

Research Article

A Mathematical Model for the Dynamics of Onchocerciasis With Vector Control and Mass Drug Administration

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Received 19 May 2023; Revised 7 June 2024; Accepted 10 June 2024

Academic Editor: Yansheng Liu

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In this paper, we investigate the transmission dynamics of onchocerciasis with asymptomatic infected humans using a mathematical model. The model incorporates interventions for treatment and vector control to evaluate the impact of these strategies. We analyse the model to determine the existence and stability of equilibrium points. Our results reveal that for the disease to persist in the community, the infection rate must exceed the sum of the treatment rate and the per capita death rate due to the disease. Sensitivity analysis highlights the critical role of the blackfly vector's average daily biting rate in disease transmission. Numerical simulations indicate that administering highly effective drugs to infected individuals significantly reduces the number of cases. Therefore, in addition to vector control, the use of highly efficient drugs is crucial for controlling the transmission of river blindness.

1. Introduction

Human onchocerciasis, or river blindness is a vector-borne tropical disease of major public and socioeconomical concern that disproportionately affects the poor and underprivileged populations, especially those that live in impoverished environments. It is one of the major contributors to preventable blindness globally [1]. Fenwick [2] reported about 37 million cases of human onchocerciasis worldwide. Among these, about 500,000 and 270,000 people are visually impaired and permanently blinded, respectively [3, 4].

It is a parasitic infectious disease caused by filarial nematode (worm) *Onchocerca volvulus* of the family Filariidae and transmitted to humans through the bite of an infected blackfly species of the genus *Simulium*. In Africa, it is caused primarily by *Simulium damnosum* sensu lato. *Simulium* blackfly vectors breed in fast-flowing, well-oxygenated rivers and streams with adequate nutrients. Once inside a human host, the adult female worm produces microfilariae, the first larval stage that migrates under the skin, where they can infect the next blackfly that bites that infected person [4].

The female blackflies require a blood meal for ovulation, as they ingest microfilariae and transmit infective-stage (third stage) larvae during the blood meal. The microfilariae (adult female worms) have an estimated mean reproductive life span of 911 years and reside primarily in subcutaneous and deep tissue nodules.

The only definitive host of *O. volvulus* are humans that harbour the adult worms. It is a chronic and slowly progressive disease that affects the skin and eyes. Its initial infestation often happens in childhood. Many individuals infected with *Ochocerciasis* remain asymptomatic for long periods of time [4]. The possibility of asymptomatic infections presents a major challenge in the control of river blindness since diagnosis of such cases is costly and time-consuming. Since humans are the only known significant reservoirs of the infection, long-lived infections and superinfections usually occur for individuals in endemic areas. Clinical manifestations occur when the microfilariae die and induce intense inflammatory responses in the host [5], usually within 1–2 years after initial infection as the infective larvae migrate and develop into adult worms. These range from no skin

or eye lesions (asymptomatic individuals) to severe disfiguring pruritic skin lesions, visual impairment, and blindness [6], attributed to the high variability of the incubation period [7]. There is no evidence to suggest that reinfection cannot occur for recovered individuals who have been treated by chemotherapy and direct transmission from human to human is not possible.

There are no vaccines or medicines available to prevent one from getting infected with *O. volvulus*. Personal protection measures against blackfly bites and vector control are the best prevention efforts. There has been large-scale vector control targeting breeding sites and mass drug administration with ivermectin that has been widely used in all endemic countries of Africa and the Americas. Elimination of onchocerciasis is grappling with many challenges. For example, asymptomatic individuals hardly accept skin snips in areas of low prevalence [8]. Another hurdle is the importation of the disease from endemic to nonendemic countries. Imported cases of river blindness due to migration and population mobility may create bridges between very different health care system capacities arising from the risk of conflict and civil war [9–12]. The immigrant humans may include seasoned workers, refugees, visitors, and tourists from endemic areas and may be infested with *O. volvulus*. With the potential of the reemergence of the disease into areas where the disease is considered eliminated and treatment with ivermectin has been stopped. Blackfly vectors from insufficiently controlled areas that migrate carry parasites into controlled areas [1, 13].

Mathematical models play a key role in providing insights into the transmission dynamics of disease. These may guide in the intervention programs, estimation of parameters, and investigation of evolutionary outcomes of control [14]. Unlike other vector-borne diseases like malaria and sleeping sickness, mathematical modelling of the dynamics of onchocerciasis has been poorly developed. However, models have been used to investigate the transmission and control of human onchocerciasis; for instance, Walker et al. [15] tested both EPIONCHO and ONCHOSIM models using data from Mali and Senegal. They noted that the use of mass treatment with ivermectin reduced the disease prevalence to zero after 15–17 years. Basanez and Bousinesq [16] developed a mathematical framework to assess the success and impact of the Onchocerciasis Control Programme in Africa. They derived a relationship between the basic reproduction number and the minimum vector density for parasite persistence in Cameroon. Cheke et al. [17] modified the model developed by Basanez and Bousinesq [16] to study the dynamics of vector population and the impact of temperature on simuliids and on *O. volvulus* within the simuliids. It was revealed that increasing temperatures are likely to reduce blackfly populations. The effect of larvicidal insecticides on the biting rates of *Simulium damnosum* (s.l.) was explored by Routledge et al. [18], and results revealed that vector biting rates can be potentially reduced through appropriate frequency and duration of larviciding with a high larvicidal efficacy. A slash and clear intervention mathematical model was proposed by Smith et al. [19] to investigate the potential for the vector control strategy towards

onchocerciasis elimination. It was revealed that annual drug treatments combined with slash and clear can enhance onchocerciasis elimination. A microsimulation model was used by Alley et al. [20] to examine the possibility of changing treatment to a hypothetical macrofilaricide from the microfilaricide ivermectin. It was revealed that it was possible to eliminate the disease with macrofilaricide, but it needed a very wide coverage level. Plaisier et al. [21] developed a simulation model to explore the reduction in disease due to different combinations of *Simulium* vector control and chemotherapy of humans.

This paper presents a deterministic model for the transmission and control of onchocerciasis. The model is analysed to explore and understand the complex transmission dynamics of the disease from which some insights and interpretations are derived. In addition to the vector control strategy, the model assesses the significance of treatment of exposed, symptomatic, and asymptomatic humans through mass drug administration which has not been effectively addressed in previous models. The rest of this paper is organised as follows: Section 2 gives the description and formulation of the susceptible-exposed-infectious-recovered (SEIR) model for the human population and SI model for the blackfly population under mass drug treatment of the exposed, asymptomatic, and symptomatic human population. In Section 3, we study the existence and positivity of solutions, obtain equilibrium and analyse the stability of the equilibrium points, compute the effective reproduction number, carry out the sensitivity analysis of the model parameters, and use the center manifold theory to study the conditions for the backward bifurcations. Numerical simulations are performed in Section 4, and we discuss results and derive conclusions in Section 5.

2. Model Description and Formulation

In this section, the formulation of a mathematical model for the transmission and spread of onchocerciasis between two interacting populations of humans (hosts) and blackflies (vectors) is presented. The total human and female blackfly populations at time t are denoted by $N_H(t)$ and $N_V(t)$, respectively. $N_H(t)$ is subdivided into the epidemiological classes susceptible $S_H(t)$, exposed $E_H(t)$ (defined as infected but not infectious), asymptotically infected $I_{Ha}(t)$, symptomatic infected $I_{Hs}(t)$, and recovered $R_H(t)$ population sizes, respectively. Thus, the total human population size $N_H(t) = S_H(t) + E_H(t) + I_{Ha}(t) + I_{Hs}(t) + R_H(t)$.

