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Neglected Tropical Diseases with Specific Emphasis on Analytically Investigating the Influence of Age-Structure on the Transmission Dynamics of Hookworm.

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ABSTRACT

This study examined the relationship between public debt and economic growth in Nigeria using annual time-series data from 1990 to 2022. Real Gross Domestic Product was used as the measure of economic growth, while the Autoregressive Distributed Lag (ARDL) modelling approach was employed for analysis. The Augmented Dickey–Fuller (ADF) test was used to examine the stationarity of the variables, while the ARDL bounds test assessed the existence of a long-run equilibrium relationship. An Error Correction Model (ECM) was subsequently employed to examine short-run dynamics. The findings from the ADF test confirmed that the variables were stationary. The ARDL results indicated that government borrowing had no significant short-run effect on economic growth. However, the bounds test confirmed the existence of a long-run equilibrium relationship between public debt and economic growth. The study concludes that government borrowing does not significantly promote short-run economic growth but has significant long-run implications for Nigeria's economy.

1. Introduction

The menace of Neglected Tropical Diseases (NTDs) in advancing poverty and death in the world cannot be overemphasized, especially in poor countries of Africa and Asia. The United Nations (UN) through the World Health Organization (WHO) has identified thirteen major NTDs, which include but are not limited to Human African Trypanosomiasis, Soil-Transmitted Helminths, Dracunculiasis, Trachoma, Lymphatic Flariasis, Onchocerciasis, Buruli Ulcer, Leprosy, Schistosomiasis, and Dengue Fever [1], [2], [3], [4].

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This has greatly impacted the economies of these countries. Efforts at eradicating these diseases have gulped billions of dollars, with the bulk of these funds coming from developed economies [5], [6]. There are speculations in some quarters that these supposed attempts by these developed economies may not be genuine after all, owing to the fact that the bulk of these funds go into large pharmaceutical companies located in these developed countries for research into providing solutions for these diseases. The impression is that these funds also circulate within these developed countries, which are major contributors of these funds. The poor countries are usually left out to wallow in poverty and mysteries arising from these diseases. Another school of thought holds the view that these diseases will continue to ravage mankind despite the supposed efforts by these developed economies because these diseases are mainly endemic in the tropical rainforest and not common to the temperate regions.

Attempts at total eradication of these NTDs have not only been left in the hands of the pharmaceutical companies and health sectors. Mathematicians over the years have formulated various Mathematical models aimed at understanding the behavioural dynamics of these diseases and have also made many recommendations for bringing down the burden of these diseases. Several Mathematical models have been developed and analyzed for understanding the dynamics of some of these NTDs [8], [12], [13], [14], [15], [16],[17],[9],[11],[10].

This investigation, however, focuses on the mechanics of hookworm transmission. Since both the larvae and adult worms reside in the human small intestine and can cause intestinal illness, the hookworm is considered an intestinal parasite of humans. Ancylostoma duodenale and Necator americanus are the two primary hookworm species that infect humans. It is one of the most prevalent soil-transmitted helminth (STH) infections globally. It is estimated to affect over 500 million people, especially in the tropical regions in Africa and Asia. The signs and symptoms of this intestinal parasite include itching and a localized rash, abdominal pain, diarrhea, loss of appetite, weight loss, fatigue, and anemia [7].

