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*Original Article*

# Analytical Investigation of the Impact of Failed Treatments on the Transmission Dynamics of Onchocerciasis

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***Abstract*** *- The drug Ivermectin is considered the medicine of choice in combating onchocerciasis. However, treatment needs to be repeated once annually or biannually within a period of 10-15 years which covers the worm's adult life cycle. Therefore, a model was designed to evaluate the impact of failure to complete treatment on the dynamics of onchocerciasis within the whole number of human inhabitants domiciled in an environment. The backward bifurcation phenomenon, via the model, was induced by human deaths caused by Onchocerciasis and the bifurcation range was also shown to be affected by the proportion of the infected population who complete their treatment. Numerical study of the model reveals that the proportion of affected human individuals who complete their treatment as well as the relative infectiousness of humans who failed to complete their treatment have significant influence on the movement of onchocerciasis in the whole number of human inhabitants domiciled in an environment. In particular, it was seen that an increase in the percentage of individuals who did not complete their structured medical care has significant impact on the backward bifurcation range. It was also shown that while increasing the treatment rate of infectious humans is important, control strategies that would encourage people to stay through the treatment period should also be implemented alongside failure to do this will undermine the gains of improved treatment rates.*

***Keywords*** *- Neglected Tropical Diseases, Onchocerciasis, Failed Treatment, Bifurcation, Quantifying, Transmission dynamics.*

## 1 Introduction

Neglected diseases of tropical origin (NTDs) of the which Human-Blackfly Onchocerciasis is one are a medically dissimilar collection of chronic disabling tropical ailments which are mostly abundant in extremely poverty-stricken populations in emergent countries of certain regions of the Americas, Asia and the African continent, exert influence on more than one billion people the world over [1]. These diseases are precipitated by a diversity of pathogens such as viruses, bacteria, protozoa and helminths and various organizations have classified the set of ailments differently [1 - 3].

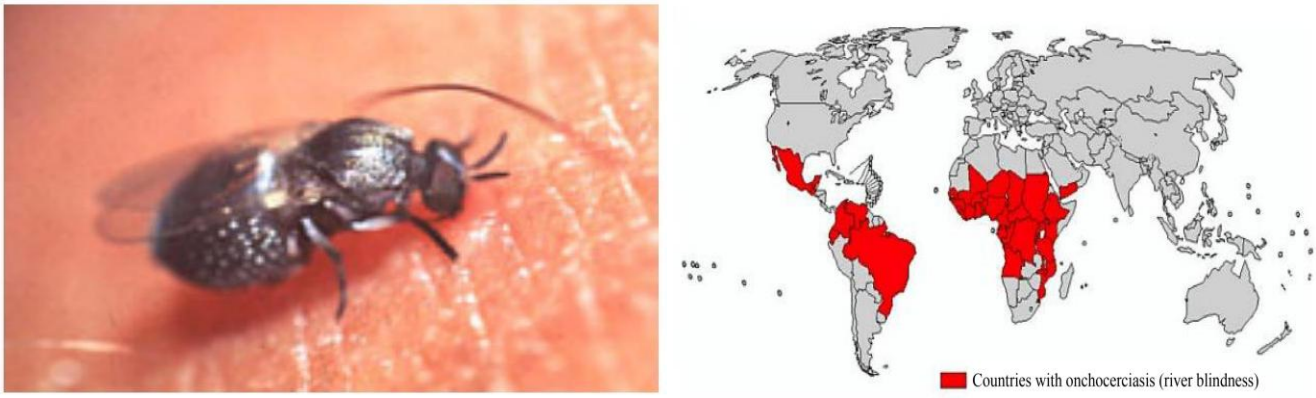
These NTDs affected approximately 1 billion persons globally with an estimated **90**% of the gross ailment burden domiciled in sub-Saharan Africa and these ailments are juxtaposed with the great three ailments (HIV/AIDS, Malaria and Tuberculosis), which usually receive substantial treatment and scientific study support [1 - 4]. However, these NTDs can aggravate HIV/AIDS and Tuberculosis and make them deadlier [5 – 6]].

Seventeen NTDs have been identified and prioritized by World Health Organization (WHO) and these diseases are common in about one hundred and forty-nine developing poor countries of the world, affecting over one billion people (with about half of these being children) and costing emerging economies billions of dollars yearly [2, 7]. These diseases resulted in about one hundred and forty-two thousand deaths in 2013, down from two hundred and four thousand deaths in 1990 [7 – 8].

About thirteen out of the seventeen NTDs identified and prioritized by the WHO are recurrent in region below the Sahara in Africa, Asia with certain neighbourhoods of the Americas. These recurrent ailments comprise of African Trypanosomiasis of Humans, Soil Transmitted Helminthes, Guinea worm disease, Granular conjunctivitis, Lymphatic Flariasis, **Onchocerciasis**, Buruli Ulcer, Leprosy, Schistosomiasis with Dengue Fever [3, 9, 10].

A lot of works has been done in providing clinical solutions to arresting the trend but combating these diseases seems insurmountable. Various Mathematical models have been formulated and analyzed to answer specific questions in order to understand the dynamics of these diseases terrorizing poor tropical nations of the world [4, 8, 11 – 22].

This paper however focuses on Onchocerciasis or river blindness, which is a dangerous nonlethal parasitic disease that leads to blindness, human hardship, and austere socioeconomic mishaps [23]. It is a major precipitate of clinical and epidemiological concern of skin irritation and disease in Africa [24 – 28]. Onchocerciasis is precipitated by a filarial worm (*Onchocerca volvulus*); it is passed on to humans via the bite of tainted black flies *Simulium damnosum* (an adult female black fly) as seen in Figure 1(a) [24 - 28].




**(a) An adult female blackfly** **(b) World demography of Onchocerciasis**

**Fig. 1 Picture of *Simulium damnosum*: an adult female black fly and the world map showing the demography of Onchocerciasis. Source: Climate Policy Watcher, 2016**

Black-flies reproduce in fast moving water bodies and this is due to the aquatic nature of the pre-adult phases of *Simulium damnosum*, which of course is responsible for the popular name of the disease, "River Blindness" [24 – 28]. The disease results in a number of skin infections and serious visual disabilities, which includes lifelong blindness, and a possible reduction of life expectancy by as much as 15 years [24, 27]. Onchocerciasis is a world-wide public health problem [24]. About forty million humans are tainted with Onchocerciasis worldwide and with nearly two million cases of blindness [24,26, 28]. About eighty-six million people in thirty-five countries live in areas of high endemicity [22 – 28]. Figure 1 (b) shows the world demography of Onchocerciasis disease where the highly endemic countries are in red ink. The infection is endemic in about thirty countries in Africa, six countries in the Americas, and in Yemen [22]. Currently, about seven to ten million Nigerians are tainted with *Onchocerca volvulus*, and over 120,000 cases of blindness as a result of Onchocerciasis have been so reported in Nigeria [3] and thousands of humans suffering incapacitating impediments of Onchocerciasis [5]. In the world, the disease is the second known cause of severe visual impairment and blindness [22].

The World Health Organisation (WHO) recommends treating onchocerciasis with ivermectin at least once yearly for 10 to 15 years [3]. Ivermectin (Mectizan) is administered orally on a maximum dose of 12mg every 6 to 12 months till the symptoms of the infection completely dies out [24]. The drug has been shown to reduce the severity of skin symptoms as well as the number of visual impairments [24]. The drug destroys the microfilariae (larvae), but it is inactive against the adult worms [24]. The group-directed medication using ivermectin (CDTI), espoused by the WHO via the African Program for Onchocerciasis Control (APOC), for mass treatments against onchocerciasis has made some significant impact in fulfilling the WHO objective of eradication [60]. But, given the duration of the therapy, certain people or groups might consistently disobey over time, continuing to serve as a focal point for the spread of the disease.

For this reason, in order to help sustain annual treatment, the Consultative Technical Committee of APOC ordered research to determine which variables can be linked to alignment over a period of time. From there, relevant enlightenment and treatment might be devised [29]. The study in [29] showed that when compliance for an eight-year period was checked only 42.9% took ivermectin amounting to $6 - 8$ times annually. Moreover, the study revealed that more than 25% of the age-qualified individuals in the group were minimal compliers, acting as a receptacle for the ongoing spread of onchocerciasis [29]. Therefore, in order to boost compliance, it was advised that CDTI program administrators target population segments with health education [29].

Some researchers have made an effort to examine the mathematical dynamics of onchocerciasis. Omondi *et al.* (2018) constructed a model that mathematically examined both established and non-established mass drug management with the drug Ivermectin. Their results revealed that: (i) disease eradication cannot be achieved without reducing the transmission levels to the barest minimum or engaging in serious and effective vector control, (ii) the disease can be controlled but not completely eradicated with treatment at established intervals and (iii) treatment at non established patterns may result in disease outbreak [17]. Omade *et al*. (2015) modeled the dynamics of Onchocerciasis using an SIR disease modelling pattern with demography in Mubi settlement of Gombe state, Nigeria [30]. Results from their work suggest that within a period of 14 days, $52\%$ of the Mubi residence were at risk of the the disease and about $50\%$ tainted rate was recorded amongst the residents. Recovery rate was reported to be about $37\%$ and that the disease constituted a serious risk to the community.

