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Modelling the Impact of Vaccination on Malaria Transmission

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Abstract

Vaccination offers a potential contribution to malaria control, particularly for vulnerable populations such as children under five. Mathematical modeling provides a structured approach to examining how vaccination may influence transmission and disease burden over time. This study aimed to investigate the impact of vaccination on malaria transmission dynamics by incorporating vaccination as a control measure into a modified mathematical model. Analytical and numerical approaches were employed to examine the model's behavior and assess the implications of vaccination for disease transmission. Linearization and the next-generation matrix approach were used to investigate stability and transmission dynamics, while numerical simulations illustrated changes in susceptibility and disease incidence over time. The model results indicated that vaccination reduced the number of susceptible individuals, contributing to lower transmission and a subsequent decline in modeled incidence. These findings suggest that vaccination can influence the progression of malaria within the conditions represented by the model. The analysis provides a theoretical basis for examining vaccination as part of malaria control planning and highlights its potential relevance to protecting populations at elevated risk. However, the results represent model-based predictions, and their practical interpretation depends on the underlying assumptions and parameter values. Further research should calibrate the model using setting-specific epidemiological data, evaluate parameter uncertainty, and compare alternative vaccination strategies. Additional investigation could also

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examine how vaccination interacts with other malaria control measures and whether predicted reductions correspond to observed outcomes. The study concludes that incorporating vaccination into transmission models can support the assessment of potential control benefits and inform questions for subsequent empirical evaluation. Its contribution lies in linking vaccination-related changes in susceptibility with the modeled trajectory of malaria transmission and incidence.

Keywords: Malaria Transmission; Vaccination; Mathematical Modeling; Stability Analysis; Numerical Simulation

Introduction

Malaria is a vector-borne disease transmitted during blood meal by infectious Anopheles mosquitoes via insertion of sporozoites in the blood of susceptible humans (Pimenta *et al.* 2015). The worldwide casualties of malaria are huge, particularly in sub-Sahara Africa, and especially in the children population (Anjorin *et al.*, 2023). The World Health Organization (WHO) estimated about 241 million malaria cases globally in 2020 that resulted in 627, 000 deaths. The tropical and sub-tropical regions (mainly Africa) carry a disproportionately high share of the global malaria burden with as much as 95% of malaria cases, 96% of malaria deaths with about 80% of these deaths are children less than 5 years of age (WHO, 2022). Symptoms of malaria include fever, headache and chills usually appear in 10–15 days after the infective mosquito bite and may be mild and difficult to recognize as malaria. In order to control the spread of malaria, preventive measures include mosquito-reduction strategies and self-protection against mosquito bites (via the use of insecticide treated bed nets (ITNs); intermittent preventive treatment (IPT), and the reduction of vector population through the destruction of their breeding sites (Oke *et al.,* 2020). Studies also revealed other intervention strategies, such as the use of indoor residual spraying (IRS) for killing infected indoors mosquitoes through the use of sterile insect technique Muller *et al.,* (2023) and the use of anti-malaria drugs to regulate malaria Oke *et al.,* (2020). While several prevention and therapeutic measures have been implemented to fight against this deadly disease, the recent groundbreaking malaria vaccine for children now recommended by the WHO is expected to help strengthen the fight against malaria infection (WHO, 2022). Also, the development of Transmission Blocking Drugs (TBDs) conferring protection against malaria will play a significant role in mitigating morbidity and mortality in malaria-prone regions (Tchoumi *et al.* 2022). It is expected that the widespread

use of the RTS malaria vaccine (trade name Mosquirix), a recombinant protein-based malaria vaccine in children in sub-Saharan Africa and other areas with moderate or high transmission of *Plasmodium falciparum* malaria along with other preventive measures could help mitigate the spread and eventually the eradication of the disease (WHO 2022)

Statement of the Research Problem.

Malaria is a vector-borne disease that poses major health challenges globally, with the highest burden in children less than 5 years old. Prevention and treatment have been the main interventions measures until the recent groundbreaking highly recommended malaria vaccine by WHO for children below five years which call for concern hereby developing mathematical model to enable gain more insight into the effectiveness of this vaccine.

Aim and Objectives of the Study.

To modify the model formulated by Segun *et al.,* (2020) we incorporate vaccination as another control strategies to enable us gain more insight into the transmission dynamics of malaria in the population. The objectives are as follow;

i. To determine the basic reproduction number of the modified model
ii. To study the existence and stability of the free equilibrium state of the modified model
iii. To carry out numerical studies on the modified model
iv. To make appropriate recommendation base on the results obtained.

Literature Review

Mathematical models for transmission dynamics of malaria are useful in providing better insights into the transmission dynamics of the disease. The models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of malaria. According to Tchoumi *et al.* (2022), the study on malaria using mathematical modeling began in 1911 with Ross (1911) in his work title "The prevention of malaria", He introduced the first deterministic two-dimensional model with one variable representing human and the other representing mosquitoes where it was shown that reduction of mosquito population below a certain threshold was sufficient to eradicate malaria.

In Elliott (2018), the Ross's model was modified by considering the latency period of the parasites in mosquitoes and their survival during that period. However, in this case, it was shown that reducing the number of mosquitoes is an inefficient control strategy that would have little effect on the epidemiology of malaria in areas of intense transmission.

