doi:10.20944/preprints201809.0214.v2

Article

Mathematical Analysis of Transfusion—Transmitted Malaria Model with Optimal Control

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Abstract: An SIRS (Susceptible–Infected–Removed-Susceptible) mathematical model for the transmission dynamics of the Transfusion–Transmitted Malaria (TTM) model with optimal control pair $u_1(t)$ and $u_2(t)$ was developed and studied in this research work. The model Transfusion–Transmitted Malaria disease–free equilibrium and endemic equilibriums points were determined. The model exhibited two equilibriums; disease-free and endemic equilibrium. It is shown that the disease–free equilibrium was locally asymptotically stable if the associated basic reproduction numbers R_0 is less than unity while the disease persists if R_0 is greater than unity. The global stability of the Transfusion–Transmitted Malaria model at the disease-free equilibrium was established using the comparison method. The optimality system was derived and an optimal control model of blood screening and drug treatment for the Transfusion–Transmitted Malaria model was investigated. Conditions for the optimal control were considered using Pontryagin's Maximum Principle and solved numerically using the Forward and Backward Finite Difference Method (FBDM). Numerical results obtained are in perfect agreement with our analytical results.

Keywords: malaria; transfusion–transmitted; basic reproduction number; stability; equilibrium; optimal control

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1. Introduction

Transfusion–transmitted malaria (TTM) was first documented in 1911 [5]. The global incidence and occurrence of TTM based on available data indicates that over hundred cases are reported annually, mostly restricted to endemic countries [2]. The chances of TTM due to donor blood in Sub Saharan African countries is increased due to malaria prevalence in donor blood sample [8]. In countries where malaria is endemic, differentiating cases of TTM from natural infection still remain a challenge as malaria infection occurrence after transfusion may be as a result of either natural infection (infection through bites from an infected female *anopheles* mosquito) or transfusion transmitted (TT). This explains the reason the number of TTM cases in endemic countries is under-reported. Acquisition of malaria parasite due to donor exposure is an increasing problem as a result in global travelling and immigration. Thus, it is more challenging to develop an optimal strategy to reduce the risk of TTM in endemic countries without unnecessary exclusion of blood donation which remain a subject of debate. In [10,11], a general overview of current strategies in non-endemic countries was considered. The strict donor deferral system which is based on travel history of individual has been adopted by most countries, However, this strategy is not optimal due to many healthy donors are differed which may result in donation loss because lengthy deferrals may discourage the donors from coming back [5]. Consequently, the optimal control strategy for a given country or location may vary according to the background level of malaria risk faced by the donor and the recipient population viz-a-viz the resources available. Thus, we aim to study in this work mathematical analysis of transfusion–transmitted malaria TTM) model with optimal control

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2. Model Formation

We formulate the mathematical model for the Transfusion–Transmitted Malaria by considering the dynamical system of equation with optimal control analysis for human population only. The human population is divided into three sub-groups: Susceptible S(t)-Infected (I(t)) –Removal (R(t)). Thus, we assume that the total populations of humans is N(t) = S(t)+I(t)=R(t). Individual are recruited into the population at rate b and die naturally at rate μ. Recovered humans become susceptible again due to loss of immunity at rate δ. Our model also includes the rate of transfusion of infected blood and transmitted rate of the disease with malaria induced death rate.

For our dynamical equations, we define the following variables and parameters as follows:

Table 1. Description of variables and parameters of the model.

Parameters and Variables	Description
$S(t)$	Susceptible human population
$I(t)$	Infected human population
$R(t)$	Recovered human population
α	Rate of transfusion of infected blood with plasmodium to Susceptible humans
π	Recovery rate of humans
β	Rate of transmission of the diseases
b	Recruitment rate
μ	Natural death rate of humans
σ	Malaria induced death rate
δ	Rate of loss of immunity

The dynamical equations for the transfusion–transmitted malaria model are given as follows:

$$\left.\begin{aligned} S_h'(t) &= bN_h(t) - \frac{\alpha\beta S_h(t) I_h(t)}{N_h(t)} - \mu S_h(t) + \delta R_h(t) \\ I_h'(t) &= \frac{\alpha\beta S_h(t) I_h(t)}{N_h(t)} - (\sigma + \pi + \mu) I_h(t) \\ R_h'(t) &= \pi I_h(t) - (\mu + \delta) R_h(t) \end{aligned}\right\} \quad (1)$$