The blackfly vector population size, N_V comprises of the susceptible and infected classes denoted by S_V and I_V , respectively. The total blackfly vector population at time t is given by $S_V(t) + I_V(t) = N_V(t)$.

Recruitment into the human population is assumed to be at a constant rate Λ_h and corresponds to births or immigration. Let a_1 be the average daily biting rate on susceptible human by a single infectious blackfly, with b_1 as the probability that a bite infects a susceptible human. The average daily biting rate on infected human by a single susceptible blackfly is denoted by a_2 with b_2 as the probability that a bite infects a susceptible blackfly. The interaction of humans and female blackflies is modeled by standard incidence as used in

TABLE 1: Model parameters and their interpretations.

Parameter	Description
Λ_h	Recruitment rate for human hosts
Λ_v	Recruitment rate for blackfly vectors
a_1	Average daily biting rate on susceptible human by a single infectious blackfly
a_2	Average daily biting rate on infected human by a single susceptible blackfly
b_1	Probability that a bite infects a susceptible human host
b_2	Probability that a bite infects a susceptible blackfly
ϵ	Fraction of asymptomatic infected humans
γ_v	Efficacy of the vector control strategy
δ	Progression rate of exposed humans to the infected class
μ_h	Per capita natural death rate of humans
μ_v	Per capita natural death rate of blackflies
μ_{vc}	Blackfly vector death rate due to vector control
α	Per capita death rate of infected humans
m	Number of female blackflies per human host
τ_e	Treatment rate of exposed individuals
τ_a	Treatment rate of asymptomatic infected infectious individuals
τ_s	Treatment rate of symptomatic infectious individuals
γ_e	Efficiency of treatment of exposed individuals
γ_a	Efficiency of treatment of asymptomatic infectious individuals
γ_s	Efficiency of treatment of symptomatic infectious individuals

Tumwiine et al. [22], with the terms $a_1 b_1 S_H I_V / N_H$ representing the rate at which susceptible human hosts S_H are infected by infective blackfly vectors I_V and $a_2 b_2 S_V (I_{Ha} + I_{Hs}) / N_H$ denoting the rate at which the susceptible blackflies S_V are infected by infected humans ($I_{Ha} + I_{Hs}$). Let a fraction ϵ of infected humans become asymptomatic and the remaining fraction $(1 - \epsilon)$ be infected humans who are symptomatic. The mass drug administration targets infected individuals at a constant treatment rate with efficiency γ . Thus, τ_e , τ_a , and τ_s are the cure rates due to the treatment of individuals in the exposed class, asymptomatic, and symptomatic classes, respectively. The efficiency of treatment in the respective classes are γ_e , γ_a and γ_s . The exposed humans progress to the infected class at a rate δ . There is a nondisease-related per capita natural death rate μ_h in all humans and an additional disease-related death rate α in the asymptomatic and symptomatic infected classes. The blackfly population has the per capita birth rate and per capita death rate Λ_v and μ_v , respectively. It is assumed that vector control results into an additional death rate μ_{vc} in the blackfly population. The description of model parameters is presented in Table 1 below.

We use SEIR model for the human population and SI (susceptible-infectious) model for the blackfly vector population. The blackfly vector population has no recovered class (black flies never recover from the infection and carry the infection throughout their life span). Thus, there is no recovered class for the blackfly vectors because of their short life cycle. Figure 1 presents the progression of onchocerciasis in the human and blackfly vector populations.

The dynamics of river blindness is given by the following coupled system of nonlinear ordinary differential equations depicted in System (1) below:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_h N_H - \frac{a_1 b_1 S_H I_V}{N_H} - \mu_h S_H, \\
\frac{dE_H}{dt} &= \frac{a_1 b_1 S_H I_V}{N_H} - (\gamma_e \tau_e + \delta + \mu_h) E_H, \\
\frac{dI_{Ha}}{dt} &= \epsilon \delta E_H - (\gamma_a \tau_a + \alpha + \mu_h) I_{Ha}, \\
\frac{dI_{Hs}}{dt} &= (1 - \epsilon) \delta E_H - (\gamma_s \tau_s + \alpha + \mu_h) I_{Hs}, \\
\frac{dR_H}{dt} &= \gamma_e \tau_e E_H + \gamma_a \tau_a I_{Ha} + \gamma_s \tau_s I_{Hs} - \mu_h R_H, \\
\frac{dS_V}{dt} &= \Lambda_v N_V - \frac{a_2 b_2 S_V I_{Ha}}{N_H} - \frac{a_2 b_2 S_V I_{Hs}}{N_H} - (\mu_v + \gamma_v \mu_{vc}) S_V, \\
\frac{dI_V}{dt} &= \frac{a_2 b_2 S_V I_{Ha}}{N_H} + \frac{a_2 b_2 S_V I_{Hs}}{N_H} - (\mu_v + \gamma_v \mu_{vc}) I_V,
\end{aligned} \tag{1}$$

together with $N_H = S_H + E_H + I_{Ha} + I_{Hs} + R_H$, and $N_V = S_V + I_V$, this implies that

$$\begin{aligned}
\frac{dN_H}{dt} &= (\Lambda_h - \mu_h) N_H - \alpha (I_{Ha} + I_{Hs}), \\
\frac{dN_V}{dt} &= (\Lambda_v - \mu_v - \gamma_v \mu_{vc}) N_V,
\end{aligned}$$

and initial conditions

$$\begin{aligned}
S_H(0) &= S_{H0}, E_H(0) = E_{H0}, I_{Ha}(0) = I_{Ha0}, I_{Hs}(0) \\
&= I_{Hs0}, R_H(0) = R_{H0}, S_V(0) = S_{V0}, I_V(0) = I_{V0}.
\end{aligned}$$

3. Model Analysis

System (1) is analysed to determine the existence and stability of the equilibria. Given the dynamic nature of the human and vector population, fractional variables are employed which are obtained by dividing each population by the total population. Consequently, we define $S_h = S_H / N_H$, $E_h = E_H / N_H$, $I_{ha} = I_{Ha} / N_H$, $I_{hs} = I_{Hs} / N_H$ and $R_h = R_H / N_H$, for the human population and $S_v = S_V / N_V$, $I_v = I_V / N_V$ for the

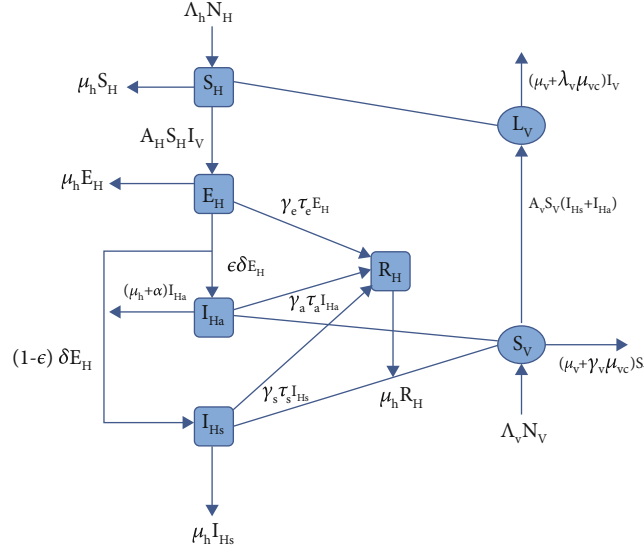


FIGURE 1: A compartmental diagram for Model (1). Lines with arrows show movement of individuals from one state to another, whereas lines without arrows show interactions between infectious humans and susceptible blackflies or infectious blackflies and susceptible humans, where $A_H = a_1 b_1 / N_H$ and $A_v = a_2 b_2 / N_H$.