Oguoma and Mbah (2014) formulated a treatment model for Onchocerciasis for tropical countries. Their simulation results confirmed that the drugs such as Ivermectin and Mectizan should be used continually to prevent reinfections [30]. Poolman and Galvani (2006) modeled the impact of concerted Ivermectin intervention for controlling river blindness. They found that the uniform population treated with ivermectin experienced geography-dependent percentage reductions in the average worm load. [22]. Basanez and Ricardez-Esquinca (1999) formulated a mathematical model to investigate the various interventions combining the removal of adult worms and their microfilaricidal and the sterilizing effect of Ivermectin on human Onchocerciasis [31]. They defined a threshold condition based on the fundamental reproduction number and the rate of vector biting for disease control. They calculated that each person would receive 7665 bites from *Simulium Onchraceum* s.l. yearly. [31]. They showed disease elimination in Central America is possible whenever the reproduction is less than one (i.e. $\mathcal{R}_0 < 1$) and In West Africa, the annual threshold biting frequency for endemic onchocerciasis varies from 288 to 720 bites per person. [4].

These models have conveyed a great deal of understanding about the infection dynamics of Onchocerciasis but they have not had much of an influence on examining the effects of a possible failed treatment on the transmission of Onchocerciasis. Hence, we suggest a mathematical model for Onchocerciasis which will be deployed in the investigation of the effect of failed treatments on the infection transitions of Human Onchocerciasis since the adult worm's life cycle is between 10 to 15 years, repeated Ivermectin treatments must be given over a period of 10 to 15 years. [32]. Considering the long time involved in the treatment of Onchocerciasis, it therefore becomes necessary to investigate the impact of a possible failure to complete treatment on the infection dynamics of the malady as the study in [8] defines compliance as not just the degree to which a patient conforms with dose of and dosing regimen of ivermectin but also the degree to which a patient follows the recommended interval or duration of treatment.

## 2. Model Formulation

We make the following assumptions for developing this model: the population is uniform, well-mixed, and both individuals and black flies carry the same risk of infection, and it is thought that the frequency of interactions between susceptible humans and black flies will determine the number of effective contacts that lead to an infection [33, 34]. The total population of the Human-Blackfly Onchocerciasis model is partitioned into two subpopulations; humans (host) and black flies (vector) which comprises of eight non-overlapping compartments (five compartments for the human subpopulation and three compartments for the black flies subpopulation).

The compartments of the models are: susceptible humans not infected with Onchocerciasis $(S_H)$; latently infected humans exposed to Onchocerciasis through bite from black flies but not infectious $(E_H)$; infected humans $(I_H)$; humans who failed to complete treatment with Ivermectin due to the time involved in completing treatment $(T_F)$; humans who completed treatment with Ivermectin $(T_C)$; susceptible black flies which are not infected with Onchocerciasis (black flies that have not yet acquired microfilariae but could do so if they feed on the blood of an infected person) ( $S_V$ ); black flies that are latently infected which have acquired microfilariae after a blood meal from infected humans but not infectious $(E_V)$ and infected black flies that are infectious and are capable of transmitting Onchocerciasis $(I_V)$. Hence the total population for humans and black flies for all time $t$, are expressed thus

$$\begin{aligned} N_H(t) &= S_H(t) + E_H(t) + I_H(t) + T_F(t) + T_C(t) \text{ and} \\ N_V(t) &= S_V(t) + E_V(t) + I_V(t), \end{aligned} \tag{1}$$

respectively.

Thus, the following deterministic system of nonlinear ODEs describe the model.:

$$
\begin{aligned}
\dot{S}_H &= \Lambda_H - \frac{\beta_H I_V}{N_H} S_H + \sigma T_C - \mu_H S_H, \\
\dot{E}_H &= \frac{\beta_H I_V}{N_H} S_H - (\alpha_H + \mu_H) E_H, \\
\dot{I}_H &= \alpha_H E_H + \gamma T_F - (\tau + \delta + \mu_H) I_H, \\
\dot{T}_F &= (1-p)\tau I_H - (\gamma + \mu_H) T_F, \\
\dot{T}_C &= p\tau I_H - (\sigma + \mu_H) T_C, \\
\dot{S}_V &= \Lambda_V - \frac{\beta_V (I_H + \eta T_F)}{N_H} S_V - \mu_V S_V, \\
\dot{E}_V &= \frac{\beta_V (I_H + \eta T_F)}{N_H} S_V - (\alpha_V + \mu_V) E_V, \\
\dot{I}_V &= \alpha_V E_V - \mu_V I_V,
\end{aligned}
\tag{2}
$$

Figure 2 shows the schematics, that is, a visual depiction of how human individuals circulate between the various system classes. Table 1 and Table 2 present the state variables cum the parameters applied in the mathematical formulation, respectively.

**Table 1. The meaning of the model's state variables**

| Variable | Description |
|---|---|
| $S_H$ | Population of susceptible individuals |
| $E_H$ | Exposed (latent) individuals |
| $I_H$ | Infectious individuals |
| $T_F$ | Individuals with failed (incomplete) treatment |
| $T_C$ | Individuals who completed treatment |
| $S_V$ | Susceptible vectors |
| $E_V$ | Exposed (infected) vectors |
| $I_V$ | Infectious vectors |

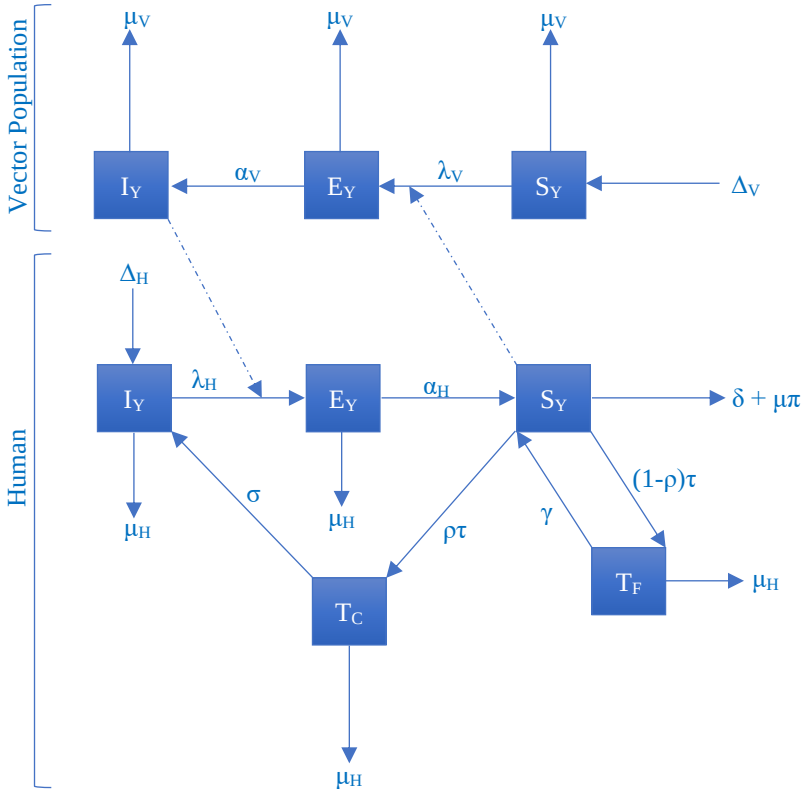



**Fig. 2 Schematics of the infection dynamics of the Human Onchocerciasis model**

**Table 2. Description of parameters of model**

| Parameter | Description |
|---|---|
| $\Lambda_H$ | Human recruitment rate. |
| $\tau$ | treatment rate. |
| $\mu_H$ | Natural death rate for humans. |
| $\alpha_H$ | Progression rate from $E_H$ to $I_H$ |
| $\delta$ | Disease induced death. |
| $\mu_V$ | Natural death rate for vectors. |
| $\alpha_V$ | Progression rate from $E_V$ to $I_V$. |
| $\eta$ | Modification parameter that calibrates the proportionate capacity of persons in |
| | group $T_F$ to engineer fresh infections proportionate to the ones in $I_H(0 \leq \eta \leq 1)$. |
| $\gamma$ | Proportion of treated individuals who completed treatment. |
| $\sigma$ | Rate at which individuals who failed treatment become re-infected. |
| $\Lambda_v$ | Recovery rate. |
| $\beta_H$ | Vector recruitment rate |
| $\beta_V$ | Human infectious rate. |

## 3. Analysis of the Model

The model's qualitative characteristics will be examined in this section.

### *3.1. Basic Properties*

First, the boundedness of the equations in system (2) will be examined. Then the state variables will be shown to be positively bounded for every given time, $t$, since the equation in system (2) describes human and Blackfly populations.

