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Effect of Environmental Immunity on Mathematical Modeling of Malaria Transmission between Vector and Host Population

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ABSTRACT: The effect of environmental immunity on the mathematical modeling of malaria transmission between vector and host population is investigated in this study using appropriate standard procedures. We develop a mathematical SIR-SI model incorporating environmental immunity parameters to describe the dynamics transmission rates of both humans and vectors with the assumption that an individual develops environmental immunity on the infected and recovered classes. The model is analyzed by the reproduction number derived using the next-generation matrix method and its stability is checked by Jacobian matrix. We demonstrate that the disease-free equilibrium is locally asymptotically stable if $R_o < 1$ (R_o – reproduction number) and is unstable if $R_o > 1$. Numerical simulation indicates that, with acquired environmental immunity due to nutrition and medicinal herbs, the spread of malaria can be significantly impacted by increasing the *recovered* class and lowering the infected class.

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Malaria transmission rates have been investigated by numerous researchers to study the exchange of the disease between host and vector populations. Malaria is endemic to many countries in the tropics, including some parts of Latin America, Africa, and Asia. It is one of the leading causes of death globally, especially among children of 5 years and under. In 2021, it was reported that an estimated 247 million cases of malaria accounted for about 619,000 deaths worldwide. Ninety-five percent of these cases occurred in Africa. Children under 5 accounted for about 80 percent of these deaths WHO (2023). A parasite known as Plasmodium is responsible for causing Malaria, which can infect human beings and female anopheles' mosquitoes alike Kipkirui *et al*., (2020). There are four primary varieties of Plasmodium: Plasmodium falciparum, Plasmodium malaria, Plasmodium oval, and Plasmodium vivax. Plasmodium falciparum is the most prevalent type and is responsible for most fatalities in Africa and Southeast Asia Mandal *et al*., (2011). The parasite enters the bloodstream when a female mosquito bites a human and can be picked up by any mosquito, whether infected or not. The parasites then invade the liver and blood cells, going through multiple developmental stages. Immunity to the parasites can be temporary with continuous exposure, while acquired immunity is developed as the immune system fights against the protozoa. However, the Plasmodium parasite uses techniques to evade the immune system that causes damage to the liver and blood cells. These techniques begin when the parasites enter the body through the skin and enable them to

survive and multiply in the liver. Due to the ability of many parasites to be destroyed in the skin, only a few manage to reach the liver where they can cause destruction using their ability to evade the immune system Aguas *et al.*, (2008). Over time, the protozoan has developed the ability to mutate using various biological markers, making it challenging for the immune system and some malaria-resistant drugs to be effective. Various measures were implemented to control malaria before the introduction of the RTS, S vaccine, such as sanitation policies, the use of treated mosquito nets, and antimalarial drugs Keegan and Dushoff (2013), Wan and Cui (2009). Despite various measures taken, eradicating malaria has proven to be a challenging task due to the issue of immunity. Mathematical models have been used to study immunity in recent research on infectious diseases. This particular study focuses on the impact of environmental immunity on the spread of malaria. Malaria is a serious global health concern that spreads through mosquito bites, with plasmodium parasites invading hepatocytes and erythrocytes in the mammalian host Liehl *et al.*, (2015). The immune system of hosts reacts complexly to several life stages of a parasite called gametocytes, triggering immune responses that vary widely based on the host's age, genetics, and exposure history. Numerous studies have been conducted on how the immune system responds to and reacts to both liver and blood-stage infections of malaria. Li *et al.,* (2011) focused on incorporating red blood cells, malaria parasitemia and immune effectors into a mathematical model with nonlinear bounded Michaelis Menten-Monod functions describing how immune cells interact with infected red blood cells and merozoites. Li *et al.,* (2011) conducted a separate study to explore how blood and liver stage infection trigger resistance to reinfection. Their research revealed that liver infection activates a specific and highly effective type I interferon (IFN) response, which contributes to the resistance observed in regions of malaria hyperendemicity. It is still uncertain whether acquired immunity to malaria can reduce the duration of infections, as some infections clear up in the first few days or weeks of appearing in the blood while others persist for many months across all age groups. Although exposure-based immunity increases with age, there seems to be no noticeable increase in the rate of infection termination Bretscher *et al.,* (2015). The processes that determine the acquisition of immunity to malaria parasites are poorly understood, in part because of a lack of validated immunological markers of protection. Environmental factors are more significant than genetics in determining a person's immunity, according to scientific studies NIH (2023). Behavior, nutrients, pollutants, weather and climatic factors, and microbes comprise environmental factors which can affect how an individual's immune system develops with time. Some research has explored the biological impact of climate, weather, and pollutants on mosquito populations and malaria transmission Castrol (2017), Endo and Eltatic (2016), Kibret *et al.*, (2019). Additionally, some mathematical models describe immunity in general. The goal of this study is to create a mathematical model that considers the impact of environmental immunity due to nutrients, supplements, and herbs. Unfortunately, this topic has not been widely discussed. This study specifically focuses on the impact of nutrition, supplements, and herbs consumed by susceptible individuals on their immune systems. Consuming healthy organic diets, regulated medicinal herbs, and supplements that contain active ingredients that boost the immune system can result in an extended partial immunity duration. Conversely, a poor diet or one containing toxin will shorten the duration of partial immunity.

