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Modelling the Impact of Trapping Blackfly Vectors on the Transmission of Onchocerciasis

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Abstract. Onchocerciasis, usually referred to as river blindness, a skin and eye parasitic infestation is caused by the filarial nematode *Onchocerca volvulus*. Current control and eradication efforts are being frustrated by the continued existence and thriving of blackflies which are the disease transmitting vectors that breed along the banks of fast flowing and highly oxygenated rivers and streams. This study aims at assessing the effect of using vector traps on the transmission and control of onchocerciasis. A host-vector deterministic model which incorporates vector trapping by use of a system of ordinary differential equations is developed. The model is analysed for steady states and the basic reproduction number is obtained using the next generation method. It is found that the disease free steady state is stable if the basic reproduction number $\mathcal{R}_0 < 1$. There exists a unique endemic equilibrium which is locally and globally asymptotically stable if $\mathcal{R}_0 > 1$. Numerical simulations show that trapping the blackfly vectors has an effect on the spread and control of the disease. However, it is discovered that using traps alone is not a sufficient strategy and needs to be combined with other methods if the disease is to be completely wiped out of the population.

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1. Introduction

Onchocerciasis, usually referred to as river blindness, is a skin and eye parasitic infestation caused by the filarial nematode *Onchocerca volvulus* [5] that is carried

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on to humans by the bites of infected blackflies which are of the genus *Simulium* [8, 37]. John O'Neil, an Irish surgeon, first observed the microfilariae of *Onchocerca volvulus* in 1874 [18]. These blackflies breed around fast-flowing and highly oxygenated rivers and streams [36] hence the name river blindness [34]. The eggs are laid in the rivers because the larva and pupa stages of the blackflies are aquatic [30]. This leads to a high prevalence of the disease in areas located along rivers where the breeding of the blackflies take place [37]. Regardless of the various strategies that have been used by different national and international organisations to eradicate the disease, recent examination data from the World Health Programme and Onchocerciasis Control Programme (OCP) indicate that more than 17.7 million people are infected globally [29]. Of these, 500, 000 are visually impaired and 270, 000 are blind [29]. About 99% of the infected persons are in Africa [37]. The symptoms of the disease may not manifest in the infected individual at the onset of infection and this is based on the ability of the larvae to migrate through the human body without triggering a response from the immune system. However, most of the symptoms are as a result of the body's inflammatory response to dead or dying larvae [29]. Symptoms of onchocerciasis include pruritis, papular dermatitis, lymphadenopathy, severe skin atrophy, leopard skin and papular onchodermatitis [15]. The effect of the inflammation caused by dead microfilariae in the eye is that there are initial reversible lesions on the cornea which when not treated, advance to permanent clouding of the cornea thereby leading to blindness [8].

Since ivermectin does not kill adult *O. volvulus*, the current treatment strategies include annual (or biannual) mass ivermectin distribution to keep the microfilarial densities low in high transmission settings [13, 21, 23] and introduce a number of alternative strategies, including other microfilaricidal therapies (such as moxidectin), macrofilaricidal (anti-wolbachial) treatments or drug combinations with a higher effect on *Onchocerca volvulus* than ivermectin [24], focal vector control [4]. In many areas of Africa that are endemic for eyeworm or with high densities of vector blackfly, ivermectin mass drug administration needs additional interventions to acheieve disease eliminate [6, 20, 26]. The process of collecting adult female black flies using different types of traps and baits which attract vectors and remove them from the population is referred to as trapping [27]. The Esperanza Window trap (EWT) is a simple trap originally developed to replace human landing collections for entomological surveillance of *O. volvulus* transmission [26]. It is useful in the vector control as it reduces human landing rates which approximate the biting rate of the blackflies [19, 32]. The impact of trapping and killing the blackflies is therefore of much importance in the transmission and control of onchocerciasis.

Mathematical models have been developed over the years to provide an insight into the dynamics of onchocerciasis transmission and control in a bid to eradicat the disease. Saporu [33] developed a model that described the interaction of the human and blackfly populations where the probability that all blackflies were infected was obtained and proved that it was clearly independent of the initial number of infected blackflies. Results showed that the death rate of the blackflies less than one was unlikely to produce the proportion of blackflies ultimately infected and that even when birth was allowed in the blackfly vector population, the proportion dying before becoming infected tended to increase with increasing birth rate.

Alley et al. [2] used a microsimulation mathematical model of the dynamics of onchocerciasis transmission to explore the potential of a hypothetical macrofilaricidal drug for the elimination of onchocerciasis under different epidemiological conditions as characterised by previous intervention strategies, that is, vectorial capacity and levels of coverage. Results showed that with a high vector biting rate and poor coverage, a very effective macrofilaricide would appear to have a

substantially higher potential for achieving elimination of the parasite than does ivermectin. Basáñez et al. [3] formulated a mathematical model of transmission intensity and the pattern of *Onchocerca volvulus* infection in human communities. Their focus was on possible constraints upon *Onchocerca volvulus* establishment in humans in relation to exposure rates to infective larvae as measured by the annual transmission potential (ATP). They discovered that relationships between microfilarial prevalence and both microfilariae and transmission intensity were non-linear. This similarity extended to the relationship between microfilarial intensity and ATP. Filipe et al. [17] developed a model that described the human infection patterns and heterogeneous exposure in river blindness and it was shown that parasite establishment in humans is determined by exposure to infective stages and host immunological responsiveness to parasites. Omondi et al. [30] presented an onchocerciasis transmission model with treatment that involved the application of optimal control. The model was found to exhibit a backward bifurcation implying that $\mathcal{R}_0 < 1$ was not sufficient to eliminate the disease from the population and the need was to lower $\mathcal{R}_0$ below a certain threshold, $\mathcal{R}_0^c$ for effective disease control. Oguntolu et al. [29] formulated a mathematical model that was used to study the dynamics of onchocerciasis. Their study showed that the disease free equilibrium point was locally asymptotically stable if the effective reproduction number $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Hassan and Shaban [22] used a deterministic model to study the effects of mass treatment using ivermectin drugs, public health education and vector control of onchocerciasis disease dynamics. They implemented larviciding and trapping strategies in the vector population with the aim of controlling population growth of blackflies. Their study revealed that the dynamics of onchocerciasis and growth of blackfly vector population are best controlled when all the control strategies are implemented simultaneously.

In this paper, the effects of trapping the blackflies on the transmission and control dynamics in an onchocerciasis transmission and control model are investigated. The exposed class in the blackfly population is neglected. The rest of this paper is organised as follows: In Section 2, the model description and formulation are presented. In Section 3, model analysis is carried out. Numerical simulation of the model is illustrated in Section 4. The paper is concluded with discussion of results and conclusion in Section 5.

2. Model description and formulation

A host-vector model is formulated for the transmission and control of onchocerciasis using a system of ordinary differential equations. The total human population at time t denoted by $N_H(t)$ is divided into four epidemiological classes: Susceptible humans, $S_H(t)$, referring to individuals that are not infected with onchocerciasis but are at risk of infection at time t, infected-acute humans, $I_A(t)$, referring to individuals that have been exposed to the disease through blackfly bites and have microfilariae in their bodies at time t, infected-chronic humans, $I_C(t)$, referring to individuals infested with *Onchocerca volvulus* worms at time t and recovered humans, $R_H(t)$, referring to individuals that have recovered from the disease and gain temporary immunity at time t. Thus the total human host population at time t denoted by $N(t)$ is given by: $N_H(t) = S_H(t) + I_A(t) + I_C(t) + R_H(t)$. Individuals are recruited into the susceptible class through a constant birth rate Λ_H. It is assumed that there is no vertical transmission and therefore all newborns in the human population are susceptible. Humans in all the four classes die due to natural causes at a constant rate μ_H. When treatment is administered to humans in the

infected-acute class, some respond to treatment and recover at a rate ν while others progress to the infected-chronic class at a rate ρ. In this stage, they either die a natural death or due to the disease at a rate ϵ. The model assumes that there is no permanent immunity and therefore, the recovered humans lose their immunity at a rate ψ.

The total blackfly population at time t, denoted by $N_V(t)$ is divided into the susceptible blackfly population, $S_V(t)$, referring to blackflies that are at risk of being infected upon biting an infected human at time t and the infected blackfly vectors, $I_V(t)$, referring to blackflies that are infected with the disease and are capable of transmitting it to humans at time t. The total blackfly vector population at time t is given by: $N_V(t) = S_V(t) + I_V(t)$. There is no recovered class for the blackflies because they do not live long enough to recover from onchocerciasis. It is also assumed that the blackflies do not die from onchocerciasis. The susceptible blackflies become infected after interacting with infected humans at a biting rate α. The model assumes that blackflies in both classes die either naturally at a rate μ_V or are trapped and killed at a rate θ. The proportion of trapped susceptible blackflies is represented by a whereas that of trapped infected blackflies is represented by b. It is assumed that all variables represented in each compartment are differentiable with respect to time and all parameters are non negative.