Further extension was described by Blackwood and Childs (2018), where the latency of infection in humans was introduced by making the additional exposed class in humans. This modification further reduces the long term prevalence of both the infected humans and mosquitoes. Thus, all other models that exist for malaria dynamics are developed from the three basic models explained earlier by incorporating different factors to make them biologically more realistic in explaining disease prevalence and prediction Eikenberry and Gumel (2018). For examples, a number of epidemiological studies considered the inclusion of the recovered class which incorporates a time dependent immunity developed on recovery from infection in humans. Their models take into account that acquired immunity to malaria depends on continuous exposure to reinfection. Moreover, some models have integrated other factors such as: environmental effects Yang *et al.*, (2000), mosquito's resistance to insecticides and resistance of some parasite strains to anti-malaria drugs Okosun *et al., (2011).*

Since the eighteen century, the development of mathematical models has been critical to provide framework and understanding of the dynamics of infectious diseases (Ndamuzi and Gahungu, 2021). Several mathematical models of malaria dynamics investigating various aspects of the disease have flourished in the literature Collins and Duffy (2022). While mosquito's population fluctuates between climatic seasons, seasonal factor tends to impact on the dynamics of infected mosquitoes and human populations in regions with hot climate (Olaniyi *et al.,* 2018). Because of mathematical tractability and convenience, though important, we will not account for seasonality in the birth rate of mosquitoes. For several decades, concerted global stringent efforts have been underway to develop effective and safe vaccine for use against malaria in humans (Zhao *et al.*, 2022), with several candidate vaccines targeting different stages of the malaria parasite's lifecycle.

An overview of integrated mathematical models for predicting the epidemiologic and economic effects of malaria vaccines on the clinical epidemiology and natural history of Plasmodium falciparum malaria both at the individual and population level has been reported in Abioye *et al.* (2020). It is noted that these models provide a unique platform for

predicting both the short- and long-term effects of malaria vaccines on the burden of disease, allowing for the temporal dynamics of effects on immunity and transmission. With mathematical models being increasingly used to inform decisions throughout product development pathways from pre-clinical studies to country implementation of novel health interventions, Galactionova *et al.* (2021) illustrate the utility of simulation approaches by reviewing malaria vaccine modeling studies. A mathematical model of vaccine combination adapted to murine malaria studies based on simple probabilistic assumptions was developed in Atcheson *et al.* (2019).

Transmission-blocking vaccines of malaria have been investigated in Zhao *et al.* (2022) and Takashima *et al.*, (2021) where as expected, vaccination has a positive impact on reducing the disease burden, while malaria could be controlled if the duration of efficacy is in the order of a human life-span (Blackwood and Childs, 2018).

Public health impact of dynamics model of a transmission-blocking vaccine alongside existing interventions suggests that school-aged children are an attractive population to target for vaccination Challenger *et al.* (2021). That is, benefit of vaccination distributed across the population averts the greatest number of cases in younger children. Even an imperfect anti-malaria vaccine (with a modest efficacy and coverage rate) can lead to effective disease control (Teboh-Ewungkem *et al.* 2010).

Handari *et al.* (2022) analyze an optimal control model of malaria incorporating pre-erythrocytic vaccine and transmission-blocking treatment. Previous studies have been hypothetical because there was no approved/licensed malaria vaccine, and also vaccination was applied to the entire population. As predicted in Challenger *et al.* (2021) and Hill (2011), the first approved malaria vaccine is for children age five and below, and consequently, we propose a mathematical model of malaria transmission dynamic by extending our previous work Tchoumi *et al.* (2022), incorporating vaccination in the class of children less than 5 years old, but with no seasonal birth rate (Herdicho *et al.* 2021). The population of children 5 months (minimum age to take the vaccine) to 5 years are vaccinated at a certain rate. The vaccinated class comprises fully vaccinated children (i.e., those who have taken all the 4 doses of the S/AS01 (RTS, S) vaccine). It is important to note that in Tchoumi *et al.* (2022), the authors studied a two-group malaria model structured by age with no vaccination, while herein, to realistically capture what is currently known about the state of the vaccine development, we incorporate vaccination for children

less than 5 years old only to capture the recently approved children vaccine. To the best of our knowledge, this is the first mathematical model investigating the impact of the newly approved children malaria vaccine on the dynamics of the disease.

Over the years, several numbers of mathematical models on the transmission dynamics of malaria have been examined. Following the simple S-I-R malaria model of (Ross, Sang *et al.,* 2012) and Macdonald *et al.,* (2014), many researchers have elaborated these models by incorporating different features associated to malaria transmission dynamics and its control. This includes modeling the effect of age groups on the transmission of malaria, as shown by (Addawe *et al.,* 2012). In their research "mathematical model for malaria transmission dynamics in human and mosquito populations" also revealed that an increase in the number of disease-induced death rate of the mosquito reduces the number of infectious human. Therefore, humans need to boost their antibodies production to be able to subdue the invasion of parasites in the bloodstream. The immunity state of the individual, that is, the general health and nutritional status of the infected individual, is a factor for preventing or aiding the occurrence of malaria. Thus, leading a healthy lifestyle and eating right foods can help boost the level of antibodies in humans. It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of malaria. Hence, we can achieve a malaria-free state by scaling down mosquito biting rate through the regular indoor residual spraying with insecticides, the use of insecticide-treated bed nets, closing of doors and windows against mosquitoes, clearing of stagnant water and drainages, and the use of mosquito repellant lotions, which are all regarded as vector control measures.

However, recent work has shown that some of the factors, as the age structure of mosquito's population and climate effects are very important for a better understanding of malaria transmission global dynamics, Beck-Johnson *et al.*, (2013). Indeed, mosquitoes undergo complete metamorphosis going through four distinct stages of development during a lifetime: egg, larva, pupa, and adult. While it is appropriate to assume that only adult mosquitos are involved in the malaria transmission, the dynamics of the juvenile stages (larvae and pupae) has significant effects on the dynamics of the mosquito population, and then the malaria transmission global dynamics. Motivated by this work, and using the malaria model in Ducrot *et al.,* (2009) as our baseline model, we include the four distinct metamorphic stages of mosquito to formulate a mosquito-stage-structured autonomous model of malaria spread in a more general setting.

In complementing anti-malarial methods, the European Medicines Agency (EMA 2015) approved the use of a malaria vaccine known as RTS,S/AS01, or Mosquirix. This vaccine candidate is intended for use against malaria caused by the P. falciparum parasite to reduce severe cases of the disease in infants aged from six weeks to 17 months in areas where malaria is dominant (EMA 2015).

