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MATHEMATICAL MODELLING TO ASSESS THE IMPACT OF A HYPOTHETICAL VACCINE ON THE DYNAMICS OF HUMAN ONCHOCERCIASIS

MUSAH KONLAN[1,*], BAABA ABASSAWAH DANQUAH[1], ABUBAKARI IDDRISU[1,2]

[1]Department of Mathematics and Statistics, University of Energy and Natural Resources, Sunyani, Ghana

[2]Department of Mathematics, Dormaa Senior High School, Dormaa Ahenkro, Ghana

Abstract. Human Onchocerciasis (River Blindness) is a neglected tropical disease with no cure. Over the years, its control and elimination had relied on Ivermectin mass drug administration. However, some public health experts and other researchers have pointed out that Onchocerciasis complete elimination may not be achievable or may required a very long time with Ivermectin drug alone. Hence, the need for more research into other alternative control strategies to assist public health authorities. The aim of this study is to construct a mathematical model to examine the influence of a hypothetical vaccine as an additional control measure for the elimination of human Onchocerciasis. We computed the disease reproductive number with and without vaccination campaign and further derived a reduction factor by which a vaccination campaign will reduce Onchocerciasis spread in a community. The proposed model disease-free equilibrium is shown to be stable when the controlled $R_o < 1$. An equation for the disease endemic condition was derived and the conditions for the existence of two endemic equilibria when $R_o < 1$ or a globally asymptotically unique equilibrium when $R_o > 1$ were established. Local sensitivity revealed that black fly removal rate is the most significant parameter for controlling human Onchocerciasis. To examine the evolution of the model sub-classes, numerical simulations were carried out.

Keywords: mathematical model; onchocerciasis vaccine; stability analysis; local sensitivity analysis; numerical simulations.

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*Corresponding author

E-mail address: musah.konlan@uenr.edu.gh

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1. INTRODUCTION

Human Onchocerciasis is one of the most important Neglected Tropical Disease targeted for eradication by 2030 [1]. It is a parasitic infection caused by the filarial worms Onchocerca volvulus and transmitted among humans through bites from infected black flies of the genus Simulium. The disease is popularly called river blindness because it is common in remote villages located near well oxygenated fast flowing streams or rivers, where black fly vectors breed [2, 3, 4, 5]. Despite the unprecedented successes chalked by both past and present Onchocerciasis control and elimination programmes, truncating Onchocerciasis transmission chain is still a public health challenge in its endemic regions (sub-Saharan Africa, Eastern Mediterranean and some isolated foci in Latin America). Over the years, the control of human Onchocerciasis has mainly relied on Ivermectin mass drug administration (IMDA). However, several studies including mathematical modelling works have suggested that the complete eradication of Onchocerciasis may not be achievable or may require a very long time with IMDA alone than anticipated [6]. It has been reported by the World Health Organization (WHO) that, in 2023, out of about 249,543,961 people who were in need of IMDA, globally, only 172,195,090 individuals could be treated (WHO, 2023). More than 99% of the people infected with river blindness live in Tropical Africa [5, 7, 8, 9]. Some key challenges hindering the control/elimination of human Onchocerciasis especially in Sub-Saharan Africa include but not limited to: the continued existence of black fly vectors, the difficulties in administering Ivermectin drug in areas where Onchocerciasis and Loa-loa are co-endemic, the issue of cross border transmission, the lack of proper knowledge about Onchocerciasis disease management in some endemic areas, the interruption of Ivermectin drug distribution often due to the eruption of conflicts and the re-emergence of disease outbreak such as Ebola and the current Covid 19 pandemic, the emergence of Ivermectin drug resistance, the limited effect of Ivermectin drug on the adult stages of Onchocerciasis parasite (Onchocerca volvulus), the emergence of onchocerciasis-associated epilepsy, the difficulties in conveying community health personnel and other logistics to remote isolated disease endemic villages/settlements [2, 5, 9, 10, 11, 12, 13]. In the face of these challenges, alternative and complementary control strategies including the deployment of an

Onchocerciasis immunization programme are highly required if human Onchocerciasis elimination targets are to be fulfilled. Concerning Onchocerciasis vaccine, a global initiative known as TOVA, the Onchocerciasis Vaccine for Africa was launched in 2015 and up to date significant progress has been achieved towards the vaccine development as a complementary Onchocerciasis control measure [14, 15, 16, 17].

Through, mathematical modelling, the most significant ecological and epidemiological factors behind an epidemic can be unveiled. For this reason, mathematical modelling has become an effective tool box in providing insight into infectious disease dynamics and in decision making process regarding disease control strategies [5, 18]. The following are some few mathematical models that have been formulated to evaluate the implementation of some alternative control measures regarding Onchocerciasis. Researchers in [11] formulated a host-vector deterministic model and assessed the effect of trapping Onchocerciasis vectors on the transmission process and control of the disease. The authors concluded from their simulation results that even though trapping of black fly vectors can reduce the spread of the disease, trapping alone is not sufficient to eliminate Onchocerciasis disease. Hence it was recommended that the implementation of trapping of black fly vectors with other control measures may achieve Onchocerciasis elimination in the society. In [19], the authors explored the combined effect of implementing four control measures against the transmission and spread of Onchocerciasis disease. They considered Ivermectin treatment and education as controls in the human sub-population while larvae control and vector trapping were implemented to control the black fly numbers in the community. Smith et al. [20] formulated a mathematical model for the "slash and clear" intervention (clearing vegetation from black fly breeding sites) and concluded that supplementing annual IMDA programme with "slash and clear" vector control could significantly accelerate the Onchocerciasis elimination process in a sustainable manner. Routledge et al. [21] modeled the positive impact of larviciding on the biting rates of Onchocerciasis vectors in Africa. Their study revealed that focal vector control can reduce vector biting rates if high larviciding efficacy can be attained and appropriate frequency and duration of larvicidal applications can be maintained. Omondi et al. [4] proposed an optimal control model for river blindness to determine how effective the simultaneous combination of personal protection, Ivermectin treatment

and vector control strategies can help mitigate the disease. The study recommended that the elimination of Onchocerciasis in the population will depend on Ivermectin treatment and vector control. Konlan et al. [5] analyzed a human Onchocerciasis control model in the presence of infective immigrants. It was demonstrated that if infected migrants are allowed into the system, the model endemic equation is a cubic polynomial, which reduces to a polynomial of degree two in the absence of infected immigrants. Turner et al. [6] used EPIONCHO (Onchocerciasis transmission model) to investigate the long term effects of using an Onchocerciasis vaccine to control the disease in areas where Ivermictin cannot be administered. To the best of our knowledge, except for the work conducted in [6] on the impact of Onchocerciasis vaccination campaign using a microsimulation model, no deterministic model has ever been formulated to evaluate the influence of a vaccine on the transmission dynamics of river blindness. Hence the need for this study to fill that gap.