2.1 *Equations of the model*

The human and blackfly populations are governed by the following system of ordinary differential equations:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \alpha\beta_H \frac{S_H I_V}{N_H} + \psi R_H - \mu_H S_H,\\
\frac{dI_A}{dt} &= \alpha\beta_H \frac{S_H I_V}{N_H} - (\mu_H + \rho + \nu) I_A,\\
\frac{dI_C}{dt} &= \rho I_A - \mu_H I_C - \epsilon I_C,\\
\frac{dR_H}{dt} &= \nu I_A - \psi R_H - \mu_H R_H,\\
\frac{dS_V}{dt} &= \Lambda_V - \alpha\beta_V \frac{(I_A + I_C) S_V}{N_H} - (\mu_V + \theta a) S_V,\\
\frac{dI_V}{dt} &= \alpha\beta_V \frac{(I_A + I_C) S_V}{N_H} - (\mu_V + \theta b) I_V,
\end{aligned} \tag{1}$$

together with

$$N_H(t) = S_H(t) + I_A(t) + I_C(t) + R_H(t), \tag{2}$$

and

$$N_V(t) = S_V(t) + I_V(t). \tag{3}$$

2.2 *Basic properties of the model*

In this subsection, the basic properties of the model are studied. The non-negativity of solutions of system (1) are described as follows:

2.2.1 Positivity and boundedness of the solutions

Theorem 2.1 *Consider system (1) with initial conditions* $\{(S_{H_0}, I_{A_0}, I_{C_0}, R_{H_0}, S_{V_0}, I_{V_0}) \geqslant 0\} \in \mathbb{R}^6_+$. *Then the solution set* $\{(S_H(t), I_A(t), I_C(t), R_H(t), S_V(t), I_V(t)\}$ *of system (1) is non-negative for all* $t > 0$.

Proof From the first equation of system (1),

$$\frac{dS_H}{dt} = \Lambda_H - \alpha\beta_H \frac{S_H I_V}{N_H} + \psi R_H - \mu_H S_H \geqslant -\Big(\alpha\beta_H \frac{I_V}{N_H} + \mu_H\Big) S_H.$$

That is,

$$\frac{dS_H}{dt} \geqslant -\Big(\alpha\beta_H \frac{I_V}{N_H} + \mu_H\Big) S_H. \tag{4}$$

By separating variables, equation (8) is integrated with initial conditions $S(0) = S_0$ as follows:

$$\int \frac{dS_H}{S_H} \geqslant -\int \Big(\alpha\beta_H \frac{I_V}{N_H} + \mu_H\Big) dt,$$

which yields

$$S_H(t) \geqslant S_0 e^{-\int (\alpha\beta_H \frac{I_V}{N_H} + \mu_H) dt} > 0.$$

In a similar way, it is shown that the remaining equations of system (1) are also positive for all $t > 0$. Thus, the solutions of the model are non-negative for all values of $t > 0$. ■

2.2.2 Invariant region

Theorem 2.2 *The region* $\mathcal{D} = \{(S_H, I_A, I_C, R_H) \in \mathbb{R}^4_+ : 0 \leqslant N_H \leqslant \frac{\Lambda}{\mu_H}, (S_V, I_V) \in \mathbb{R}^2_+ : 0 \leqslant N_V \leqslant \frac{\Lambda_V - \theta a S_V}{\mu_V}\}$ *is positively invariant and attracting with respect to the model.*

Proof Let $\{(S_H(t), I_A(t), I_C(t), R_H(t), S_V(t), I_V(t)\}$ be any solution of system (1) with non-negative initial conditions given by $\{(S_{H_0}, I_{A_0}, I_{C_0}, R_{H_0}, S_{V_0}, I_{V_0})$. We obtain the region the total human population of susystem (1) is bounded by differentiating equation (2) to obtain

$$\frac{dN_H}{dt} = \Lambda_H - \mu_H N_H - \epsilon I_C. \tag{5}$$

It is noted that in the absence of infected humans, that is, when $I_C = 0$, then

$$\frac{dN_H}{dt} \leqslant \Lambda - \mu_H N_H. \tag{6}$$

Solving equation (6) gives

$$N_H(t) \leqslant \frac{\Lambda}{\mu_H} + \left(N_H(0) - \frac{\Lambda}{\mu_H}\right) exp(-\mu_H t).$$

Evaluating as $t \to \infty$, shows that $N_H(t) \to \frac{\Lambda}{\mu_H}$. Therefore the total human population is bounded by $\frac{\Lambda}{\mu_H}$, and the solution is bounded in $\mathcal{D}_H = \{(S_H, I_A, I_C, R_H) \in \mathbb{R}^4_+ : 0 \leqslant N_H \leqslant \frac{\Lambda}{\mu_H}\}$.
Similarly, for the blackfly vector population, differentiating equation (3) and substituting for $\frac{dS_V}{dt}$ and $\frac{dI_V}{dt}$ from system (1) gives

$$\frac{dN_V}{dt} = \Lambda_V - [\mu_V N_V + \theta a S_V + \theta b I_V]. \tag{7}$$

It is noted that in the absence of infected blackflies, that is, when $I_V = 0$, then

$$\frac{dN_V}{dt} \leqslant \Lambda_V - \mu_V N_V - \theta a S_V. \tag{8}$$

Solving equation (8) gives

$$N_V(t) = \frac{\Lambda_V - \theta a S_V}{\mu_V} + \frac{\theta a}{\mu_V} \exp(-\mu_V t) \int \exp(\mu_V t) \frac{dS_V}{dt} dt.$$

As $t \to \infty$, $N_V(t) \to \frac{\Lambda_V - \theta a S_V}{\mu_V}$ and thus, the total blackfly population is bounded by $\frac{\Lambda_V - \theta a S_V}{\mu_V}$, and the solution is bounded in $\mathcal{D}_V = \{(S_V, I_V) \in \mathbb{R}^2_+ : 0 \leqslant N_V \leqslant \frac{\Lambda_V - \theta a S_V}{\mu_V}\}$. Therefore, the solution set of system (1) is bounded in $\mathcal{D} = \{(S_H, I_A, I_C, R_H) \in \mathbb{R}^4_+ : 0 \leqslant N_H \leqslant \frac{\Lambda}{\mu_H}, (S_V, I_V) \in \mathbb{R}^2_+ : 0 \leqslant N_V \leqslant \frac{\Lambda_V - \theta a S_V}{\mu_V}\}$. The model is thus epidemiologically and mathematically well posed. ■

3. Model analysis

3.1 *Equilibria of the model*

It is easier to analyse system (1) in terms of proportions of quantities instead of actual population sizes. This is done by having the ratio of the population of each subgroup to the total species population. Let $s_h = \frac{S_H}{N_H}$, $i_a = \frac{I_A}{N_H}$, $i_c = \frac{I_C}{N_H}$, $r_h = \frac{R_H}{N_H}$, $s_v = \frac{S_V}{N_V}$ and $i_v = \frac{I_V}{N_V}$ be the proportions of the subgroups S_H, I_A, I_C, R_H, S_V and I_V respectively. Note that the ratio of the total vector population to the total human population is denoted as $m = \frac{N_V}{N_H}$. The ratio m is taken as a constant because it is well known that a vector takes a fixed number of blood meals per unit time independent of the population density of the host [35]. Therefore, by differentiating with respect to time t, it is clear that s_h, i_a, i_c, r_h, s_v and i_v satisfy the following

system of differential equations:

$$\begin{aligned}
\frac{ds_h}{dt} =&\frac{\Lambda_H}{N_H}(1-s_h) - \alpha\beta_H s_h i_v m + \psi r_h + \epsilon i_c s_h,\\
\frac{di_a}{dt} =&\alpha\beta_H s_h i_v m - i_a\Big(\frac{\Lambda_H}{N_H} - \epsilon i_c + \nu + \rho\Big),\\
\frac{di_c}{dt} =&\rho i_a - \epsilon i_c - i_c\frac{\Lambda_H}{N_H} + \epsilon i_c^2,\\
\frac{dr_h}{dt} =&\nu i_a - \psi r_h - \frac{\Lambda_H}{N_H} r_h + \epsilon i_c r_h,\\
\frac{ds_v}{dt} =&, \theta a s_v^2 - s_v\Big(\theta a + \alpha\beta_V(i_a+i_c) - \theta b i_v + \frac{\Lambda_V}{N_V}\Big) + \frac{\Lambda_V}{N_V},\\
\frac{di_v}{dt} =&\alpha\beta_V(i_a+i_c)s_v - \Big(\frac{\Lambda_V}{N_V} + \theta b - \theta a s_v\Big)i_v + \theta b i_v^2
\end{aligned} \tag{9}$$

where $s_h + i_a + i_c + r_h = 1$ and $s_v + i_v = 1$. And

$$\frac{dN_H}{dt} = \Big(\frac{\Lambda_H}{N_H} - \mu_H - \epsilon i_c\Big)N_H. \tag{10}$$

Consider the equation (10). At equilibrium, $\frac{dN_H}{dt} = 0$ and since $N_H \neq 0$, then this implies that $\frac{\Lambda_H}{N_H} = \mu_H + \epsilon i_c$.