blackfly population. Here the ratio $m = N_V / N_H$ is the number of female blackfly vectors per human host. The resulting system of differential equations is

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h(1 - S_h) - a_1 b_1 m S_h I_v + \alpha S_h (I_{ha} + I_{hs}), \\
 \frac{dE_h}{dt} &= a_1 b_1 m S_h I_v - (\Lambda_h + \gamma_e \tau_e + \delta) E_h + \alpha E_h (I_{ha} + I_{hs}), \\
 \frac{dI_{ha}}{dt} &= \epsilon \delta E_h - (\Lambda_h + \gamma_a \tau_a + \alpha) I_{ha} + \alpha I_{ha} (I_{ha} + I_{hs}), \\
 \frac{dI_{hs}}{dt} &= (1 - \epsilon) \delta E_h - (\Lambda_h + \gamma_s \tau_s + \alpha) I_{hs} + \alpha I_{hs} (I_{ha} + I_{hs}), \\
 \frac{dR_h}{dt} &= \gamma_e \tau_e E_h + \gamma_a \tau_a I_{ha} + \gamma_s \tau_s I_{hs} - \Lambda_h R_h + \alpha R_h (I_{ha} + I_{hs}), \\
 \frac{dS_v}{dt} &= \Lambda_v(1 - S_v) - a_2 b_2 S_v I_{ha} - a_2 b_2 S_v I_{hs}, \\
 \frac{dI_v}{dt} &= a_2 b_2 S_v I_{ha} + a_2 b_2 S_v I_{hs} - \Lambda_v I_v,
 \end{aligned} \tag{2}$$

together with $S_h + E_h + I_{ha} + I_{hs} + R_h = 1$ and $S_v + I_v = 1$, and the initial conditions

$$\begin{aligned}
 S_h(0) &= S_{h0}, E_h(0) = E_{h0}, I_{ha}(0) = I_{ha0}, I_{hs}(0) = I_{hs0}, R_h(0) \\
 &= R_{h0}, S_v(0) = S_{v0}, I_v(0) = I_{v0}.
 \end{aligned} \tag{3}$$

3.1. Existence and Positivity of Solutions. It is important to establish whether System (2) is well-posed and biologically meaningful. This is studied in the invariant region in which

the solution to System (2) makes biological sense. It is assumed that all the state variables and parameters of (2) are nonnegative for all $t \geq 0$.

The following results guarantee that the river blindness model given by System (2) is epidemiologically and mathematically well-posed. They are stated as follows:

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_v \subset \mathbb{R}_+^5 \times \mathbb{R}_+^2,$$

where

$$\begin{aligned}
 \mathcal{D}_h &= \{ (S_h, E_h, I_{ha}, I_{hs}, R_h) \in \mathbb{R}_+^5 : 0 \leq S_h + E_h + I_{ha} + I_{hs} + R_h \leq 1 \} \\
 \mathcal{D}_v &= \{ (S_v, I_v) \in \mathbb{R}_+^2 : 0 \leq S_v + I_v \leq 1 \}.
 \end{aligned}$$

Theorem 1. *There exists a bounded domain $\mathcal{D}$ that contains the solution set $\{S_h, E_h, I_{ha}, I_{hs}, R_h, S_v, I_v\}$.*

Proof 1. Consider the solution set $\{S_h, E_h, I_{ha}, I_{hs}, R_h, S_v, I_v\}$ with positive initial conditions (3). Let

$$V_1(S_h, E_h, I_{ha}, I_{hs}, R_h) = S_h(t) + E_h(t) + I_{ha}(t) + I_{hs}(t) + R_h(t) \tag{4}$$

$$V_2(S_v, I_v) = S_v(t) + I_v(t). \tag{5}$$

The derivatives V'_1 and V'_2 along solutions to System (2) for the human population and blackfly vector population are, respectively, obtained from the equations

$$\begin{aligned}
 \frac{dV_1}{dt} &= \frac{dV_1}{dS_h} \frac{dS_h}{dt} + \frac{dV_1}{dE_h} \frac{dE_h}{dt} + \frac{dV_1}{dI_{ha}} \frac{dI_{ha}}{dt} + \frac{dV_1}{dI_{hs}} \frac{dI_{hs}}{dt} + \frac{dV_1}{dR_h} \frac{dR_h}{dt} \\
 \frac{dV_2}{dt} &= \frac{dV_2}{dS_v} \frac{dS_v}{dt} + \frac{dV_2}{dI_v} \frac{dI_v}{dt}.
 \end{aligned}$$

From Equation (4), $dV_1/dS_h = (dV_1)/(dE_h) = (dV_1)/(dI_{ha}) = (dV_1)/(dI_{hs}) = (dV_1)/(dR_h) = 1$. Similarly, from (5), $(dV_2)/(dS_v) = (dV_2)/(dI_v) = 1$.

$(dV_1)/dt$ and $(dV_2)/dt$ can be obtained by adding the first 5 equations of System (2) and the last 2 equations of System (2), respectively. It follows that

$$\begin{aligned}\frac{dV_1}{dt} &= \Lambda_h(1 - S_h) - \Lambda_h = -\Lambda_h S_h \leq 0 \\ \frac{dV_2}{dt} &= \Lambda_v - (S_v + I_v)\Lambda_v = 0.\end{aligned}$$

Clearly, $V'_1 \leq 0$ and $V'_2 \leq 0$. Integrating the two inequalities gives

$$V_1 \leq V_1(S_{h0}, E_{h0}, I_{ha0}, I_{hs0}, R_{h0}) = S_{h0} + E_{h0} + I_{ha0} + I_{hs0} + R_{h0} \leq 1$$

and

$$\begin{aligned}V_2 &\leq V_2(S_{v0}, I_{v0}) = S_{v0} + I_{v0}, \\ &\leq 1.\end{aligned}$$

This shows that the normalized System (2) of the formulated model exists in the region given by

$$\begin{aligned}\mathcal{D} = \{ (S_h, E_h, I_{ha}, I_{hs}, R_h, S_v, I_v) \in \mathbb{R}_+^7 : 0 \leq S_h + E_h + E_{ha} \\ + I_{hs} + R_h \leq 1 ; 0 \leq S_v + I_v \leq 1 \}.\end{aligned}$$

Since the model represents human host and black fly vector populations, it is necessary to show that the solution of System (2) is nonnegative in the feasible region $\mathcal{D}$ for all $t \geq 0$. $\square$

Theorem 2. Let $\{S_{h0}, E_{h0}, I_{ha0}, I_{hs0}, R_{h0}, S_{v0}, I_{v0} \geq 0\} \in \mathcal{D}$ in Equation (3) be the initial data for System (2). Then, the solution set $\{S_h, E_h, I_{ha}, I_{hs}, R_h, S_v, I_v\}$ of System (2) is positive for all $t > 0$.