2. HUMAN ONCHOCECIASIS CONTROL MODEL FORMULATION

To proposed a mathematical model for the control of human Onchocerciasis, we consider two interacting sub-models consisting of humans and black flies. The human sub-population is made up of five sub-classes: susceptible (S_h), vaccinated (V_h), exposed (E_h), infected (I_h) and treated (T_h) sub-classes. This implies that at any point in time, the total human population is:

$$N_h(t) = S_h(t) + V_h(t) + E_h(t) + I_h(t) + T_h(t) \tag{1}$$

For this model, the susceptible human class is generated at a constant rate π_h consisting of new births and immigrants. Some of these susceptible humans are vaccinated against the disease and move to the vaccinated class at a rate φ or become exposed to river blindness infections through effective contact with infected black fly at a rate λ_h. It is also assumed here that the vaccine is not 100% efficient and also does not provide long-life protection. Hence vaccinated humans either become exposed to the disease but at a reduced rate $(1-\sigma)\lambda_h$ or return back to susceptible class at a rate ω, where $\sigma \in (0,1)$ is the efficacy of the vaccine. Some Exposed humans receive Ivermectin drug and move to treated class at rate ϕ while others progress to infected class at rate γ_h. Infected humans receive Ivermectin treatment and move to treatment class at rate ψ. The density of each human compartment is further reduced at a constant rate μ_h. Human

Onchocerciasis is not fatal, hence there is no Onchocerciasis induced death associated with the model. According to [22], some individuals under Ivermectin treatment are still transmitting the disease. Thus, the degree of infectiousness of a human under Ivermectin drug is denoted by ρ. The black fly sub-population on the other hand, is stratified into three classes: susceptible (S_b), exposed (E_b) and infected (I_b) vectors. The susceptible black fly population is generated at a rate π_b and move to exposed vector class at a rate λ_b. The exposed black fly vectors (E_b) become infected at rate γ_b. The population of each black fly vector class is reduced due to natural death at rate μ_b. Thus, at any time t, the black fly population under study (N_b) satisfies:

$$N_b(t) = S_b(t) + E_b(t) + I_b(t) \tag{2}$$

$\lambda_h = \frac{\theta\beta_h I_b}{N_h}$ is the force of infection for the human while that of the black fly is denoted by $\lambda_b = \frac{\theta\beta_b(I_h+\rho T_h)}{N_h}$. The infection dynamics of human Onchocerciasis is illustrated by the diagram in Figure 1. The table below defines the model parameters.

TABLE 1. Onchocerciasis Model Parameter Description

Parameter	**Epidemiological Description**	**Value**	**Source**	**Unit**
π_h	Human recruitment rate	0.031	[23]	$/day$
φ	Progression rate from susceptible humans to vaccinated class	0.5	Assumed	–
ω	Progression rate from vaccinated class to susceptible humans	0.3	Assumed	–
σ	Efficacy of the vaccine	0.5	Assumed	–
μ_h	Natural mortality rate of human	1/23178	[23]	$/day$
β_h	Probability of Onchocerciasis disease being transmitted from an infected/treated human to a susceptible black fly	0.073	[23]	–
γ_h	Progression rate from exposed to infected humans	0.0037	[4]	$/day$
ϕ	Progression rate from exposed humans to treatment class	0.0233	[4]	$/day$

Table 1 (continued)

Parameter	Epidemiological Description	Value	Source	Unit
ψ	Progression rate from infected humans to treatment class	0.0217	[5]	$/day$
ρ	Degree of infectiousness of treated humans	0.0276	[4]	–
π_b	Black fly recruitment rate	0.73	[23]	$/day$
θ	Black fly biting rate	0.0855	[23]	$/day$
β_b	Probability of transmitting Onchocerciasis infections from an infected black fly to a susceptible human	0.080	[23]	–
γ_b	Progression rate of exposed black fly to infected black fly	0.13384	[4]	$/day$
μ_b	Natural mortality rate of black fly	0.02897	[4]	$/day$

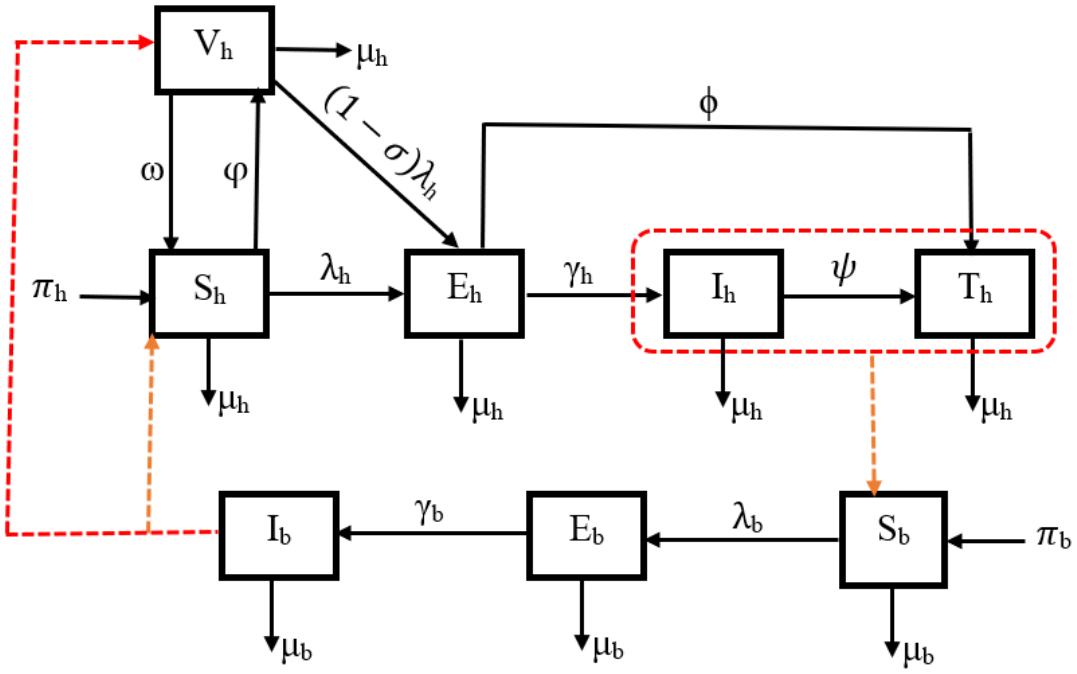

FIGURE 1. Schematic Diagram for Onchocerciasis Dynamics

The differential equations describing Figure 1 above are as follows:

$$
\begin{cases}
\dfrac{dS_h}{dt} = \pi_h + \omega V_h - \lambda_h S_h - (\varphi + \mu_h) S_h \\
\dfrac{dV_h}{dt} = \varphi S_h - (1-\sigma)\lambda_h V_h - (\omega + \mu_h) V_h \\
\dfrac{dE_h}{dt} = \lambda_h S_h + (1-\sigma)\lambda_h V_h - (\phi + \gamma_h + \mu_h) E_h \\
\dfrac{dI_h}{dt} = \gamma_h E_h - (\psi + \mu_h) I_h \\
\dfrac{dT_h}{dt} = \phi E_h + \psi I_h - \mu_h T_h \\
\dfrac{dS_b}{dt} = \pi_b - \lambda_b S_b - \mu_b S_b \\
\dfrac{dE_b}{dt} = \lambda_b S_b - (\gamma_b + \mu_b) E_b \\
\dfrac{dI_b}{dt} = \gamma_b E_b - \mu_b I_b
\end{cases} \tag{3}
$$

To ease our computations, the following notations will be used in the subsequent sections:

$Q_0 = (\varphi + \mu_h), \quad Q_1 = (\omega + \mu_h), \quad Q_2 = (\phi + \gamma_h + \mu_h),$