The Model: The SIR-SI model has been developed as an improved version of Ross's SIS model, which was used for studying disease spread. In this model, the relationship between the human-host compartment and the vector (mosquito) compartments has been incorporated. There are three classes for humans, namely susceptible, infectious, and recovery, while vectors have two classes, susceptible and infectious. Unlike the previous model, individuals in this model do not become susceptible again as they develop immunity.

Model Formulation: The model employed is the SIR-SI model that consists of five compartments named S_h, I_h, R_h, S_v and I_v. It is adapted from the model of Gebremeskel and Krogstad (2015) and Ochomba (2013). The environmental immunity parameter was, however, incorporated into the infectious and recovered class to see its effect on the basic reproduction number and the equilibrium points.

Model Diagram: *The model comprises of susceptible humans S_h, infected humans I_h, recovered humans R_h, susceptible vectors S_v, and infected vectors I_v each with their unique parameters* that can vary. Unlike other infectious diseases that provide permanent immunity, the recovered class will eventually lose their immunity due to exposure, nutrition, and supplement intake. This loss of immunity is represented by the immunity parameter $m_h I_h$. Figure 1 illustrates the relationship between the host and vector. At time t, the total human and vector population is

$$N_h = S_h + I_h + R_h \quad (1)$$
$$N_v = S_v + I_v$$

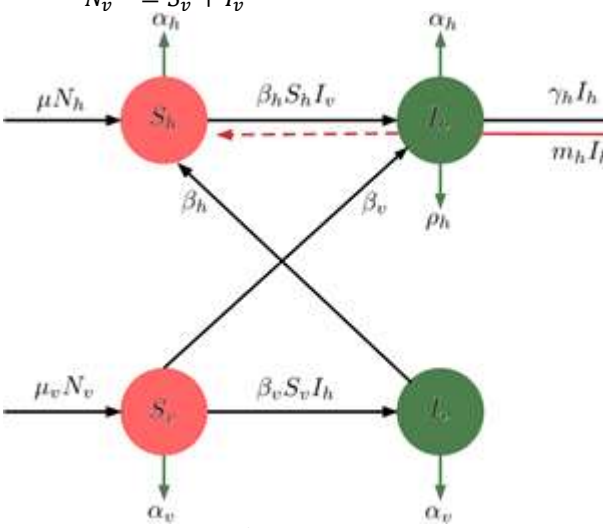

Fig. 1. Diagram of the Extended SIR-SI Model

Model Equation: The rate of change in each compartment expressed in the non-linear ordinary differential equation is given as this:

$$\begin{aligned}
\frac{dS_h}{dt} &= \mu_h N_h - \beta_h S_h I_v - \alpha_h S_h + \tau_h I_h \\
\frac{dI_h}{dt} &= \beta_h S_h I_v - \alpha_h I_h - \gamma_h I_h - \rho_h I_h - m_h I_h - \tau_h I_h \\
\frac{dR_h}{dt} &= \gamma_h I_h - \alpha_h R_h + m_h I_h \\
\frac{dS_v}{dt} &= \mu_v N_v - \beta_v S_v I_h - \alpha_v S_v \\
\frac{dI_v}{dt} &= \beta_v S_v I_h - \alpha_v I_v
\end{aligned} \quad (2)$$