Formal recommendations were issued by the (WHO 2022) on the pilot implementations of the four-dose schedule of Mosquirix in children below the age of five years. This pilot project was carried out in three Sub-Saharan African countries, namely Ghana, Kenya and Malawi, where three doses were given to children between one and 59 months old, at least four weeks apart from the first dose, with the fourth dose being issued 15–18 months after the third dose (WHO 2016, 2018).

Methodology

Model Formulation and Description

According to Segun *et al* (2020), the model sub-divides the total human population at time (t), denoted by $N_h(t)$, into susceptible humans (S_h), infected humans (I_h), and recovered humans (R_h).

$$N_h(t) = S_h + I_h + R_h \tag{1}$$

The model also sub-divides the total vector population at time (t), denoted by $N_v(t)$, into immature mosquitoes (A_v), susceptible mosquitoes (S_v), and infected mosquitoes (I_v). Thus, the total vector population is given by

$$N_v(t) = A_v + S_v + I_v \tag{2}$$

As the disease progresses, individuals move from one class to the other. Individuals are recruited into the susceptible human population by either via birth or immigration at the rate πh, and also by the loss of immunity of recovered humans at a rate ψ. The human population susceptible is depopulated by infection subsequent to contact with infectious vectors at a rate $\lambda_h(b)$, defined as

$$\lambda_h(b) = \frac{\beta_{hv}\varepsilon(b)I_v}{N_h} \tag{3}$$

The parameter β_{hv} is the probability of effective transmission from human to mosquitoes, following the contact rate of mosquito to human by the rate $\varepsilon(b)$. Furthermore, the population of susceptible humans is decreased by natural death, at a rate μ. Thus, the rate of change of the population of susceptible human is given by

$$\frac{dS_h}{dt} = \pi_h + \psi R_h - \lambda_h(b) S_h - \mu_h S_h \tag{4}$$

The population of infected humans individuals is generated by the infection of susceptible humans (at the rate $\lambda_h(b)$) and is decreased by recovery of infected individuals (at a rate τ_h), natural death (at a rate μ_h) and disease induced death (at a rate δ_h), so that

$$\frac{dI_h}{dt} = \lambda_h(b) S_h - (\tau_h + \mu_h + \delta_h) I_h \tag{5}$$

Finally, on human population, the population of recovered humans is generated by the recovery of infected humans (at a rate τ_h). It is decreased by loss of immunity and natural death (at a rate ψ and μ_h respectively). Thus, the recovered human population is given by

$$\frac{dR_h}{dt} = \tau_h I_h - (\psi + \mu_h) R_h \tag{6}$$

The immature mosquitoes are populated by mosquito egg deposition (at a rate $\pi_v(b)$), and reduced by maturation of immature mosquitoes (at a rate γ_v) and natural mortality (at a rate $\mu_v(q)$). Thus, the immature mosquito population is given by

$$\frac{dA_v}{dt} = \pi_v(q) - (\gamma_v + \mu_v(q)) A_v \tag{7}$$

The population of susceptible mosquitoes is generated by the maturation of immature mosquitoes (at a rate γ_v). It is further depopulated by infection following effective contact with infectious humans at a rate $\lambda_v(b)$, defined as

$$\lambda_v(b) = \frac{\beta_{vh} \varepsilon(b) I_h}{N_h} \tag{8}$$

The parameter βvh is the probability of effective transmission from mosquitoes to humans, following the contact rate of mosquito to human by the rate $\varepsilon(b)$. Furthermore,

the population of susceptible vectors is decreased by natural death, at a rate μv. Thus, the rate of change of the population of susceptible vectors is given by

$$\frac{dS_v}{dt} = \gamma_v A_v - \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))S_v \qquad (9)$$

Finally, the population of infected vectors is generated by the infection of susceptible vectors (at a rate $\lambda_v(b)$) and is decreased by natural death of mosquitoes (at a rate μv). The rate of change of the population of infected vectors is given by

$$\frac{dI_v}{dt} = \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))I_v \qquad (10)$$

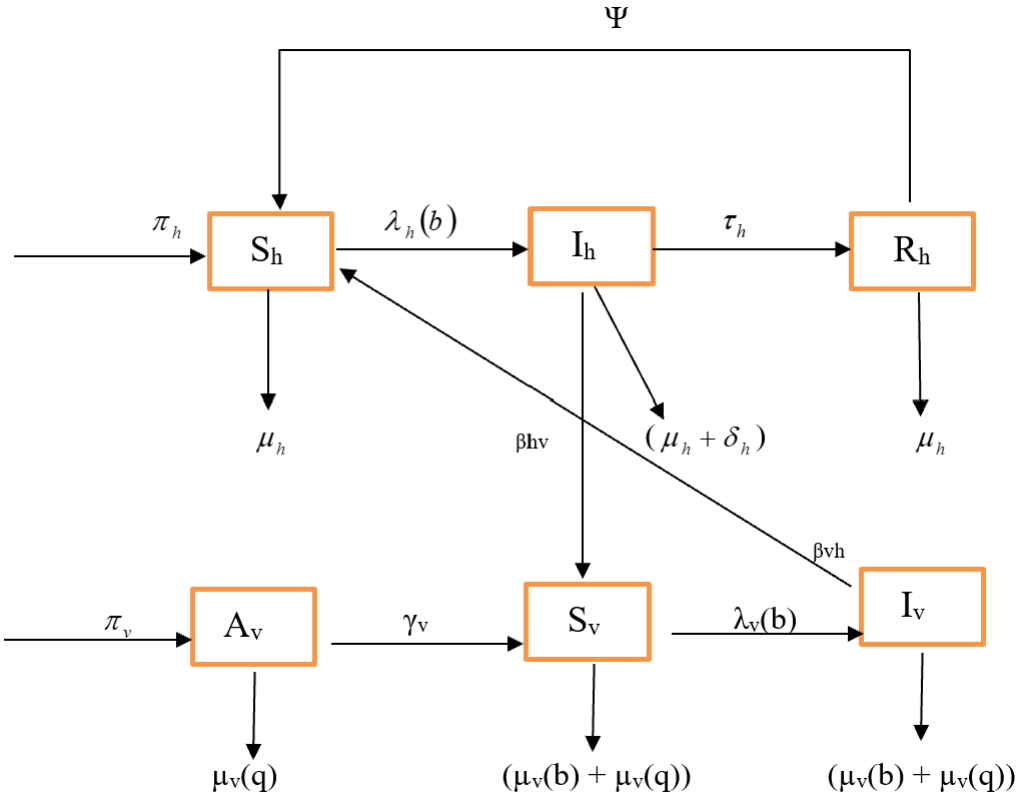


Fig 1 The flowchart of malaria disease with control strategy

Existing Model Equation by Segun *et al.*, (2020)