$Q_3 = (\psi + \mu_h) \quad \text{and} \quad Q_4 = (\gamma_b + \mu_b)$

2.1. Boundedness of Model Solutions and Model Invariant Region.

The basic qualitative properties of model (3) are summarized below:

Theorem 1. *The solution set of system (3):* $\{S_h(t),\, V_h(t),\, E_h(t),\, I_h(t),\, T_h(t),\, S_b(t),\, E_b(t)\, I_b(t)\}$ *is positive and bounded for all time* $t > 0$ *whenever each element of the initial condition set* $\{S_h(0),\, V_h(0),\, E_h(0),\, I_h(0),\, T_h(0),\, S_b(0),\, E_b(0)\, I_b(0)\}$ *is non-negative.*

Proof. Consider

$$
\frac{dS_h}{dt} = \pi_h + \omega V_h - \lambda_h S_h - Q_0 S_h \tag{4}
$$

$$
\begin{aligned}
&\Longrightarrow \frac{dS_h}{dt} \geq -(\lambda_h + Q_0) S_h \\
&\Longrightarrow \int \frac{1}{S_h} dS_h \geq -\int (\lambda_h + Q_0) dt \\
&\Longrightarrow S_h(t) \geq S_h(0) e^{-(Q_0 t + \int_0^t \lambda_h(u) du)} \geq 0
\end{aligned} \tag{5}
$$

Similarly:

$$V_h(t) \geq V_h(0)e^{-[Q_1 t+\int_0^t (1-\sigma)\lambda_h(u)du]} \geq 0, \quad E_h(t) \geq E_h(0)e^{-Q_2 t} \geq 0,$$

$$I_h(t) \geq I_h(0)e^{-Q_3 t} \geq 0, \quad T_h(t) \geq T_h(0)e^{-\mu_h t} \geq 0, \tag{6}$$

$$S_b(t) \geq S_b(0)e^{-(\mu_b t+\int_0^t \lambda_b(u)du)} \geq 0, E_b(t) \geq E_b(0)e^{-Q_4 t} \geq 0, \quad I_b(t) \geq I_b(0)e^{-\mu_b t} \geq 0$$

The above proves that system (3) has non-negative solutions.

Theorem 2. *The non-negative set:*

$$\mathscr{D} = \mathscr{D}_h \times \mathscr{D}_b \subset \mathbb{R}^5_+ \times \mathbb{R}^3_+ \tag{7}$$

where

$$\mathscr{D}_h = \left\{ (S_h, V_h, E_h, I_h, T_h) \in \mathbb{R}^5_+ : 0 \leq N_h = S_h + V_h + E_h + I_h + T_h \leq \frac{\pi_h}{\mu_h} \right\} \tag{8}$$

and

$$\mathscr{D}_b = \left\{ (S_b,\ E_b,\ I_b) \in \mathbb{R}^3_+ :\ 0 \leq N_b = S_b + E_b + I_b \leq\ \frac{\pi_b}{\mu_b} \right\} \tag{9}$$

is the invariant region for model (3)

Proof. To determine the subset $\mathscr{D}_h$, we consider:

$$\begin{aligned} N_h &= S_h + V_h + E_h + I_h + T_h \\ \Longrightarrow \frac{dN_h}{dt} &= \pi_h - \mu_h N_h \\ \Longrightarrow \frac{dN_h}{N_h - \frac{\pi_h}{\mu_h}} &= -\mu_h dt \\ \Longrightarrow N_h - \frac{\pi_h}{\mu_h} &= \left(N_h(0) - \frac{\pi_h}{\mu_h} \right) e^{-\mu_h t} \end{aligned} \tag{10}$$

taking the limit as $t \to +\infty$, of the last equation in (10) yields: $N_h \to \frac{\pi_h}{\mu_h}$

Consequently, the following result is obtained

$$0 \leq N_h \leq \frac{\pi_h}{\mu_h} \tag{11}$$

Therefore:

$$\mathscr{D}_h = \left\{ (S_h,\ V_h,\ E_h,\ I_h,\ T) \in \mathbb{R}^5_+ : 0 \leq S_h + V_h + E_h + I_h + T_h \leq \frac{\pi_h}{\mu_h} \right\} \tag{12}$$

Next, $\mathscr{D}_b$ is determined by considering the black fly sub-population defined by:

$$\begin{aligned}
&N_b = S_b + E_b + I_b \\
&\Longrightarrow \frac{d}{dt}(N_b) = \frac{d}{dt}(S_b + E_b + I_b) \\
&\Longrightarrow \frac{dN_b}{dt} = \frac{dS_b}{dt} + \frac{dE_b}{dt} + \frac{dI_b}{dt} \\
&\Longrightarrow \frac{dN_b}{dt} = \pi_b - \mu_b N_b \\
&\Longrightarrow N_b - \frac{\pi_b}{\mu_b} = \left(N_b(0) - \frac{\pi_b}{\mu_b}\right) e^{-\mu_b t} \\
&\Longrightarrow 0 \leq N_b \leq \frac{\pi_b}{\mu_b} \quad \text{as} \quad t \to +\infty.
\end{aligned}$$

$$\text{Therefore,} \quad \mathscr{D}_b = \left\{ (S_b,\, E_b,\, I_b) \in \mathbb{R}^3_+ : \quad 0 \leq S_b + E_b + I_b \leq \frac{\pi_b}{\mu_b} \right\} \tag{13}$$

Thus, combining (12) and (13) yields the set:

$$\mathscr{D} = \mathscr{D}_h \times \mathscr{D}_b \subset \mathbb{R}^5_+ \times \mathbb{R}^3_+ \tag{14}$$

□

2.2. Onchocerciasis Disease-Free Equilibrium Point (DFE). The Onchocerciasis model's DFE state is the non-trivial solution obtained by setting the right hand side of system (3) to zero. We are reminded here that at the DFE state no one in the population is exposed, infected or treated. That is $E_h = I_h = T_h = E_b = I_b = 0$. Hence, substituting this condition into system (3), we obtain the DFE of the Onchocerciasis model is given by

$$\xi^* = (S_h^*,\, V_h^*,\, 0,\, 0,\, 0,\, S_b^*,\, 0,\, 0) \tag{15}$$

where: $S_h^* = \frac{Q_1 \pi_h}{Q_0 Q_1 - \varphi\omega}$, $\quad V_h^* = \frac{\varphi \pi_h}{Q_0 Q_1 - \varphi\omega}$ *and* $S_b^* = \frac{\pi_b}{\mu_b}$

2.3. The Basic Reproductive Number(R_o). In epidemiological modelling, R_o is a threshold parameter that provides updates on the evolution of an epidemic. It is often computed using the method of next generating matrix. Adopting this method, we decompose the disease sub-model of system (3) in the form $\frac{dX}{dt} = (\mathscr{F}_i - \mathscr{V}_i)X^T$ where X^T is the transpose of

$X = (E_h,\ I_h,\ T_h,\ E_b,\ I_b)$. Hence, $\mathscr{F}_i$ and $\mathscr{V}_i$ are respectively given as:

$$\mathscr{F}_i = \begin{pmatrix} \lambda_h S_h + (1-\sigma)\lambda_h V_h \\ 0 \\ 0 \\ \lambda_b S_b \\ 0 \end{pmatrix} \text{ and } \mathscr{V}_i = \begin{pmatrix} Q_2 E_h \\ -\gamma_h E_h + Q_3 I_h \\ -\phi E_b - \psi I_h + \mu_m T_h \\ Q_4 E_b \\ -\gamma_b E_b + \mu_b I_b \end{pmatrix} \tag{16}$$