With the following assumptions:

(1) Both recruitment rate (b) and natural death rate for humans (μ) is assumed to be equal

(2) Transmission of the plasmodium is via transfusion of blood to blood contact of infected blood with plasmodium to a susceptible individual

(3) Due to the assumption made in (2), the Vector population is excluded

(4) Susceptible individual become infected upon blood to blood contact of infected blood with plasmodium

The flow diagram for the model is given in Figure 1:

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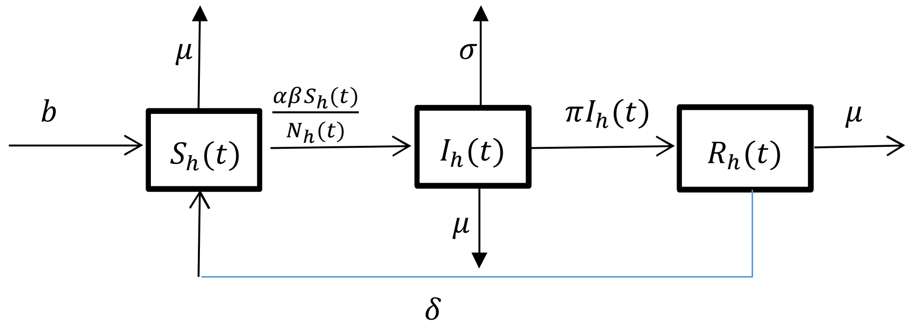

Figure 1. Flow-chart of the transfusion–transmitted malaria model showing movements amongst susceptible, infected and recovered compartments.

3. Model Analysis

System (1) is resolved by non-dimensionalizing the variables as follow by setting:

$$S(t)=\frac{S_h(t)}{N_h(t)}, I(t)=\frac{I_h(t)}{N_h(t)}, R(t)=\frac{R_h(t)}{N_h(t)} \quad (2)$$

$$S'(t)=\frac{S'_h(t)}{N_h(t)}, I'(t)=\frac{I'_h(t)}{N_h(t)}, R'(t)=\frac{R'_h(t)}{N_h(t)} \quad (3)$$

Substituting Equations (2) and (3) into (1) yields

$$\left.\begin{aligned} S'(t)&=b-\alpha\beta I(t)S(t)-\mu S(t)+\delta R(t)\\ I'(t)&=\alpha\beta I(t)S(t)-(\sigma+\pi+\mu)I(t)\\ R'(t)&=\pi I(t)-(\mu+\delta)R(t)\end{aligned}\right\} \quad (4)$$

3.1. The Population Dynamics of the Model

Let $N(t)$ represent the total human population. Thus

$$N(t)=S(t)+I(t)+R(t) \quad (5)$$

Differentiating (5) with respect to t give

$$N'(t)=b-\mu\left(S(t)+I(t)+R(t)\right)-\sigma I(t)$$

At disease free we obtain

$$N'(t)+\mu N(t)=b \quad (6)$$

Since the recruitment rate (b) is equal to the natural death rate (μ) and as $t\to\infty$, then the total human population reaches a value given as

$$N(t)=\frac{b}{\mu} \quad (7)$$

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3.2. Positivity of Solution

For the Transfusion–Transmitted Malaria model of Equation (4) to be epidemiologically well posed, we need to show that all solution with non-negative initial conditions will remain non-negative, for all $t \geq 0$.

Theorem 3.1: Let: $\eta = \eta_a \subset R_+^3$ with $\eta_a = \left\{ (S(t), I(t), R(t)) \in R_+^3 : (S(t) + I(t) + R(t)) \leq N(t) = \frac{b}{\mu} \right\}$, then the solution $(S(t), I(t), R(t))$ of the system (4) are positive $\forall t \geq 0$.