The Variables and Parameters in equation 2 are

S_h = Susceptible - number of healthy humans who are susceptible to the disease; I_h = Infected - number of infected humans; R_h = Recovered - number of recovered humans; S_v = number of susceptible mosquitoes; I_v = number of infected mosquitoes; $N_h(t)$ = Total human population at time t; $N_v(t)$ = Total vector (mosquito) population at t; μ_h = birth rate of humans; μ_v = birth rate of vectors; α_h=death rate of humans; α_v= death rate of vectors; β_h= contact/transmission rate of humans; β_v=contact/transmission rate of vectors; ρ_h= disease induced death rate; γ_h= recovery rate for humans; τ_h= rate at which infected humans enter the susceptible class; m_h= environmental immunity due to nutrition and supplement.

Assumptions: Humans and vector population are assumed to be constant. All newborns and immigrants are susceptible to infection. Population of both human and vectors are non-negative Malaria develops when infected female bites humans. Humans move from one compartment to another with respect to the disease as it evolves. Humans enter S through birth rate and migration. Humans leave S through death rate, both natural and disease induced. All mosquitoes are subject to death rate, both natural and induced. Recovered humans can reenter susceptible compartment. Recovered individuals in human population develop immunity. Recovered humans can also enter the infectious class when their immunity wanes. A person who has recovered from an infection cannot be infected again unless they become susceptible once more. It's important to mention that the model for vectors does not have an immune category since many mosquitoes do not have immunity. It should be emphasized that the model's vector component does not incorporate an immune category, given that the majority of mosquitoes do not survive the infection. Rather, their infectious period concludes with their demise owing to their brief life cycle. The stability analysis and evaluation of the basic reproduction number will be conducted based on the model equations. The outcome of the analysis will demonstrate how the loss of immunity from the recovered and infectious class affects the system.

Disease Free Equilibrium: The steady state, also known as disease-free equilibrium, occurs when there is no infection, meaning that both the exposed and infected classes are at zero. At equilibrium, the model can be normalized by setting the formulated model to zero, that is,

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0$$

Putting $I_h = I_v = 0$, the model equation (2) becomes;

$$\begin{aligned}
\frac{dS_h}{dt} &= \mu_h N_h - \alpha_h S_h \\
\frac{dR_h}{dt} &= -\alpha_h R_h \\
\frac{dS_v}{dt} &= \mu_v N_v - \alpha_v S_v
\end{aligned}$$

Solving for S_h, R_h, and S_v, gives the disease-free equilibrium as

$$D_0 = (S_h, I_h, R_h, S_v, I_v) = [\tfrac{\mu_h N_h}{\alpha_h}, 0, 0, \tfrac{\mu_v N_v}{\alpha_v}, 0]. \quad (3)$$

Endemic Equilibrium: At the endemic equilibrium state, the disease cannot be completely eliminated, but instead, it stays within the population. To maintain the disease within the population, the model requires that the S_h, I_h, R_h, S_v, I_v values are not equal to zero at the equilibrium state. The endemic equilibrium represents

a constant state of the model, where infected humans and vectors indicate the presence of the infection, $Ih = Iv \neq 0$. From the model equation. Solving for S_h, I_h, R_h, S_v, I_v, we have

$$(S_h, I_h, R_h, S_v, I_v) = \left[\frac{\mu_h N_h + \tau_h I_h}{\beta_h I_v + \alpha_h}, \frac{\beta_h s_h I_v}{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}, \frac{\gamma_h I_h + m_h I_h}{\alpha_h}, \frac{\mu_v N_v}{\beta_v I_h - \alpha_v}, \frac{\beta_v S_v I_h}{\alpha_v}\right] \quad (4)$$

Basic Reproduction Number $\boldsymbol{R_0}$: This is the expected number of secondary cases per primary area in a given population, and it is found using the next generation matrix. $R_0 = \rho(FV^{-1})$, R_0 = dominant eigenvalue of FV^{-1} F = defines terms which contains secondary infections. $V =$ contains terms that do not include secondary infections. $\rho =$ spectral radius. From the disease class $\frac{dI_h}{dt}$ and $\frac{dI_v}{dt}$, find F and V. F and V are the matrices of the partial derivative of the disease classes with respect to the dependent variables. The next generation matrix is found as follows: First, find the product of F and the inverse of V, i.e., FV^{-1}. Next, find the eigenvalues of FV^{-1} and select the dominant one which yields the R_0. From the disease classes with respect to secondary infections β_h and β_v, and non-secondary infections, the Jacobian for F and V is computed.