Computing the Jacobian matrices of $\mathscr{F}_i$ and $\mathscr{V}_i$ with respect to E_h, I_h, T_h, E_b, I_b at the DFE, we obtain:

(17)

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\theta\beta_h}{N_h^*}\left(S_h^* + (1-\sigma)V_h^*\right) \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\theta\beta_b S_b^*}{N_h^*} & \frac{\rho\theta\beta_b S_b^*}{N_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} Q_2 & 0 & 0 & 0 & 0 \\ -\gamma_h & Q_3 & 0 & 0 & 0 \\ -\phi & -\psi & \mu_h & 0 & 0 \\ 0 & 0 & 0 & g_4 & 0 \\ 0 & 0 & 0 & -\gamma_b & \mu_b \end{pmatrix}$$

It follows from (17) that:

$$V^{-1} = \begin{pmatrix} \frac{1}{Q_2} & 0 & 0 & 0 & 0 \\ \frac{\gamma_h}{Q_2 Q_3} & \frac{1}{Q_3} & 0 & 0 & 0 \\ \frac{\gamma_h\psi + \phi Q_3}{Q_2 Q_3 \mu_h} & \frac{\psi}{Q_3\mu_h} & \frac{1}{\mu_h} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{Q_4} & 0 \\ 0 & 0 & 0 & \frac{\gamma_b}{Q_4\mu_b} & \frac{1}{\mu_b} \end{pmatrix} \tag{18}$$

Substituting $S_h^* = \frac{Q_1\pi_h}{Q_0Q_1 - \varphi\omega}$, $V_h^* = \frac{\varphi\pi_h}{Q_0Q_1-\varphi\omega}$, $S_b^* = \frac{\pi_b}{\mu_b}$ *and* $N_h^* = \frac{\pi_h}{\mu_h}$ into the expression of F in (17) and multiplying it with V^{-1}, gives the next generation matrix FV^{-1} as:

(19)
$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{\theta\beta_h\gamma_b[Q_1+(1-\sigma)\varphi]}{Q_4\mu_b(Q_0Q_1-\omega\varphi)} & \frac{\theta\beta_h[Q_1+(1-\sigma)\varphi]}{\mu_b(Q_0Q_1-\omega\varphi)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\theta\beta_b\pi_b[\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)]}{Q_2Q_3\pi_h\mu_b} & \frac{\theta\beta_b\pi_b(\rho\psi+\mu_h))}{Q_3\pi_h\mu_b} & \frac{\rho\theta\beta_b\pi_b}{\pi_h\mu_b} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

Solving for the eigenvalue of FV^{-1}, we obtain:

$\lambda_1 = -\sqrt{\frac{\theta^2\beta_b\beta_h\gamma_b\pi_b\mu_h(Q_1+(1-\sigma)\varphi)(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))}{Q_2Q_3Q_4\pi_h\mu_b^2(Q_0Q_1-\omega\varphi)}}$ $\lambda_2 = \lambda_3 = \lambda_4 = 0$ and

$\lambda_5 = +\sqrt{\frac{\theta^2\beta_b\beta_h\gamma_b\pi_b\mu_h(Q_1+(1-\sigma)\varphi)(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))}{Q_2Q_3Q_4\pi_h\mu_b^2(Q_0Q_1-\omega\varphi)}}$

Now, according to [24], the disease reproductive ratio is defined as $R_o = \Gamma(FV^{-1})$, where $\Gamma(M)$ represents the spectral radius of a given matrix M. Thus, following the above description, the reproductive number of our Onchocerciasis model is λ_5. That is:

(20)
$$R_o = \sqrt{\frac{\theta^2\beta_b\beta_h\gamma_b\pi_b\mu_h\left(Q_1+(1-\sigma)\varphi\right)\left(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)\right)}{Q_2Q_3Q_4\pi_h\mu_b^2\left(Q_0Q_1-\omega\varphi\right)}}$$

It is easy to see that our model's reproductive number can be expressed in the form: $R_o = \sqrt{R_o^h \times R_o^b}$ where:

$$R_o^h = \frac{\theta\beta_h\mu_h\left(Q_1+(1-\sigma)\varphi\right)\left(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)\right)}{Q_2Q_3\pi_h\left(Q_0Q_1-\omega\varphi\right)} \quad \text{and} \quad R_o^b = \frac{\theta\beta_b\gamma_b\pi_b}{Q_4\mu_b^2}$$

We note here that R_o^h and R_o^b quantify the individual contributions of hosts and vectors to the overall local Onchocerciasis transmission process in the community under consideration [5].

2.4. Impact of Vaccination Campaign on Onchocerciasis Spread. It is easy to see from (20) that in the absence of vaccination campaign ($\varphi = 0$), R_o becomes say

(21)
$$R_{owv}^2 = \frac{\theta^2\beta_b\beta_h\gamma_b\pi_b\left(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)\right)}{Q_2Q_3Q_4\pi_h\mu_b^2}$$

Using, (20) and (21), we obtain the relation

$$R_o = \sqrt{\frac{Q_1 + (1-\sigma)\varphi}{Q_1 + \varphi}} R_{owv} \tag{22}$$

Since, $\frac{Q_1+(1-\sigma)\varphi}{Q_1+\varphi} \leq 1$, we conclude that an Onchocerciasis vaccination campaign will reduce the disease spread by $\sqrt{\frac{Q_1+(1-\sigma)\varphi}{Q_1+\varphi}}$.

2.5. Stability Analysis of Onchocerciasis Disease-Free Equilibrium Point.

2.5.1. *Local Stability.*

Theorem 3. *The Onchocerciasis DFE state* $(\xi^*) = \left(\frac{Q_1\pi_h}{Q_0Q_1-\varphi\omega}, \frac{\varphi\pi_h}{Q_0Q_1-\varphi\omega}, 0, 0, 0, \frac{\pi_b}{\mu_b}, 0, 0\right)$ *admits a locally asymptotic stability (LAS) if* $R_o < 1$ *and is unstable if* $R_o > 1$

Proof. To investigate the stability conditions for the model DFE (ξ^*), we adopt the linearization method. Thus, we first compute the matrix of partial derivatives denoted by $J(x)$ of system (3) with respect to the model state variables: S_h, V_h, E_h, I_h, T_h, S_b, E_b and I_b and further evaluate it at (ξ^*) to obtain

$$J(\xi^*) = \begin{pmatrix} -Q_0 & \omega & 0 & 0 & 0 & 0 & 0 & -\frac{b\beta_h S_h^*}{N_h^*} \\ \varphi & -Q_1 & 0 & 0 & 0 & 0 & 0 & -\frac{(1-\sigma)\theta\beta_h V_h^*}{N_h^*} \\ 0 & 0 & -Q_2 & 0 & 0 & 0 & 0 & \frac{\theta\beta_h}{N_h^*}(S_h^* + (1-\sigma)V_h^*) \\ 0 & 0 & \gamma_h & -Q_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi & \psi & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{\theta\beta_b S_b^*}{N_h^*} & -\frac{\rho\theta\beta_b S_b^*}{N_h^*} & -\mu_b & 0 & \\ 0 & 0 & 0 & \frac{\theta\beta_b S_b^*}{N_h^*} & \frac{\rho\theta\beta_b S_b^*}{N_h^*} & 0 & -Q_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_b & -\mu_b \end{pmatrix} \tag{23}$$