Proof 2. From the first equation of System (2), it is observed that

$$\frac{dS_h}{dt} \geq -(\Lambda_h + a_1 b_1 m I_v) S_h. \quad (6)$$

Integrating Equation (6), it gives

$$S_h(t) \geq S_{h0} e^{-(\Lambda_h + a_1 b_1 m I_v)t} \geq 0.$$

Similarly, it can be shown that $E_h \geq 0, I_{ha} \geq 0, I_{hs} \geq 0, R_h \geq 0, S_v \geq 0$ and $I_v \geq 0$ for all $t \geq 0$. $\square$

3.2. Existence of Disease-Free Equilibrium Point. It is clearly seen from System (2) that R_h is a superfluous fraction variable since the rest of the fractional variables do not depend on it. Moreover, R_h is easily obtained from the equation $R_h = 1 - (S_h + E_h + I_{ha} + I_{hs})$. The variable S_v is also replaced

by $S_v = 1 - I_v$ in the sixth equation of System (2) to give the following 5-dimensional system

$$\begin{aligned}\frac{dS_h}{dt} &= \Lambda_h(1 - S_h) - a_1 b_1 m S_h I_v + \alpha S_h (I_{ha} + I_{hs}), \\ \frac{dE_h}{dt} &= a_1 b_1 m S_h I_v - (\Lambda_h + \gamma_e \tau_e + \delta) E_h + \alpha E_h (I_{ha} + I_{hs}), \\ \frac{dI_{ha}}{dt} &= \epsilon \delta E_h - (\Lambda_h + \gamma_a \tau_a + \alpha) I_{ha} + \alpha I_{ha} (I_{ha} + I_{hs}), \\ \frac{dI_{hs}}{dt} &= (1 - \epsilon) \delta E_h - (\Lambda_h + \gamma_s \tau_s + \alpha) I_{hs} + \alpha I_{hs} (I_{ha} + I_{hs}), \\ \frac{dI_v}{dt} &= a_2 b_2 (1 - I_v) I_{ha} + a_2 b_2 (1 - I_v) I_{hs} - \Lambda_v I_v,\end{aligned} \quad (7)$$

together with $S_h + E_h + I_{ha} + I_{hs} + R_h = 1$ and $S_v + I_v = 1$ and the initial conditions

$$\begin{aligned}S_h(0) &= S_{h0}, E_h(0) = E_{h0}, I_{ha}(0) = I_{ha0}, I_{hs}(0) \\ &= I_{hs0}, R_h(0) = R_{h0}, S_v(0) = S_{v0}, I_v(0) = I_{v0}.\end{aligned} \quad (8)$$

The equilibrium point for which there is no disease in the community is referred to as a disease-free equilibrium point. In the absence of the disease, $E_h = I_{ha} = I_{hs} = I_v = 0$. Hence, the disease-free equilibrium point is given by $(1, 0, 0, 0, 0)$.

3.3. Existence of the Unique Endemic Equilibrium. If the unique endemic equilibrium point $E_1 = (S_h^*, E_h^*, I_{ha}^*, I_{hs}^*, I_v^*)$ exists, then, $S_h^* > 0, E_h^* > 0, I_{ha}^* > 0, I_{hs}^* > 0$ and $I_v^* > 0$. Adding all the equations in System (7) gives

$$\begin{aligned}\Lambda_h(1 - S_h^* - E_h^* - I_{ha}^* - I_{hs}^*) + \alpha(I_{ha}^* + I_{hs}^*)(S_h^* + E_h^* + I_{ha}^* + I_{hs}^*) \\ + (a_2 b_2 (1 - I_v^*) - (\gamma_s \tau_s + \alpha)) I_{hs}^* \\ + (a_2 b_2 (1 - I_v^*) - (\gamma_a \tau_a + \alpha)) I_{ha}^* - (\gamma_e \tau_e E_h^* + \Lambda_v I_v^*) = 0.\end{aligned}$$

Rearranging gives

$$\begin{aligned}\Lambda_h(1 - S_h^* - E_h^* - I_{ha}^* - I_{hs}^*) + \alpha(I_{ha}^* + I_{hs}^*)(S_h^* + E_h^* + I_{ha}^* + I_{hs}^*) \\ + (a_2 b_2 (1 - I_v^*) - (\gamma_s \tau_s + \alpha)) I_{hs}^* \\ + (a_2 b_2 (1 - I_v^*) - (\gamma_a \tau_a + \alpha)) I_{ha}^* = (\gamma_e \tau_e E_h^* + \Lambda_v I_v^*).\end{aligned} \quad (9)$$

Clearly, the right-hand side of Equation (9) is positive. Since $1 - S_h^* - E_h^* - I_{ha}^* - I_{hs}^* > 0$, if $(a_2 b_2 (1 - I_v^*) - (\gamma_s \tau_s + \alpha)) > 0$ and $a_2 b_2 (1 - I_v^*) - (\gamma_a \tau_a + \alpha) > 0$; then, the left hand side of System (9) is positive. These two conditions imply that $I_v^* < a_2 b_2 - (\gamma_s \tau_s + \alpha)/a_2 b_2$ and $I_v^* < a_2 b_2 - (\gamma_a \tau_a + \alpha)/a_2 b_2$. Also since I_v^* lies between 0 and 1, then the endemic equilibrium point exists if $a_2 b_2 > \gamma_s \tau_s + \alpha$ and $a_2 b_2 > \gamma_a \tau_a + \alpha$ with I_v^* lying in the interval $(0, \min \{1, a_2 b_2 - \gamma_s \tau_s/a_2 b_2, a_2 b_2 - \gamma_a \tau_a/a_2 b_2\})$.

The assumptions $a_2 b_2 > \gamma_s \tau_s + \alpha$ and $a_2 b_2 > \gamma_a \tau_a + \alpha$ are of significant importance for the disease to persist. They show that the average infection rate of susceptible blackflies

should be greater than the treatment rate of asymptomatic and symptomatic individuals plus their percapita death due to the disease.

3.4. Effective Reproduction Number. The effective reproduction number of an infection, denoted $\mathcal{R}_e$, is the average number of secondary infections caused by a single infected individual at a given point as interventions are implemented. It is a key parameter that may be used to monitor the impact of the disease control programs. For the stability analysis of System (7), we obtain the threshold condition for the establishment of the disease by the method used in [23]. We consider the infected classes of System (7)

$$\begin{aligned}\frac{dE_h}{dt} &= a_1 b_1 m S_h I_v - (\Lambda_h + \gamma_e \tau_e + \delta) E_h + \alpha E_h (I_{ha} + I_{hs}), \\ \frac{dI_{ha}}{dt} &= \epsilon \delta E_h - (\Lambda_h + \gamma_a \tau_a + \alpha) I_{ha} + \alpha I_{ha} (I_{ha} + I_{hs}), \\ \frac{dI_{hs}}{dt} &= (1 - \epsilon) \delta E_h - (\Lambda_h + \gamma_s \tau_s + \alpha) I_{hs} + \alpha I_{hs} (I_{ha} + I_{hs}), \\ \frac{dI_v}{dt} &= a_2 b_2 (1 - I_v) I_{ha} + a_2 b_2 (1 - I_v) I_{hs} - \Lambda_v I_v.\end{aligned}\quad (10)$$

The following are obtained from System (10)

$$\mathcal{F} = \begin{pmatrix} a_1 b_1 m S_h I_v \\ 0 \\ 0 \\ a_2 b_2 (1 - I_v) (I_{ha} + I_{hs}) \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} (\Lambda_h + \gamma_e \tau_e + \delta) E_h - \alpha E_h (I_{ha} + I_{hs}) \\ (\Lambda_h + \gamma_a \tau_a + \alpha) I_{ha} - \epsilon \delta E_h - \alpha I_{ha} (I_{ha} + I_{hs}) \\ (\Lambda_h + \gamma_s \tau_s + \alpha) I_{hs} - (1 - \epsilon) \delta E_h - \alpha I_{hs} (I_{ha} + I_{hs}) \\ \Lambda_v I_v \end{pmatrix}.$$