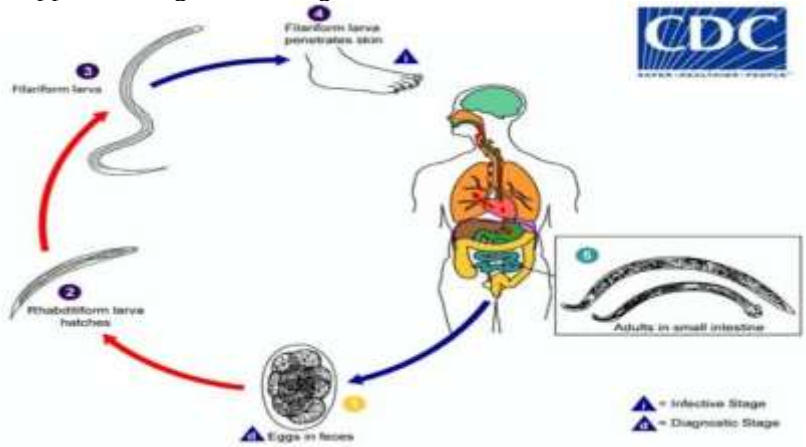

Figure 1: Life cycle of Hookworm

A lot of work has been done to study the dynamics of Hookworm. Notable amongst them are the works of Coffenga et al. (2017) used data from India to formulate two mathematical models for the dynamics of Ascaris lumbricoides and hookworm infection transmission, and the effects of mass drug administration (MDA) were compared. The additional advantages of semi-annual vs annual de-worming, targeting the entire population as opposed to just children, and the possibility of stopping transmission were all qualitatively agreed upon by their models. Also, their study illustrated that long-term predictions are sensitive to modelling assumptions about which age groups contribute most to transmission, which depended on human demography and age-patterns in exposure and contribution to the environmental reservoir of infection, the latter being notoriously difficult to empirically quantify ([18]). Pawelek et al. (2017) developed a model to study the spread of the hookworm infection at the population level, having a variety of parasite development stages, and their modelling results suggested that the de-worming programs should be maintained at least once per year to control the infection ([19]). Sabatelli et al. (2008) presented a model to assess the relative impact of chemotherapy and vaccination against human hookworm infection and investigated the implications of

potential correlations between risk of infection and vaccine efficacy. According to their modelling, the greatest impact of vaccination will occur in high-transmission settings when vaccine efficacy is highest among those with the highest worm burdens, and the reduction of morbidity is largely dependent on the degree of protection attained in the most vulnerable 8-12% of the population. They also demonstrated that if the risk of infection and vaccine protection are correlated, there is not always a direct correspondence between the reduction in worm burden and in morbidity, with the precise relationship varying according to transmission setting ([20]). Also, Mustapha et al. (2020) built a deterministic model which had two infectious classes and multiple parasite developmental stages. Their results showed that instead of a single intervention, it was very effective to combine chemotherapy with health education and sanitation. The study highlighted the importance of integrating environmental management with treatment strategies to interrupt transmission ([21]). Sangari et al. (2024) formulated a five-compartment mathematical model for hookworm transmission in Nigeria. Their result showed that disease prevalence is reduced with improved sanitation and early treatment ([22]). Shafique et al. (2025) developed an eight-compartment mathematical model to study the prevalence of hookworm. Their results showed that stochastic non-standard finite difference methods preserved important biological properties while accurately simulating transmission dynamics ([23]). Malizia et al. (2024) proposed a statistical framework linking hookworm intensity to haemoglobin concentration, thereby providing a quantitative approach for evaluating hookworm-associated anemia. Their work improved understanding of morbidity assessment and supported more accurate evaluation of intervention effectiveness in endemic settings ([24]).

However, these literatures and virtually all mathematical models studying the dynamics of Hookworm have not explored the role of an age-structured model in the transmission dynamics of hookworm. The works of Ajjampur et al. (2021), which investigated hookworm epidemiology within community settings and reported that adult populations constitute an important reservoir for continued transmission is enough biological evidence to partition the human hookworm population into children and adults in order to have mathematical evidence for their claims. Their findings further demonstrated that school-based de-worming alone is insufficient for interrupting transmission and emphasized the need for community-wide treatment programmes to achieve elimination ([25]). This paper focuses on exploring the impact of age structure on the dynamics of Hookworm. The model is cascaded into two human subpopulations: children and adults.

2 The Age-structured Hookworm Model

In formulating the age-structured Hookworm model, we make the following assumptions: the population is homogeneous, well-mixed, and all individuals have equal chances of being infected, and the number of effective contacts (resulting in an infection) is assumed to depend on the frequency of contacts between susceptible and infected water [29], Mishra. There is no vertical transmission of the disease in humans or immigration of infectious classes, and humans become infected when they come into contact with filariform larvae in aquatic settings. New individuals enter the system as children; as they age, they progress into the adult classes, and recovered children become susceptible immediately, since the disease does not confer any form of immunity. The natural death rate is constant for both children and adult populations, and treatment can also wane in children, and they move into susceptible adults. The total population of the Age-Structured Hookworm model is partitioned into nine non-overlapping compartments (four for the children subpopulation, four for the adult subpopulation, and one for the aquatic environment). The compartments of the models are: susceptible children not infected with Hookworm (S_c); latently infected children exposed to Hookworm through interaction with the aquatic environment but not infectious (E_c); infected children (I_c); recovered children (R_c); susceptible adults not infected with Hookworm (S_a); latently infected adults exposed to Hookworm through interaction with the aquatic environment but not infectious (E_a); infected adults (I_a); recovered adults (R_a) and the aquatic environment (V). Hence, the total population for the age-structured Hookworm model at all time t, is given by:

$$N(t) = N_c(t) + N_a(t) = S_c(t) + E_c(t) + I_c(t) + R_c(t) + S_a(t) + E_a(t) + I_a(t) + R_a(t). \quad (1)$$