According to the Routh-Hurwitz stability theorem, matrix $J(\xi^*)$ will be stable (have eigenvalues with negative real parts) if its trace and determinant are negative and positive respectively [25]. Now, it is not hard to see that the trace of $J(\xi^*) < 0$. And,

$$\det(J(\xi^*)) = Q_2Q_3Q_4(\mu_b\mu_h)^2(\varphi + Q_1)(1 - R_o^2) \tag{24}$$

is positive if and only if $R_o < 1$. This therefore establishes the fact that the Onchocerciasis disease-free equilibrium state ξ^* admits a local asymptotic stability when $R_o < 1$ and unstable otherwise. □

2.5.2. *Global Stability.* To investigate the long term stability status of the Onchocerciasis DFE point (ξ^*), we adopt the following from [26]

Theorem 4. *For any square Metzler matrix* **B**, *the following statements are equivalent:*

(i) *Matrix* **B** *has real negative eigenvalues*

(ii) *Matrix* **B** *is non-singular and* $-\mathbf{B}^{-1}$ *is non-negative*

Theorem 5. *The Onchocerciasis DFE state* $(\xi^*) = \left(\frac{Q_1\pi_h}{Q_0Q_1-\varphi\omega}, \frac{\varphi\pi_h}{Q_0Q_1-\varphi\omega}, 0, 0, 0, \frac{\pi_b}{\mu_b}, 0, 0\right)$ *admits a global asymptotic stability (GAS) if* $R_o < 1$ *and is unstable if* $R_o > 1$

Proof. Following the method used in [19, 27, 28, 29, 30, 31, 32], system (3) is first expressed as follows:

$$\begin{cases} \frac{dY_s}{dt} = \mathscr{A}\left(Y_s - Y_{sDFE}\right) + \mathscr{B}Y_i \\ \frac{dY_i}{dt} = \mathscr{C}Y_i \end{cases} \tag{25}$$

where, $Y_s = (S_h, V_h, S_b)^T$ $Y_i = (E_h, I_h, T_h, E_b, I_b)^T$ and $Y_{DFE} = \left(S_h^*, V_h^*, S_b^*\right) = \left(\frac{Q_1\pi_h}{Q_0Q_1-\omega\varphi}, \frac{Q_1\pi_h}{Q_0Q_1-\omega\varphi}, \frac{\pi_b}{\mu_b}\right)$

$$(Y_s - Y_{sDFE}) = \begin{cases} S_h - \frac{Q_1\pi_h}{Q_0Q_1-\omega\varphi} \\ V_h - \frac{\varphi\pi_h}{Q_0Q_1-\omega\varphi} \\ S_b - \frac{\pi_b}{\mu_b} \end{cases} \tag{26}$$

$$\mathscr{A} = \frac{\partial Y_s}{\partial(S_h,\, V_h,\, S_b)}, \quad \mathscr{B} = \frac{\partial Y_s}{\partial(E_h,\, I_h,\, T_h,\, E_b,\, I_b)}, \quad \mathscr{C} = \frac{\partial Y_i}{\partial(E_h,\, I_h,\, T_h,\, E_b,\, I_b)} \tag{27}$$

That is:

$$\mathscr{A} = \begin{pmatrix} -Q_0 & \omega & 0 \\ \varphi & -Q_0 & 0 \\ 0 & 0 & -\mu_b \end{pmatrix} \tag{28}$$

$$\mathscr{B} = \begin{pmatrix} 0 & 0 & 0 & 0 & -\frac{\theta\beta_h S_h^*}{N_h^*} \\ 0 & 0 & 0 & 0 & -\frac{(1-\sigma)\theta\beta_h V_h^*}{N_h^*} \\ 0 & -\frac{\theta\beta_b S_b^*}{N_h^*} & -\frac{\rho\theta\beta_b S_b^*}{N_h^*} & 0 & 0 \end{pmatrix} \tag{29}$$

(30)

$$\mathscr{C} = \begin{pmatrix} -Q_2 & 0 & 0 & 0 & \frac{\theta\beta_h}{N_h^*}\left(S_h^* + (1-\sigma)V_h^*\right) \\ \gamma_h & -Q_3 & 0 & 0 & 0 \\ \phi & \psi & -\mu_h & 0 & 0 \\ 0 & \frac{\theta\beta_b S_b^*}{N_h^*} & \frac{\rho\theta\beta_b S_b^*}{N_h^*} & -Q_4 & 0 \\ 0 & 0 & 0 & \gamma_b & -\mu_b \end{pmatrix} = \begin{pmatrix} -Q_2 & 0 & 0 & 0 & K_1 \\ \gamma_h & -Q_3 & 0 & 0 & 0 \\ \phi & \psi & -\mu_h & 0 & 0 \\ 0 & K_2 & \rho K_2 & -Q_4 & 0 \\ 0 & 0 & 0 & \gamma_b & -\mu_b \end{pmatrix}$$

where $K_1 = \frac{\theta\beta_h\mu_h[Q_1+(1-\sigma)\varphi]}{Q_0Q_1-\varphi\omega}$ and $K_2 = \frac{\theta\beta_b\mu_h\pi_b}{\pi_h\mu_b}$

Now, to conclude our proof, we need to establish that all the eigenvalues of matrix $\mathscr{A}$ are negative while matrix $\mathscr{C}$ is metzler stable.

Clearly, there is nothing to dispute that $-\mu_b < 0$ is one eigenvalue of matrix $\mathscr{A}$ and the other remaining two eigenvalues can be obtained from the reduced matrix:

$$\mathscr{M} = \begin{pmatrix} -Q_0 & \omega \\ \varphi & -Q_1 \end{pmatrix} \tag{31}$$

The trace of matrix $\mathscr{M} = -(Q_0+Q_1) < 0$ and its determinant is $Q_0Q_1 - \varphi\omega = \mu_h(\varphi+Q_1) > 0$. This is enough to conclude that the matrix $\mathscr{A}$ has real and negative eigenvalues.

Next, we use Theorem 4 to verify the stability of the Metzler matrix $\mathscr{C}$

$$-\mathscr{C}^{-1} = \left(\frac{1}{1-R_o^2}\right)\begin{pmatrix} C_{11} & C_{12} & C_{13} & C_{14} & C_{15} \\ C_{21} & C_{22} & C_{23} & C_{24} & C_{25} \\ C_{31} & C_{32} & C_{33} & C_{34} & C_{35} \\ C_{41} & C_{42} & C_{43} & C_{44} & C_{45} \\ C_{51} & C_{52} & C_{53} & C_{54} & C_{55} \end{pmatrix} \tag{32}$$