$$\frac{dS_h}{dt} = \pi_h + \psi R_h - \lambda_h(b)S_h - \mu_h S_h$$

$$\frac{dI_h}{dt} = \lambda_h(b)S_h - (\tau_h + \mu_h + \delta_h)I_h$$

$$\frac{dR_h}{dt} = \tau_h I_h - (\psi + \mu_h) Rh \quad (11)$$

$$\frac{dA_V}{dt} = \pi_v(q) - (\gamma_v + \mu_v(q)) Av$$

$$\frac{dS_V}{dt} = \gamma_v A_v - \lambda v(b) S_v - (\mu_v(b) + \mu_v(q)) S_v$$

$$\frac{dI_V}{dt} = \lambda_v(b) S_v - (\mu_v(b) + \mu_v(q)) I_v$$

Assumptions of the modified Model.

1. The model uses systems of ordinary differential equations.

2. We include a vaccination compartment (V) in the human population.

3. Only mature female anopheles' mosquitos (F_a) are considered

4. Susceptible human populations who are vaccinated at the rate σ_h are free from malaria disease.

5. Vaccinated individual become susceptible again at a rate α_h if stopped taking vaccine.

Table1 .modified model description of variable

Variables	Description
S_h	Susceptible individuals
I_h	Infected individuals
R_h	Recovered individuals
V_h	Vaccinated individuals
F_a	Susceptible Mosquitoes

Table 2. Description of parameters

Parameters	Description
π_h	Recruitment rate of humans
ψ	rate of loss of immunity in humans
μ_h	Natural mortality rate of humans
τ_h	Recovery rate of infectious individuals
δ_h	Disease induced death rate of humans
β_{vh}	Probability of effective transmission from mosquito to human
$\varepsilon(b)$	Contact rate of mosquito with human
b	Proportion of treated net usage
q	Proportion of insecticide spray on the environment

Parameters	Description
π_v	Egg deposition rate of mosquitoes
$\mu_v(b)$	Mortality rate of mosquitoes due to treated net
$\mu_v(q)$	Mortality rate of mosquitoes due to insecticide spray
σ_h	Vaccination rate
α_h	Rate at which vaccinated individuals become susceptible again

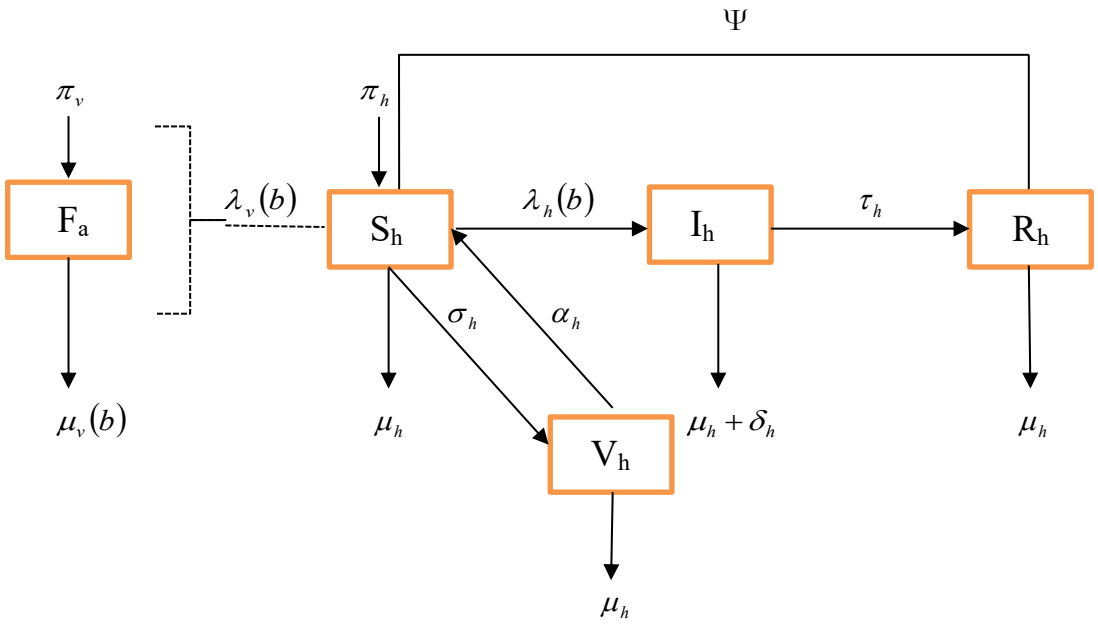


Fig 2. modified/new model flowchart

Modified Model equation

Based on our assumptions and understanding of the model flow chart above, we obtain new equations as shown below;

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \pi_h - \alpha_h S_h + \psi R_h - \lambda_h(b) I_h S_h - \mu_h S_h \\
\frac{dV_h}{dt} &= \sigma_h S_h - \mu_h V_h - \alpha_h V_h \\
\frac{dI_h}{dt} &= \lambda_h(b) I_h S_h - (\mu_h + \tau_h + \delta_h) I_h \\
\frac{dR_h}{dt} &= \tau_h I_h - (\mu_h + \psi) R_h \\
\frac{dF_a}{dt} &= \pi_v + \lambda_v(b) - \mu_v(b) F_a
\end{aligned}\right\} \qquad (12)$$

Results

Positivity and Boundedness of Solution

We have shown that the solution set to the model equation is feasible and attracts all solution in we now show that the variables of the model equation (12) are non-negative.