The above assumptions lead to a system of 9 ordinary differential equations (ODEs) describing the disease dynamics of our age-structured hookworm model. For $\mathrm{i} = \mathrm{a}, \mathrm{c}$, the equations in the model are:

$$\frac{dS_c}{dt} = \Lambda - \lambda_c S_c + \sigma_c R_c - (\mu + \eta) S_c$$
$$\frac{dE_c}{dt} = \lambda_c S_c - (\mu + \eta + \gamma_c) E_c$$
$$\frac{dI_c}{dt} = \gamma_c E_c - (\alpha_c + \delta_c + \mu + \eta) I_c$$
$$\frac{dR_c}{dt} = \alpha_c I_c - (\sigma_c + \phi_c + \mu + \eta) R_c$$
$$\frac{dS_a}{dt} = \eta S_c - \lambda_a S_a + \sigma_a R_a + \phi_c R_c - \mu S_a \quad (2)$$
$$\frac{dE_a}{dt} = \eta E_c + \lambda_a S_a - (\mu + \gamma_a) E_a$$
$$\frac{dI_a}{dt} = \eta I_c + \gamma_a E_a - (\alpha_a + \delta_a + \mu) I_a$$
$$\frac{dR_a}{dt} = \eta R_c + \alpha_a I_a - (\sigma_a + \mu) R_a$$
$$\frac{dV}{dt} = \pi_c I_c + \pi_a I_a - \chi V$$

where:

$$\lambda_c = \beta_c \frac{(I - \phi\xi)V}{k+V} \text{ and } \lambda_a = \beta_a \frac{(I - \phi\xi)V}{k+V} \quad (3)$$

Figure 2 shows the schematic representation of the age-structured hookworm model that a pictorial representation of how the children and adults move within the compartments. Table 1 and 2 shows the state variables and their interpretations and the parameters of the model and their interpretations, respectively.

Variable	Description
$S_c(t)$	Susceptible children
$E_c(t)$	Latently-infected children
$I_c(t)$	Infected children
$R_c(t)$	Recovered children
$S_a(t)$	Susceptible adult
$E_a(t)$	Latently-infected adult
$I_a(t)$	Infected adult
$R_a(t)$	Recovered adult
V	Aquatic environment

Table 1: Variables of the Age-Structured Hookworm model and their interpretation.

Parameter	Description
μ	Natural death rate
Λ	Recruitment rate of children
$\beta_i (\mathrm{i} = c, a)$	Infectious rate of i
k	Saturation constant
η	Maturation rate of children or ageing into the adult population
$\sigma_i (i = a, c)$	Loss of immunity within the same age group

$\gamma_i(i = c, a)$	Transfer rate from the latently infected class to the infected class of i
$\alpha_i(i = c, a)$	Treatment rate of i
ϕ_c	Rate at which treatment wanes for children, and they become susceptible in the adult population
$\delta_i(i = c, a)$	Disease-induced death of i
ϕ	Availability of control measure in the aquatic environment
ξ	Efficacy of control measures in the aquatic environment
$\pi_i(i = c, a)$	Concentration of worms deposited in the aquatic environment by i
χ	Natural death rate of filariform larva in the aquatic environment

Table 2: Parameters of the Age-Structured Hookworm Model and their interpretation.