**Theorem 3.1:** Let the first information of the Human-Blackfly Onchocerciasis model in system (2) be shown as $A(0) \geq 0$, where $A(t) = (S_H, E_H, I_H, T_F, T_C, S_V, E_V, I_V)$. Then the trajectories $A(t)$ of the Human-Blackfly Onchocerciasis model (2) with positive first information remains non-negative for every given time $t \geq 0$. Furthermore,

$$\lim_{t\to\infty}\sup N_H(t) = \frac{\Lambda_H}{\mu_H} \text{ and } \lim_{t\to\infty}\sup N_V(t) = \frac{\Lambda_V}{\mu_V} \tag{3}$$

where:

$$N_H(t) = S_H(t) + E_H(t) + I_H(t) + T_F(t) + T_C(t) \tag{4}$$

And

$$N_V(t) = S_V(t) + E_V(t) + I_V(t). \tag{5}$$

Proof:

Let $t_1 = \sup\{t > 0: A(t) > 0 \in [0, t]\}$. Thus for $t > 0$, it follows from the first equation of the system (2) that

$$\dot{S}_H = \Lambda_H - \frac{\beta_H I_V}{N} S_H + \sigma T_C - \mu_H S_H \tag{6}$$

which can be rewritten as

$$\left[\frac{d}{dt} + \left(\frac{\beta_H I_V}{N} + \mu_H\right)\right] S_H(t) \geq \Lambda_H \tag{7}$$

Which follows that

$\frac{d}{dt}\left[S_H(t)\exp\left(\int_0^{t_1} \frac{\beta_H I_V(\varphi)}{N(\varphi)} d\varphi + \mu_H t\right)\right] \geq \Lambda_H \exp\left(\int_0^{t_1} \frac{\beta_H I_V(\varphi)}{N(\varphi)} d\varphi + \mu_H t\right)$.

Thus,

$S_H(t_1)\exp\left(\int_0^{t_1} \frac{\beta_H I_V(\varphi)}{N(\varphi)} d\varphi + \mu_H t\right) - S_H(0) \geq \Lambda_H \int_0^{t_1} \exp\left(\int_0^{p} \frac{\beta_H I_V(\varphi)}{N(\varphi)} d\varphi + \mu_H t\right) dp$

So that,

$$S_H(t_1) \geq S_H(0)\exp\left[-\left(\int_0^{t_1}\frac{\beta_H I_V(\varphi)}{N(\varphi)}d\varphi + \mu_H t\right)\right] + \exp\left[-\left(\int_0^{t_1}\frac{\beta_H I_V(\varphi)}{N(\varphi)}d\varphi + \mu_H t\right)\right] \times \int_0^{t_1}\Lambda_H \exp\left(\int_0^{p}\frac{\beta_H I_V(\varphi)}{N(\varphi)}d\varphi + \mu_H p\right)dp \quad (8)$$

Hence $S_H(t) \geq 0, \forall t \geq 0$ since $S_H(t)$ is the sum of positive terms.
Looking at the second equation in system (2)

$$\frac{dE_H}{dt} = \frac{\beta_H I_H}{N}S_H - (\alpha_H + \mu_H)E_H \quad (9)$$

It follows from (9) that

$$\frac{dE_H}{dt} \geq -(\alpha_H + \mu_H)E_H \quad (10)$$

Integrating (10) as a function of $t$ in $[0, t_1]$, yields

$$E_H(t_1) \geq E_H(0)\exp\{-(\alpha_H + \mu_H)t_1\} > 0 \quad (11)$$

Therefore $E_H(t) > 0$ for every $t > 0$.
Looking at the third equation of system (2),

$$\frac{I_H}{dt} = \alpha_H E_H + \gamma T_F - (\tau + \delta + \mu_H)I_H, \quad (12)$$

It follows from (12) that

$$\frac{I_H}{dt} \geq -(\tau + \delta + \mu_H)I_H \quad (13)$$

Integrating (13) as a function of $t$ in $[0, t_1]$, yields

$$I_H(t_1) \geq I_H(0)\exp\{-(\tau + \delta + \mu_H)t_1\} > 0 \quad (14)$$

It follows that $I_H(t) > 0$ for all $t > 0$.
Looking at the fourth equation of system (2),

$$\frac{T_F}{dt} = (1-p)\tau I_H - (\gamma + \mu_H)T_F \quad (15)$$

It follows from (15) that

$$\frac{T_F}{dt} \geq -(\gamma + \mu_H)T_F \quad (16)$$

Integrating (16) as a function of $t$ in $[0, t_1]$, gives

$$T_F(t_1) \geq T_F(0)\exp\{-(\gamma + \mu_H)t_1\} > 0 \quad (17)$$

Therefore $T_F(t) > 0$ for all $t > 0$.
Looking at the fifth equation of system (2),

$$\frac{T_C}{dt} = p\tau I_H - (\sigma + \mu_H)T_C \quad (18)$$

It follows from (18) that

$$\frac{T_C}{dt} \geq -(\sigma + \mu_H)T_C \quad (19)$$

Integrating (19) as function of $t$ in $[0, t_1]$, yields

$$T_C(t_1) \geq T_C(0)\exp\{-(\sigma + \mu_H)t_1\} > 0 \quad (20)$$

Therefore $T_C(t) > 0$ for all $t > 0$.
And again, looking at the sixth equation of system (2)

$$\dot{S}_V = \Lambda_V - \frac{\beta_V(I_H + \eta T_F)}{N}S_V - \mu_V S_V, \quad (21)$$

where $\lambda_V = \dfrac{\beta_V(I_H + \eta T_F)}{N}$
which can be written as

$$\left[\frac{d}{dt} + \beta_V\frac{(I_V + \eta T_F}{N_V} + \mu_V\right]S_V(t) = \Lambda_V \quad (22)$$

Which follows that

$$\frac{d}{dt}\left[S_V(t)\exp\left(\int_0^{t_1}\lambda_V(\phi)d\phi + \mu_V t\right)\right] = \Lambda_V \exp\left(\int_0^{t_1}\lambda_V(\phi) + \mu_V t\right). \quad (23)$$

Thus,

$$S_V(t_1)\exp\left(\int_0^{t_1}\lambda_V(\phi)d\phi+\mu_V t\right)-S_V(0)=\Lambda_V\int_0^{t_1}\exp\left(\int_0^{p}\lambda_V(\phi)d\phi+\mu_V t\right)dp, \tag{24}$$

So that,

$$S_V(t_1)=S_V(0)\exp\left[-\left(\int_0^{t_1}\lambda_V(\phi)d\phi+\mu_V t\right)\right]+\exp\left[-\left(\int_0^{t_1}\lambda_V(\phi)d\phi+\mu_V t\right)\right]\times\int_0^{t_1}\Lambda_V\exp\left(\int_0^{p}\lambda_V(\phi)d\phi+\mu_V p\right)dp \tag{25}$$

Therefore $S_V(t_1)\geq 0$, for every $t\geq 0$.
Looking at the seventh equation of system (2),

$$\frac{E_V}{dt}=\frac{\beta_V(I_H+\eta T_F)}{N}S_V-(\alpha_V+\mu_V)E_V \tag{26}$$

It follows from (26) that

$$\frac{E_V}{dt}\geq-(\alpha_V+\mu_V)E_V \tag{27}$$

Integrating (27) as function of $t$ in $[0,t_1]$, yields

$$E_V(t_1)\geq E_V(0)\exp\{-(\alpha_V+\mu_V)t_1\}>0 \tag{28}$$

Hence $E_V(t)>0$ for all $t>0$.
Lastly, looking at the eighth equation of system (2),

$$\frac{I_V}{dt}=\alpha_V E_V-\mu_V I_V \tag{29}$$

It follows from (29) that

$$\frac{I_V}{dt}\geq-\mu_V I_V \tag{30}$$

Integrating (30) as function of $t$ in $[0,t_1]$, yields

$$I_V(t_1)\geq I_V(0)\exp\{-\mu_V t_1\}>0 \tag{31}$$

Therefore $I_V(t)>0$ for all $t>0$.

From the above, we have shown that for the Human-Blackfly Onchocerciasis model, $A(t)\geq 0$, where: $A(t)=(S_H,E_H,I_H,T_F,T_C,S_V,E_V,I_V)$. Hence the orbits A(t) engendered by the Human-Blackfly Onchocerciasis model in (2) with positive first starting points will forever remain nonnegative for every time $t>0$.

Subsequently, we have to show that every human and black-fly subpopulation is bounded (since we cannot combine all subpopulations into a single invariant set). We must also establish the bound for each subpopulation. Lastly, we must demonstrate that all the sets of these subclasses are unchanging positively and attract every positive orbit (there is a distinct orbit to the first value problem that subsists for every time) of system (2).

**Theorem 3.2:** Allow $(S_H,E_H,I_H,T_F,T_C,S_V,E_V,I_V)$ be the orbits system (2) with initial conditions given in Theorem 3.1 as well as the biologically viable area defined in the set $\mathcal{D}=\mathcal{D}_H\times\mathcal{D}_V\subset\mathbb{R}_+^5\times\mathbb{R}_+^3\subset\mathbb{R}_+^8$ where:

$$\begin{aligned}\mathcal{D}_H&=\left\{(S_H,E_H,I_H,T_F,T_C)\in\mathbb{R}_+^5: N_H\leq\frac{\Lambda_H}{\mu_H}\right\}\\ \mathcal{D}_V&=\left\{(S_V,E_V,I_V)\in\mathbb{R}_+^3: N_V\leq\frac{\Lambda_V}{\mu_V}\right\}\end{aligned} \tag{32}$$

is positively invariant and draws all the model's positive orbit.