Proof: From the first differential equation of System (1),

$$\left.\begin{aligned} \frac{dS(t)}{dt} &\geq -[\alpha\beta I + \mu]S(t) \\ \frac{dS(t)}{S(t)} &\geq -[\alpha\beta I + \mu]dt \end{aligned}\right\}$$

Integrating both sides

$$\int \frac{dS(t)}{S(t)} \geq -\int_0^t [\alpha\beta I(t) + \mu]dt$$

To obtain

$$S(t) \geq k e^{-\int_0^t [\alpha\beta I(t) + \mu]dt}, \text{ at } t \to 0$$

$$S(0) = K = S(t), \text{ hence } S(t) \geq S(0) e^{-\int_0^t [\alpha\beta I(t) + \mu]dt} \geq 0, \forall\, t > 0.$$

Similar reasoning can be used for other differential equations of Equation (4) hence, it follows that the Transfusion–Transmitted Malaria model is positive and bounded with a unique solution.■

3.3. The Local Stability of Disease-free Equilibrium, P_0

System (4) has a disease-free equilibrium (DFE) obtained by setting the right-hand side of Equation (4) to zero, given by

$$P_0 : (S, I, R) = \left(\frac{b}{\mu}, 0, 0 \right) \qquad (8)$$

The Jacobian Matrix of Equation (4) about (8) is

$$J(P_0) = \begin{pmatrix} -\mu & \frac{-\alpha\beta b}{\mu} & \delta \\ 0 & \frac{\alpha\beta b}{\mu} - (\sigma + \pi + \mu) & 0 \\ 0 & \pi & -(\mu + \delta) \end{pmatrix}$$

So that the eigenvalues λ are real and given by $\lambda_1 = -\mu$, $\lambda_2 = -(\mu + \delta)$ and $\lambda_3 = (\sigma + \pi + \mu)(R_0 - 1)$.

Introducing now the basic reproduction number R_0:

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$$R_0 = \frac{\alpha\beta b}{\mu(\sigma+\pi+\mu)} \tag{9}$$

The expression in (9) can be obtained using the next generation matrix approach by finding the dominant eigenvalues of the matrix FV^{-1} where

$$F = \begin{pmatrix} \frac{\alpha\beta b}{\mu} & 0 \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} (\alpha+\pi+\mu) & 0 \\ -\pi & (\mu+\delta) \end{pmatrix}$$

So that:

(a) If $R_0 < 1$, then the eigenvalues are all negative then P_0 is locally asymptotically stable.

(b) If $R_0 > 1$, then two eigenvalues are negative and one is positive, then P_0 is unstable.

The above result is summarized in the following theorem.

Theorem 3.2: The disease-free equilibrium (DFE) P_0 of Equation (4) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

3.4. Global Stability of Disease-Free Equilibrium (DFE)

There are conditions for global asymptotic stability (GAS) of the disease – free equilibrium to be established, one of such condition is maintaining a constant population size. Observe that the model (4) will maintain a population size with time given by

$$N(t) = \frac{b}{\mu}$$

The global stability could be proved by several methods. The Lyapunov method has been used by several researchers, but here, the comparison approach as described in Lashmkantham et al. [6] will be used. The following theorem proves the global stability of the (DFE).

Theorem 3.3: Assuming that the system of Equation (4) describes a human population at equilibrium, then the (DFE) P_0 of (2) is globally asymptotically stable (GAS) if $R_0 < 1$, otherwise unstable.

Proof:

Using the comparison approach, the rate of change of the infected and recovered compartments of Equation (4) can be written as

$$\begin{pmatrix} \frac{dI(t)}{dt} \\ \frac{dR(t)}{dt} \end{pmatrix} \le (F-V)\begin{pmatrix} I(t) \\ R(t) \end{pmatrix} \tag{10}$$

where F and V retain their original meaning, according to Castillo-Chaves and song [3], all eigenvalues of $(F-V)$ have negative real root, i.e., $\lambda_1 = -(\mu+\delta)$, $\lambda_2 = (\sigma+\pi+\mu)(R_0 - 1)$. It follows that λ_2 is real and negative provided $R_0 < 1$. Hence, the linearized differential inequality (10) is stable whenever $R_0 < 1$. Consequently, $(I(t), R(t)) \to (0,0)$ as $t \to \infty$ evaluating system

doi:10.20944/preprints201809.0214.v2

(4) at $I(t) = R(t) = 0$ makes $S(t) \to \frac{b}{\mu}$ for $R_0 < 1$. Hence, the disease-free equilibrium P_0 is globally asymptotically stable (GAS) if $R_0 < 1$.

