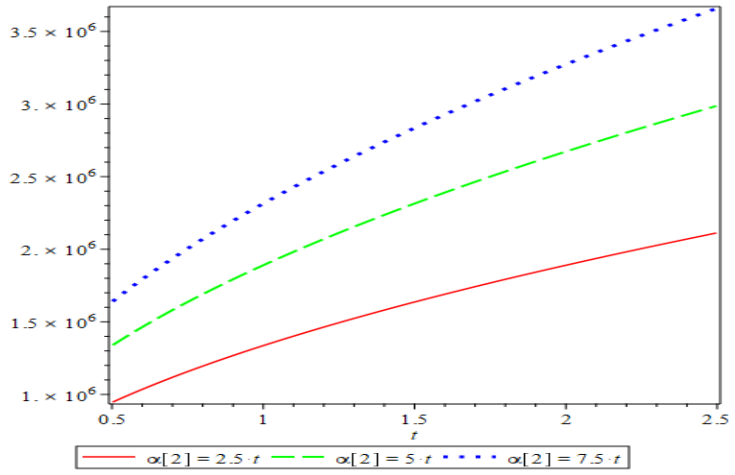


Figure 4: Graph of $R_0$ against time varying $\alpha_2$

The figure 2 represents the relationship between the reproduction number and the recovery of individual in the sickle cell compartment from malaria. The reproduction number increases with time as the rate of recovery in this compartment increases. This indicates that the recovery from malaria in SS patients leads to a decrease in infected individuals over time.

Figure 3 represents the changes in reproduction number with time, varying the infection rate in humans. The graph indicates that a reduction in the infection rate of malaria in the human population will bring the spread of the disease under control.

Figure 4 shows the relationship between the reproduction number and time while varying the contact rate between mosquitoes and humans. It was shown that for all the compartments reducing the contact of mosquitoes with humans is a sure way to bring the disease under control.

## CONCLUSION

This study presents a mathematical model that investigates the effects of malaria on humans based on genetic structure. The model's stability analysis reveals that the disease-free equilibrium is globally asymptotically stable, indicating that the disease can be controlled and eventually eliminated if the basic reproduction number is reduced below unity. The graphical representations of the model's dynamics provide valuable insights into the interactions among the compartments and the impact of genetic structure on malaria transmission. The protective effect of the sickle cell trait against malaria is inherent to the trait itself, rather than relying on standard malaria recovery rates. To effectively control malaria transmission, it's crucial to minimize infection rates ($\alpha_1$) and contact rates ($\alpha_2$) between mosquitoes and humans, regardless of sickle cell status. The findings of this study have significant implications for the development of effective control strategies against malaria. Overall, this research contributes to the understanding of malaria dynamics and provides a framework for further studies on the genetic aspects of malaria transmission.

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