$$\frac{dI_h}{dt} = \beta_h S_h I_v - \alpha_h I_h - \gamma_h I_h - \rho_h I_h - m_h I_h - \tau_h I_h = F_1$$

$$\frac{dI_v}{dt} = \beta_v S_v I_h - \alpha_v I_v = F_2$$

Let

$$F = \begin{pmatrix} \frac{\partial F_1}{\partial I_h} & \frac{\partial F_1}{\partial I_v} \\ \frac{\partial F_2}{\partial I_h} & \frac{\partial F_2}{\partial I_v} \end{pmatrix}$$

and

$$V = \begin{pmatrix} \frac{\partial F_1}{\partial I_h} & \frac{\partial F_1}{\partial I_v} \\ \frac{\partial F_2}{\partial I_h} & \frac{\partial F_2}{\partial I_v} \end{pmatrix}$$

Thus,

$$F = \begin{bmatrix} 0 & \beta_h S_h \\ \beta_v S_v & 0 \end{bmatrix}.$$

Similarly,

$$V = \begin{bmatrix} -(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h) & 0 \\ 0 & -\alpha_v \end{bmatrix}$$

So that

$$V^{-1} = \begin{bmatrix} \frac{-\alpha_v}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} & 0 \\ 0 & \frac{-(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} \end{bmatrix}$$

It follows that

$$FV^{-1} = \begin{bmatrix} 0 & \beta_h S_h \frac{-(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} \\ \beta_v S_v \frac{-\alpha_v}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} & 0 \end{bmatrix}$$

The dominant Eigenvalue or reproductive ratio is

$$|FV^{-1} - \lambda I| = 0$$

$$\begin{vmatrix} 0 - \lambda & \beta_h S_h \frac{-(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} \\ \beta_v S_v \frac{-\alpha_v}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)} & 0 - \lambda \end{vmatrix} = 0$$

$$\lambda^2 - \left[\frac{-\beta_v S_v}{(\alpha_v)(\alpha_h + \gamma_h + \rho_h I_h + m_h + \tau_h I_h)}\right]\left[\frac{-\beta_h S_h}{(\alpha_v}\right] = 0$$

$$\lambda = \sqrt{\left(\frac{\beta_v S_v (\alpha_v)}{(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}\right)\left(\frac{\beta_h S_h}{\alpha_v}\right)}$$

Therefore,

$$R_0 = \sqrt{\left(\frac{\beta_v S_v}{(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}\right)\left(\frac{\beta_h S_h}{\alpha_v}\right)} \quad (5)$$

Where S_v and S_h are at disease free equilibrium D_0 in (3).

Stability Analysis of the Disease-Free Equilibrium

Theorem 1. The disease-free equilibrium $D_0 = \left(\frac{\mu_h N_h}{\alpha_h}, 0, 0, \frac{\mu_v N_v}{\alpha_v}, 0\right)$*, exists for all non-negative values of its parameters and it is locally asymptotically stable when* $R_0 \leq 1$ *and it is unstable when* $R_0 > 1$.

The Jacobian matrix of system of equation (2) at disease free equilibrium (2) is given by

$$J = \begin{bmatrix} -\alpha_h & \tau_h & 0 & 0 & -\beta_h S_h \\ 0 & -(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h) & 0 & 0 & \beta_h S_h \\ 0 & \gamma_h + m_h & -\alpha_h & 0 & 0 \\ 0 & -\beta_v S_v & 0 & -\alpha_v & 0 \\ 0 & \beta_v S_v & 0 & 0 & -\alpha_v \end{bmatrix}$$