**Proof:** The bound for the human subpopulation is determined by calculating the rate at which the entire human population changes as defined by the system, which is the sum of vector field's right-hand side of the total human community in system (2) represented by:

$$\frac{dN_H(t)}{dt}=\Lambda_H-\mu_H N_H(t)-\delta I_H(t) \tag{33}$$

From (33), it follows that

$$\frac{dN_H(t)}{dt}\leq\Lambda_H-\mu_H N_H(t) \tag{34}$$

From equation (34), therefore, we have

$$\frac{dN_H}{dt}e^{\mu_H t} + \mu_H N_H e^{\mu_H t} \leq \Lambda_H e^{\mu_H t} \tag{35}$$

Equation (35) can be rewritten as

$$\int_0^t \frac{dN_H}{d\tau}e^{\mu_H \tau}d\tau \leq \Lambda_H \int_0^t e^{\mu_H \tau}d\tau \tag{36}$$

Integrating (36) with the initial condition $N_H(t) = N_H(0)$, we have

$$N_H(t)e^{\mu_H t} - N_H(0) \leq \frac{\Lambda_H}{\mu_H}(e^{\mu_H t} - 1). \tag{37}$$

Solving for $N_H(t)$ from (37), gives

$$N_H(t) \leq N_H(0)e^{-\mu_H t} + \frac{\Lambda_H}{\mu_H}(1 - e^{-\mu_H t}) \tag{38}$$

It implies that $N_h(t) \leq \frac{\Lambda_H}{\mu_H}$ if $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$. Thus under the orbits of the system, the domain $\mathcal{D}_H$ is unchanging positively. Furthermore, if $N_H(0) > \frac{\Lambda_H}{\mu_H}$, then $N_H(t)$ asymptotically approaches $\frac{\Lambda_H}{\mu_H}$ as $t \to \infty$ or the trajectories go into the set $\mathcal{D}_H$ in finite time. As a result, every trajectory is drawn to the domain $\mathcal{D}_H$, and no trajectory leaves any $\mathcal{D}_H$ border in $\mathbb{R}_+^5$. It follows that $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$ if $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$.Thus, under the system's orbits, the domain $\mathcal{D}_H$ is unchanging positively. Furthermore, if $N_H(0) > \frac{\Lambda_H}{\mu_H}$., then either $N_H(t)$ asymptotically approaches $\frac{\Lambda_H}{\mu_H}$ as t→∞ or the orbits enter the domain $\mathcal{D}_H$in finite time. As a result, every trajectory is drawn to the domain $\mathcal{D}_H$, and no trajectory ever leaves its boundaries.

The Blackfly population bound can be obtained by summing the vector field's right-hand side of the Blackfly community in system (2). Which becomes

$$\frac{dN_V(t)}{dt} \leq \Lambda_V - \mu_V N_V(t) \tag{39}$$

From equation (39), thus, we have

$$\frac{dN_V}{dt}e^{\mu_V t} + \mu_V N_V e^{\mu_V t} \leq \Lambda_V e^{\mu_V t} \tag{40}$$

Equation (40) can be rewritten as

$$\int_0^t \frac{dN_V}{d\tau}e^{\mu_V \tau}d\tau \leq \Lambda_V \int_0^t e^{\mu_V \tau}d\tau \tag{41}$$

Integrating (41) while employing the initial starting point $N_V(t) = N_V(0)$, we have

$$N_V(t)e^{\mu_V t} - N_V(0) \leq \frac{\Lambda_V}{\mu_V}(e^{\mu_V t} - 1) \tag{42}$$

Solving for $N_V(t)$ from (42), gives

$$N_V(t) \leq N_V(0)e^{-\mu_V t} + \frac{\Lambda_V}{\mu_V}(1 - e^{-\mu_V t}) \tag{43}$$

It follows that $N_V(t) \leq \frac{\Lambda_V}{\mu_V}$ if $N_V(0) \leq \frac{\Lambda_V}{\mu_V}$. Therefore, under the system's flow, the domain $\mathcal{D}_V$is positively invariant. Furthermore, given the condition that $N_V(0) > \frac{\Lambda_V}{\mu_V}$, then either $N_V(t)$ asymptotically approaches $\frac{\Lambda_V}{\mu_V}$ as $t \to \infty$, or the orbits enter the domain $\mathcal{D}_V$in finite time. As a result, every trajectory is drawn to the domain $\mathcal{D}_V$, and no trajectory leaves any $\mathcal{D}_V$boundary in $\mathbb{R}_+^3$.

Since $\mathcal{D} = \mathcal{D}_H \times \mathcal{D}_V$,, it follows that set $\mathcal{D}$ is also positively-unchanging and an attractor, meaning that none of the orbits exits through any boundary of $\mathcal{D}$. Our demonstration that $\mathcal{D}_H$ and $\mathcal{D}_V$ are invariantly positive is complete.

$$\mathcal{D} = \begin{cases} (S_H, E_H, I_H, T_F, T_C) \in \mathbb{R}_+^5 : N_H \leq \dfrac{\Lambda_H}{\mu_H} \\ (S_V, E_V, I_V) \in \mathbb{R}_+^3 : N_V \leq \dfrac{\Lambda_V}{\mu_V} \end{cases} \tag{44}$$

The right-hand side of system (2) must consequently be smooth in order for the initial data problem to have a distinct solution that lasts forever. Thus, when viewed from an epidemiological and mathematical perspective, the system is well stated, and it is enough to examine the movement of the trajectories that the system in $\mathcal{D}$ generates.

### *3.2. Local Asymptotic Stability of Disease-Free Equilibrium*

By setting the diseased classes (that is, state variables of the individuals with infections) and the right-hand side of the system's equations to zero and solving the resulting system, the Disease-Free Equilibrium (DFE) of the system can be determined. The model's DFE is given by

$$\mathcal{E}_0 = (S_H^0, E_H^0, I_H^0, T_F^0, T_C^0, S_V^0, E_V^0, I_V^0) = \left(\frac{\Lambda_H}{\mu_H}, 0,0,0,0, \frac{\Lambda_V}{\mu_V}, 0,0\right) \tag{45}$$

The next generation matrix operator approach is used to evaluate the Local Asymptotic Stability (LAS) of the DFE [34]. The matrices F and V representing the new infection terms and the existing transfer terms, respectively, are provided by using notations similar to those in [34]

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \beta_H \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_V \Lambda_V \mu_V}{\Lambda_H \mu_H} & \frac{\eta \beta_V \Lambda_V \mu_V}{\Lambda_H \mu_H} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} p_1 & 0 & 0 & 0 & 0 \\ -\alpha_H & p_2 & -\gamma & 0 & 0 \\ 0 & -(1-p)\tau & p_3 & 0 & 0 \\ 0 & 0 & 0 & p_4 & 0 \\ 0 & 0 & 0 & -\alpha_V & \mu_V \end{pmatrix}$$

The reproduction number $\mathcal{R}_0 = \rho(FV^{-1})$, with $\rho$ being the spectral radius of $FV^{-1}$ is given by

$$\mathcal{R}_0 = \sqrt{\frac{\beta_H \alpha_H \mu_H ((1-p)\eta\tau + p_3)}{p_1(p_2 p_3 - (1-p)\gamma\tau)\Lambda_H} \cdot \frac{\beta_V \alpha_V \Lambda_V}{p_5 \mu_V^2}} = \sqrt{\mathcal{R}_{0H} \cdot \mathcal{R}_{0V}} \tag{46}$$

where

$$\mathcal{R}_{0H} = \frac{\beta_H \alpha_H \mu_H ((1-p)\eta\tau + p_3)}{p_1(p_2 p_3 - (1-p)\gamma\tau)\Lambda_H},\ \mathcal{R}_{0V} = \frac{\beta_V \alpha_V \Lambda_V}{p_5 \mu_V^2} \tag{47}$$

$$p_1 = \alpha_H + \mu_H,\ p_2 = \tau + \delta + \mu_H,\ p_3 = \gamma + \mu_H,\ p_4 = \sigma + \mu_H,$$
$$p_5 = \alpha_V + \mu_V$$

Using Theorem 2 in [35] we claim the following result:

**Lemma 3.1:** The DFE of system (2) is LAS in $\mathcal{D}$ if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.
Epidemiologically, **Lemma 3.1** implies Onchocerciasis eradication from the population when $\mathcal{R}_0 < 1$, if the first magnitudes of the subclasses of the population of system (2) dwell in the basin of attraction of the DFE. Also, the implication of this is that given the condition that a minute number of infectious cases come into such population, their presence will not result in a large epidemic outbreak in the population even though a fraction of infected individual fails treatment.

#### *3.2.1. Analysis of the Reproduction Number*

The sensitiveness of threshold quantity, $\mathcal{R}_0$ to specific key parameters that describes the impact of failure to complete the treatment regimen by infected humans is analyzed. The parameter values used for creating this plot are derived from Table 3. Figure 3 shows that for onchocerciasis to be controlled, over $67\%$ of the infected persons will need to complete their treatment even if the infected persons who failed to complete their treatment is $50\%$ as infectious as the infected persons who were nevertreated.