$$
\begin{cases}
C_{11} = Q_3Q_4\mu_b\mu_h, \quad C_{12} = K_1K_2\gamma_b(\rho\psi+\mu_h), \quad C_{13} = \rho\gamma_bK_1K_2Q_3, \quad C_{14} = \gamma_bK_1Q_3\mu_h \\
C_{15} = K_1Q_3Q_4\mu_h \quad C_{21} = Q_4\gamma_h\mu_b\mu_h \quad C_{22} = Q_2Q_4\mu_b\mu_h(1-H_1) \quad C_{23} = \rho\gamma_b\gamma_hK_1K_2 \\
C_{24} = \gamma_b\gamma_hK_1\mu_h, \quad C_{25} = K_1Q_4\gamma_h\mu_h, \quad C_{31} = Q_4\mu_b(\psi\gamma_h+\phi Q_3), \quad C_{32} = Q_2Q_4\psi\mu_b + K_1K_2\phi\gamma_b \\
C_{33} = Q_2Q_3Q_4\mu_b(1-R_{owt}^2) \quad C_{34} = K_1Q_4(\phi Q_3+\psi\gamma_h), \quad C_{35} = K_1\gamma_b(\phi Q_3+\psi\gamma_h) \\
C_{41} = K_2\mu_b[\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)] \quad C_{42} = K_2Q_2\mu_b(\mu_h+\rho\psi) \quad C_{43} = K_2\rho\mu_bQ_2Q_3 \\
C_{44} = \mu_b\mu_hQ_2Q_3 \quad C_{45} = K_1K_2[\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h)] \quad C_{51} = K_2\gamma_b\gamma_h(\mu_h+\phi Q_3+\rho\psi) \\
C_{52} = K_2\gamma_bQ_2(\mu_h+\rho\psi) \quad C_{53} = \rho K_2\gamma_bQ_2Q_3 \quad C_{54} = \gamma_bQ_2Q_3\mu_h \quad C_{55} = Q_2Q_3Q_4\mu_h \\
\\
H_1 = \frac{\phi\rho\theta^2\beta_b\beta_h\gamma_b\pi_b\mu_h(Q_1+(1-\sigma)\varphi)}{Q_2Q_4\pi_h\mu_b^2(Q_0Q_1-\omega\varphi)}, \quad R_{owt}^2 = \frac{\theta^2\beta_b\beta_h\gamma_b\gamma_h\pi_b\mu_h^2(Q_1+(1-\sigma)\varphi)}{Q_2Q_3Q_4\pi_h\mu_b^2(Q_0Q_1-\omega\varphi)}
\end{cases} \tag{33}
$$

Now, $-\mathscr{C}^{-1} \geq 0$ if each entry of the matrix in (33) is non-negative and $R_o < 1$. Owing to the non-negativity of the model parameters, all the entries of matrix $-\mathscr{C}^{-1}$ are obviously non-negative if $R_o < 1$ except for C_{22} and C_{33}.

Before we proceed, we need to establish the signs of C_{22} and C_{33}. To examine the positivity of C_{22}, we expressed R_o in the form

$$R_o = \sqrt{H_0 + H_1} \tag{34}$$

where

$$H_o = \frac{\theta^2\beta_b\beta_h\gamma_b\pi_b\mu_h\,(Q_1+(1-\sigma)\varphi)\,(\gamma_h\mu_h+\rho\psi\gamma_h)}{Q_2Q_3Q_4\pi_h\mu_b^2\,(Q_0Q_1-\omega\varphi)} \tag{35}$$

Since $H_o > 0$ it follows that $R_o < 1 \implies H_1 < 1$. Thus, $C_{22} > 0$ whenever $R_o < 1$.

Also, it is not hard to see that $R_{owt} < R_o$. Thus, $R_o < 1 \implies R_{owt} < 1$, and this proves the positivity of C_{33}.

Therefore, $-\mathscr{C}^{-1} \geq 0$ if and only if $R_o < 1$. Hence, the Onchocerciasis free equilibrium state admits a GAS whenever $R_o < 1$. □

Here, it is important to observe that R_{owt} is the value of R_o in the absence of transmission of infections from the humans under Ivermectin drug treatment ($\rho = 0$). Therefore, this analysis stressed on the importance of effective Ivermectin treatment in the control of Onchocerciasis epidemic.

2.6. Existence of Onchocerciasis Endemic Equilibrium (EEP). In the study of dynamical systems, it is well known that any perturbed system will definitely settle at an equilibrium point as time evolves. Likewise, the evolution of an epidemic will admit an endemic equilibrium state. Thus, in this section, we explore the conditions for the existence of Onchocerciasis endemic equilibrium point.

Let $\xi^{**} = (S_h^{**}, V_h^{**}, E_h^{**}, I_h^{**}, T_m^{**}, S_b^{**}, E_b^{**}, I_b^{**})$ denote the endemic equilibrium point (EEP) of the proposed model. At the EEP, system (3) takes the following form:

$$
\begin{cases}
\pi_h + \omega V_h^{**} - (\lambda_h^{**} + Q_0) S_h^{**} = 0 \\
\varphi S_h^{**} - \left[(1-\sigma)\lambda_h^{**} + Q_1\right] V_h^{**} = 0 \\
\lambda_h^{**} S_h^{**} + (1-\sigma)\lambda_h^{**} V_h^{**} - Q_2 E_h = 0 \\
\gamma_h E_h^{**} - Q_3 I_h^{**} = 0 \\
\phi E_h^{**} + \omega E_h^{**} - \mu_h T_h^{**} = 0 \\
\pi_b - (\lambda_b^{**} + \mu_b) S_b^{**} \\
\lambda_b^{**} S_b^{**} - Q_4 E_b^{**} = 0 \\
\gamma_b E_b^{**} - \mu_b I_b^{**} = 0
\end{cases} \tag{36}
$$

Solving for the non-trivial solution of (36), yields the following system:

$$
\begin{cases}
S_h^{**} = \frac{\pi_h\left(Q_1+(1-\sigma)\lambda_h^{**}\right)}{(Q_0+\lambda_h^{**})[Q_1-(1-\sigma)\lambda_h^{**}]-\varphi\omega} \\
V_h^{**} = \frac{\varphi\pi_h}{(Q_0+\lambda_h^{**})[Q_1-(1-\sigma)\lambda_h^{**}]-\varphi\omega} \\
E_h^{**} = \frac{\pi_h\left(Q_1+(1-\sigma)(\varphi+\lambda_h^{**})\right)\lambda_h^{**}}{Q_2(Q_0+\lambda_h^{**})[Q_1-(1-\sigma)\lambda_h^{**}]-Q_2\varphi\omega} \\
I_h^{**} = \frac{\gamma_h\pi_h\left(Q_1+(1-\sigma)(\varphi+\lambda_h^{**})\right)\lambda_h^{**}}{Q_2Q_3(Q_0+\lambda_h^{**})[Q_1-(1-\sigma)\lambda_h^{**}]-Q_2Q_3\varphi\omega} \\
T_h^{**} = \frac{\phi E_h^{**}+\psi I_h^{**}}{\mu_h} \\
S_b^{**} = \frac{Q_3\pi_b\pi_h}{Q_3\pi_h\mu_b+\theta\beta_b(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))E_h^{**}} \\
E_b^{**} = \frac{\theta\beta_b\pi_b(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))E_h^{**}}{Q_4\left(Q_3\pi_h\mu_b+\theta\beta_b(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))E_h^{**}\right)} \\
I_b^{**} = \frac{\theta\beta_b\gamma_b\pi_b(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))E_h^{**}}{Q_4\mu_b\left(Q_3\pi_h\mu_b+\theta\beta_b(\gamma_h\mu_h+\rho(\phi Q_3+\psi\gamma_h))E_h^{**}\right)}
\end{cases} \tag{37}
$$