3.5. The Local Asymptotical Stability of the Endemic Equilibrium

Observe that Equation (4) have the endemic equilibrium point P^* defined as $P^* = (S^*(t), I^*(t), R^*(t))$ such that

$$S^*(t) = \frac{b}{\mu R_0}, I^*(t) = A(R_0 - 1), R^*(t) = \frac{\pi A(R_0 - 1)}{(\delta + \mu)]}$$

where

$$A = \frac{\mu(\delta + \mu)(\sigma + \pi + \mu)}{\alpha\beta[\delta(\sigma + \mu) + \mu(\sigma + \pi + \mu)]}$$

The Jacobian matrix of Equation (4) at P^* is

$$J(P^*) = \begin{pmatrix} -\left(\mu + A(R_0 - 1)\right) & \frac{-\alpha\beta b}{\mu R_0} & \delta \\ A(R_0 - 1) & 0 & 0 \\ 0 & \pi & -(\mu + \delta) \end{pmatrix} \quad (11)$$

It follows from Routh-Hurwitz condition that:

(i) The Trace of (*) = −(+ (0 − 1)) − (+) < 0, if 0 > 1

(ii) The determinant of (*) = (1 − (+)(+ +) /) (0 − 1) if 0 > 1 and (+)(+ +) / < 1

It follows that all the eigenvalues of $J(P^*)$ are real negative roots if $R_0 > 1$ and $\frac{(\delta+\mu)(\sigma+\pi+\mu)}{\pi\delta} < 1$ which implies that the endemic equilibrium point P^* is locally asymptotically stable. The foregoing discussion is summarized as follows:

Theorem 3.4: The endemic equilibrium point P^* of System (4) is locally asymptotically stable if $R_0 > 1$ and $\frac{(\delta+\mu)(\sigma+\pi+\mu)}{\pi\delta} < 1$, otherwise unstable.

3.6. Impact of Transfusion Rate (α) on Malaria Transmission

To analyze the effect of transfusion rate on malaria, we begin by expressing R_0 in terms of α as follows:

$$R_0(\alpha) = \frac{\alpha\beta b}{\mu(\sigma + \pi + \mu)} \quad (12)$$

Differentiating $R_0(\alpha)$, partially with respect to α leads to

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$$\frac{\partial R_0(\alpha)}{\partial \alpha} = \frac{\beta b}{\mu(\sigma + \pi + \mu)} \tag{13}$$

If Equation (13) is greater than zero, then an increase in transfusion rate result in an increase in the number of malaria cases. However, if Equation (13) is equal to zero, then the transfusion rate α does not have any significant effect on the transmission dynamics of malaria.

3.7. Analysis of Optimal Control

This section focus on the optimal control analysis of model equation (4), using the Pontryagin's Maximum Principle [9] to analyze and determine the necessary conditions for the optimal control of transfusion –transmitted malaria. Time dependent preventive and treatment control are introduced into the model (4) to determine the optimal strategy for controlling the disease. Thus, we have

$$\left.\begin{aligned} S'(t) &= b - (1-u_1)\beta I(t)S(t) - \mu S(t) + \delta R(t) \\ I'(t) &= (1-u_1)\beta I(t)S(t) - (\sigma + u_2 + \mu)I(t) \\ R'(t) &= u_2 I(t) - (\mu + \delta)R(t) \end{aligned}\right\} \tag{14}$$

Our aim is to minimize the number of infected humans to malaria due to TT, and the cost of applying preventive and treatment controls $u_1(t)$ and $u_2(t)$. Thus, we consider the objective functional

$$J(u_1, u_2) = \int_0^{t_f} \left(w_1 I(t) + w_2 u_1^2(t) + w_3 u_2^2(t) \right) dt \tag{15}$$

The control function, $u_1(t)$ and $u_2(t)$ are bounded, Lebesque integrable functions. The controls $u_1(t)$ and $u_2(t)$ denotes the effects on preventing transfusion of infected blood with plasmodium through effective blood screening and treatment of malaria infected individuals respectively. The coefficients w_1, w_2 and w_3 are the balancing cost factors of the three parts of the objective function while t_f is the final time.

We then seek to find an optimal control, $u_1^*(t)$ and $u_2^*(t)$ such that

$$J(u^*_1, u^*_2) = \min \left\{ J(u_1, u_2) \mid_{(u_1, u_2)}, u_1, u_2 \in u \right\} \tag{16}$$

where $u = \{u_1, u_2 : \textit{are measurable with } 0 \le u_1 \le 1, 0 \le u_2 \le 1\}$ is the control set.