Table 3 shows the impact of all parameters of system (2) on the effective reproduction number $\mathcal{R}_0$ by calculating the elasticity indices of $\mathcal{R}_0$ to the model parameters with values given in Table 3. The effective reproduction number $\mathcal{R}_0$ shows the highest sensitivity to the natural vector mortality. In addition, the basic reproduction number is highly sensitive to the proportion of humans who complete their treatment. We see that an increase of $1\%$ in the proportion of humans who complete their treatment will lead to about $0.9\%$ reduction in the reproduction number. It is also important to state that the relative infectiousness of humans who do not complete their treatment has significant impact on the basic reproduction number. These results show that failure to complete treatment by infected humans has significant impact on the control and possible eradication of onchocerciasis in a population.

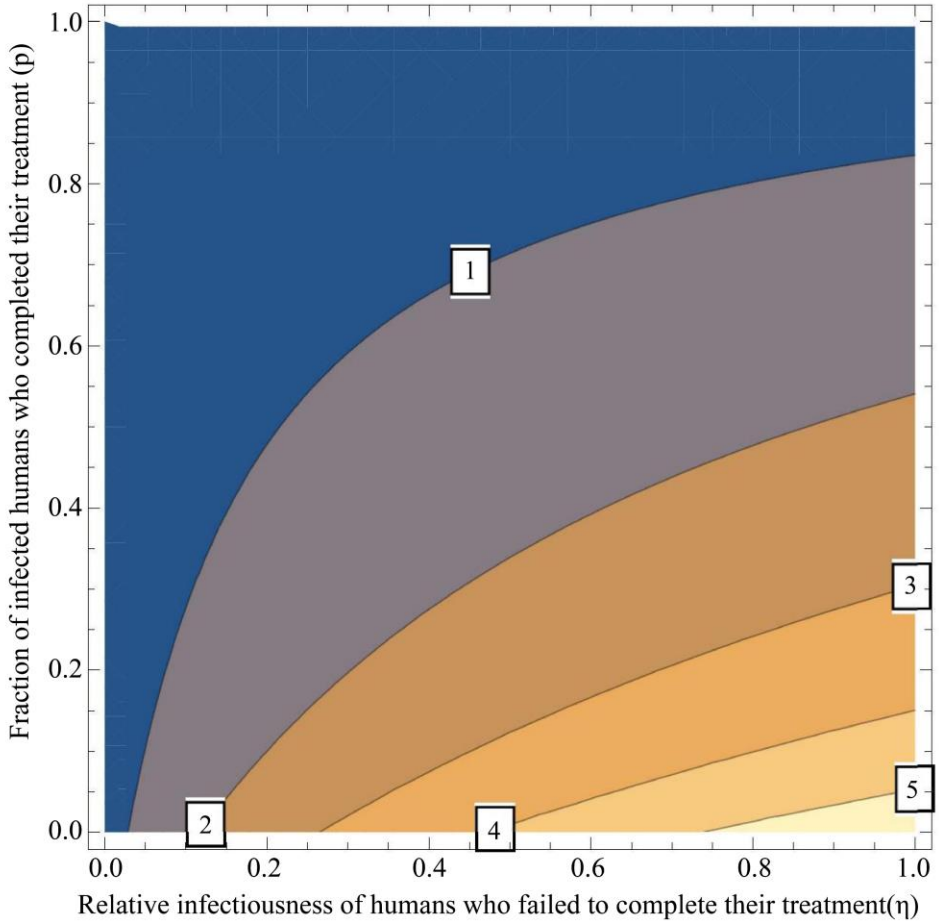



**Fig. 3 Contour plot of $\mathcal{R}_0$ as a function of the relative infectiousness of humans who failed to complete their treatment ($\eta$) and the proportion of humans who completed treatment ($p$)**

**Table 3. Elasticity indices of parameter in the basic reproduction number $\mathcal{R}_0$.**

| Parameter | Elasticity index |
|---|---|
| $\mu_V$ | -1.23288 |
| $p$ | -0.92578 |
| $\eta$ | 0.5 |
| $\Lambda_H$ | -0.5 |
| $\Lambda_V$ | 0.5 |
| $\beta_H$ | 0.5 |
| $\beta_V$ | 0.5 |
| $\gamma$ | -0.393655 |
| $\mu_H$ | 0.331853 |
| $\alpha_V$ | 0.2332877 |
| $\tau$ | 0.0424375 |
| $\alpha_H$ | 0.0193634 |

### *3.3. Presence of Endemic Equilibrium Point (EEP) for model (2)*

The EEP is the critical point for which the malady persists in the community. Let

$$\mathcal{E}_1 = (S_H^{**}, E_H^{**}, I_H^{**}, T_F^{**}, T_C^{**}, S_V^{**}, E_V^{**}, I_V^{**}) \tag{48}$$

be the EEP for system (2), gotten by resolving the right-flank of the equations in system (2) as a function of the forces of infection at the EEP where $\lambda_H^{**} = \frac{\beta_H I_V^{**}}{N_H^{**}}$, and $\lambda_V^{**} = \frac{\beta_V (I_H^{**} + \eta T_F^{**})}{N_V^{**}}$.

$$
\begin{aligned}
S_H^{**} &= \frac{p_1 p_4 G_1 \Lambda_H}{\lambda_H^{**} G_2 + p_1 p_4 \mu_H G_1},\ E_H^{**} = \frac{p_4 G_1 \lambda_H^{**} \Lambda_H^{**}}{G_2 \lambda_H^{**} + p_1 p_4 \mu_H G_1} \\
I_H^{**} &= \frac{\alpha_H p_3 p_4 \lambda_H^{**} \Lambda_H^{**}}{G_2 \lambda_H^{**} + p_1 p_4 \mu_H G_1},\ T_F^{**} = \frac{(1-p)\tau \alpha_H p_4 \lambda_H^{**} \Lambda_H^{**}}{G_2 \lambda_H^{**} + p_1 p_4 \mu_H G_1}, \\
T_C^{**} &= \frac{p\tau \alpha_H p_3 \lambda_H^{**} \Lambda_H^{**}}{G_2 \lambda_H^{**} + p_1 p_4 \mu_H G_1},\ S_V^{**} = \frac{\Lambda_V}{\lambda_V^{**} + \mu_V} \\
E_V^{**} &= \frac{\lambda_V^{**} \Lambda_V}{p_5(\lambda_V^{**} + \mu_V)},\ I_V^{**} = \frac{\alpha_V \lambda_V^{**} \Lambda_V}{p_5 \mu_V (\lambda_V^{**} + \mu_V)}
\end{aligned} \tag{49}
$$

where $G_1 = p_2 p_3 - \gamma\tau(1-p) > 0,\ G_2 = p_1 p_4 G_1 - \sigma p \tau \alpha_H p_3 > 0.$

After performing numerous algebraic modifications, it becomes obvious that the EEP of system (2) satisfies the given polynomial at the equilibrium point when the equations for the EEP in (49) are substituted into the force of infection.:

$$A_2 \lambda_H^{**2} + A_1 \lambda_H^{**} + A_0 = 0 \tag{50}$$

where

$$
\begin{aligned}
A_2 =& p_5 \Lambda_H \mu_V \big(G_1 p_4 \mu_V + p_4(1-q)\alpha_H(\beta_V \eta + \mu_V)\tau + p_3 \alpha_H (p_4(\beta_V + \mu_V) + p\mu_V \tau)\big) \\
& \Big(G_1 p_4 + \alpha_H \big(p_4(1-p)\tau + p_3(p_4 + p\tau)\big)\Big) \\
A_1 =& 2 G_1 p_1 p_4 p_5 \Lambda_H \mu_V^2 \Big(G_1 p_4 + \alpha_H \big(p_4(1-p)\tau + p_3(p_4 + p\tau)\big)\Big) \\
& + G_1 p_1 p_4^2 p_5 \beta_V \Lambda_H \mu_V \alpha_H (p_3 + (1-p)\eta\tau) - G_2 p_4 \alpha_H \alpha_V \beta_H \beta_V \Lambda_V (p_3 + (1-p)\eta\tau), \\
A_0 =& \Lambda_H \mu_V^2 p_1^2 p_4^2 p_5 G_1^2 (1 - \mathcal{R}_0^2).
\end{aligned} \tag{51}
$$

The components of the EEP are then obtained by solving for $\lambda_H{}^{**}$ from the polynomial (50), and substituting the positive values of $\lambda_H{}^{**}$ into the expressions in (49).

Moreover, the number of positive roots of the polynomial (50) depends on the sign change between the coefficients $A_1$ and $A_0$.

The above results can be summed in the theorem below.

**Theorem 3.3: The model (2) has**

1. two endemic equilibria if $A_1 < 0$ and $\mathcal{R}_0 < 1$,
2. unique endemic equilibria if $A_1 > 0$ and $\mathcal{R}_0 > 1$,
3. no endemic equilibrium otherwise, when $\mathcal{R}_0 < 1$.

It is significant to note that the first item in **Theorem 3.3** suggests the possible presence of a backward bifurcation in the system (2). The backward bifurcation phenomenon is marked by the parallel-existence of a disease-free state cum an endemic equilibrium that are both stable whenever the corresponding effective reproduction number is below unity. The implication of this is that the standard requirement for disease control (i.e., $\mathcal{R}_0 < 1$ ) is no longer sufficient for effective disease control in a population, although it is a necessary requirement. In this case, fruitful strategies for disease regulation will be consequent on the first conditions of divers classes of the system under examination [36]. In a population (where individuals failed to complete the treatment regimen because of the long time required), the fundamental requirement of having the effective reproduction number below unity, being necessary, is no longer sufficient for effective Onchocerciasis regulation.