Lemma2. Let $\{S_h(t), I_h(t), V_h(t), R_h(t), F_a(t)\}$ be initial state variable values, and then the set of solutions of equation (12) is positive for all $t \geq 0$

Proof

Let's consider the system of equation (12)

From equation (12a), if we assume that

$$\frac{dS_h}{dt} = \pi_h + \alpha_h S_h + \psi R_h - \lambda_h(b) I_h S_h - \mu_h S_h - \sigma_h S_h \geq \alpha_h S_h - \lambda_h(b) I_h S_h - \mu_h S_h \tag{13}$$

$$\frac{dS}{dt} \geq -(\alpha_h + \lambda_h(b) I_h + \mu_h) S_h$$

$$\frac{dS}{S_h} \geq -(\alpha_h + \lambda_h(b) I_h + \mu_h) dt$$

Solving the equation above for $S_h(t)$ we have,

$\ln S_h(t) \geq -(\alpha_h + \lambda_h(b) I_h + \mu_h)t + k_3$, where k_3 is a constant

$$S_h(t) \geq C_1 e^{-(\alpha_h + \lambda_h(b) I_h + \mu_h)t}$$

When $t = 0$ we have,

$S_h(0) = C_1$, substituting into the above,

$$S_h(t) \geq S_h(0) e^{-(\alpha_h + \lambda_h(b) I_h) + \mu_h)t} \geq 0 \tag{14}$$

Therefore, $S_h(t) \geq 0$

In like manner, from equation (12b),

$$\frac{dV_h}{dt} = \sigma_h S_h - \mu_h V_h - \alpha_h V_h \geq -(\mu_h + \alpha_h)V_h \tag{15}$$

$$\frac{dV_h}{dt} \geq -(\mu_h + \alpha_h)V_h$$

$$\frac{dV_h}{V_h} \geq -(\mu_h + \alpha_h)dt$$

Solving the equation above for I(t) we have,

$$\ln V_h(t) \geq -(\mu_h + \alpha_h)\, t + k_4$$

$$V_h(t) \geq C_2 e^{-(\mu_h+\alpha_h)t}$$

When $t = 0$ we have,

$V_h(0) \geq C_2$ Substituting into the above,

$$V_h(t) \geq V_h(0)\, e^{-(\mu_h+\alpha_h)t} \geq 0 \tag{16}$$

Therefore, $V_h(t) \geq 0$

In the same, from equation (12c),

$$\frac{dI_h}{dt} = \lambda_h(b)I_h S_h - (\mu_h + \tau_h + \delta_h)I_h \geq -(\mu_h + \tau_h + \delta_h - \lambda_h(b)S_h)I_h \tag{17}$$

$$\frac{dI_h}{dt} \geq -(\mu_h + \tau_h + \delta_h - \lambda_h(b)S_h)I_h$$

$$\frac{dI_h}{I_h} \geq -(\mu_h + \tau_h + \delta_h - \lambda_h(b)S_h)dt$$

Solving the equation above for $I_h(t)$ we have,

$$\ln I_h(t) \geq -(\mu_h + \tau_h + \delta_h - \lambda_h(b)S_h)\, t + k_5$$

$$I_h(t) \geq C_3 e^{-(\mu_h+\tau_h+\delta_h-\lambda_h(b)S_h)t}$$

When $t = 0$ we have,

$I_h(0) \geq C_3$ Substituting into the above,

$$I_h(t) \geq I_h(0)\, e^{-(\mu_h+\tau_h+\delta_h-\lambda_h(b)S_h))t} \geq 0 \quad (18)$$

Therefore, $I_h(t) \geq 0$

Also, from equation (12d),

$$\frac{dR_h}{dt} = \tau_h I_h - (\mu_h + \psi)R_h \geq -(\mu_h + \psi)R_h \quad (19)$$

$$\frac{dR_h}{dt} \geq -(\mu_h + \psi)R_h$$

$$\frac{dR_h}{R_h} \geq -(\mu_h + \psi)dt$$

Solving the equation above for $R_h(t)$ we have,

$$\ln R_h(t) \geq -(\mu_h + \psi)\, t + k_6$$

$$R_h(t) \geq C_4 e^{-(\mu_h+\psi)\, t}$$

When $t = 0$ we have,

$R_h(0) \geq C_4$ Substituting into the above,

$$R_h(t) \geq R_h(0)\, e^{-(\mu_h+\psi)\, t} \geq 0 \quad (20)$$

Therefore, $R_h(t) \geq 0$

Thus, we have established that all state variables are positive $\forall\, t \geq 0$

Finally, from equation (12d),

$$\frac{dF_a}{dt} = \pi_v + \lambda_v(b) - \mu_v(b)F_a \geq -\mu_v(b)F_a \quad (21)$$

$$\frac{dF_a}{dt} \geq -\mu_v(b)F_a$$

$$\frac{dF_a}{F_a} \geq -\mu_v(b)dt$$

Solving the equation above for $F_a(t)$ we have,

$$\ln F_a(t) \geq -\mu_v(b)t + k_7$$

$$F_a(t) \geq C_4 e^{-\mu_v(b)t}$$

When $t = 0$ we have,

$F_a(0) \geq C_4$ Substituting into the above,

$$F_a(t) \geq F_a(0)\, e^{-\mu_v(b)t} \geq 0 \tag{22}$$

Therefore, $F_a(t) \geq 0$

Thus, we have established that all state variables are positive $\forall\, t \geq 0$

Local Stability of the Disease Free Equilibrium (DFE)