Considering the conditions that an optimal solution must satisfy as it was given by Pontryagin Maximum Principle [9], this principle helps to convert (14) and (15) to a minimization problem with respect to the controls $u_1(t)$ and $u_2(t)$ on a point-wise Hamiltonian H defined thus,

$$\begin{aligned} H = {} & w_1 I(t) + w_2 u_1^2 + w_3 u_2^2 + \lambda_1 \left[b - (1-u_1)\beta I(t)S(t) - \mu S(t) + \delta R(t) \right] + \\ & \lambda_2 \left[(1-u_1)\beta I(t)S(t) - (\sigma + u_2 + \mu) I(t) \right] \\ & + \lambda_3 \left[u_2 I(t) - (\mu + \delta) R(t) \right] \end{aligned}$$

where λ_1, λ_2 and λ_3 are the adjoint variables (co-state variables).

Theorem 3.5: Consider an optimal control u^*_1, u^*_2 and solutions of *S(t)*, *I(t)*, *R(t)* with the corresponding state system (14) and (15) that minimizes $J(u_1, u_2)$ over *u*. Then there exist adjoint variables λ_1, λ_2 and λ_3 satisfying.

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$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial S(t)}, \qquad \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial I(t)}, \qquad \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial R(t)} \tag{17}$$

with transversality conditions

$$\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = 0 \tag{18}$$

and

$$u_1^* = \min\left\{1, \max\left(0, \frac{(\lambda_2 - \lambda_1)\alpha\beta I(t)S(t)}{2w_2}\right)\right\} \tag{19}$$

$$u_2^* = \min\left\{1, \max\left(0, \frac{(\lambda_2 - \lambda_3)I(t)}{2w_3}\right)\right\} \tag{20}$$

Proof:

Corollary 4.1 of Fleming and Rishel [4] established the existence of an optimal control due to the convexity of the integrand of J with respect to $u_1(t)$ and $u_2(t)$, a priori boundedness of the state variable solutions and the Lipschitz property of the state system with respect to the state variables. The differential equations governing the adjoint variables are obtained as follows:

$$\left.\begin{aligned}
\frac{d\lambda_1}{dt} &= (\lambda_1 - \lambda_2)(1 - u_1)\beta I(t) + \mu\lambda_1 \\
\frac{d\lambda_2}{dt} &= (\lambda_1 - \lambda_2)(1 - u_1)\beta S(t) + (\sigma + u_2 + \mu)\lambda_2 - u_2\lambda_3 - w_1 \\
\frac{d\lambda_3}{dt} &= (\mu + \delta)\lambda_3 - \delta\lambda_1
\end{aligned}\right\} \tag{21}$$

Solving for u^*_1 and u^*_2, subject to the constraints, the characterization (19) and (20) can be derived as follows:

At the very minimum

$$\frac{\partial H}{\partial u_1} = 2w_3u_1 + \lambda_1\beta I(t)S(t) - \lambda_2\beta I(t)S(t) = 0$$

$$\frac{\partial H}{\partial u_2} = 2w_3u_2 - \lambda_2 I(t) + \lambda_3 I(t) = 0$$

Thus,

$$\left.\begin{aligned}
u_1^* &= \frac{(\lambda_2 - \lambda_1)\beta I(t)S(t)}{2w_2} = \varepsilon_1 \\
u_2^* &= \frac{(\lambda_2 - \lambda_3)I(t)}{2w_3} = \varepsilon_2
\end{aligned}\right\} \tag{22}$$

By standard control arguments involving the bounds on the controls, we have that

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$$u_i^* = \begin{cases} 0, \ if \ \ \varepsilon_i^* \leq 0 \\ \varepsilon_i^*, \ if \ 0 \leq \varepsilon_i^* < 1 \\ 1 \ , \ if \ \ \ \varepsilon_i^* \geq 1 \end{cases} \tag{23}$$

where $i = 1,2$. Conclusively we can re-write (23) as

$$\left. \begin{aligned} u_1^* &= \min\{1, \max(0, \varepsilon_1)\} \\ u_2^* &= \min\{1, \max(0, \varepsilon_2)\} \end{aligned} \right\} \tag{24}$$

4. Numerical Simulations and Discussion of Results

The numerical solutions are illustrated using MAPLE 18 program with computation times of 3.52 s on a windows 7 operating system. The optimality system, consist of the state system, adjoint system, initial conditions for the state system and the transversality conditions for the adjoint system. The state system is solved by the forward finite difference scheme using the current iterations solutions of the state equations. The adjoint system is solved by the backward finite difference scheme using the current iterations solutions of the state equations because of the transversality conditions. Then the control are updated by using a convex combination of the previous controls and the value from the characterization (19) and (20). Thus, the process is repeated and the iterations are stopped at the final time t_f. The table of parameter descriptions and values used in the numerical simulation of the model are given in Table 2.