Finding the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ above at point E_0 gives

$$\mathbf{F} = \begin{bmatrix} 0 & 0 & 0 & a_1 b_1 m \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & a_2 b_2 (1 - I_v) & a_2 b_2 & 0 \end{bmatrix}, \quad (11)$$

$$\mathbf{V} = \begin{bmatrix} \Lambda_h + \gamma_e \tau_e + \delta & 0 & 0 & 0 \\ -\epsilon \delta & \Lambda_h + \gamma_a \tau_a + \alpha & 0 & 0 \\ -(1 - \epsilon) \delta & 0 & \Lambda_h + \gamma_s \tau_s + \alpha & 0 \\ 0 & 0 & 0 & \Lambda_v \end{bmatrix}, \quad (12)$$

$$\mathbf{V}^{-1} = \begin{bmatrix} (\Lambda_h + \gamma_e \tau_e + \delta)^{-1} & 0 & 0 & 0 \\ \epsilon \delta (\Lambda_h + \gamma_e \tau_e + \delta)^{-1} (\Lambda_h + \gamma_a \tau_a + \alpha)^{-1} & (\Lambda_h + \gamma_a \tau_a + \alpha)^{-1} & 0 & 0 \\ (1 - \epsilon) \delta (\Lambda_h + \gamma_e \tau_e + \delta)^{-1} (\Lambda_h + \gamma_s \tau_s + \alpha)^{-1} & 0 & (\Lambda_h + \gamma_s \tau_s + \alpha)^{-1} & 0 \\ 0 & 0 & 0 & \Lambda_v^{-1} \end{bmatrix}. \quad (13)$$

The effective reproduction number is given by

$$\mathcal{R}_e = \sqrt{\frac{a_2 b_2 [a_1 b_1 m \epsilon \delta (\Lambda_h + \gamma_s \tau_s + \alpha) + (1 - \epsilon) \delta a_1 b_1 m (\Lambda_h + \gamma_a \tau_a + \alpha)]}{\Lambda_v (\Lambda_h + \gamma_e \tau_e + \delta) (\Lambda_h + \gamma_a \tau_a + \alpha) (\Lambda_h + \gamma_s \tau_s + \alpha)}}. \quad (14)$$

It is noted that $\mathcal{R}_e$ is the geometric mean of

$$\mathcal{R}_v = \frac{a_2 b_2}{\Lambda_v}$$

$$\mathcal{R}_h = \frac{a_1 b_1 m \delta [\epsilon (\Lambda_h + \gamma_s \tau_s + \alpha) + (1 - \epsilon) (\Lambda_h + \gamma_a \tau_a + \alpha)]}{(\Lambda_h + \gamma_e \tau_e + \delta) (\Lambda_h + \gamma_a \tau_a + \alpha) (\Lambda_h + \gamma_s \tau_s + \alpha)}.$$

TABLE 2: Parameter values.

Parameter	Symbol	Value	Ref.
Recruitment rate of humans	Λ_h	0.031/day	[25]
Recruitment rate of blackflies	Λ_v	0.73/day	[26]
Per-capita natural death rate of humans	μ_h	1/23178/day	[25]
Per-capita natural death rate of blackflies	μ_v	0.0476/day	[27]
Death rate of blackflies due to vector control	μ_{vc}	0.05/day	Assumed
Biting rate by infectious blackfly on susceptible human	a_{-1}	0.0855/day	[28]
Biting rate by susceptible blackfly on an infectious human	a_{-2}	0.0855/day	[28]
Probability that a bite infects a susceptible human	b_{-1}	0.073	[28]
Probability that a bite infects a susceptible blackfly	b_{-2}	0.080	[28]
Fraction of asymptomatic infected humans	ϵ	0–0.9	Assumed
Progression rate to the infective class	δ	1/240/day	[29]
Recovery rate of exposed individuals	τ_e	0.00950/day	[29]
Recovery rate of asymptomatic infected individuals	τ_a	0.0222/day	[30]
Recovery rate of symptomatic infected individuals	τ_s	0.0222/day	[30]
Disease-induced death rate	α	0.00001438/day	[31]

3.5. Local Stability of the Disease-Free Equilibrium. System (7) admits the disease-free equilibrium whose local stability is established by evaluating the Jacobian matrix of the system at the disease-free equilibrium point. The Jacobian matrix for System (7) gives

$$J = \begin{bmatrix} -J_{11} & 0 & \alpha S_h & \alpha S_h & -a_1 b_1 m S_h \\ a_1 b_1 m I_v & -J_{22} & \alpha E_h & \alpha E_h & a_1 b_1 m S_h \\ 0 & \epsilon \delta & -J_{33} & 0 & 0 \\ 0 & (1 - \epsilon) \delta & 0 & -J_{44} & 0 \\ 0 & 0 & a_2 b_2 (1 - I_v) & a_2 b_2 (1 - I_v) & -J_{55} \end{bmatrix}.$$

$$\begin{aligned} J_{11} &= \Lambda_h + a_1 b_1 m I_v - \alpha(I_{ha} + I_{hs}), \\ J_{22} &= \Lambda_h + \gamma_e \tau_e + \delta - \alpha(I_{ha} + I_{hs}), \\ J_{33} &= \Lambda_h + \gamma_a \tau_a + \alpha - \alpha(2I_{ha} + I_{hs}), \\ J_{44} &= \Lambda_h + \gamma_s \tau_s + \alpha - \alpha(2I_{hs} + I_{ha}), \\ J_{55} &= \Lambda_v - a_2 b_2 (I_{ha} + I_{hs}). \end{aligned} \quad (15)$$

The Jacobian matrix J evaluated at the disease-free equilibrium $E_0(1, 0, 0, 0, 0)$ gives

$$J_{E_0} = \begin{bmatrix} -\Lambda_h & 0 & \alpha & \alpha & -a_1 b_1 m \\ 0 & -(\Lambda_h + \gamma_e \tau_e + \delta) & 0 & 0 & a_1 b_1 m \\ 0 & \epsilon \delta & -(\Lambda_h + \gamma_a \tau_a + \alpha) & 0 & 0 \\ 0 & (1 - \epsilon) \delta & 0 & -(\Lambda_h + \gamma_s \tau_s + \alpha) & 0 \\ 0 & 0 & a_2 b_2 & a_2 b_2 & -\Lambda_v \end{bmatrix}. \quad (16)$$

We use the stability test based on the Gershgorin circles [24] (Proposition 3.1) to establish the local stability of E_0 . Since all the diagonal entries of J_{E_0} are negative, then the first hypothesis of [24] (Proposition 3.1) is satisfied. The second hypothesis is satisfied if and only if the following inequalities

$$a_1 b_1 m + 2\alpha < \Lambda_h, \quad (17)$$

$$a_1 b_1 m < \Lambda_h + \gamma_e \tau_e + \delta, \quad (18)$$

$$\epsilon \delta < \Lambda_h + \gamma_a \tau_a + \alpha, \quad (19)$$

$$(1 - \epsilon) \delta < \Lambda_h + \gamma_s \tau_s + \alpha, \quad (20)$$

$$2a_2 b_2 < \Lambda_v, \quad (21)$$

hold. Therefore, if (17)–(21) are taken, then E_0 is locally asymptotically stable.