The malaria model (12) has a disease free equilibrium obtained by setting the right-hand sides of the equations in the model to zero and solving at $I_h=0$. Thus, the disease free equilibrium is given by;

$$\frac{dS_h(t)}{dt} = \frac{dV_h(t)}{dt} = \frac{dI_h(t)}{dt} = \frac{dT(t)}{dt} = \frac{dR(t)}{dt} = \frac{dF_a(t)}{dt} = 0 \tag{23}$$

Equation (12) becomes,

Use

$$\left.\begin{aligned} &\pi_h - \lambda_h(b)I_h S_h + \psi R_h - \lambda_h(b)S_h - \mu_h S_h = 0 \\ &\sigma_h S_h - \mu_h V_h - \alpha_h V_h = 0 \\ &\lambda_h(b)I_h S_h - (\mu_h + \tau_h + \delta_h)I_h = 0 \\ &\tau_h I_h - (\mu_h + \psi)R_h = 0 \\ &\pi_v + (\lambda_v(b) - \mu_v(b)F_a = 0 \end{aligned}\right\} \tag{24}$$

In absence of infection (that is I_h=0, V_h=0, R_h=0), adding the individual equations in the system (14) and we have

$$\pi_h - \mu_h S_h = 0 \Rightarrow \mu_h S_h = \pi_h \Rightarrow S_h = \frac{\pi_h}{\mu_h} \tag{25}$$

In like manner, solving equation (13e) for F_a, yields

$$\pi_v + (\lambda_v(b) - \mu_v(b) F_a = 0$$

$$\mu_v(b) F_a = \pi_v + (\lambda_v(b)$$

$$F_a = \frac{\pi_v + (\lambda_v(b)}{\mu_v(b)} \tag{26}$$

Therefore, the disease-free equilibrium (DFE) point of the system of model equation (12) denoted E^0 exist and is express as,

$$E^0 = (S_h{}^0, V_h{}^0, I_h{}^0, R_h{}^0, F_a{}^0) = \left(\frac{\pi_h}{\mu_h}, 0, 0, \ 0, \frac{\pi_v + (\lambda_v(b)}{\mu_v(b)} \right) \tag{27}$$

Basic Reproduction Number

We compute the basic reproduction number R_0 for the model (12). This will prove in analyzing the stability of the equilibrium points. R_0 is a very important dimensionless epidemiological parameter which measures the average number of secondary cases generated by a primary case when the infected individual is introduced into a population of completely susceptible individuals. We will determine R_0 for system (12) by applying the next generation matrix approach as laid out by Van den and James (2002). The first step to calculating R_0 is to rewrite the infective classes of the model equations in the form:

$$f = \begin{bmatrix} \sigma_h S_h \\ \lambda_h(b) I_h S_h \end{bmatrix} \tag{28}$$

And

$$v = \begin{bmatrix} -\mu_h V_h - \alpha_h V_h \\ -(\mu_h + \tau_h + \delta_h) I_h \end{bmatrix} \tag{29}$$

$$F = \begin{bmatrix} 0 & 0 \\ 0 & \lambda_h(b)\left(\frac{\pi_h}{\mu_h}\right) \end{bmatrix} = \begin{bmatrix} 0 & 0 \\ 0 & \frac{\lambda_h(b)\pi_h}{\mu_h} \end{bmatrix} = \begin{bmatrix} 0 & 0 \\ 0 & \frac{\lambda_h(b)\pi_h}{\mu_h} \end{bmatrix} \tag{30}$$

$$V = \begin{bmatrix} c_2 & 0 \\ 0 & c_3 \end{bmatrix} \tag{31}$$

Here the matrices F and V denote the new infection terms and the remaining transfer terms, respectively. With the notations $c_2 = -(\mu_h+\alpha_h)$ and $c_3 = -(\mu_h+\tau_h+\delta_h)$, we determine the following next generation matrices as the Jacobian matrices of $\mathcal{F}(t, x(t))$ and $\mathcal{V}(t, x(t))$, respectively, evaluated at the malaria free equilibrium. Next, we have

$$V^{-1} = \frac{1}{c_3 c_2}\begin{bmatrix} c_3 & 0 \\ 0 & c_2 \end{bmatrix} \tag{32}$$

From $R_0 = FV^{-1}$, we need to find

$$FV^{-1} = \frac{1}{c_3 c_2}\begin{bmatrix} 0 & 0 \\ 0 & \frac{\lambda_h(b)\pi_h}{\mu_h} \end{bmatrix}.\begin{bmatrix} c_3 & 0 \\ 0 & c_2 \end{bmatrix} \tag{33}$$

$$FV^{-1} = \frac{1}{c_3 c_2}\begin{bmatrix} 0 & 0 \\ 0 & \frac{\lambda_h(b)\pi_h c_2}{\mu_h} \end{bmatrix} \tag{34}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 \\ 0 & \frac{\lambda_h(b)\pi_h}{\mu_h c_3} \end{bmatrix} \tag{35}$$

$$FV^{-1} - \lambda I = \begin{bmatrix} 0-\lambda & 0 \\ 0 & \frac{\lambda_h(b)\pi_h}{\mu_h c_3} - \lambda \end{bmatrix}$$

$$\left|FV^{-1} - \lambda I\right| = \frac{\lambda_h(b)\pi_h \lambda}{\mu_h c_3} - \lambda^2$$

$$\frac{\lambda_h(b)\pi_h}{\mu_h c_3} - \lambda = 0$$

$$\lambda = \frac{\lambda_h(b)\pi_h}{\mu_h c_3}$$

Thus, the basic reproduction number R_0 is obtained as the spectral radius FV^{-1}, that is,

$$R_0 = \frac{\lambda_h(b)\pi_h}{\mu_h(\mu_h + \tau_h + \delta_h)} \tag{36}$$

Local stability of the malaria free equilibrium

To examine the local stability of the malaria free equilibrium, we first obtain the following Jacobian matrix of the system (12) at $\mathbb{E}_0$. Next, we then establish the following stability result.

$$J_{E0} = \begin{pmatrix} -d_1 & \alpha_h & 0 & \psi_h & 0 \\ \sigma_h & -d_2 & 0 & 0 & 0 \\ \lambda_h(b) & 0 & -d_3 & 0 & 0 \\ 0 & 0 & \tau_h & -d_4 & 0 \\ 0 & 0 & 0 & 0 & -d_5 \end{pmatrix} \tag{37}$$

$d_1 = \sigma_h + \mu_h$, $d_2 = \alpha_h + \mu_h$, $d_3 = \mu_h + \sigma_h + \tau_h$, $d_4 = \psi_h + \mu_h$, $d_5 = \frac{\beta_{vh}\varepsilon(b)I_h}{N_h} + \mu_v(b)$

Theorem Two

The malaria free equilibrium $\mathbb{E}_0$ is locally asymptotically stable if $R_0 < 1$, otherwise it is unstable.