Also, from equation $\frac{dN_V}{dt} = \big(\frac{\Lambda_V}{N_V} - \mu_V - \theta a s_v - \theta b i_v\big)N_V$, $\frac{dN_V}{dt} = 0$ at equilibrium and since $N_V \neq 0$, then this implies $\frac{\Lambda_V}{N_V} = \mu_v + \theta a s_v + i_v\theta b$. Substituting for $\frac{\Lambda_H}{N_H}$, $\frac{\Lambda_V}{N_V}$ and the proportion $r_h = 1 - (s_h + i_a + i_c)$ in system (9) above to reduce the dimension of the system gives

$$\begin{aligned}
\frac{ds_h}{dt} =&\mu_H + \epsilon i_c + \psi(1 - i_a - i_c) - s_h(\mu_H + \psi + \alpha\beta_H i_v m),\\
\frac{di_a}{dt} =&\alpha\beta_H s_h i_v m - (\mu_H + \rho + \nu)i_a,\\
\frac{di_c}{dt} =&\rho i_a - i_c(\epsilon + \mu_H),\\
\frac{ds_v}{dt} =&(\mu_v + \theta b) - s_v\Big(\alpha\beta_V(i_a+i_c) + \mu_V + \theta b\Big),\\
\frac{di_v}{dt} =&\alpha\beta_V(i_a+i_c) - i_v\Big(\alpha\beta_V(i_a+i_c) + \mu_V + \theta b)\Big).
\end{aligned} \tag{11}$$

Theorem 3.1 *System (11) has two equilibrium points. The unique disease free equilibrium* $E_0(s_h, i_a, i_c, s_v, i_v) = [1, 0, 0, 1, 0]$ *and the endemic equilibrium*

$$E_1(s_h, i_a, i_c, s_v, i_v) = \Bigg[\frac{\mu_H + \epsilon i_c + \psi(1 - i_a - i_c)}{\mu_H + \psi + \alpha\beta_H m\Big(\frac{\alpha\beta_V(i_a+i_c)}{\mu_V + \theta b + \alpha\beta_V(i_a+i_c)}\Big)}, \frac{\alpha\beta_H m g f}{\mu_H + \rho + \nu}, \frac{\rho i_a}{(\epsilon + \mu_H)},$$
$$\frac{\mu_V + \theta b}{\mu_V + \theta b + \alpha\beta_V(i_a+i_c)}, \frac{\alpha\beta_V(i_a+i_c)}{\mu_V + \theta b + \alpha\beta_V(i_a+i_c)}\Bigg],$$

where $f = s_h$ *and* $g = i_v$.

Proof Setting the right hand sides of equations in system (11) equal to zero for all infected variables set to zero, namely $i_a = i_c = i_v = 0$ gives $s_h = s_v = 1$. Therefore, there exists a disease free equilibrium point $E_0(s_h, i_a, i_c, s_v, i_v) = [1, 0, 0, 1, 0]$. For $i_a \neq 0$ and $i_c \neq 0$, we determine the endemic equilibrium point by expressing each of s_h, s_v and i_v in terms of i_a and/or i_c. Therefore, there exists an endemic equilibrium point

$$E_1(s_h, i_a, i_c, s_v, i_v) = \left[\frac{\mu_H + \epsilon i_c + \psi(1 - i_a - i_c)}{\mu_H + \psi + \alpha\beta_H m\left(\frac{\alpha\beta_V(i_a+i_c)}{\mu_V+\theta b+\alpha\beta_V(i_a+i_c)}\right)}, \frac{\alpha\beta_H mgf}{\mu_H + \rho + \nu}, \frac{\rho i_a}{(\epsilon + \mu_H)}, \right.$$
$$\left. \frac{\mu_V + \theta b}{\mu_V + \theta b + \alpha\beta_V(i_a + i_c)}, \frac{\alpha\beta_V(i_a + i_c)}{\mu_V + \theta b + \alpha\beta_V(i_a + i_c)} \right],$$

where $f = s_h$ and $g = i_v$. ■

3.2 *Basic reproduction number*

According to Diekmann et al. [14], the basic reproduction number, $\mathcal{R}_0$ is the expected number of secondary cases produced by a typically infected individual during its entire period of infectiousness in a completely susceptible population.
The basic reproduction number is obtained using the next generation matrix method as described in Diekmann et al. [14].
Let

$$\frac{dx_i}{dt} = X_i(x_1 \dots x_n), i = 1, \dots, n \tag{12}$$

be a system of differential equations governing the spread of an epidemic and suppose that

$$\frac{dx_j}{dt} = X_j(x_1 \dots x_n), j = 1, \dots, m$$

is the infected subsystem of system (12).
In order to compute $\mathcal{R}_0$, it is important to distinguish new infections that enter the population from others in the population. Consider $\mathcal{F}_j$ to be a vector of new infections and $\mathcal{V}_j$ to be a vector formed by other transfers. $\mathcal{F}_j$ and $\mathcal{V}_j$ are assumed to be continuously differentiable. The basic reproduction number is the spectral radius of the next generation matrix FV^{-1}, where $F = \partial\mathcal{F}_j$ and $V = \partial\mathcal{V}_j$ computed at the disease free equilibrium point of system (9).

$$\mathcal{R}_0 = \sigma(FV^{-1}).$$

Consider the infected subsystem of system (11) below

$$\frac{di_a}{dt} = \alpha\beta_H s_h i_v m - (\mu_H + \rho + \nu)i_a,$$
$$\frac{di_c}{dt} = \rho i_a - i_c(\epsilon + \mu_H),$$
$$\frac{di_v}{dt} = \alpha\beta_V(i_a + i_c)s_v - (\mu_V + \theta b)i_v.$$

The vector of new infections $\mathcal{F}$ and the vector formed by other transfers $\mathcal{V}$ are given by

$$\mathcal{F} = \begin{bmatrix} \alpha\beta_H s_h i_v m \\ 0 \\ \alpha\beta_V (i_a + i_c) s_v \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} (\mu_H + \rho + \nu) i_a \\ -\rho i_a + (\epsilon + \mu_H) i_c \\ (\mu_V + \theta b) i_v \end{bmatrix}.$$

For the disease free equilibrium point E_0, matrices F_0 and V_0 are computed as follows:

$$F_0 = \begin{bmatrix} 0 & 0 & \alpha\beta_H m \\ 0 & 0 & 0 \\ \alpha\beta_V & \alpha\beta_V & 0 \end{bmatrix} \quad \text{and} \quad V_0 = \begin{bmatrix} \rho + \nu + \mu_H & 0 & 0 \\ -\rho & (\mu_H + \epsilon) & 0 \\ 0 & 0 & \mu_V + \theta b \end{bmatrix}.$$

Using row operations, the inverse of matrix V_0 is obtained from $V_0 I = I V_0^-$, where I is the identity matrix.

$$V_0^{-1} = \begin{bmatrix} \frac{1}{\mu_H+\rho+\nu} & 0 & 0 \\ \frac{\rho}{(\epsilon+\mu_H)(\mu_H+\nu+\rho)} & \frac{1}{\mu_H+\epsilon} & 0 \\ 0 & 0 & \frac{1}{\mu_V+\theta b} \end{bmatrix}.$$

Thus, the next generation matrix is given by

$$F_0 V_0^{-1} = \begin{bmatrix} 0 & 0 & \frac{\alpha\beta_H m}{\mu_V+\theta b} \\ 0 & 0 & 0 \\ \frac{\alpha\beta_V(\mu_H+\rho+\epsilon)}{(\mu_H+\epsilon)(\mu_H+\rho+\nu)} & \frac{\alpha\beta_V}{\mu_H+\epsilon} & 0 \end{bmatrix}.$$

The eigenvalues of $F_0 V_0^{-1}$ are

$$0, -\sqrt{\frac{\alpha^2\beta_H\beta_V m(\mu_H+\epsilon+\rho)}{(\mu_V+\theta b)(\mu_H+\epsilon)(\mu_H+\rho+\nu)}} \text{ and } \sqrt{\frac{\alpha^2\beta_H\beta_V m(\mu_H+\epsilon+\rho)}{(\mu_V+\theta b)(\mu_H+\epsilon)(\mu_H+\rho+\nu)}}.$$

Therefore, the basic reproduction number $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \sqrt{\frac{\alpha^2\beta_H\beta_V m(\mu_H+\epsilon+\rho)}{(\mu_V+\theta b)(\mu_H+\epsilon)(\mu_H+\rho+\nu)}}.$$

3.3 *Sensitivity analysis*

Onchocerciasis control and eradication strategies should target important parameters which have a high impact on the basic reproduction number. A sensitivity analysis of $\mathcal{R}_0$ to each of the 8 different parameters is derived and presented in Table 1 below. The basic reproduction number is explicitly determined by the parameters $\alpha, \beta_H, \beta_V, \mu_H, \epsilon, \rho, \nu$ and μ_V. The sensitivity index of $\mathcal{R}_0$ to each of

the parameters is computed using the approach by Chitnis *et al.* [10].