### *3.4. Backward Bifurcation Analysis*

The existence of backward bifurcation in the model (2) is examined via the Center Manifold Theory [37 - 39]. We claim the following results.

**Theorem 3.4:** The model (2) exhibits backward bifurcation at $\mathcal{R}_0 = 1$ given that a bifurcation coefficient, described by a $> 0$.
**Proof:** Let

$$\mathcal{E}_a = (S_H^{**}, E_H^{**}, I_H^{**}, T_F^{**}, T_C^{**}, S_V^{**}, E_V^{**}, I_V^{**}, T^{**}) \tag{52}$$

represent an arbitrary EEP of the model (2). It is easy to carry out the ensuing switch of variables.

Let $S_H = x_1, E_H = x_2, I_H = x_3, T_F = x_4, T_C = x_5, S_V = x_6, E_V = x_7$, and $I_V = x_8$. It follows, that system (2) can be re-written as

$$\begin{aligned}
\dot{x}_1 &\equiv f_1 = \Lambda_H - \lambda_H x_1 + \sigma x_5 - \mu_H x_1,\\
\dot{x}_2 &\equiv f_2 = \lambda_H x_1 - (\alpha_H + \mu_H)x_2,\\
\dot{x}_3 &\equiv f_3 = \alpha_H x_2 + \gamma x_4 - (\tau + \delta + \mu_H)x_3,\\
\dot{x}_4 &\equiv f_4 = (1-p)\tau x_3 - (\gamma + \mu_H)x_4,\\
\dot{x}_5 &\equiv f_5 = p\tau x_3 - (\sigma + \mu_H)x_5,\\
\dot{x}_6 &\equiv f_6 = \Lambda_V - \lambda_V x_6 - \mu_V x_6,\\
\dot{x}_7 &\equiv f_7 = \lambda_V x_6 - (\alpha_V + \mu_V)x_7,\\
\dot{x}_8 &\equiv f_8 = \alpha_V x_7 - \mu_V x_8,
\end{aligned} \tag{53}$$

where

$$\lambda_H = \frac{\beta_H x_8}{\sum_{i=1}^{5} x_i} \text{ and } \lambda_V = \frac{\beta_V (x_3 + \eta x_4)}{\sum_{i=1}^{5} x_i} \tag{54}$$

are the forces of infection corresponding to human and blackfly populations respectively. Suppose $\beta_H = \beta_H^*$ is chosen as a bifurcation parameter for the system (53). Solving for $\beta_H = \beta_H^*$ from $\mathcal{R}_0 = 1$ yields

$$\beta_H = \beta_H^* = \frac{p_1 p_5 \Lambda_H \mu_V^2 G_1}{\alpha_H \alpha_V \Lambda_V \mu_H \beta_V (p_3 + (1-p)\eta\tau)} \tag{55}$$

where $G_1 = p_2 p_3 - (1-p)\gamma\tau$. The Jacobian of the transformed system (53) at the DFE with $\beta_H = \beta_H^*$, is given by:

$$J_{\beta_H^*} = J(\mathcal{E}_0)|_{\beta_H = \beta_H^*} = \begin{pmatrix}
-\mu_H & 0 & 0 & 0 & \sigma & 0 & 0 & -\beta_H^* \\
0 & -p_1 & 0 & 0 & 0 & 0 & 0 & \beta_H^* \\
0 & \alpha_H & -p_2 & \gamma & 0 & 0 & 0 & 0 \\
0 & 0 & (1-p)\tau & -p_3 & 0 & 0 & 0 & 0 \\
0 & 0 & p\tau & 0 & -p_4 & 0 & 0 & 0 \\
0 & 0 & -\frac{\beta_V x_6^*}{x_1^*} & -\frac{\beta_V x_6^* \eta}{x_1^*} & 0 & 0 & -p_5 & 0 \\
0 & 0 & \frac{\beta_V x_6^*}{x_1^*} & \frac{\beta_V x_1^* \eta}{x_1^*} & 0 & 0 & -p_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \alpha_V & \mu_V
\end{pmatrix} \tag{56}.$$

The matrix $J_{\beta_H^*}$ has a simple zero eigenvalue (i.e., a center) and all other eigenvalues possess negative real parts. Consequently, the Center Manifold Theorem can be applied. For the case $\mathcal{R}_0 = 1$, it can be shown that the Jacobian $\left(J_{\beta_H}\right)$ of the system (53) at $\beta_H = \beta_H^*$ possesses a right eigenvector given by $\mathrm{w} = [w_1, w_2, \dots, w_8]^T$, where

$$\begin{aligned}
&w_1 = \frac{(\sigma p\tau p_3 \alpha_H - p_1 p_4 G_1)}{p_3 p_4 \mu_H \alpha_H} w_3,\ w_2 = \frac{G_1}{p_3 \alpha_H} w_3,\ w_3 = w_3 > 0,\\
&w_4 = \frac{(1-p)\tau}{p_3} w_3,\ w_5 = \frac{p\tau}{p_4} w_3,\ w_6 = -\frac{\beta_V \Lambda_V \mu_H (p_3(1-p)\eta\tau)}{\Lambda_H p_3 \mu_V^2} w_3,\\
&w_7 = \frac{\beta_V \Lambda_V \mu_H (p_3(1-p)\eta\tau)}{\Lambda_H p_3 p_5 \mu_V} w_3,\ w_8 = \frac{\beta_V \alpha_V \Lambda_V \mu_H (p_3(1-p)\eta\tau)}{\Lambda_H \mu_V p_3 p_5} w_3.
\end{aligned} \tag{57}$$

Similarly, $J_{\beta_H^*}$ has a left eigenvector $\mathbf{v} = (v_1, v_2, \dots, v_8)$, satisfying $\mathbf{v} \cdot \mathbf{w} = 1$ , where

$$\begin{aligned}
&v_1 = 0,\ v_2 = \frac{\alpha_H}{p_1} v_3,\ v_3 = v_3 > 0,\ v_4 = \frac{(\gamma x_1^* p_1 p_5 \mu_V + \beta_H^* \beta_V \alpha_H \alpha_V \eta x_6^*)}{x_1^* \mu_V p_1 p_3 p_5} v_3,\\
&v_5 = 0,\ v_6 = 0,\ v_7 = \frac{\beta_H^* \alpha_H \alpha_V}{p_1 p_5 \mu_V} v_3\ v_8 = \frac{\beta_H^* \alpha_H}{p_1 \mu_V} v_3
\end{aligned} \tag{58}.$$

We also calculate the related non-zero partial derivatives of the right flanks of the transformed system (53), (evaluated at the DFE with $\beta_H = \beta_H^*$)

$$\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_2\,\partial x_8}=\frac{\partial^2 f_2}{\partial x_3\,\partial x_8}=\frac{\partial^2 f_2}{\partial x_4\,\partial x_8}=\frac{\partial^2 f_2}{\partial x_5\,\partial x_8}=-\frac{\beta_H^*}{x_1^*},\\
&\frac{\partial^2 f_7}{\partial x_1\,\partial x_3}=\frac{\partial^2 f_7}{\partial x_2\,\partial x_3}=\frac{\partial^2 f_7}{\partial x_3\,\partial x_4}=\frac{\partial^2 f_7}{\partial x_3\,\partial x_5}=-\frac{\beta_V x_6^*}{x_1^{*2}},\\
&\frac{\partial^2 f_7}{\partial x_1\,\partial x_4}=\frac{\partial^2 f_7}{\partial x_2\,\partial x_4}=\frac{\partial^2 f_7}{\partial x_3\,\partial x_4}=\frac{\partial^2 f_7}{\partial x_4\,\partial x_5}=-\frac{\beta_V x_6^*\eta}{x_1^{*2}},\\
&\frac{\partial^2 f_7}{\partial x_3\,\partial x_3}=-\frac{2\beta_V x_6^*}{x_1^{*2}},\ \frac{\partial^2 f_7}{\partial x_3\,\partial x_6}=\frac{\beta_V}{x_1^*},\\
&\frac{\partial^2 f_7}{\partial x_4\,\partial x_4}=-\frac{2\beta_V x_6^*\eta}{x_1^{*2}},\ \frac{\partial^2 f_7}{\partial x_4\,\partial x_6}=\frac{\beta_V\eta}{x_1^*}\ \frac{\partial^2 f_2}{\partial x_8\beta_H^*}=1.
\end{aligned}\tag{59}$$

The bifurcation coefficients, $a$ and $b$ given below as

$$a=\sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i\,\partial x_j}(0,0),\ \text{and}\ b=\sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i\,\partial \beta_H^*}(0,0)\tag{60}$$