Proof

The characteristic polynomial of the matrix J_{E_0} is obtained as

$$J_{E0} = \begin{pmatrix} d_1 & \alpha_h & 0 & \psi_h & 0 \\ \sigma_h & d_2 & 0 & 0 & 0 \\ \frac{\beta_{hv}\varepsilon(b)I_v}{N_h} & 0 & d_3 & 0 & 0 \\ 0 & 0 & \tau_h & d_4 & 0 \\ 0 & 0 & 0 & 0 & d_5 \end{pmatrix} \tag{38}$$

$$J_{E0} = \begin{pmatrix} d_1-\lambda & \alpha_h & 0 & \psi_h & 0 \\ \sigma_h & d_2-\lambda & 0 & 0 & 0 \\ \frac{\beta_{hv}\varepsilon(b)I_v}{N_h} & 0 & d_3-\lambda & 0 & 0 \\ 0 & 0 & \tau_h & d_4-\lambda & 0 \\ 0 & 0 & 0 & 0 & d_5-\lambda \end{pmatrix} \tag{39}$$

$$Det(J_{E_0} - \lambda I) = (d_1 - \lambda)(d_2 - \lambda))(d_3 - \lambda))(d_4 - \lambda))(d_5 - \lambda) = 0 \tag{40}$$

$$(d_1 - \lambda)(d_2 - \lambda))(d_3 - \lambda))(d_4 - \lambda))(d_5 - \lambda) = 0 \tag{41}$$

The malaria free equilibrium $\mathbb{E}_0$ is locally asymptotically stable if and only if all roots of the above characteristic polynomial have negative real parts. Obviously, all the eigenvalues

$$\lambda_1 = d_1 = -(\sigma_h + \mu_h), \quad \lambda_2 = d_2 = -(\alpha_h + \mu_h), \lambda_3 = d_3 = -(\mu_h + \sigma_h + \tau_h), \lambda_4 = d_4 = -(\psi_h + \mu_h), \lambda_5 = d_5 = -(\frac{\beta_{vh}\varepsilon(b)I_h}{N_h} + \mu_v(b))$$

Hence, the malaria free equilibrium is locally asymptotically stable if $R_0 < 1$.

Remark

Theorem 2 implies that malaria can be eradicated from the population (when $R_0 < 1$) if the initial sizes of the sub-populations of the model (12) are in the basin of attraction of the malaria free equilibrium. To ensure that eradication of the plasmodium does not depend on the initial sizes of the sub-populations, it is important to establish that the malaria free equilibrium globally asymptotically stable.

Numerical Simulation

In this section, we performed a numerical simulation to give more insights to our analytical results. Some of the parameters used are taken from Oke *et al.* (2023) and the remaining parameters were estimated and assumed. Using MAPLE, we performed the numerical simulation of the system of model equation (12) subject to the initial conditions given as; $S_h(0) = 125, V_h(0) = 15, I_h(0) = 70, R_h(0) = 10, F_a(0) = 30$, and different set of parameter values as are captured in the table below at final time $t_f = 30\ days$

Table 3. Parameter used in model simulations

Parameter	Values	Source
π_h	0.0024	Assumed
ψ	0.0005275	(Oke *et al.*, 2023)
μ_h	0.007	Assumed
τ_h	0.0092	(Oke *et al.*, 2023)
δ_h	0.0003454	Assumed
$\boldsymbol{\alpha_h}$	0.05	(Oke *et al.*, 2023)
λ_h	0.00343	assumed
σ_h	0.40	assumed
π_v	0.0225	assumed
λ_v	0.008	assumed
μ_v	0.094	assumed