Now, Solving the eigen values of the Jacobian matrix $|J - \lambda I| = 0$, we have

$$\begin{bmatrix} -\alpha_h - \lambda & \tau_h & 0 & 0 & -\beta_h S_h \\ 0 & -(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h) - \lambda & 0 & 0 & \beta_h S_h \\ 0 & \gamma_h + m_h & -\alpha_h - \lambda & 0 & 0 \\ 0 & -\beta_v S_v & 0 & -\alpha_v - \lambda & 0 \\ 0 & \beta_v S_v & 0 & 0 & -\alpha_v - \lambda \end{bmatrix} = 0$$

$$\begin{aligned} \lambda_1 &= -\alpha_h \\ \lambda_2 &= -\alpha_h \\ \lambda_3 &= -\alpha_v \\ \lambda_4 &= -\frac{1}{2}(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h) \\ &\quad -\frac{1}{2}\left(\sqrt{(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2 - 4(m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)}\right) \\ \lambda_5 &= -\frac{1}{2}(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h) \\ &\quad +\frac{1}{2}\left(\sqrt{(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2 - 4(m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)}\right). \end{aligned} \quad (6)$$

Since λ_5 is the dominant eigenvalue in (6), it is $\lambda_5 < 0$. The dominant eigenvalue is the R_o threshold which determines whether the disease will persist or die out. That is, if $\lambda_5 < 0$, then $R_o < 1$. It follows that

$$-\frac{1}{2}(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)$$
$$+\frac{1}{2}\left(\sqrt{(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2 - 4(m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)}\right) < 0$$
$$\frac{1}{2}\left(\sqrt{(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2 - 4(m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)}\right)$$
$$< \frac{1}{2}(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)$$
$$(m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2 - 4(m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)$$
$$< (m_h + \alpha_h + \alpha_v + \gamma_h + \rho_h + \tau_h)^2.$$

This implies

$$\begin{aligned} m_h\alpha_v + \alpha_h\alpha_v - s_h s_v \beta_h \beta_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h &> 0 \\ -s_h s_v \beta_h \beta_v &> -(m_h\alpha_v + \alpha_h\alpha_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h) \\ \frac{s_h s_v \beta_h \beta_v}{(m_h\alpha_v + \alpha_h\alpha_v + \alpha_v\gamma_h + \alpha_v\rho_h + \alpha_v\tau_h)} &< 1 \quad (7) \end{aligned}$$

Therefore R_o in equation (5) can obtained by squaring both sides of (7) as

$$R_0 = \sqrt{\left(\frac{\beta_v S_v}{(\alpha_h + \gamma_h + \rho_h + m_h + \tau_h I_h)}\right)\left(\frac{\beta_h S_h}{\alpha_v}\right)} < 1, \quad (8)$$

Where S_v and S_h are at disease free equilibrium D_0 in (3).

Clearly, the condition of Theorem 1 has been satisfied which means that all the eigen values of the Jacobian matrix have negative real part. Moreover, since $R_o < 1$, it follows that from (8) that the disease-free equilibrium point is stable.

Numerical Simulations and Results: The evaluation of the model involved a numerical analysis. Through simulations, it was possible to observe the impact of the parameters. The software used for the simulations

was Wolfram Mathematica. The values for the parameters of the SIR-SI were obtained from Dorner and Mosleh (2020).

Table 1: Values of the Parameters for Fig. 2, Fig. 3, Fig. 4, Fig. 5, Fig. 6 and Fig. 7

Parameters	*Values*	*Sources*
$S_h(0)$	100	Dorner and Mosleh (2020)
$I_h(0)$	10	Dorner and Mosleh (2020)
$R_h(0)$	30	Dorner and Mosleh (2020)
$S_v(0)$	30	Dorner and Mosleh (2020)
$I_v(0)$	15	Dorner and Mosleh (2020)
μ_h	0.004	Dorner and Mosleh (2020)
μ_v	0.07	Dorner and Mosleh (2020)
α_h	0.00006	Dorner and Mosleh (2020)
α_v	0.067	Dorner and Mosleh (2020)
β_h	0.2	Dorner and Mosleh (2020)
β_v	0.09	Dorner and Mosleh (2020)
ρ_h	3	Estimated value
γ_h	1.2	Estimated value
τ_h	0.02	Estimated value
m_h	2	Estimated value

In the simulations from Fig. 2 through Fig. 7, the human recovery rate increased and then remain stable for a long time as a result of the environmental immunity developed due to access to organic nutritional diets, medicinal herbs and supplements while the number of infected and susceptible humans reduced drastically.