Definition 3.2 The sensitivity index of a variable u, that depends continuously on a parameter p is defined as

$$\Upsilon_p^u = \frac{\partial u}{\partial p}.\frac{p}{u},$$

where u is a differentiable function of p.

Thus by the definiton above, the formula used to derive an expression for the sensitivity of $\mathcal{R}_0$ to a parameter p is

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p}.\frac{p}{\mathcal{R}_0}.$$

The sensitivity indices of β_H and β_V are given by

$$\Upsilon_{\beta_H}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_H}.\frac{\beta_H}{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_V}.\frac{\beta_V}{\mathcal{R}_0} = 0.5.$$

The same approach is used to obtain the indices of other parameters, that is, α,

Table 1. Numerical values of sensitivity indices of $\mathcal{R}_0$.

Parameter symbol	**Sensitivity index**
β_H	0.5
β_V	0.5
ϵ	-0.12354
ρ	0.41437
ν	-0.42037
μ_H	-0.36909
μ_V	-0.09615
α	1

μ_H, ν, ϵ, ρ and μ_V. The parameter values used are $\alpha = 0.0855$, $\mu_H = 1/23178$, $\nu = 1/45$, $\epsilon = 0.00001438$, $\rho = 1/240$ and $\mu_V = 1/21$.

Table 1 shows the sensitivity indices of $\mathcal{R}_0$ to the parameters. A positive value in the sensitivity index shows that if the parameter is increased when all other parameters are kept constant, then the value of $\mathcal{R}_0$ increases, while for a negative sensitivity index, when the parameter value is increased with all other parameters kept constant, the value of $\mathcal{R}_0$ decreases.

3.4 *Local stability of the disease free equilibrium*

To determine the local stability of the disease free equilibrium point E_0, the Jacobian matrix for system (11) evaluated at the disease free equilibrium point E_0 given by $E_0(s_h, i_a, i_c, s_v, i_v) = [1, 0, 0, 1, 0]$ to obtain the Jacobian matrix $\mathcal{J}_{E_0}$ given

below. The local stability of the disease free equilibrium using the Jacobian matrix

$$\mathcal{J}_{E_0} = \begin{bmatrix} -(\mu_H+\psi) & -\psi & -\psi+\epsilon & 0 & -\alpha\beta_H m \\ 0 & -(\mu_H+\rho+\nu) & 0 & 0 & \alpha\beta_H m \\ 0 & \rho & -(\mu_H+\epsilon) & 0 & 0 \\ 0 & -\alpha\beta_V & -\alpha\beta_V & -(\mu_V+\theta b) & 0 \\ 0 & \alpha\beta_V & \alpha\beta_V & 0 & -(\mu_V+\theta b) \end{bmatrix}.$$

is done as follows:

The eigenvalues of $\mathcal{J}_{E_0}$ are $-(\mu_H+\psi)$, $-\mu_V$ and the eigenvalues of the polynomial;

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{13}$$

in which λ is the eigenvalue, where

$$a_1 = 2\mu_H + \mu_V + \epsilon + \rho + \nu + \theta b, \tag{14}$$

$$a_2 = (\mu_H+\rho+\nu)(\mu_H+\mu_V+\theta b+\epsilon) + (\mu_H+\epsilon)(\mu_V+\theta b) - \alpha^2\beta_H\beta_V m, \tag{15}$$

and

$$a_3 = (\mu_V+\theta b)(\mu_H+\rho+\nu)(\mu_H+\epsilon) - \alpha^2\beta_H\beta_V m(\rho+\mu_H+\epsilon). \tag{16}$$

By the Routh-Hurwitz criteria for a polynomial of degree 3 , the eigenvalues of the characteristic equation (13) have negative real parts if and only if the following conditions $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 > a_3$ hold for the coefficients of the characteristic equation (13). From equation (14), it is clear that $a_1 > 0$. From equation (15), $a_2 > 0$ if

$$(\mu_H+\rho+\nu)(\mu_H+\mu_V+\theta b+\epsilon) + (\mu_H+\epsilon)(\mu_V+\theta b) > \alpha^2\beta_H\beta_V m.$$

From equation (16), $a_3 > 0$ if

$$(\mu_V+\theta b)(\mu_H+\rho+\nu)(\mu_H+\epsilon) > \alpha^2\beta_H\beta_V m(\rho+\mu_H+\epsilon).$$

This gives the expression

$$\begin{aligned} &\frac{(\mu_V+\theta b)(\mu_H+\rho+\nu)(\mu_H+\epsilon)}{\alpha^2\beta_H\beta_V m(\rho+\mu_H+\epsilon)} > 1 \\ &\Rightarrow \frac{1}{\mathcal{R}_0^2} > 1 \\ &\Rightarrow \mathcal{R}_0 < 1 \end{aligned}$$

where $\mathcal{R}_0 = \sqrt{\frac{\alpha^2\beta_H\beta_V m(\mu_H+\epsilon+\rho)}{(\mu_V+\theta b)(\mu_H+\epsilon)(\mu_H+\rho+\nu)}}$.

This implies that $a_3 > 0$ if $\mathcal{R}_0 < 1$. The condition $a_1a_2 - a_3 > 0$ is also checked to verify whether it holds since $a_1 > 0, a_2 > 0$ and $a_3 > 0$ are true.
$a_1a_2 - a_3 = (2\mu_H + \mu_V + \epsilon + \rho + \nu + \theta b) \times \Big((\mu_H + \rho + \nu)(\mu_H + \mu_V + \theta b + \epsilon) + (\mu_H + \epsilon)(\mu_V + \theta b) - \alpha^2 \beta_H \beta_V m \Big) - \Big((\mu_V + \theta b)(\mu_H + \rho + \nu)(\mu_H + \epsilon) - \alpha^2 \beta_H \beta_V m(\rho + \mu_H + \epsilon) \Big) > 0.$
Since $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 - a_3 > 0$, then the characteristic polynomial above has negative real parts. Hence, the disease free equilibrium point E_0 is locally asymptotically stable when $\mathcal{R}_0 < 1$. Thus, E_0 is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$, and thus the following theorem has been established:

Theorem 3.3 *The disease-free equilibrium E_0 is locally stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

3.5 *Global stability of the disease free equilibrium*

The global stability of the disease free equilibrium is investigated using a theorem by Castillo-Chavez and Song [7] . System (11) can be re-written as

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z), \\ \frac{dZ}{dt} &= G(X, Z), G(X, 0) = 0, \end{aligned} \tag{17}$$

where $X \in \Re^m$ denotes the number of uninfected individuals and $Z \in \Re^n$ denotes the number of infected individuals. The disease free equilibrium of the model is denoted by $U = (X^*, 0)$. For the point $U = (X^*, 0)$ to be globally asymptotically stable, the following conditions (H_1) and (H_2) must be satisfied provided $\mathcal{R}_0 < 1$.

(H_1) For $\frac{dX}{dt} = F(X, 0), X^*$
(H_2) $G(X, Z) = MZ - \widehat{G}(X, Z), \widehat{G}(X, Z) \geqslant 0$ for $(X, Z) \in \Omega$,
where $M = D_Z G(X^*, 0)$ is an M-matrix and Ω is the region where the model makes biological sense. If system (11) satisfy the above two conditions, then the following theorem holds.