One of the ways of controlling the spread of malaria disease is through blood screening of donors; however, lack of information or ignorance may affect the impact blood screening can have on malaria transmission.

The behaviour of the total human populations is investigated over time in Figure 2. It was observed for threshold parameter $R_0 < 1$, the asymptotic nature of the population is established. The number of susceptible individuals increases with time and infected humans recovered while the infected humans' decreases asymptotically over time. However, for $R_0 > 1$, the unstable nature of the population became evident as depicted in Figure 3.

Consequently, optimal control strategies using the combination of screening donor's blood $u_1(t)$ and treatment for those infected $u_2(t)$ were used on the model to control the transmission of malaria. The following scenarios were considered:

Table 2. Values of Parameters for system (4).

Parameters	Baseline Value	Source
α	0.1	Assumed
π	0.5	[10]
β	0.001	[10]
b	1/(70 × 365)	[11]
μ	1/(70 × 365)	[11]
σ	0.01	[11]
δ	0.1	Assumed

(a) Optimal Control Using Screening of Donor's Blood $u_1(t)$ and Treatment $u_2(t)$

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In this case, two control are used to optimize the objective function J. It was observed in Figure 4 that the combination of both controls resulted in significant decrease in the number of infected humans (green solid line) as against the drastic increase observed in the uncontrolled case (red dotted line).

(b) Optimal Control Using Treatment $u_2(t)$ Only

Here, the objective function J is optimized using control $u_2(t)$ while the control on blood screening was set to zero. It was observed that number of infected humans showed significant reduction while there is an increase in the number of infected humans in the uncontrolled case as shown in Figure 5

(c) Optimal Control Using Screening of Donor's Blood $u_1(t)$ Only

The objective functional J is optimized in this case by setting the control on treatment $u_2(t)$ to zero. The result of this strategy clearly underline that screening of blood before transfusion is carried out is important as the number of infected humans that would have been infected with malaria reduces as a result of blood screening while the number of infected individuals increases as a result of no blood screening as depicted in Figure 6.

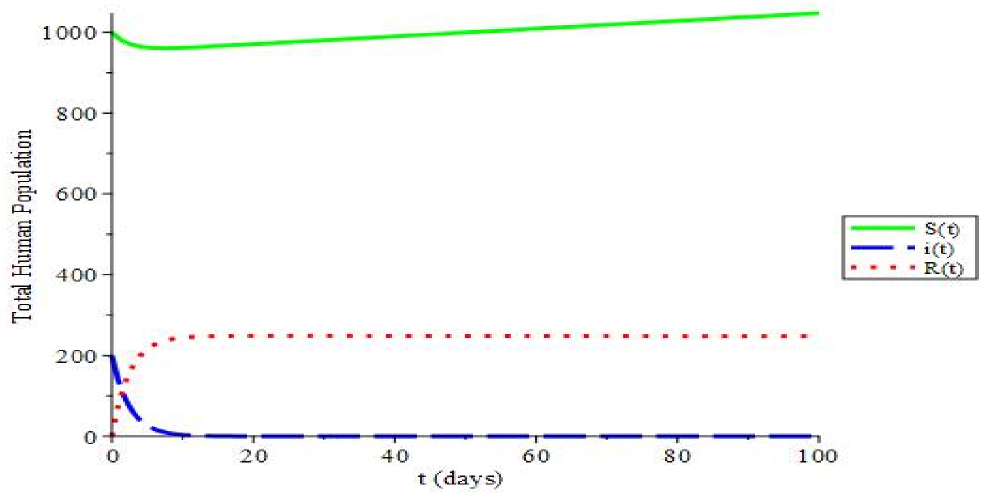

Figure 2. The graph of the total human population for $R_0 < 1$.