3.6. Sensitivity Analysis. In this subsection, we establish the most influential parameters in river blindness transmission through sensitivity analysis of the effective reproduction number $\mathcal{R}_e$ using parameter values given in Table 2. Sensitivity analysis is an important tool in determining the extent to which a perturbation in model parameters changes in the effective reproduction number [32]. The results of this analysis may guide public health intervention policies for the efficient use of limited resources. A parameter is referred to as a sensitivity factor if a small change in a parameter value can cause a big change in the value of the effective reproduction number; otherwise, it is referred to as an insensitive factor. Table 3 shows the sensitivity indices of $\mathcal{R}_e$ with respect to the parameters influencing transmission of onchocerciasis. We note from the sensitivity indices given in Table 3 that

TABLE 3: Sensitivity indices of $\mathcal{R}_e$ with respect to parameters.

Parameter	Sensitivity index
Λ_h	-0.8284
a_{-1}	0.5000
a_{-2}	0.5000
b_{-1}	0.5000
b_{-2}	0.5000
m	0.5000
Λ_v	-0.5000
δ	0.4534
τ_e	-0.1064
τ_a	-0.1043
τ_s	-0.1043
α	-0.0004
ϵ	0.0000

the value of $\mathcal{R}_e$ increases when the parameter values a_1, a_2, b_1, b_2 and m increase while other parameter values are kept fixed. This indicates that an increase in the endemicity of onchocerciasis since the indices have positive signs.

3.7. Bifurcation Analysis. We use the method first formulated by Castillo-Chavez and Song [33] to provide the necessary and sufficient conditions for backward bifurcation. In order to check whether the model undergoes a backward bifurcation, we proceed as follows: Let the new variables be $x_1 = S_h, x_2 = E_h, x_3 = I_{ha}, x_4 = I_{hs}, x_5 = I_v$. System (7) becomes

$$\begin{aligned}
\frac{dx_1}{dt} &= \Lambda_h(1 - x_1) - a_1 b_1 m x_1 x_5 + \alpha x_1(x_3 + x_4) = f_1, \\
\frac{dx_2}{dt} &= a_1 b_1 m x_1 x_5 - (\Lambda_h + \gamma_e \tau_e + \delta) x_2 + \alpha x_2(x_3 + x_4) = f_2, \\
\frac{dx_3}{dt} &= \epsilon \delta x_2 - (\Lambda_h + \gamma_a \tau_a + \alpha) x_3 + \alpha x_3(x_3 + x_4) = f_3, \\
\frac{dx_4}{dt} &= (1 - \epsilon) \delta x_2 - (\Lambda_h + \gamma_s \tau_s + \alpha) x_4 + \alpha x_4(x_3 + x_4) = f_4, \\
\frac{dx_5}{dt} &= a_2 b_2(1 - x_5) x_3 + a_2 b_2(1 - x_5) x_4 - \Lambda_v x_5 = f_5,
\end{aligned} \tag{22}$$

Taking a_1 as the bifurcation parameter while considering the case $\mathcal{R}_e = 1$ gives

$$a_1 = a_1^* = \frac{\Lambda_v(\Lambda_h + \gamma_e \tau_e + \delta)(\Lambda_h + \gamma_a \tau_a + \alpha)(\Lambda_h + \gamma_s \tau_s + \alpha)}{a_2 b_1 b_2 m \delta (\epsilon(\Lambda_h + \gamma_s \tau_s + \alpha) + (1 - \epsilon)(\Lambda_h + \gamma_a \tau_a + \alpha))}.$$

The linearisation matrix of System (22) evaluated at the disease-free equilibrium $E_0(1, 0, 0, 0, 0)$ is given by

$$J(E_0, a_1^*) = \begin{bmatrix} -\Lambda_h & 0 & \alpha & \alpha & -a_1^* b_1 m \\ 0 & -(\Lambda_h + \gamma_e \tau_e + \delta) & 0 & 0 & a_1^* b_1 m \\ 0 & \epsilon \delta & -(\Lambda_h + \gamma_a \tau_a + \alpha) & 0 & 0 \\ 0 & (1 - \epsilon) \delta & 0 & -(\Lambda_h + \gamma_s \tau_s + \alpha) & 0 \\ 0 & 0 & a_2 b_2 & a_2 b_2 & -\Lambda_v \end{bmatrix}. \tag{23}$$

The right eigenvector $w = (w_1, w_2, w_3, w_4, w_5)^T$ corresponding to the zero eigenvalue is computed using $J(E_0, a_1^*) w = 0$, which yields

$$\begin{aligned}
-\Lambda_h w_1 + \alpha w_3 + \alpha w_4 - a_1^* b_1 m w_5 &= 0, \\
-(\Lambda_h + \gamma_e \tau_e + \delta) w_2 + a_1^* b_1 m w_5 &= 0, \\
\epsilon \delta w_2 - (\Lambda_h + \gamma_a \tau_a + \alpha) w_3 &= 0, \\
(1 - \epsilon) \delta w_2 - (\Lambda_h + \gamma_s \tau_s + \alpha) w_4 &= 0, \\
a_2 b_2 w_3 + a_2 b_2 w_4 - \Lambda_v w_5 &= 0.
\end{aligned}$$

Setting $w_3 = 1$ gives

$$\begin{aligned}
w_2 &= \frac{(\Lambda_h + \gamma_a \tau_a + \alpha)}{\epsilon \delta}, \\
w_4 &= \frac{(1 - \epsilon)(\Lambda_h + \gamma_a \tau_a + \alpha)}{\epsilon(\Lambda_h + \gamma_s \tau_s + \alpha)}, \\
w_5 &= \frac{1}{\Lambda_v} \left(a_2 b_2 + \frac{a_2 b_2 (1 - \epsilon)(\Lambda_h + \gamma_a \tau_a + \alpha)}{\epsilon(\Lambda_h + \gamma_s \tau_s + \alpha)} \right), \\
w_1 &= \frac{1}{\Lambda_h} \left(\alpha + \frac{\alpha(1 - \epsilon)(\Lambda_h + \gamma_a \tau_a + \alpha)}{\epsilon(\Lambda_h + \gamma_s \tau_s + \alpha)} - \frac{a_1^* b_1 m}{\Lambda_v} \left(a_2 b_2 + \frac{a_2 b_2 (1 - \epsilon)(\Lambda_h + \gamma_a \tau_a + \alpha)}{\epsilon(\Lambda_h + \gamma_s \tau_s + \alpha)} \right) \right).
\end{aligned}$$

The left eigenvector $v = (v_1, v_2, v_3, v_4, v_5)$ corresponding to the zero eigenvalue is computed using $v J(E_0, a_1^*) = 0$, which yields

$$\begin{aligned}
-\Lambda_h v_1 &= 0, \\
-(\Lambda_h + \gamma_e \tau_e + \delta) v_2 + \epsilon \delta v_3 + (1 - \epsilon) \delta v_4 &= 0, \\
\alpha v_1 - (\Lambda_h + \gamma_a \tau_a + \alpha) v_3 + a_2 b_2 v_5 &= 0, \\
\alpha v_1 - (\Lambda_h + \gamma_s \tau_s + \alpha) v_4 + a_2 b_2 v_5 &= 0, \\
a_1^* b_1 m v_1 + a_1^* b_1 m v_2 - \Lambda_v v_5 &= 0.
\end{aligned}$$

It follows that $v_1 = 0$, and setting $v_3 = 1$ gives

$$\begin{aligned}
v_5 &= \frac{(\Lambda_h + \gamma_a \tau_a + \alpha)}{a_2 b_2}, \\
v_2 &= \frac{\Lambda_v(\Lambda_h + \gamma_a \tau_a + \alpha)}{a_1^* b_1 a_2 b_2 m}, \\
v_4 &= \frac{(\Lambda_h + \gamma_a \tau_a + \alpha)}{(\Lambda_h + \gamma_s \tau_s + \alpha)}.
\end{aligned}$$