Theorem 3.4 *The point $U = (X^*, 0)$ is globally asymptotically stable equilibrium provided that $\mathcal{R}_0 < 1$ and the conditions (H_1) and (H_2) are satisfied.*

Proof From system (11),

$$F(X, Z) = \begin{bmatrix} \mu_H + \epsilon i_c + \psi(1 - i_a - i_c) - s_h(\mu_H + \psi + \alpha\beta_H i_v m) \\ (\mu_V + \theta b) - s_v(\mu_V + \theta b + \alpha\beta_V(i_a + i_c)) \end{bmatrix}$$

and

$$G(X, Z) = \begin{bmatrix} \alpha\beta_H s_h i_v m - i_a(\mu_H + \rho + \nu) \\ \rho i_a - i_c(\epsilon + \mu_H) \\ \alpha\beta_V(i_a + i_c) - i_v(\mu_V + \theta b + \alpha\beta_V(i_a + i_c)) \end{bmatrix}.$$

To investigate condition (H_1),

$$F(X, 0) = \begin{bmatrix} (1 - s_h)(\mu_H + \psi) \\ (1 - s_v)(\mu_V + \theta b) \end{bmatrix}.$$

Since $s_h \leqslant 1$ and $s_v \leqslant 1$, then $F(X,0) \geqslant 0$ and therefore, there is convergence in Ω. Therefore, X^* is globally asymptotically stable.
To investigate condition (H_2) for the disease free equilibrium point E_0,

$$M = \begin{bmatrix} -(\mu_H + \rho + \nu) & 0 & \alpha\beta_H m \\ \rho & -(\mu_H + \epsilon) & 0 \\ \alpha\beta_V & \alpha\beta_V & -(\mu_V + \theta b) \end{bmatrix},$$

$$\widehat{G}(X,Z) = \begin{bmatrix} \alpha\beta_H i_v m(1 - s_h) \\ 0 \\ \alpha\beta_V(i_a + i_c)(1 - s_v) \end{bmatrix}.$$

Since $s_h \leqslant 1$ and $s_v \leqslant 1$, then $\widehat{G}(X,Z) \geqslant 0$ and thus equilibrium E_0 is globally asymptotically stable. ■

3.6 *Local stability of the endemic equilibrium*

Theorem 3.5 *For non-negative initial conditions and $\mathcal{R}_0 > 1$, the endemic equilibrium E_1 of system (11) is locally asymptotically stable whenever it exists.*

Proof The Jacobian matrix of system (11) evaluated at the endemic equilibrium point E_1 is

$$\mathcal{J}_{E_1} = \begin{bmatrix} -a_{11} & -a_{12} & a_{13} & 0 & -a_{15} \\ a_{21} & -a_{22} & 0 & 0 & a_{25} \\ 0 & a_{32} & -a_{33} & 0 & 0 \\ 0 & -a_{42} & -a_{43} & -a_{44} & 0 \\ 0 & a_{52} & a_{53} & 0 & -a_{55} \end{bmatrix},$$

where $a_{11} = (\mu_H + \alpha\beta_H i_v^* m + \psi)$, $a_{12} = \psi$, $a_{13} = -\psi + \epsilon$, $a_{15} = \alpha\beta_H s_h m$, $a_{21} = \alpha\beta_H i_v m$, $a_{22} = \mu_H + \rho + \nu$, $a_{25} = \alpha\beta_H s_h m$, $a_{32} = \rho$, $a_{33} = (\mu_H + \epsilon)$, $a_{42} = \alpha\beta_V s_v$, $a_{43} = \alpha\beta_V s_v$, $a_{44} = \alpha\beta_V(i_a + i_c) + (\mu_V + \theta b)$, $a_{52} = \alpha\beta_V s_v$, $a_{53} = \alpha\beta_V s_v$, $a_{55} = (\mu_V + \theta b) + \alpha\beta_V(i_a + i_c)$.

The characteristic equation of the Jacobian matrix $\mathcal{J}_{E_1}$ is given by

$$\mathcal{Z}^5 + a_1\mathcal{Z}^4 + a_2\mathcal{Z}^3 + a_3\mathcal{Z}^2 + a_4\mathcal{Z} + a_5 = 0, \tag{18}$$

where

$$a_1 = a_{11} + a_{22} + a_{33} + a_{44} + a_{55},$$

$$\begin{aligned} a_2 = {} & a_{11}a_{22} + a_{12}a_{21} + a_{11}a_{33} + a_{11}a_{44} + a_{22}a_{33} + a_{11}a_{55} + a_{22}a_{44} \\ & - a_{25}a_{52} + a_{22}a_{55} + a_{33}a_{44} + a_{33}a_{55} + a_{44}a_{55}, \end{aligned}$$

$$a_3 = a_{11}a_{22}a_{33} - a_{13}a_{21}a_{32} + a_{12}a_{21}a_{33} + a_{11}a_{22}a_{44} + a_{12}a_{21}a_{44} - a_{11}a_{25}a_{52} + a_{15}a_{21}a_{51} + a_{11}a_{22}a_{55} + a_{11}a_{33}a_{44} + a_{12}a_{21}a_{55} + a_{11}a_{33}a_{55} + a_{22}a_{33}a_{44} - a_{25}a_{32}a_{53} + a_{11}a_{44}a_{55} - a_{25}a_{33}a_{52} + a_{22}a_{33}a_{55} + a_{25}a_{42}a_{54} - a_{25}a_{44}a_{52} + a_{22}a_{44}a_{55} + a_{33}a_{44}a_{55},$$

$$a_4 = a_{15}a_{21}a_{32}a_{53} - a_{11}a_{25}a_{32}a_{53} - a_{11}a_{25}a_{33}a_{52} - a_{13}a_{21}a_{32}a_{55} + a_{15}a_{21}a_{33}a_{52} + a_{11}a_{22}a_{33}a_{55} + a_{12}a_{21}a_{33}a_{55} + a_{11}a_{25}a_{42}a_{54} - a_{11}a_{25}a_{44}a_{52} - a_{15}a_{21}a_{42}a_{54} + a_{15}a_{21}a_{42}a_{52} + a_{11}a_{22}a_{44}a_{55} + a_{12}a_{21}a_{44}a_{55} + a_{11}a_{33}a_{44}a_{55} + a_{25}a_{32}a_{43}a_{54} - a_{25}a_{32}a_{44}a_{53} + a_{25}a_{33}a_{42}a_{54} - a_{25}a_{33}a_{44}a_{52} + a_{22}a_{33}a_{44}a_{55} - a_{13}a_{21}a_{32}a_{44} + a_{11}a_{22}a_{33}a_{44} + a_{12}a_{21}a_{33}a_{44},$$

$$a_5 = a_{11}a_{25}a_{32}a_{43}a_{54} - a_{11}a_{25}a_{32}a_{44}a_{53} - a_{15}a_{21}a_{32}a_{43}a_{54} + a_{15}a_{21}a_{32}a_{44}a_{52} + a_{11}a_{25}a_{33}a_{42}a_{54} - a_{11}a_{25}a_{33}a_{44}a_{52} - a_{13}a_{21}a_{32}a_{44}a_{55} - a_{15}a_{21}a_{33}a_{42}a_{54} + a_{15}a_{21}a_{33}a_{44}a_{52} + a_{11}a_{22}a_{33}a_{44}a_{55} + a_{12}a_{21}a_{33}a_{44}a_{55}.$$

The characteristic equation (18) has eigenvalues with negative real parts provided the coefficients satisfy the following Routh-Hurwitz criteria $a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_5 > 0, a_1a_2a_3 > a_3^2 + a_1^2a_4$ and $(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 - a_1^2a_4) > a_5(a_1a_2 - a_3)^2 + a_1a_5^2$.
If the above Routh-Hurwitz criteria are satisfied, then the endemic equilibrium E_1 is locally asymptotically stable whenever it exists. ■

3.7 *Global stability of the endemic equilibrium*

Theorem 3.6 *The endemic equilibrium point E_1 for the system (11) is globally asymptotically stable in $\mathcal{D}$ if $\mathcal{R}_0 > 1$.*

Proof Consider a Lyapunov function of the form

$$L(t) = \left(s_h - s_h^* - s_h^* ln(\frac{s_h}{s_h^*})\right) + \left(i_a - i_a^* - i_a^* ln(\frac{i_a}{i_a^*})\right) + \left(i_c - i_c^* - i_c^* ln(\frac{i_c}{i_c^*})\right) + \left(s_v - s_v^* - s_v^* ln(\frac{s_v}{s_v^*})\right) + \left(i_v - i_v^* - i_v^* ln(\frac{i_v}{i_v^*})\right).$$