Impact of Vaccination on Malaria Transmission

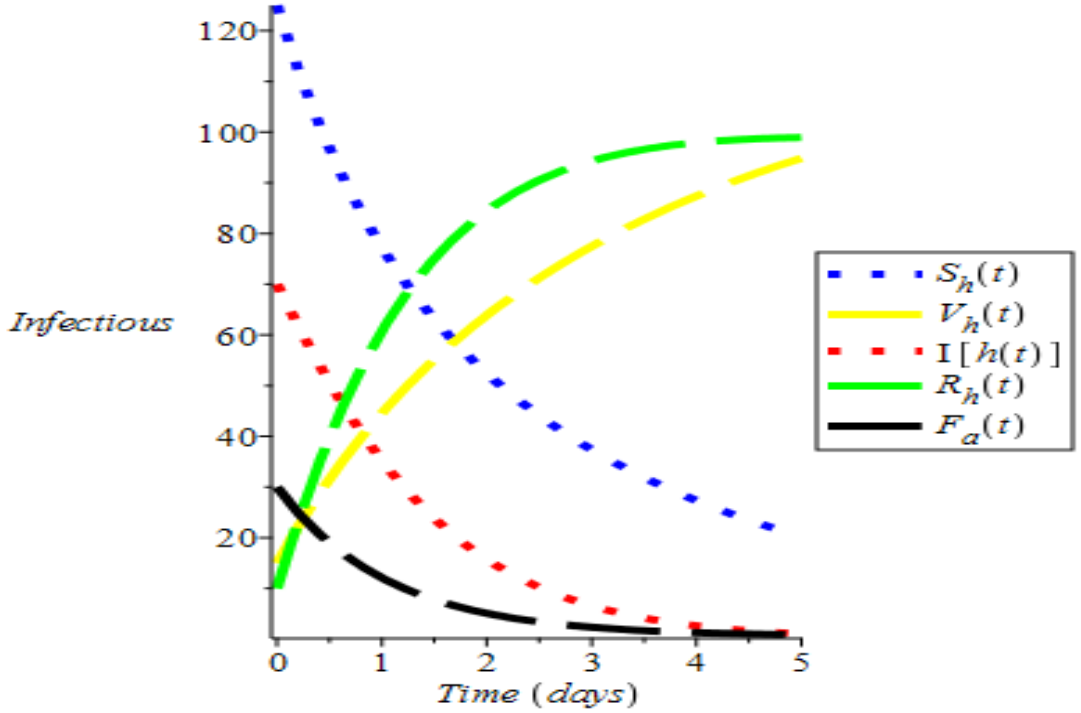


Figure 3. The effect of vaccination on malaria transmission

Discussion of Numerical Simulation

The lemma presented in this section establishes that the solution set to the model equations remains positive for all time. This is crucial in epidemiological modelling as negative solutions would have no physical interpretation. By analyzing each compartment of the model equations individually, the lemma demonstrates that under certain conditions, all state variables ($S_h(t)$, $V_h(t)$, $I_h(t)$, $R_h(t)$, and $F_a(t)$) remain positive. The proof employs mathematical manipulation to derive expressions for each state variable, ensuring positivity. Through careful analysis of the model equations, it was seen that the solutions are bounded and remain non-negative, which is essential for the validity and interpretation of the model.

Local stability of the DFE is crucial for understanding the long-term behaviour of the model. The Disease Free Equilibrium (DFE) represents a state where there are no infected individuals in the population. By analyzing the Jacobian matrix of the model equations evaluated at the DFE, we assesses whether small perturbations from this equilibrium will lead to convergence back to the DFE (stability) or diverge away from it (instability). It was observed that, the DFE is locally asymptotically stable, since the basic reproduction number (R_0) was found to be less than one (1). This result is significant as it

indicates that malaria can be eradicated if R_0 is kept below one (1), emphasizing the importance of intervention strategies such as vaccination.

The numerical simulation provides additional insights into the model's behavior and validates the analytical results obtained earlier. By employing parameter values from previous studies and assuming values for parameters not explicitly stated, the simulation illustrates the impact of vaccination on malaria transmission dynamics. The presented graph (Figure 2) depicts the reduction in malaria transmission due to the introduction of vaccination. This visual representation reinforces the theoretical findings and offers a practical understanding of how vaccination can contribute to malaria control efforts.

The results reveal the profound impact of vaccination on malaria transmission dynamics. Through mathematical modelling, it is demonstrated how the introduction of vaccination leads to a reduction in the number of susceptible individuals, thereby lowering the force of infection and decreasing the overall burden of malaria within the population. Graphical representations and numerical simulations illustrate the decline in malaria incidence rates over time due to vaccination campaigns.

Discussion

This research aimed to modify an existing mathematical model of malaria transmission dynamics of Oke *et al.* (2023) by incorporating vaccination as a control strategy. The study began by highlighting the significance of mathematical modelling in understanding malaria dynamics and the potential impact of vaccination model on disease control. The methodology involved modifying the existing equations to include vaccination compartments and conducting analytical and numerical analyses to assess the effects of vaccination on malaria transmission.

The modified model equations were analyzed to ensure the positivity and boundedness of solutions, establishing that all state variables remain non-negative throughout the dynamics. The local stability of the disease-free equilibrium (DFE) was investigated, with the basic reproduction number (R_0) computed to assess the potential for malaria eradication. Numerical simulations were performed using Maple 16 software application to validate the analytical results and provide insights into the impact of vaccination on malaria transmission dynamics.

Results demonstrated that vaccination significantly reduces malaria transmission by lowering the number of susceptible individuals, thereby decreasing the force of infection

within the population. The model predicted a decline in malaria incidence rates over time due to the introduction of vaccination, highlighting the potential of vaccination as an effective control measure against malaria.

Conclusion

This study contributes to the understanding of malaria transmission dynamics and the potential impact of vaccination on disease control. By modifying an existing mathematical model by Oke *et al.* (2023) to incorporate vaccination, the research provides insights into how vaccination can reduce malaria transmission and contribute to efforts aimed at disease eradication. The positivity and boundedness of solutions ensure the validity and interpretation of the model, while the analysis of the DFE stability indicates that malaria eradication is feasible if R_0 remains below one. Numerical simulations further support the effectiveness of vaccination in reducing malaria incidence rates, emphasizing the importance of vaccination as a key intervention strategy. Overall, this research underscores the significance of mathematical modeling in informing public health policies and interventions for malaria control.

Recommendations

Based on the findings of this study, the following recommendations are proposed:

1. Continued surveillance and monitoring of malaria transmission dynamics to assess the effectiveness of vaccination campaigns and other control measures.
2. Further research to refine and validate the modified mathematical model using empirical data from vaccination programs and epidemiological studies.
3. Implementation of comprehensive malaria control strategies that integrate vaccination with existing interventions such as vector control and access to treatment.
4. Collaboration between policymakers, researchers, and healthcare providers to ensure equitable access to vaccination and other malaria control measures, particularly in high-burden regions.
5. Investment in research and development of new vaccines and innovative control strategies to address emerging challenges in malaria control and elimination.

By implementing these recommendations, stakeholders can work towards achieving the goal of malaria eradication and improving public health outcomes in malaria-endemic regions.

Suggestions for Further Studies

Scholars can research on the feasibility and effectiveness of different strategies for implementing the malaria vaccine within existing healthcare systems. Compare strategies such as routine immunization programs, mass vaccination campaigns, and targeted interventions in high-burden areas to identify the most efficient approaches for achieving high vaccine coverage and impact.

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