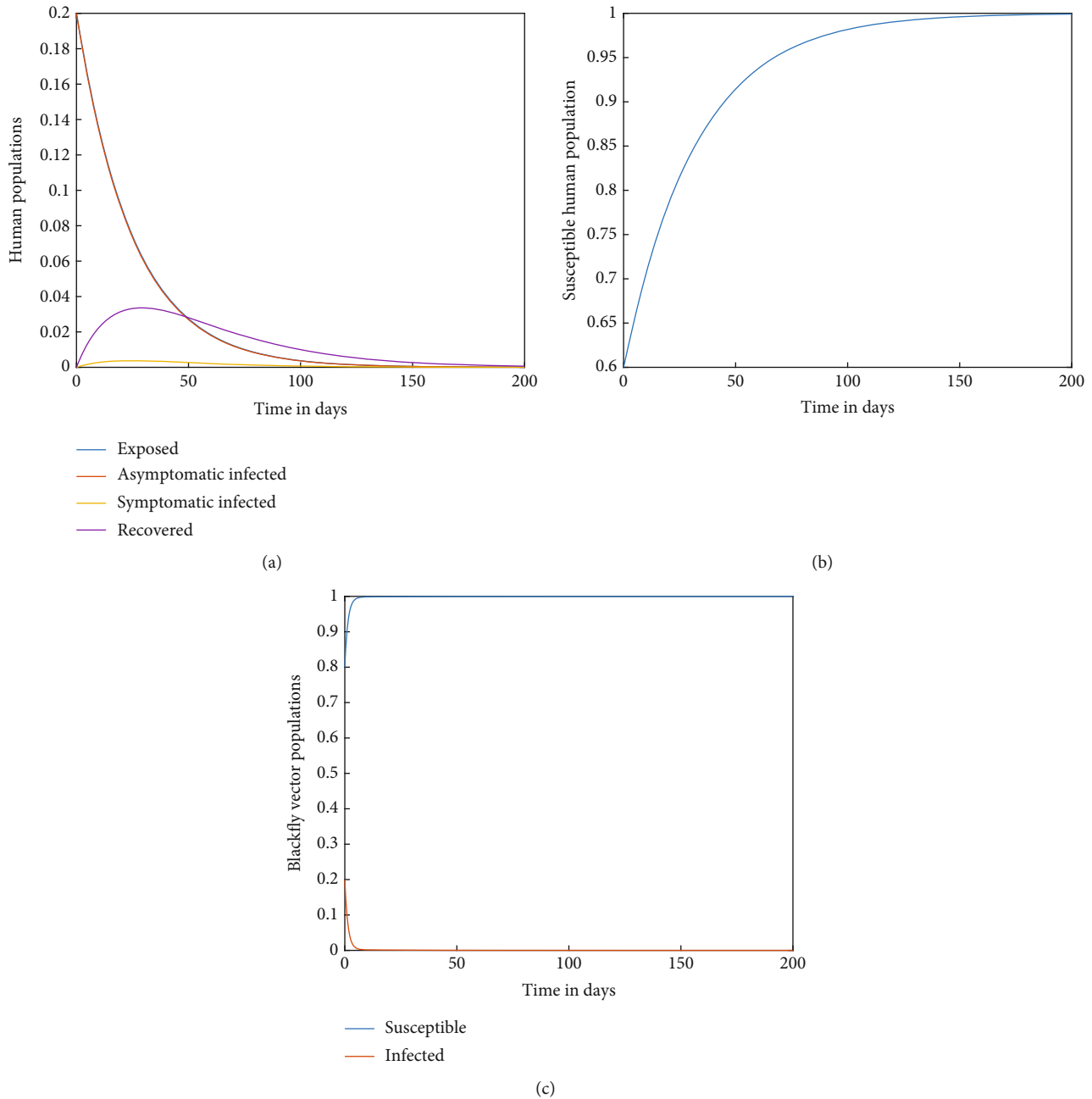


FIGURE 2: (a) The variation in the exposed, asymptomatic infected, and symptomatic infected human population with time over a period of 200 days. (b) The variation in susceptible human population over time for a period of 200 days. (c) The variation in blackfly population with time over a period of 200 days.

The nonzero partial second derivatives computed at E_0 $(1, 0, 0, 0, 0)$ are

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_3 \partial x_4} = \alpha,$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_5} = ma_1 b_1,$$

$$\frac{\partial^2 f_3}{\partial x_3 \partial x_3} = \frac{\partial^2 f_4}{\partial x_4 \partial x_4} = 2\alpha,$$