Differentiating $L(t)$ with respect to time t gives

$$\frac{dL}{dt} = \left(\frac{s_h - s_h^*}{s_h}\right)\frac{ds_h}{dt} + \left(\frac{i_a - i_a^*}{i_a}\right)\frac{di_a}{dt} + \left(\frac{i_c - i_c^*}{i_c}\right)\frac{di_c}{dt} + \left(\frac{s_v - s_v^*}{s_v}\right)\frac{ds_v}{dt} + \left(\frac{i_v - i_v^*}{i_v}\right)\frac{di_v}{dt}.$$

$$\frac{dL}{dt} = \left(\frac{s_h - s_h^*}{s_h}\right)[\mu_H + \epsilon i_c + \psi(1 - i_a - i_c) - s_h(\mu_H + \psi + \alpha\beta_H i_v m)]$$
$$+ \left(\frac{i_a - i_a^*}{i_a}\right)[\alpha\beta_H s_h i_v m - (\mu_H + \rho + \nu)i_a]$$
$$+ \left(\frac{i_c - i_c^*}{i_c}\right)[\rho i_a - i_c(\epsilon + \mu_H)]$$
$$+ \left(\frac{s_v - s_v^*}{s_v}\right)[(\mu_V + \theta b) - s_v((\mu_V + \theta b) + \alpha\beta_V(i_a + i_c))]$$
$$+ \left(\frac{i_v - i_v^*}{i_v}\right)[\alpha\beta_V(i_a + i_c) - i_v((\mu_V + \theta b) + \alpha\beta_V(i_a + i_c))]\,.$$

$$\frac{dL}{dt} = \left(\frac{s_h - s_h^*}{s_h}\right)[\mu_H + \epsilon(i_c - i_c^*) + \psi(1 - (i_a - i_a^*) - (i_c - i_c^*))$$
$$- (s_h - s_h^*)(\mu_H + \psi + \alpha\beta_H(i_v - i_v^*)m)]$$
$$+ \left(\frac{i_a - i_a^*}{i_a}\right)[\alpha\beta_H(s_h - s_h^*)(i_v - i_v^*)m - (\mu_H + \rho + \nu)(i_a - i_a^*)]$$
$$+ \left(\frac{i_c - i_c^*}{i_c}\right)[\rho(i_a - i_a^*) - (i_c - i_c^*)(\epsilon + \mu_H)]$$
$$+ \left(\frac{s_v - s_v^*}{s_v}\right)[(\mu_V + \theta b) - (s_v - s_v^*)((\mu_V + \theta b) + \alpha\beta_V((i_a - i_a^*) + (i_c - i_c^*)))]$$
$$+ \left(\frac{i_v - i_v^*}{i_v}\right)[\alpha\beta_V((i_a - i_a^*) + (i_c - i_c^*)) - (i_v - i_v^*)((\mu_V + \theta b)$$
$$+ \alpha\beta_V((i_a - i_a^*) + (i_c - i_c^*)))].$$

$$\frac{dL}{dt} = \frac{\mu_H(s_h - s_h^*)}{s_h} + \frac{\epsilon i_c(s_h - s_h^*)}{s_h} - \frac{\epsilon i_c^*(s_h - s_h^*)}{s_h} + \frac{\psi(s_h - s_h^*)}{s_h} - \frac{\psi i_a(s_h - s_h^*)}{s_h}$$
$$+ \frac{\psi i_a^*(s_h - s_h^*)}{s_h} - \frac{\psi i_c(s_h - s_h*)}{s_h} + \frac{\psi i_c^*(s_h - s_h*)}{s_h} - \frac{\mu_H(s_h - s_h^*)^2}{s_h}$$
$$- \frac{\psi(s_h - s_h^*)^2}{s_h} - \frac{\alpha\beta_H m i_v(s_h - s_h^*)^2}{s_h} + \frac{\alpha\beta_H m i_v^*(s_h - s_h^*)^2}{s_h}$$
$$+ \frac{\alpha\beta_H m s_h i_v(i_a - i_a^*)}{i_a} - \frac{\alpha\beta_H m s_h i_v^*(i_a - i_a^*)}{i_a} - \frac{\alpha\beta_H m s_h^* i_v(i_a - i_a^*)}{i_a}$$
$$+ \frac{\alpha\beta_H m s_h^* i_v^*(i_a - i_a^*)}{i_a} - \frac{\mu_H(i_a - i_a^*)^2}{i_a} - \frac{\rho(i_a - i_a^*)^2}{i_a} - \frac{\nu(i_a - i_a^*)^2}{i_a} + \frac{\rho i_a(i_c - i_c^*)}{i_c}$$
$$- \frac{\rho i_a^*(i_c - i_c^*)}{i_c} - \frac{(\epsilon + \mu_H)(i_c - i_c^*)^2}{i_c} + \frac{(\mu_V + \theta b)(s_v - s_v^*)}{s_v} - \frac{(\mu_V + \theta b)(s_v - s_v^*)^2}{s_v}$$
$$- \frac{\alpha\beta_V i_a(s_v - s_v^*)^2}{s_v} + \frac{\alpha\beta_V i_a^*(s_v - s_v^*)^2}{s_v} - \frac{\alpha\beta_V i_c(s_v - s_v^*)^2}{s_v} + \frac{\alpha\beta_V i_c^*(s_v - s_v^*)^2}{s_v}$$

$$+\frac{\alpha\beta_V i_a(i_v-i_v^*)}{i_v}-\frac{\alpha\beta_V i_a^*(i_v-i_v^*)}{i_v}+\frac{\alpha\beta_V i_c(i_v-i_v^*)}{i_v}-\frac{\alpha\beta_V i_c^*(i_v-i_v^*)}{i_v}$$
$$-\frac{(\mu_V+\theta b)(i_v-i_v^*)^2}{i_v}-\frac{\alpha\beta_V i_a(i_v-i_v^*)^2}{i_v}+\frac{\alpha\beta_V i_a^*(i_v-i_v^*)^2}{i_v}-\frac{\alpha\beta_V i_c(i_v-i_v^*)^2}{i_v}$$
$$+\frac{\alpha\beta_V i_c^*(i_v-i_v^*)^2}{i_v}$$

Collecting positive terms together and negative parts together gives

$$\frac{dL}{dt}=A-B,$$

where

$$A=\frac{\mu_H(s_h-s_h^*)}{s_h}+\frac{\epsilon i_c(s_h-s_h^*)}{s_h}+\frac{\psi(s_h-s_h^*)}{s_h}+\frac{\psi i_a^*(s_h-s_h^*)}{s_h}+\frac{\psi i_c^*(s_h-s_h*)}{s_h}$$
$$+\frac{\alpha\beta_H m i_v^*(s_h-s_h^*)^2}{s_h}+\frac{\alpha\beta_H m s_h i_v(i_a-i_a^*)}{i_a}+\frac{\alpha\beta_H m s_h^* i_v^*(i_a-i_a^*)}{i_a}+\frac{\rho i_a(i_c-i_c^*)}{i_c}$$
$$+\frac{(\mu_V+\theta b)(s_v-s_v^*)}{s_v}+\frac{\alpha\beta_V i_a^*(s_v-s_v^*)^2}{s_v}+\frac{\alpha\beta_V i_c^*(s_v-s_v^*)^2}{s_v}+\frac{\alpha\beta_V i_a(i_v-i_v^*)}{i_v}$$
$$+\frac{\alpha\beta_V i_c(i_v-i_v^*)}{i_v}+\frac{\alpha\beta_V i_a^*(i_v-i_v^*)^2}{i_v}+\frac{\alpha\beta_V i_c^*(i_v-i_v^*)^2}{i_v}.$$

and

$$B=\frac{\epsilon i_c^*(s_h-s_h^*)}{s_h}+\frac{\psi i_a(s_h-s_h^*)}{s_h}+\frac{\psi i_c(s_h-s_h*)}{s_h}+\frac{\mu_H(s_h-s_h^*)^2}{s_h}+\frac{\psi(s_h-s_h^*)^2}{s_h}$$
$$+\frac{\alpha\beta_H m i_v(s_h-s_h^*)^2}{s_h}+\frac{\alpha\beta_H m s_h i_v^*(i_a-i_a^*)}{i_a}+\frac{\alpha\beta_H m s_h^* i_v(i_a-i_a^*)}{i_a}$$
$$+\frac{\mu_H(i_a-i_a^*)^2}{i_a}+\frac{\rho(i_a-i_a^*)^2}{i_a}+\frac{\nu(i_a-i_a^*)^2}{i_a}+\frac{\rho i_a^*(i_c-i_c^*)}{i_c}+\frac{(\epsilon+\mu_H)(i_c-i_c^*)^2)}{i_c}$$
$$+\frac{(\mu_V+\theta b)(s_v-s_v^*)^*}{s_v}+\frac{\alpha\beta_V i_a(s_v-s_v^*)^2}{s_v}+\frac{\alpha\beta_V i_c(s_v-s_v^*)^2}{s_v}+\frac{\alpha\beta_V i_a^*(i_v-i_v^*)}{i_v}$$
$$+\frac{\alpha\beta_V i_c^*(i_v-i_v^*)}{i_v}+\frac{(\mu_V+\theta b)(i_v-i_v^*)^2}{i_v}-\frac{\alpha\beta_V i_a(i_v-i_v^*)^2}{i_v}+\frac{\alpha\beta_V i_c(i_v-i_v^*)^2}{i_v}.$$

Hence if $A<B$, then $\frac{dL}{dt}\leqslant 0$. Note that $\frac{dL}{dt}=0$ if and only if $s_h=s_h^*$, $i_a=i_a^*$, $i_c=i_c^*$, $s_v=s_v^*$, $i_v=i_v^*$ and thus the largest compact invariant set in $[(s_h^*,i_a^*,i_c^*,s_h^*,i_v^*)\in\mathcal{D}:\frac{dL}{dt}=0]$ is the singleton set E_1. By Lasalle's invariant principles [25], this implies that E_1 is globally asymptotically stable in $\mathcal{D}$ if $A<B$. ■

In the next section, the numerical solutions of system (11) for the parameter values given in Table 2 together with initial conditions are carried out. The parameter values are gleaned from literature within realistic ranges for a typical scenario in a rural community for the purpose of illustration. Where applicable, the unit of measure is day^{-1}.