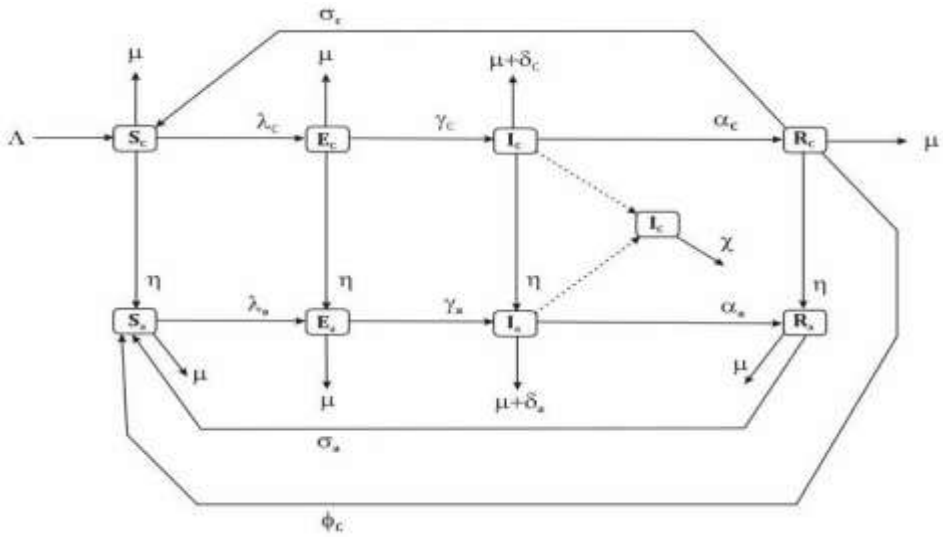

Figure 2: Schematic representation of the age-structured hookworm model.

3 Hookworm Model Analysis

This section considers the qualitative properties of the model.

3.1 Positivity and Boundedness of Solutions of the Age-Structured Hookworm Model

The age-structured Hookworm model will be analyzed in an epidemiologically feasible region. We shall show that all the feasible solutions are bounded uniformly in a proper subset

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_V \subset \mathbb{R}_+^8 \times \mathbb{R}_+ \subset \mathbb{R}_+^9 \tag{4}$$

where:

$$\mathcal{D}_h = \left\{(S_c(t), S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t)) \in \mathbb{R}_+^8 : N_h \leq \frac{\Lambda}{\mu}\right\} \tag{5}$$

and

$$\mathcal{D}_V = \left\{V \in \mathbb{R}_+ : V \leq \frac{\Lambda(\pi_c + \pi_a)}{\mu\chi}\right\} \tag{6}$$

For the Age-Structured Hookworm model to be biologically meaningful, it is important to prove that all its state variables are non-negative and bounded for all time (t), and that $\mathcal{D}$ is indeed bounded. We also showed that the orbits generated by the Age-Structured Hookworm model are positively invariant for all time, t. In line with the above, we claim the following.

Theorem 1. Let the initial data for the age-structured hookworm model be given as $X(0) \geq 0$ where $X(t) = (S_c(t), S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t), V)$. Then the trajectories $X(t)$ of the age structured hookworm model with positive initial conditions will remain nonnegative for all time $t > 0$.

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

$$\frac{dS_c(t)}{dt} = \Lambda_-(\lambda_c S_c + \mu + \eta)S_c(t) + \sigma_c R_c(t)$$

Which can be rewritten as

$$\left[\frac{d}{dt} + (\lambda_c + \mu + \eta)\right] S_c(t) \geq \Lambda \tag{7}$$

which follows that,

$$\frac{d}{dt}\left[S_c(t)\exp\left\{(\mu+\eta)t + \int_0^t \lambda_c(\tau)d\tau\right\}\right] \geq \Lambda\exp\left\{(\mu+\eta)t + \int_0^t \lambda_c(\tau)d\tau\right\} \tag{8}$$

as a result,

$$S_c(t_1)\exp\left\{(\mu+\eta)t_1 + \int_0^{t_1} \lambda_c(\tau)d\tau\right\} - S_c(0) \geq \int_0^{t_1} \Lambda_c[\exp\{(\mu+\eta)y + \int_0^y \lambda_c(\tau)d\tau\}]dy$$

So that, $S_c(t_1) \geq S_c(0)\exp\left[-(\mu+\eta)t_1 - \int_0^{t_1} \lambda_c(\tau)d\tau\right] + [\exp\{-(\mu+\eta)t_1 - \int_0^{t_1} \lambda_c(\tau)d\tau\}]\int_0^{t_1} \Lambda_c[\exp\{(\mu_h+\eta)y + \int_0^y \lambda_c(\tau)d\tau\}]dy \geq 0.$

Hence $S_c(t) \geq 0, \quad \forall \quad t > 0.$

Similarly, it can be shown that $S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t), V(t)$ are $\geq 0, \forall \quad t > 0.$

Hence, the trajectories X(t) generated by the Age-Structured Hookworm model with non-negative initial data/conditions will always be non-negative $\forall \ t > 0$.