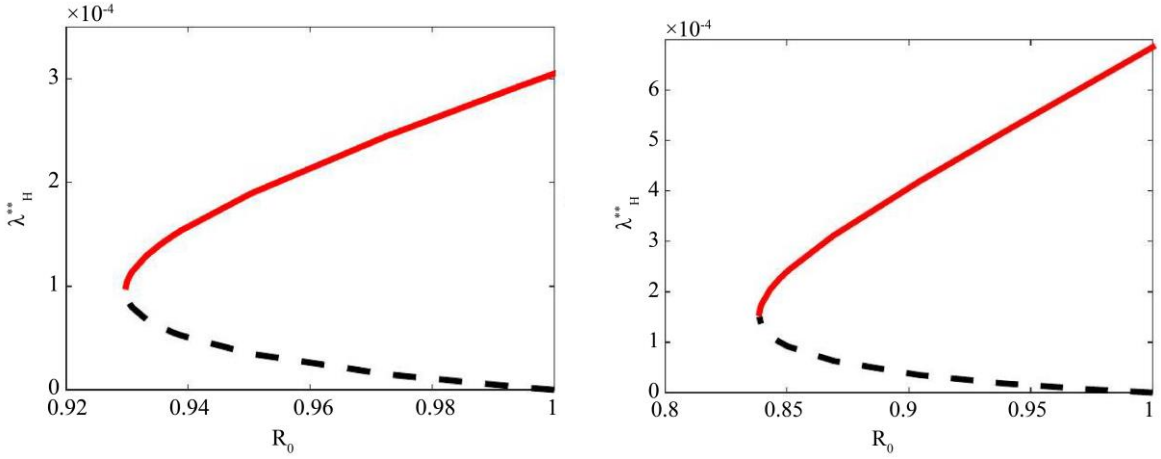
are also computed.

$$\begin{aligned}
a=&-\frac{2v_3w_3^2}{x_1^{*2}p_3^2p_4\alpha_H\mu_H}[G_1x_1^*\mu_H(p_4G_1+p_3p_4\alpha_H+p_4\alpha_H(1-p)\tau+\alpha_Hp_3p\tau)\\
&+G_1x_1^*p_4\alpha_H(p_3+(1-p)\tau)-\beta_H^*\alpha_H^2\alpha_V\beta_Vx_6^*\delta p_3p_4(p_3+(1-p)\tau)]\\
b=&\frac{v_3w_3\beta_V\alpha_V\alpha_H\Lambda_V\mu_H(p_3(1-p)\eta\tau)}{\Lambda_H\mu_V^2p_1p_3p_5}>0.
\end{aligned}\tag{61}$$

Obviously $b>0$ for all biologically feasible parameter values. Hence, if $\delta=0$ it implies that $a<0$. For the non-existence of backward bifurcation, we consider the special case of the transformed model (53) with negligible disease-induced deaths $\delta=0$. Then the backward bifurcation coefficient, $a$, given in (61) reduces to:

$$\begin{aligned}
a=&-\frac{2v_3w_3^2}{x_1^{*2}p_3^2p_4\alpha_H\mu_H}[G_1x_1^*\mu_H(p_4G_1+p_3p_4\alpha_H+p_4\alpha_H(1-p)\tau+\alpha_Hp_3p\tau)\\
&+G_1x_1^*p_4\alpha_H(p_3+(1-p)\tau)]<0.
\end{aligned}\tag{62}$$

Though this study has confirmed that the presence of onchocerciasis induced death will cause a backward bifurcation in the transmission dynamics of the disease, we are also interested in the impact of the fraction who completed their treatment on the bifurcation range. A graphical description of the backward bifurcation phenomenon is given in Figure 4 shows that a decrease in the fraction of humans who completed their treatment increases the backward bifurcation range, making disease control more challenging. Thus, public health control strategies should not just focus on increasing treatment rates but also making sure that a good percentage of the treated population complete their treatment.




(a) $p=0.8$ (b) $p=0.2$

**Fig. 4 A backward bifurcation diagram for the onchocerciasis model (2), showing force of infection against the reproduction number $\mathcal{R}_0$ with $\tau=\delta=0.0005$**

### *3.5. Universal stability of DFE when* $\delta = 0$

Here, we show that the DFE of the system (2) is universally asymptotically stable (GAS), when the malady-induced death is inconsequential; i.e., $\delta = 0$. Making use of this assumption, the resulting reduced model is given by

$$\begin{aligned}
\dot{S}_H &= \Lambda_H - \frac{\beta_H I_V}{N} S_H + \sigma T_C - \mu_H S_H, \\
\dot{E}_H &= \frac{\beta_H I_H}{N} S_H - (\alpha_H + \mu_H) E_H, \\
\dot{I}_H &= \alpha_H E_H + \gamma T_F - (\tau + \mu_H) I_H, \\
\dot{T}_F &= (1-p)\tau I_H - (\gamma + \mu_H) T_F, \\
\dot{T}_C &= p\tau I_H - (\sigma + \mu_H) T_C, \\
\dot{S}_V &= \Lambda_V - \frac{\beta_V (I_H + \eta T_F)}{N} S_V - \mu_V S_V, \\
\dot{E}_V &= \frac{\beta_V (I_H + \eta T_F)}{N} S_V - (\alpha_V + \mu_V) E_V, \\
\dot{I}_V &= \alpha_V E_V - \mu_V I_V,
\end{aligned} \tag{63}$$

We declare the ensuing result:

**Theorem 3.5:** The DFE of the model (2), with negligible disease-induced death (i.e., $\delta = 0$ ) is $GAS$ in $\mathcal{D}$ if $\mathcal{R}_0 \leq 1$ and not stable if $\mathcal{R}_0 > 1$.

**Proof:**

Look at the Lyapunov function given by:

$$\mathcal{T} = Q_1 E_H + Q_2 I_H + Q_3 T_F + Q_4 E_V + Q_5 I_V \tag{64}$$

where

$$\begin{aligned}
Q_1 &= \frac{\beta_V^* S_V^* \alpha_V \alpha_H (p_3 + (1-p)\eta\tau)}{p_1 p_5 \mu_V (p_2 p_3 - (1-p)\gamma\tau)}, \\
Q_2 &= \frac{\beta_V^* S_V^* \alpha_V (p_3 + (1-p)\eta\tau)}{p_5 \mu_V (p_2 p_3 - (1-p)\gamma\tau)}, \\
Q_3 &= \frac{\beta_V^* S_V^* \alpha_V (\gamma + \eta p_2)}{p_5 \mu_V (p_2 p_3 - (1-p)\gamma\tau)}, \\
Q_4 &= \frac{\alpha_V \mathcal{R}_0^m}{\mu_V p_5},\ Q_5 = \frac{\mathcal{R}_0^m}{\mu_V}.
\end{aligned} \tag{65}$$

including Lyapunov derivatives (where a dot depicts a time derivative)

$$\dot{\mathcal{T}} = Q_1 \dot{E}_H + Q_2 \dot{I}_H + Q_3 \dot{T}_F + Q_4 \dot{E}_V + Q_5 \dot{I}_V \tag{66}$$

Placing the right flank of system (2) in (66), we have

$$\begin{aligned}
\dot{\mathcal{T}} &= Q_1 \lambda_H S_H + Q_4 \lambda_V S_V - [Q_1 p_1 + Q_2 \alpha_H] E_H - [Q_2 p_2 - Q_3 (1-p)\tau] I_H \\
&\quad - [Q_3 p_3 - Q_2 \gamma] T_F - [Q_4 p_5 - Q_5 \alpha_V] E_v - Q_5 \mu_V I_V \\
&= \frac{\lambda_H S_H \mathcal{R}_0^{m^2}}{\beta_H S_H^*} + \frac{\lambda_V S_V \alpha_V \mathcal{R}_0}{\mu_V p_5} - \frac{\lambda_V S_V^* \alpha_V}{\mu_V p_5} - \frac{\lambda_H \mathcal{R}_0^m}{\beta_H} \\
&= \frac{\lambda_H \mathcal{R}_0^m}{\beta_H} \left( \frac{S_H \mathcal{R}_0^m}{S_H^*} - 1 \right) + \frac{\lambda_H S_V^* \alpha_V}{p_5 \mu_V} \left( \frac{S_V \mathcal{R}_0^m}{S_V^*} - 1 \right) \\
&\leq \frac{\lambda_H \mathcal{R}_0^m}{\beta_H} (\mathcal{R}_0^m - 1) + \frac{\lambda_V S_V^* \alpha_V}{p_5 \mu_V} (\mathcal{R}_0^m - 1) \\
&= \left( \frac{\lambda_H \mathcal{R}_0^m}{\beta_H} + \frac{\lambda_V S_V^* \alpha_V}{p_5 \mu_V} \right) (\mathcal{R}_0^m - 1)
\end{aligned} \tag{67}$$

Hence, $\dot{\mathcal{T}} \leq 0$ whenever $\mathcal{R}_0 \leq 1$ alongside $\dot{\mathcal{T}} = 0$ if and only if $I_V = I_H = T_F = 0$. Thus, $\mathcal{T}$ is a Lyapunov function in $\mathcal{D}$. Thus, we can safely conclude based on LaSalle's Invariance Principle [22] that:

$$(E_H(t), I_H(t), T_F(t), E_V(t), I_v(t)) \to (0,0,0,0,0,0) \text{ as } t \to \infty. \quad (68)$$

Hence, all orbits of the equations of system (2), with $\delta = 0$, approach the DFE of system (2), as $t \to \infty$ for $\mathcal{R}_0 \leq 1$. This outcome shows that in a community (where individuals are most likely to fail treatment), with the assumption of negligible disease induced death ($\delta = 0$), then the DFE of the system (2) will be universally asymptotically stable (GAS) given the condition that $\mathcal{R}_0 \leq 1$. Therefore, Onchocerciasis is certain to be eliminated from the population regardless of the first magnitudes of the sub-population given the condition that $\mathcal{R}_0 \leq 1$.