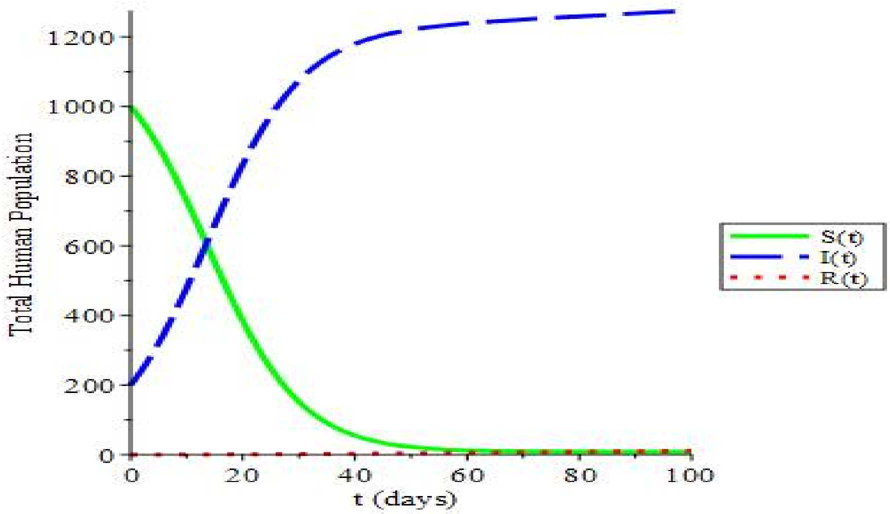

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Figure 3. The graph of the total human population for $R_0 > 1$.

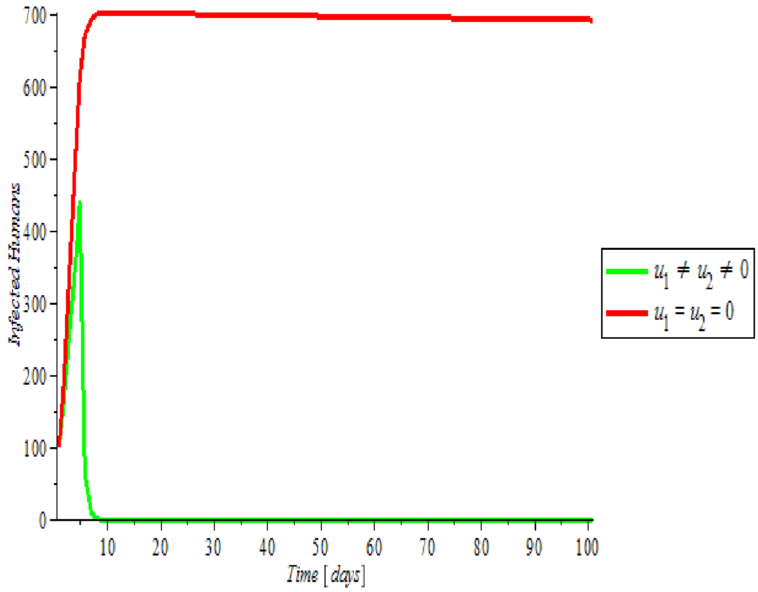

Figure 4. The variation of proportion of malaria infected population using $u_1(t)$ and $u_2(t)$ as controls.