It is not hard to see that with $\lambda_h^{**} = 0$, system (37) becomes nothing but ξ^*, while $\lambda_h^{**} \neq 0$ generates the quadratic equation:

$$A_2\lambda_h^{**2} + A_1\lambda_h^{**} + A_0 = 0 \tag{38}$$

Where:

$$A_0 = (Q_0Q_1 - \omega\varphi)Q_2Q_3Q_4\pi_h\mu_b^2\left(1 - R_o^2\right) \tag{39}$$

$$A_1 = [(1-\sigma)Q_0 + Q_1]Q_2Q_3Q_4(\pi_h\mu_b)^2 - (1-\sigma)\theta^2\beta_b\beta_h\gamma_b\pi_b\pi_h\mu_h\left(\gamma_h\mu_h + \rho(\phi Q_3 + \psi\gamma_h)\right) \tag{40}$$

$$A_2 = (1-\sigma)Q_3\pi_h^2\mu_b\{Q_2Q_4\mu_b + \theta\beta_b\left(\gamma_h\mu_h + \rho(\phi Q_3 + \psi\mu_h)\right)\} \tag{41}$$

From (38), we establish the following results:

Theorem 6. *The proposed Onchocerciasis control model (3) may have:*

(i) *One unique EEP when* $A_0 < 0$ $(R_o > 1)$

(ii) *One unique EEP when* $A_1 < 0$, *and* $R_o = 1$ *or* $A_1^2 - 4A_0A_2 = 0$

(iii) *Two EEP when* $A_1 < 0$ *and* $A_0 > 0$ $(R_o < 1)$ *or* $A_1^2 - 4A_0A_2 > 0$

(iv) *No EEP otherwise.*

Case (iii) of theorem 6 provides the conditions for system (3) to have two positives endemic equilibrium when $R_o < 1$. This suggests that the model may undergo backward bifurcation. In this case, just keeping R_o below one does not guarantee the elimination of Onchocerciasis in the community. By equating the discriminant (Δ) of $A_2\lambda_h^{**2} + A_1\lambda_h^{**} + A_0 = 0$ to zero, we determine R_o^c, the critical value of R_o for which backward bifurcation occurs,

$$\Delta = 0 \implies A_1^2 - 4A_0A_2 = 0$$

$$\implies R_o^c = \sqrt{1 - \frac{A_1^2}{4(Q_0Q_1 - \omega\varphi)Q_2Q_3Q_4\pi_h\mu_b^2A_2}}$$

Hence, for values of R_o between $R_o^c < R_o < 1$, system (3) will experience backward bifurcation. This also implies that for the disease to be eliminated, R_o needs to pushed below R_o^c. Case (i) of theorem 6 suggests that system (3) admits a unique EEP when $R_o > 1$. Thus, in the next section, we use a Lyapunov function to explore the stability status of this EEP within its global neighborhood.

2.7. Global Stability of the Onchocerciasis Endemic Equilibrium Point (EEP).

Theorem 7. *The unique EEP* (ξ^{**}) *of system (3) admits a global asymptotic stability.*

Proof. As in Goswami et al. [33], we consider a positive definite function $\mathscr{L}$ defined by:

$$\mathscr{L}(x^{**}) = \sum_{i=1}^{n} \frac{1}{2}(x_i - x_i^{**})^2 \quad i = i^{th} \text{ sub-model compartment}$$

Now,

$$\begin{aligned} \mathscr{L}(\xi^{**}) &= \frac{1}{2}\left(S_h - S_h^{**} + V_h - V_h^{**} + E_h - E_h^{**} + I_h - I_h^{**} + T_h - T_h^{**}\right)^2 \\ &+ \frac{1}{2}\left(S_b - S_b^{**} + E_b - E_b^{**} + I_b - I_b^{**}\right)^2 \end{aligned} \tag{42}$$

$$\begin{aligned} \Longrightarrow \frac{d\mathscr{L}(\xi^{**})}{dt} &= \left(S_h - S_h^{**} + V_h - V_h^{**} + E_h - E_h^{**} + I_h - I_h^{**} + T_h - T_h^{**}\right)\frac{dN_h}{dt} \\ &+ \left(S_b - S_b^{**} + E_b - E_b^{**} + I_b - I_b^{**}\right)\frac{dN_b}{dt} \\ &= (N_h - N_h^{**})(N_h - \mu_h N_h) + (N_b - N_b^{**})(N_b - \mu_b N_b) \\ &= -\mu_h(N_h - N_h^{**})^2 - \mu_b(N_b - N_b^{**})^2 \\ &= -\left[\mu_h(N_h - N_h^{**})^2 + \mu_b(N_b - N_b^{**})^2\right] \end{aligned} \tag{43}$$

We conclude from (43) that $\frac{d\mathscr{L}}{dt} < 0$ and $\frac{d\mathscr{L}}{dt} = 0$ when $N_h = N_h^{**}$ and $N_b = N_b^{**}$ that is $s_h^{**} = S_h$, $V_h^{**} = V_h$, $E_h^{**} = E_h$, $I_h^{**} = I_h$, $T_h^{**} = T_h$, $S_b^{**} = S_b$, $E_b^{**} = E_b$, and $I_b^{**} = I_b$. Thus, is follows from [34], that the Onchocerciasis unique endemic equilibrium will admit a GAS whenever it exists. □

3. Sensitivity Analysis of Model Parameters

To ensure cost effectiveness for disease control, the most contributing parameters to the disease spread are targeted first if possible. To ascertain the contributions of the model parameters to the spread of the Onchocerciasis, we perform local sensitivity analysis. This help identify the parameters with higher contribution to (R_o). Using the formula:

$$\Gamma_p^{R_o} = \frac{\partial R_o}{\partial p} \times \frac{p}{R_o} \tag{44}$$

where P denotes a given parameter of R_o, we generate Table (2) below.

TABLE 2. Sensitivity Indices of R_o Parameters

Parameter	**Sensitivity index**
θ	$+1.0000$
β_h	$+0.5000$
π_h	-0.5000
μ_h	$+0.0024$
ω	$+0.0779$
φ	-0.0779
σ	-0.2273
ϕ	-0.0034
γ_h	$+0.0042$
ψ	-0.0047
ρ	$+0.4967$
β_b	$+0.5000$
π_b	$+0.5000$
γ_b	$+0.0890$
μ_b	-1.0899

The sign against an index describes the kind of relationship between the corresponding parameter and R_o. A positive sign means that increasing (decreasing) the value of the corresponding parameter will result in an increase (decrease) in the value of R_o. On the other hand, a negative sign means there is an inverse relation between the corresponding parameter and R_o [5]. Applying this explanation to Table 2 suggests that increasing the black fly removal rate from the community will yield the highest reduction in Onchocerciasis spread.