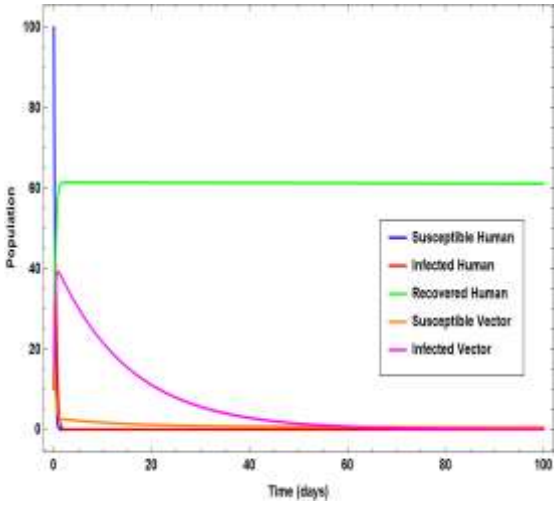

Fig. 2. Recovered human class when m_h=0 which is attributed to the partial immunity acquired due to long exposure to the disease.

The figure 3 shows that the recovered human class when $m_h = 0$ which is attributed to the partial immunity acquired due to long exposure to the disease. The figure 4 showed a significant increase in the recovered class when $m_h > 0$. This shows the effect of environmental immunity on the model. While figure 4 show both partial and environmental immunity decreased the infected class. It is worth noting that with environmental immunity $m_h > 0$, the infected human remained constant at zero level time but partial immunity wanes off with time which might trigger sudden increase in the infected class.

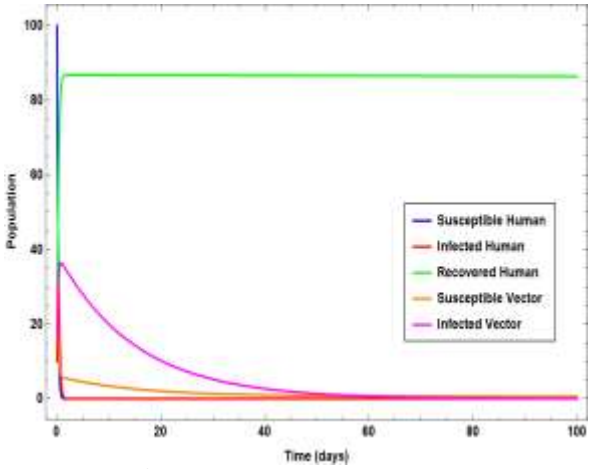

Fig. 3. A significant increase in the recovered class when m_h >0. This shows the effect of environmental immunity on the model.

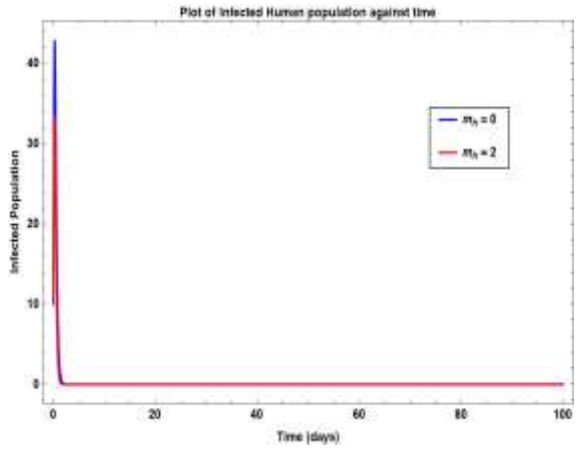

Fig. 4. Both partial and environmental immunity decreased the infected class.

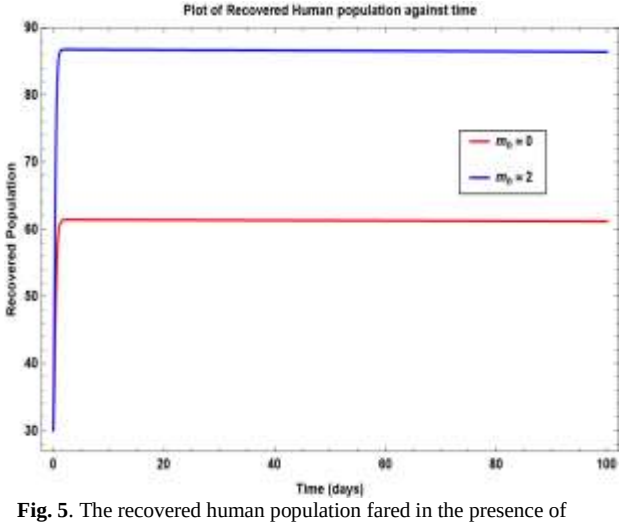

Fig. 5. The recovered human population fared in the presence of environmental immunity