4. Numerical simulations

To explore the dynamical behaviour of the onchocerciasis model in the human and blackfly populations and illustrate the analytical results, numerical simulations to system (11) are obtained using MATLAB computer software program given in the Appendex. Numerical simulations are performed using parameter values indicated in Table 2 together with the initial conditions in terms of proportions: $s_h(0) = 0.5$, $i_a(0) = 0.1$, $i_c(0) = 0.1$, $s_v(0) = 0.75$ and $i_v(0) = 0.25$ corresponding to $S_H(0) = 500$, $I_A(0) = 100$, $I_C(0) = 100$, $S_V(0) = 150$ and $I_V(0) = 50$.

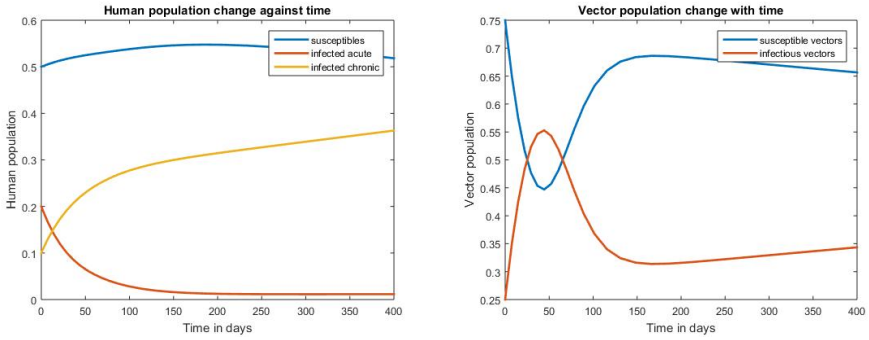
Table 2. Parameters values.

Parameter	Symbol	Value	Reference
Recruitment rate of humans	Λ_H	0.031	[31]
Recruitment rate of blackflies	Λ_V	0.73	[16]
Natural death rate of humans	μ_H	1/23178	[31]
Natural death rate of blackflies	μ_V	1/21	[9]
Trapping rate of blackflies	θ	$0 - 0.9$	[28]
Biting rate of blackflies	α	0.0855	[4]
Rate of loss of immunity	ψ	1/105	[12]
Probability that a blackfly becomes infective after biting an infected human host	β_V	0.80	[4]
Proportion of infective bites on a human host	β_H	0.073	[4]
Recovery rate of infected acute humans	ν	1/45	[38]
Progression rate to the infected chronic class	ρ	1/240	[1]
Disease induced death rate	ϵ	0.00001438	[11]

Figure 1 shows the numerical simulations of the model without blackfly trapping. It is observed that from Figures 1(a) and 1 (b), the endemic equilibrium point is stable in both the human and blackfly populations. This shows that the endemic equilibrium is locally stable. In Figure 1(a), the infected chronic human population is seen to increase as the infected acute human population falls. The fall in the infected acute human population is due to recovery, natural death and progression to the infected chronic human population. It is observed that the decrease in the infected acute human population is largely due to the progression to the infected chronic human population.

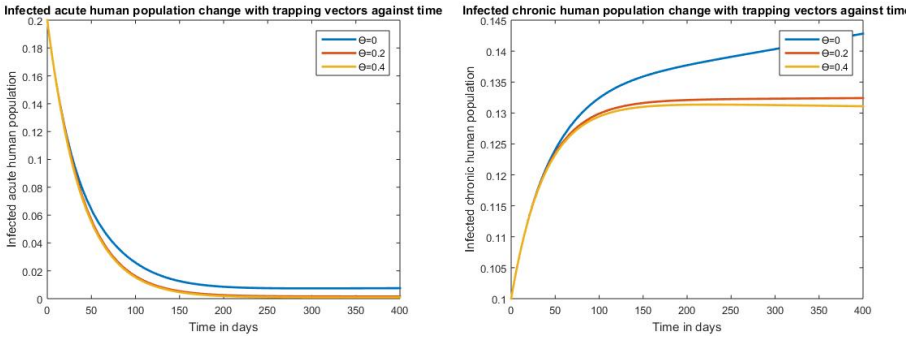
Figure 1(b) shows that in the initial stages of the disease, the infectious blackflies increase rapidly as the susceptible blackflies decrease. This is shown by a sharp rise in the curve for the infectious blackflies within the first 50 days. The infection in the blackfly vector population reaches its peak, then falls and later gains stability. This shows that transmission of onchocerciasis is more rapid during the early days of the outbreak.

Figure 2 shows the effect of trapping blackflies on the disease transmission dynamics. Figure 2(a) shows the effect of trapping blackflies on the infected acute human population and Figure 2(b) shows the effect of trapping blackflies on the infected chronic human population. It is observed that the infection in the infected acute human population is highest at the lowest value of θ ($\theta = 0$) and lowest at the highest value of θ ($\theta = 0.4$). This shows that trapping of blackflies reduces the infected acute human population with time. Similarly, it is observed in 2(b) that the infected chronic human population is highest at the lowest value of θ ($\theta = 0$)



(a) (b)

Figure 1. Simulation results showing change in population with time in the absence of trapping interventions (a) Human population, and Blackfly population.



(a) (b)

Figure 2. Simulation results showing the effect of variation of trapping on (a) infected acute human population, and (b) infected chronic human population.

and lowest at the highest value of θ ($\theta = 0.4$). This shows that trapping blackflies decreases the infected chronic human population with time. The change in the infected chronic human population is observed to begin after about 35 days. This is because the infected acute humans who were infected before trapping still progress to the infected chronic human class at the same rate within the first days of trapping.

A greater fall is observed in the infected chronic human population as compared to the fall in the infected acute human population due to blackfly trapping, especially for $\theta = 0.2$. This shows that trapping blackflies affects the infected chronic human population more than the infected acute human population after a long time. This is because of the possibility of super infections caused by bites of infected blackflies on the infected acute human population. However, with trapping of the disease transmitting blackflies, this possibility is mitigated as the blackflies are stopped from biting the humans.

5. Discussion and conclusion

In this paper, an onchocerciasis transmission and control model with blackfly trapping is formulated. The model is firstly shown to be mathematically and epidemiologically well posed followed by the investigation of its equilibria. Analysis of the

model shows the existence of two equilibria with one being the disease free equilibrium E_0 and the other being the endemic equilibrium E_1. The basic reproduction number $\mathcal{R}_0$ of the model is determined and its value is seen to be dependent on the interaction coefficient between the human host and blackfly vector populations. Sensitivity analysis of the parameters highlights the biting rate of the blackflies as the most sensitive parameter. Interventions should therefore be focussed on reducing the contact rate between the humans and the blackflies which in turn reduces the blackfly biting rate α and consequently also reduces the value of $\mathcal{R}_0$ which is a necessary condition for mitigating the disease. It is shown that the disease free equilibrium point is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$. This means that if an infected individual is introduced to the system, the disease wanes if $\mathcal{R}_0 < 1$ otherwise it spreads. The disease free equilibrium E_0 is also shown to be globally asymptotically stable if $\mathcal{R}_0 < 1$. There exists a unique endemic equilibrium if $\mathcal{R}_0 > 1$. The necessary and sufficient conditions for endemic local stability provided by Routh-Hurwitz criteria confirm that E_1 is locally asymptotically stable. A Lyapunov function is used to investigate global stability of the endemic equilibrium E_1. It is therefore established that the endemic equilibrium E_1 is locally and globally asymptotically stable when $\mathcal{R}_0 > 1$ implying that the disease persists in the population as long as $\mathcal{R}_0 > 1$. Thus, it is important to keep the basic reproduction number below unity.