### *3.6. Global Stability of an Endemic Steady State*

Consider a unique case of the system (2) when there is no waning of treatment received by infected individuals (i.e. the disease confers life long immunity), meaning once treated, the individuals remain treated for life ($\sigma = 0$). This leads to the following reduced model.

$$\begin{aligned}
\dot{S}_H &= \Lambda_H - \frac{\beta_H I_V}{N} S_H - \mu_H S_H, \\
\dot{E}_H &= \frac{\beta_H I_H}{N} S_H - (\alpha_H + \mu_H) E_H, \\
\dot{I}_H &= \alpha_H E_H + \gamma T_F - (\tau + \mu_H) I_H, \\
\dot{T}_F &= (1-p)\tau I_H - (\gamma + \mu_H) T_F, \\
\dot{T}_C &= p\tau I_H - \mu_H T_C, \\
\dot{S}_V &= \Lambda_V - \frac{\beta_V (I_H + \eta T_F)}{N} S_V - \mu_V S_V, \\
\dot{E}_V &= \frac{\beta_V (I_H + \eta T_F)}{N} S_V - (\alpha_V + \mu_V) E_V, \\
\dot{I}_V &= \alpha_V E_V - \mu_V I_V,
\end{aligned} \quad (69)$$

Furthermore, let the stable manifold of the DFE of the model (2) (with $\sigma = 0$ ) be given by

$$\mathcal{D}_0 = \{(S_H, E_H, I_H, T_F, T_C, S_V, E_V, I_V) \in \mathcal{D} : E_H = I_H = T_F = E_V = I_V = 0\}. \quad (70)$$

We declare the ensuing result.

Theorem 3.6 The EEP, $\mathcal{E}_1$(with $\sigma = 0$ ) is $GAS$ in $\mathcal{D} \setminus \mathcal{D}_0$ whenever $\mathcal{R}_0 > 1$.

Proof: Let $\mathcal{Q}$ be a Lyapunov function expressed as

$$\begin{aligned}
\mathcal{Q} = {} & S_H - S_H^{**} \ln\left(\frac{S_H}{S_H^{**}}\right) + E_H - E_H^{**} \ln\left(\frac{E_H}{E_H^{**}}\right) + B_1 \left(I_H - I_H^{**} \ln\frac{I_H}{I_H^{**}}\right) \\
& + B_2 \left(T_F - T_F^{**} \ln\frac{T_F}{T_F^{**}}\right) + S_V - S_V^{**} \ln\left(\frac{S_V}{S_V^{**}}\right) + E_V - E_V^{**} \ln\left(\frac{E_V}{E_V^{**}}\right) \\
& + B_3 \left(I_V - I_V^{**} \ln\frac{I_V}{I_V^{**}}\right),
\end{aligned} \quad (71)$$

where: $B_1 = \frac{p_1}{\alpha_H}$, $B_2 = \frac{p_1 p_2}{\alpha_H (1-p)\tau}$, $B_3 = \frac{p_5}{\alpha_V}$, and derivatives with respect to time of the Lyapunov functional, $\mathcal{Q}$ defined by

$$\begin{aligned}
\dot{\mathcal{Q}} = {} & \left(1 - \frac{S_H^{**}}{S_H}\right) \dot{S}_H + \left(1 - \frac{E_H^{**}}{E_H}\right) \dot{E}_H + B_1 \left(1 - \frac{I_H^{**}}{I_H}\right) \dot{I}_H + B_2 \left(1 - \frac{T_F^{**}}{T_F}\right) \dot{T}_F \\
& + \left(1 - \frac{S_V^{**}}{S_V}\right) \dot{S}_V + \left(1 - \frac{E_V^{**}}{E_V}\right) \dot{E}_V + B_3 \left(1 - \frac{I_V}{I_V^{**}}\right) \dot{I}_V
\end{aligned} \quad (72)$$

Putting the right flanks of the equations in Equation (69) representing $\dot{S}_H, \dot{E}_H, \dot{I}_H, \dot{T}_F, \dot{S}_V, \dot{E}_V$, and $\dot{I}_V$ into (72), after several algebraic calculations gives:

$$\begin{aligned}\dot{Q} \leq\ & \mu_H S_H^{**}\left(2-\frac{S_H^{**}}{S_H}-\frac{S_H}{S_H^{**}}\right)+\mu_V S_V^{**}\left(2-\frac{S_V^{**}}{S_V}-\frac{S_V}{S_V^{**}}\right)\\ &+\beta_H I_V^{**} S_H^{**}\left(5-\frac{S_H^{**}}{S_H}-\frac{E_H^{**} S_H}{E_H S_H^{**}}-\frac{E_H I_H^{**}}{E_H^{**} I_H}-\frac{T_F}{T_F^{**}}-\frac{T_F^{**} I_H}{T_F I_H^{**}}\right)\\ &+\frac{\gamma\beta_H I_V^{**} S_H^{**} T_F^{**}}{\alpha_H E_H^{**}}\left(1-\frac{I_H^{**}}{I_H}\right)\\ &+\beta_V I_H^{**} S_V^{**}\left(4-\frac{S_V^{**}}{S_V}-\frac{E_V^{**} S_V}{E_V S_V^{**}}-\frac{I_V}{I_V^{**}}-\frac{I_V^{**} E_V}{I_V E_V^{**}}\right)\\ &+\beta_V \eta T_F^{**} S_V^{**}\left(4-\frac{S_V^{**}}{S_V}-\frac{E_V^{**} S_V}{E_V S_V^{**}}-\frac{I_V}{I_V^{**}}-\frac{I_V^{**} E_V}{I_V E_V^{**}}\right).\end{aligned} \tag{73}$$

Due to the fact that the arithmetic mean is exceeds the geometric mean, the corresponding inequalities hold

$$\begin{aligned}&1-\frac{I_H^{**}}{I_H}\leq 0\\ &2-\frac{S_H^{**}}{S_H}-\frac{S_H}{S_H^{**}}\leq 0,\ 2-\frac{S_V^{**}}{S_V}-\frac{S_V}{S_V^{**}}\leq 0,\\ &4-\frac{S_V^{**}}{S_V}-\frac{E_V^{**} S_V}{E_V S_V^{**}}-\frac{I_V}{I_V^{**}}-\frac{I_V^{**} E_V}{I_V E_V^{**}}\leq 0\\ &5-\frac{S_H^{**}}{S_H}-\frac{E_H^{**} S_H}{E_H S_H^{**}}-\frac{E_H I_H^{**}}{E_H^{**} I_H}-\frac{T_F}{T_F^{**}}-\frac{T_F^{**} I_H}{T_F I_H^{**}}\leq 0\end{aligned} \tag{74}$$

Thus, $\dot{Q}\leq 0$ whenever $\mathcal{R}_0>1$.

Since the corresponding variables in the equations for $T_C$ are at the endemic equilibrium, these can be transferred into the equation for $T_C$ in the model (2) (with $\sigma=0$ ), so that:

$$T_C(t)\to T_C^{**}\ \text{ as }\ t\to\infty \tag{75}$$

Thus, $Q$ is a Lyapunov function in $\mathcal{D}\setminus\mathcal{D}_0$. Epidemiologically, the result showed that in a population (where individuals are most likely to fail treatment), Onchocerciasis induced death and waning of treatment are negligible (i.e., $\delta=\sigma=0$ ), the endemic critical point is guaranteed to be universally asymptotically stable (GAS) given that $\mathcal{R}_0>1$ implying that Onchocerciasis will thrive in the community under consideration no matter the first sizes of the sub-population given that $\mathcal{R}_0>1$.

## 4. Discussions and Conclusion

An Onchocerciasis mathematical model incorporating humans who failed to complete their treatment is formulated and analyzed. The disease-free equilibrium of system (2) was shown to be locally asymptotically stable in $\mathcal{D}$ given that $\mathcal{R}_0<1$, which implied that Onchocerciasis can be eradicated from the population given that the first sizes of the sub-classes of the system (2) lie in the basin of attraction of the DFE and that a small influx of infected humans with Onchocerciasis into the community where individuals failed treatment would not generate large outbreaks, and unstable and if $\mathcal{R}_0>1$. Analyzing the reproduction number $(\mathcal{R}_0)$, it was seen that if the infected persons who failed to complete their treatment is $50\%$ as infectious as the infected persons who did not get to be treated, then over $67\%$ of the infected persons will need to complete their treatment for effective disease control. The system (2) was also shown to undergo the backward bifurcation phenomenon induced by human deaths induced by onchocerciasis. The backward bifurcation range was also seen to increase by a decrease in the fraction of humans who completed their treatment. The equilibrium at the infection free state was shown to be globally asymptotically stable when there is negligible Onchocerciasis induced human deaths. Also, the endemic equilibrium was shown to be globally asymptotically stable for the unique case given that there is negligible disease induced death and permanent immunity upon completion of treatment.

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