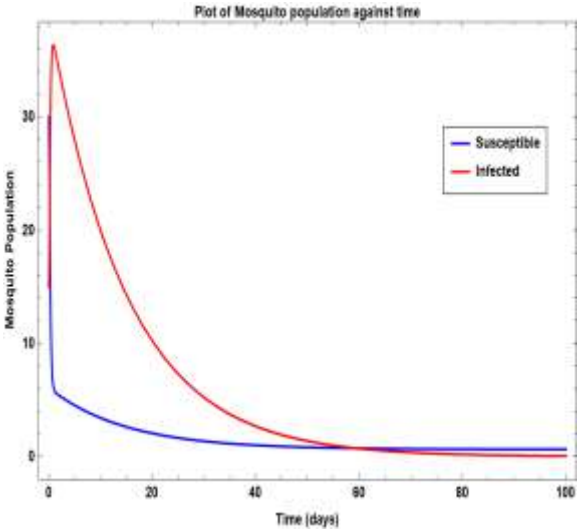

Fig. 6. This graph shows that the mosquitoes do not develop immunity

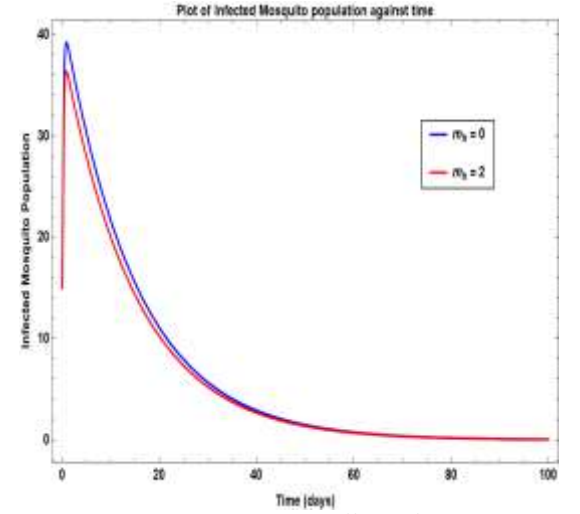

Fig. 7. How environmental immunity affects infected mosquitoes.

The figure 5 specifically looks at how the recovered human population fared in the presence of environmental immunity. That is, when $m_h = 0$ versus when $m_h > 0$. It can be seen that when $m_h > 0$, there was a significant increase in the recovered human population.

Figure 6 shows that that the mosquitoes do not develop immunity, so that even though $m_h > 0$, it does not show any significant difference as to when $m_h = 0$ because it is assumed that mosquitoes do not develop immunity, either partial or environmental. It can be seen that the infected mosquitoes' peaks and then drop. This could be due to death through various means such as vector-controlled measures like insecticides, as they do not develop immunity. It is possible that they can also die from being eaten by other predators.

Figure 7 evaluates how environmental immunity affected mosquitoes. Even though immunity has no impact on the vector population from the model equation and diagram (Fig 7), the graph here shows that when $m_h > 0$, the infected mosquitoes decreased further. This is a result of decrease in infected humans due to environmental immunity. Infected mosquito will also decrease because there are fewer humans capable of infecting uninfected mosquito. It is evident that environmental immunity due to nutrition, supplements and medicinal herbs has a positive effect in boosting the immune system, thus increasing the recovered class and decreasing the infected class.

Conclusion: Controlling malaria involves understanding its transmission with reproduction number as a factor. Numerical simulations showed the effect of different values of the parameters. The result obtained showed that as environmental immunity increases for the recovered class the number of infected humans reduces drastically. And this subsequently reduces the number of infected mosquitoes since they are fewer humans capable of infecting uninfected mosquitoes. The significant impact of this eventually led to $R_o < 1$ resulting in decreased malaria prevalence.

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