4. NUMERICAL SIMULATIONS

To examine the evolution of the model sub-classes in time, we simulated system (3) using Matlab ode45. The following values: $S_h(0) = 200,\ V_h(0) = 100,\ E_h(0) = 120,\ I_h(0) =$

70, $T_h(0) = 0$, $S_b(0) = 1000$, $E_b(0) = 150$, $I_b(0) = 30$ and the numerical values of the model parameters from Table 1 are used. The results are shown from Figure 2 to Figure 9 below

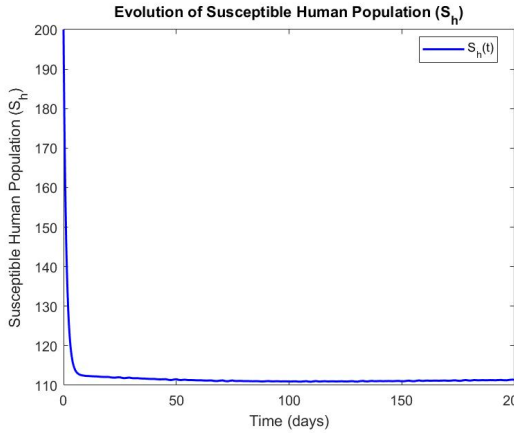

FIGURE 2. Susceptible Humans

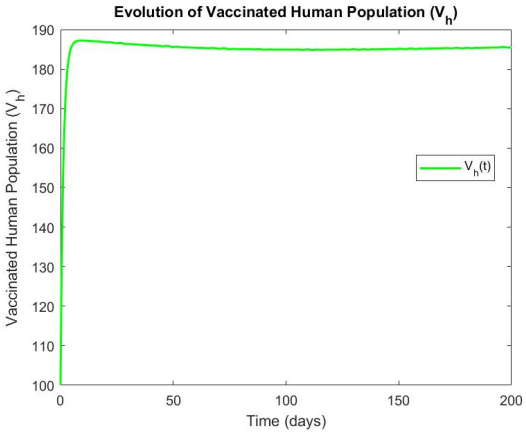

FIGURE 3. Vaccinated Humans

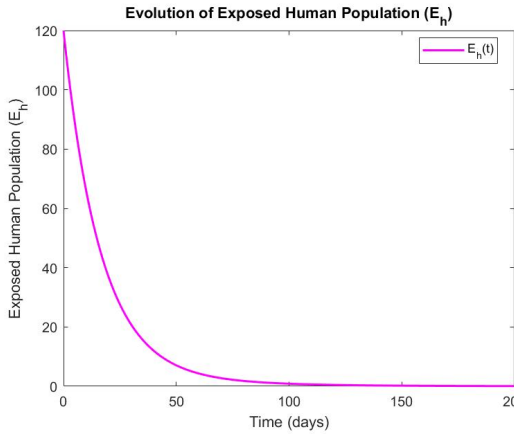

FIGURE 4. Exposed Humans

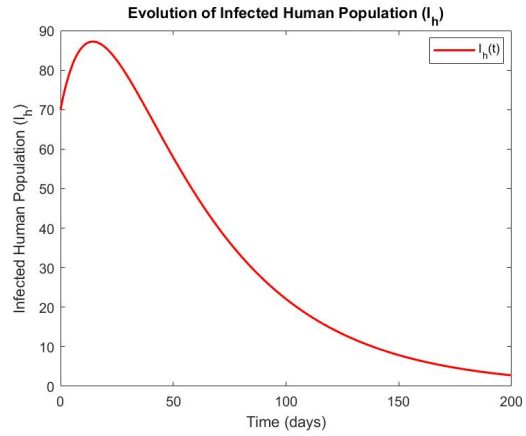

FIGURE 5. Infected Humans

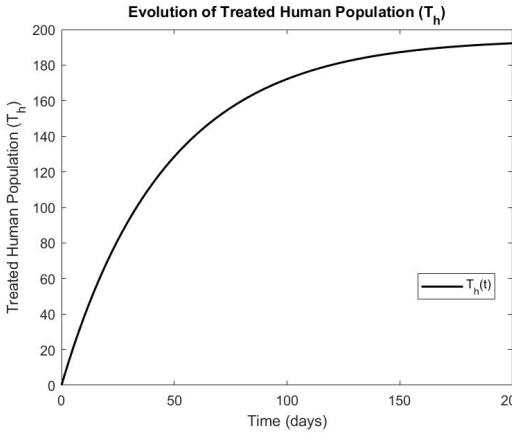

FIGURE 6. Treated Humans

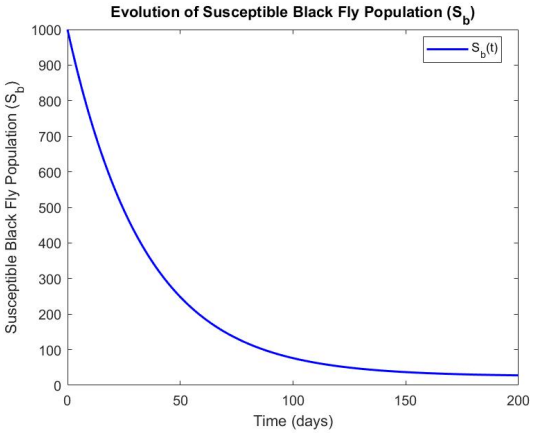

FIGURE 7. Susceptible Black Fly

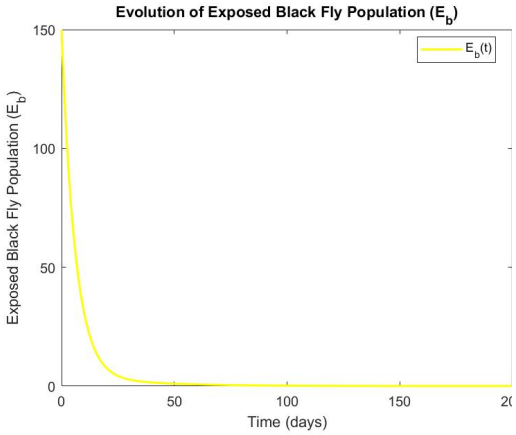

FIGURE 8. Exposed Black Fly

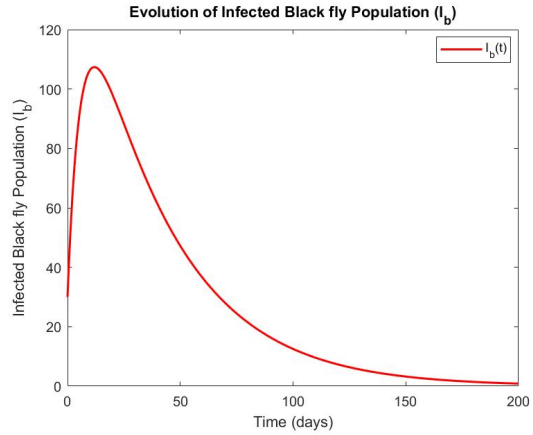

FIGURE 9. Infected Black Fly

5. CONCLUSION

In this study, we formulated a mathematical model for the control of human Onchocerciasis using a combination of Ivermectin drug and a hypothetical vaccine as an alternative control measure. The significance of the vaccine is demonstrated through the computation of a reduction factor in the disease spread that will result from launching a vaccination campaign in a community. The Onchocerciasis disease-free equilibrium state is shown to admit a locally and globally stability when $R_o < 1$. Also the conditions for the model to admit a unique and globally asymptotically stable disease persistent equilibrium or two endemic equilibria have been established. The contributions of the model parameters to the overall disease spread is established through sensitivity analysis which pointed out that Onchocerciasis vector control should not be undermined if the disease is to be eliminated. The evolution of the model sub-classes is ascertained through numerical simulations.

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DATA AVAILABILITY STATEMENT

The data used to support our numerical analysis is taken from related published Onchocerciasis works and are respectively cited.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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