$$\frac{\partial^2 f_5}{\partial x_3 \partial x_5} = -a_2 b_2,$$

$$\frac{\partial^2 f_5}{\partial x_4 \partial x_5} = -a_2 b_2,$$

$$\frac{\partial^2 f_2}{\partial x_5 \partial a_1} = ma_1 b_1.$$

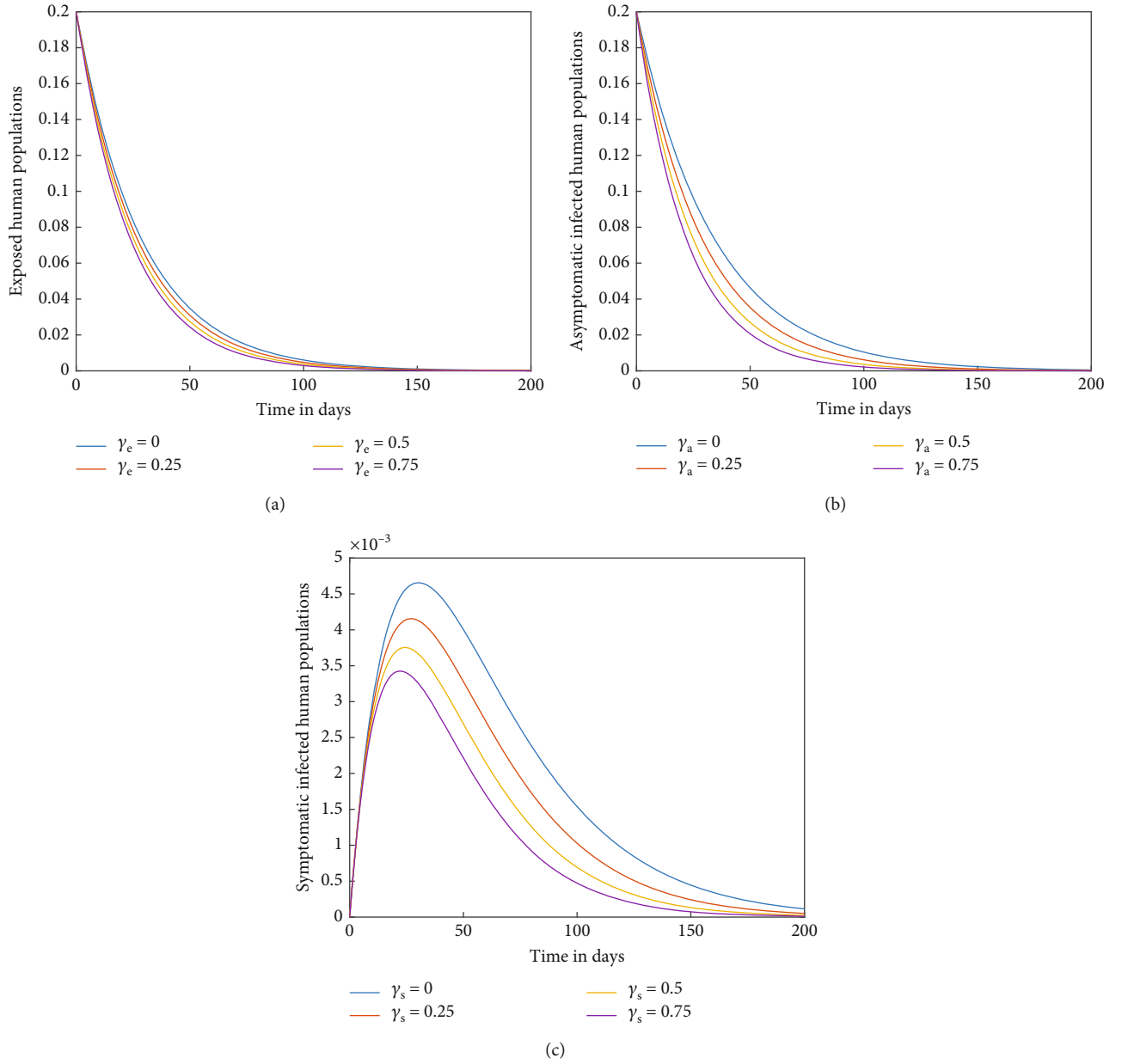


FIGURE 3: (a) The effect of treatment in the exposed class with efficacy $\gamma_e = 0$, $\gamma_e = 0.25$, $\gamma_e = 0.5$, and $\gamma_e = 0.75$. (b) The effect of treatment in the asymptomatic infected class with efficacy $\gamma_a = 0$, $\gamma_a = 0.25$, $\gamma_a = 0.5$, and $\gamma_a = 0.75$. (c) The effect of treatment in the symptomatic infected class with efficacy $\gamma_s = 0$, $\gamma_s = 0.25$, $\gamma_s = 0.5$, and $\gamma_s = 0.75$.

Since $v_1 = 0$, we have

$$\begin{aligned}
 a &:= \sum_{i,j=1}^5 v_2 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + \sum_{i,j=1}^5 v_3 w_i w_j \frac{\partial^2 f_3}{\partial x_i \partial x_j} \\
 &\quad + \sum_{i,j=1}^5 v_4 w_i w_j \frac{\partial^2 f_4}{\partial x_i \partial x_j} + \sum_{i,j=1}^5 v_5 w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}, \\
 &= v_2 (2w_1 w_5 a_1 b_1 m + w_2 w_3 \alpha + w_2 w_4 \alpha) + 2v_3 w_3 \alpha (w_3 + w_4) \\
 &\quad + v_4 w_4 \alpha (w_3 + 2w_4) - v_5 w_5 (2w_3 a_2 b_2)
 \end{aligned}$$

$$\begin{aligned}
 b &:= \sum_{i=1}^5 v_2 w_i \frac{\partial^2 f_2}{\partial x_i \partial a_1^*} + \sum_{i=1}^5 v_3 w_i \frac{\partial^2 f_3}{\partial x_i \partial a_1^*} + \sum_{i=1}^5 v_4 w_i \frac{\partial^2 f_4}{\partial x_i \partial a_1^*} \\
 &\quad + \sum_{i=1}^5 v_5 w_i \frac{\partial^2 f_5}{\partial x_i \partial a_1^*}. \\
 &= v_2 (mb_1 w_1 x_5 + mb_1 w_5 x_1) > 0.
 \end{aligned}$$

Since $b > 0$, then a backward bifurcation occurs when $a > 0$; otherwise, the system exhibits a forward bifurcation

TABLE 4: The effect of drug efficacy to the effective reproduction number.

Efficacy level	Effective reproduction number
0.0	0.0149
0.25	0.0133
0.5	0.0120
0.75	0.0110
1.0	0.0101

at $\mathcal{R}_e = 1$, giving local asymptotic stability of the endemic equilibrium point when $\mathcal{R}_e > 1$ and close to 1 (see [33]).

4. Numerical Simulations

This section presents illustrations of the dynamical behaviour of onchocerciasis in the human and blackfly populations using numerical simulations of System (2). The MATLAB computer software program is used to perform numerical simulations. The parameter values are shown in Table 2 together with the approximated initial data in terms of proportions $S_h(0) = 0.6$, $E_h(0) = 0.2$, $I_{ha}(0) = 0.2$, $I_{hs}(0) = 0$, $R_h(0) = 0$, $S_v = 0.8$, and $i_v(0) = 0.2$ are used. An efficacy level of $\gamma_e = \gamma_a = \gamma_s = 0.5$ is assumed for the simulation in Figure 2. We observe that the proportion of the susceptible human population Figure 2(b) and the susceptible blackfly population Figure 2(c) increase with time due to recruitment. The interaction between the blackflies and human causes an increase in the proportion of the exposed human population that declines together with the asymptomatic and symptomatic humans due to treatment as seen in Figure 2(a). These simulations also show that the disease-free equilibrium is locally stable, in agreement with the results from the theoretical analysis in Subsection 3.5.

In Figures 3(a), 3(b), and 3(c), we note a significant decrease in the proportion of exposed, asymptomatic, and symptomatic individuals as the effectiveness of the treatment regimen rises. This underscores the importance of supplying efficacious medications to combat the spread of onchocerciasis within the community. By doing so, we can substantially reduce onchocerciasis transmission rates, consequently diminishing its prevalence and incidence within the locality. It is also important to note that the symptomatic class responds more to higher levels of drug efficacy compared to the asymptomatic and exposed classes.

We have estimated the effective reproduction number using the parameters given in Table 4, with varying levels of treatment efficacy. For simplicity, we assume the same drug efficacy level for the exposed, asymptomatic infected, and symptomatic infected individuals. The results are shown in Table 4. We observe a decline in the effective reproduction number as the drug efficacy increases. This suggests that the transmission of river blindness can be significantly reduced with highly effective medications.

5. Discussion and Conclusion

The transmission and control of onchocerciasis in a human population and blackfly vector population have been investigated using a mathematical model. The model includes a class of humans that are asymptomatic infected. Mathematical and epidemiological well posedness was established through analysis. Further analysis revealed that the model has a disease-free equilibrium, and the conditions for the existence of the endemic equilibrium have been derived. The conditions for local stability of the disease-free equilibrium have been obtained using the Gershgorin circles theorem. We have observed that for the endemic equilibrium to exist, the infection rate should be greater than the treatment rate plus the per capita death due to the disease. This result is of significant importance since it shows that the treatment level should be kept above the infection rate to avoid an endemic state. The next-generation approach was used to obtain the effective reproduction number $\mathcal{R}_e$. Sensitivity analysis was carried out, and it was established that the blackfly vector average biting rate per day is crucial in the disease transmission. It is, therefore, important to use control measures that reduce the number of blackfly bites on humans.

Numerical simulations of the model were carried out, and results revealed that intervention strategies need to focus on the treatment of infected individuals and the reduction of contact between blackflies and humans. We further note that the infected human population is reduced as the efficiency of the treatment varies; thus, the provision of effective drugs that curtail the development and instantaneous elimination of all living microfilariae is crucial in order to reduce transmission of the disease in the population.

Data Availability Statement

All data used in the study is available in the articles cited in the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

The authors received no specific funding for this work.

Acknowledgments

This manuscript has been prepared in memory of the late Professor Julius Tumwiine who conceived the idea of this study.

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