Numerical simulations show that the disease persists in the community with continued existence of the blackflies. This is shown by a fall in the infected acute human population with a consequent rise in the infected chronic human population as the infected blackfly population also increases as shown in Figure 1. This can be explained to be as a result of super infections brought about by blackfly bites which increase the microfilariae densities in the infected acute human population thereby leading to a rapid progression to the infected chronic human population class. With trapping of the disease transmitting blackflies, numerical simulations show a decrease in the infected acute and infected chronic human populations with increase in the trapping rate θ as shown in Figure 2. However, it is observed that there is a slight decrease in the infected acute and infected chronic human populations with increasing trapping rate. This therefore suggests that using traps alone may not be a sufficient strategy in eradicating the disease.

In conclusion, the significance of trapping the blackflies needs to be recognised in the spread and control dynamics of onchocerciasis. However, it should be combined with other strategies to ensure that the disease is reduced and eventually eradicated from the population. In this study, we assumed the use of a microfilaricide as the mode of treatment. However, in future studies, the model can be extended to investigate the effect of using both a micofilaricide and a macrofilaricide as the mode of treatment. The model can also be extended to investigate the effect of using an onchocerciasis vaccine in a bid to eradicate the disease.

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Appendix

Matlab codes used in the numerical simulation of the model equations
For the change in human populations with time shown in Figure 1(a)

```
function FV = funtraped(t,x);
Lambda_H=0.03;
Lambda_V=0.73;
beta_H=0.073;
beta_V=0.80;
mu_H=1/(63.5*365);
phi=1/(105);
rho=1/240;
nu=1/45;
mu_V=1/21;
epsilon=1/69534;
alpha=0.0855;
m=0.25;
FV(1,1)=(1-x(1))*Lambda_H/x(7)-alpha*beta_H*x(1)*x(6)*m+phi*x(4)+epsilon*x(3)*x(1);
FV(2,1)= alpha*beta_H*x(1)*x(6)*m-((Lambda_H/x(7))+nu+rho-epsilon*x(3))*x(2);
FV(3,1)=rho*x(2)-((Lambda_H/x(7))+epsilon-epsilon*x(3))*x(3);
FV(4,1)=nu*x(2)-((Lambda_H/x(7))+phi-epsilon*x(3))*x(4);
FV(5,1)=(1-x(5))*Lambda_V/x(8)-alpha*beta_V*(x(2)+x(3))*x(5);
FV(6,1)=alpha*beta_V*(x(2)+x(3))*x(5)-Lambda_V*x(6)/x(8);
FV(7,1)=((Lambda_H/x(7))-mu_H-epsilon*x(3))*x(7);
FV(8,1)=((Lambda_V/x(8))-mu_V)*x(8);

>> [t,xV] = ode23('funtraped',[0 400],[0.5,0.2,0.1,0.1,0.75,0.25,1000,250]);
>> plot(t,xV(:,1),t,xV(:,2),t,xV(:,3),'Linewidth',2)
>> legend('susceptibles','infected acute','infected chronic')
>> xlabel('Time in days'),ylabel('Human population')
>> title('Human population change against time')
```

For the change in the blackfly populations with time shown in Figure 1(b)

```
function FV = funtraped(t,x);
Lambda_H=0.03;
Lambda_V=0.73;
beta_H=0.073;
beta_V=0.80;
mu_H=1/(63.5*365);
phi=1/(105);
rho=1/240;
nu=1/45;
mu_V=1/21;
epsilon=1/69534;
alpha=0.0855;
m=0.25;
FV(1,1)=(1-x(1))*Lambda_H/x(7)-alpha*beta_H*x(1)*x(6)*m+phi*x(4)+epsilon*x(3)*x(1);
FV(2,1)= alpha*beta_H*x(1)*x(6)*m-((Lambda_H/x(7))+nu+rho-epsilon*x(3))*x(2);
FV(3,1)=rho*x(2)-((Lambda_H/x(7))+epsilon-epsilon*x(3))*x(3);
FV(4,1)=nu*x(2)-((Lambda_H/x(7))+phi-epsilon*x(3))*x(4);
FV(5,1)=(1-x(5))*Lambda_V/x(8)-alpha*beta_V*(x(2)+x(3))*x(5);
FV(6,1)=alpha*beta_V*(x(2)+x(3))*x(5)-Lambda_V*x(6)/x(8);
FV(7,1)=((Lambda_H/x(7))-mu_H-epsilon*x(3))*x(7);
FV(8,1)=((Lambda_V/x(8))-mu_V)*x(8);

>> [t,xV] = ode23('funtraped',[0 400],[0.5,0.2,0.1,0.1,0.75,0.25,1000,250]);
>> plot(t,xV(:,5),t,xV(:,6),'Linewidth',2)
>> legend('susceptible vectors','infectious vectors')
>> xlabel('Time in days'),ylabel('Vector population')
>> title('Vector population change with time')
```

For the change in the infected acute human population with blackfly trapping against time shown in Figure 2(a)

```
function FV = funtraped2(t,x);
Lambda_H=0.03;
Lambda_V=0.73;
beta_H=0.073;
beta_V=0.8;
mu_H=1/(63.5*365);
phi=1/(105);
rho=1/240;
nu=1/45;
epsilon=1/69534;
alpha=0.0855;
m=0.25;
for mu_V = [0.05,0.07,0.09];
FV(1,1)=(1-x(1))*Lambda_H/x(7)-alpha*beta_H*x(1)*x(6)*m+phi*x(4)+epsilon*x(3)*x(1);
FV(2,1)= alpha*beta_H*x(1)*x(6)*m-((Lambda_H/x(7))+nu+rho-epsilon*x(3))*x(2);
FV(3,1)=rho*x(2)-((Lambda_H/x(7))+epsilon-epsilon*x(3))*x(3);
FV(4,1)=nu*x(2)-((Lambda_H/x(7))+phi-epsilon*x(3))*x(4);
FV(5,1)=(1-x(5))*Lambda_V/x(8)-alpha*beta_V*(x(2)+x(3))*x(5);
FV(6,1)=alpha*beta_V*(x(2)+x(3))*x(5)-Lambda_V*x(6)/x(8);
FV(7,1)=((Lambda_H/x(7))-mu_H-epsilon*x(3))*x(7);
FV(8,1)=((Lambda_V/x(8))-mu_V)*x(8);
end

>> [t,xV] = ode23('funtraped2',[0 400],[0.5,0.2,0.1,0.1,0.75,0.25,
1000,250,0.5,0.2,0.1,0.1,0.75,0.25,1000,250,0.5,0.2,0.1,0.1,0.75,0.25,1000,250]);
>> plot(t,xV(:,2),t,xV(:,10),t,xV(:,18),'Linewidth',2)
>> legend('theta=0','theta=0.2','theta=0.4')
>> xlabel('Time in days'),ylabel('Infected acute human population')
>> title('Infected acute human population change with trapping vectors against time')
```

For the change in the infected chronic human population with blackfly trapping against time shown in Figure 2(b)

```
function FV = funtraped2(t,x);
Lambda_H=0.03;
Lambda_V=0.73;
beta_H=0.073;
beta_V=0.8;
mu_H=1/(63.5*365);
phi=1/(105);
rho=1/240;
nu=1/45;
epsilon=1/69534;
alpha=0.0855;
m=0.25;
for mu_V = [0.05,0.07,0.09];
FV(1,1)=(1-x(1))*Lambda_H/x(7)-alpha*beta_H*x(1)*x(6)*m+phi*x(4)+epsilon*x(3)*x(1);
FV(2,1)= alpha*beta_H*x(1)*x(6)*m-((Lambda_H/x(7))+nu+rho-epsilon*x(3))*x(2);
FV(3,1)=rho*x(2)-((Lambda_H/x(7))+epsilon-epsilon*x(3))*x(3);
FV(4,1)=nu*x(2)-((Lambda_H/x(7))+phi-epsilon*x(3))*x(4);
FV(5,1)=(1-x(5))*Lambda_V/x(8)-alpha*beta_V*(x(2)+x(3))*x(5);
FV(6,1)=alpha*beta_V*(x(2)+x(3))*x(5)-Lambda_V*x(6)/x(8);
FV(7,1)=((Lambda_H/x(7))-mu_H-epsilon*x(3))*x(7);
FV(8,1)=((Lambda_V/x(8))-mu_V)*x(8);
end

>> [t,xV] = ode23('funtraped2',[0 400],[0.5,0.2,0.1,0.1,0.75,0.25,
1000,250,0.5,0.2,0.1,0.1,0.75,0.25,1000,250,0.5,0.2,0.1,0.1,0.75,0.25,1000,250]);
>> plot(t,xV(:,3),t,xV(:,11),t,xV(:,19),'Linewidth',2)
>> legend(theta=0','theta=0.2','theta=0.4')
>> xlabel('Time in days'),ylabel('Infected chronic human population')
>> title('Infected chronic human population change with trapping vectors against time')
```