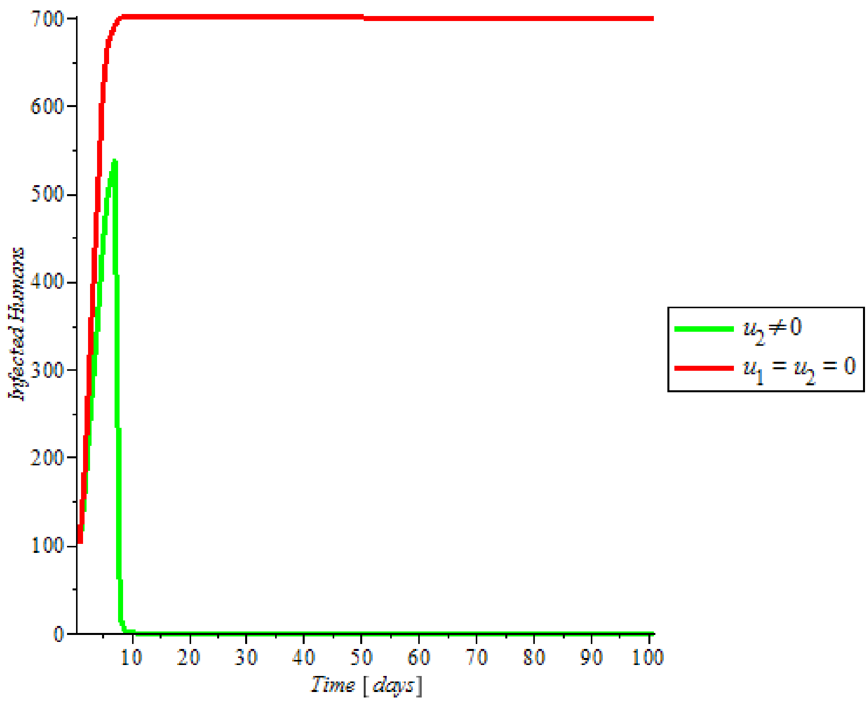

Figure 5. The variation of proportion of malaria infected population using $u_2(t)$ as control.

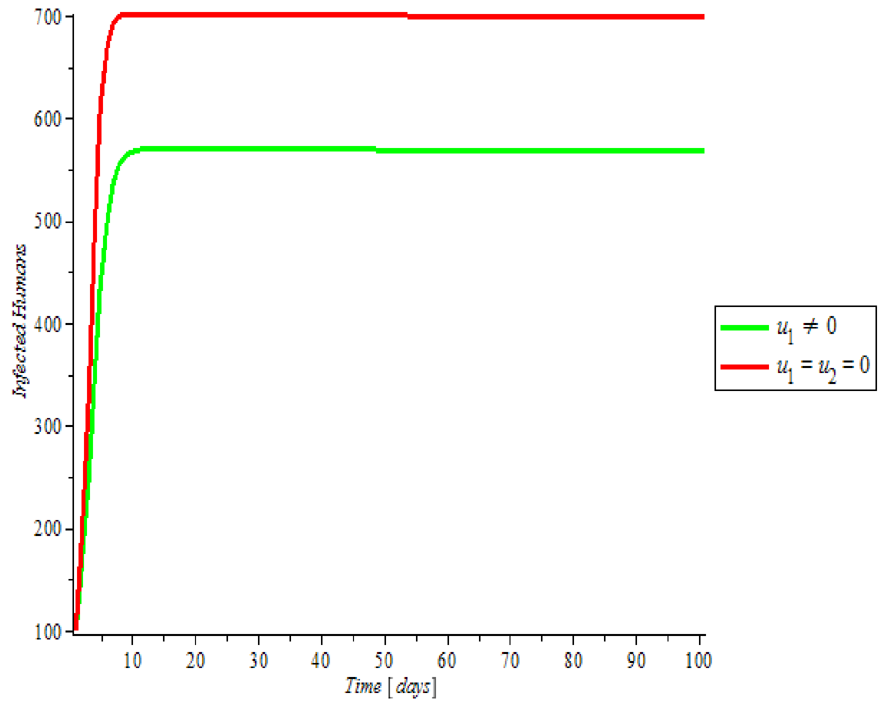

Figure 6: The variation of proportion of malaria infected population using $u_1(t)$ as control.

5. Conclusions

In this study, we used a mathematical model to examine the Transfusion–transmitted malaria (TTM) on the spread of malaria. Although screening of donor's blood is not the only means of controlling the disease, we demonstrated that blood screening of donor's has a positive impact in reducing the disease burden. The derivative of the reproduction number R_0 with respect to rate of transfusion of infected blood α revealed that more individual is likely to become infected as it has a positive impact on R_0, this led to the introduction of controls $u_1(t)$ and $u_2(t)$ in the optimal control model. The control model was analyzed using Pontryagin's Maximum Principle. The result of the analysis revealed that the combination of using both controls yielded the best result.

Conclusively, lack of screening donor's blood may have an adverse effect in the control of malaria transmission especially in malaria endemic regions. It is also clear from our optimal control analysis that screening of donors blood and treatment of infected individual will help to reduce the number of malaria cases, however, we submit that more robust models must be developed to include the dynamics of vector populations, information on human nature and behavior towards blood screening and other interventions in order to give realistic estimates on malaria dynamics. This shall form the basis for a separate research.

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