Next, we show that the Age-Structured Hookworm model is bounded and also determine the bound and subsequently show that the domains of the Age-Structured Hookworm model are positively-invariant and attract all the positive trajectories (there exists a unique solution to the initial value problem, and the solution exists for all time) of the model.

Lemma 1: Let $(S_c(t), S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t), V)$ be the orbits of the Age Structured Hookworm model with initial conditions given in Theorem 1 and the biological feasible region given by the set

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_V \subset \mathbb{R}_+^8 \times \mathbb{R}_+ \subset \mathbb{R}_+^9$$

where:

$$\mathcal{D}_h = \left\{(S_c(t), S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t)) \in \mathbb{R}_+^8 : N_h \leq \frac{\Lambda}{\mu}\right\}$$

and

$$\mathcal{D}_V = \left\{V \in \mathbb{R}_+ : V \leq \frac{\Lambda(\pi_c + \pi_a)}{\mu\chi}\right\}.$$

The region $\mathcal{D} = \mathbb{R}_+^9$ is positively invariant and attracts all the non-negative trajectories of the Age-Structured Hookworm model with initial condition in $\mathbb{R}_+^9$.

Proof: Adding up the equations in system 2 for humans in the Age-Structured Hookworm model gives

$$\frac{dN_h(t)}{dt} = \Lambda - \mu N_h(t) - \delta_c I_c(t) - \delta_a I_a(t) \leq \Lambda - \mu N_h(t). \tag{9}$$

Hence, $\frac{dN_h(t)}{dt} \leq 0$ if $N_h(t) \geq \frac{\Lambda}{\mu}$. A standard comparison theorem in [27] can be used to show that $N_h(t) \leq N_h(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t})$. If $N_h(0) \leq \frac{\Lambda}{\mu}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ for all $t > 0$. Hence, the set $\mathcal{D}_h$ is positively invariant.

Moreover, if $N_h(0) > \frac{\Lambda}{\mu}$, then either the orbits enter the domain $\mathcal{D}_h$ in finite time or $N_h(t)$ asymptotically approaches $\frac{\Lambda}{\mu}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_h$ attracts all trajectories in $\mathbb{R}_+^8$.

Similarly, for the ninth equation in system 2 , we have:

$\frac{dV(t)}{dt} = \pi_c I_c(t) + \pi_a I_a(t) - \chi V(t)$ which can be written as $\frac{dV(t)}{dt} \leq (\pi_c + \pi_a)\frac{\Lambda}{\mu} - \chi V(t)$. Since we shown that $N_h(t) = S_c + S_a + E_c + E_a + I_c + I_a + R_c + R_a \leq \frac{\Lambda}{\mu} \Longrightarrow I_c \leq \frac{\Lambda}{\mu}$ and $I_a \leq \frac{\Lambda}{\mu}$.

Hence, $\frac{dV(t)}{dt} \leq 0$ if $V(t) \geq (\pi_c + \pi_a)\frac{\Lambda}{\mu\chi}$. A standard comparison theorem in [27] can be used to show that $V(t) \leq V(0)e^{-\chi t} + \frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}(1 - e^{-\chi t})$. If $V(0) \leq \frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}$, then $V(t) \leq \frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}$ for all $t > 0$. Hence, the set $\mathcal{D}_V$ is positively invariant.

Moreover, if $V(0) > \frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}$, then either the orbits enter the domain $\mathcal{D}_V$ in finite time or $V(t)$ asymptotically approaches $\frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_V$ attracts all trajectories in $\mathbb{R}_+$.

Since the domain $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_V$ is positively-invariant, it is enough to study the dynamics of the flows generated by the system Age Structured Hookworm Model in $\mathcal{D}$.

$$\mathcal{D} = \begin{cases} (S_c(t), S_a(t), E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t)) \in \mathbb{R}_+^8 : N_h \leq \dfrac{\Lambda}{\mu} \\ V \in \mathbb{R}_+ : V \leq \dfrac{\Lambda(\pi_c + \pi_a)}{\mu\chi} \end{cases}$$

We conclude, therefore, that the model Age Structured Hookworm Model is both mathematically and epidemiologically well posed. Hence, it is sufficient to consider the dynamics of the flow generated by the Age Structured Hookworm model in $\mathcal{D}$. In this region, the model can be considered as being epidemiologically and mathematically well-posed [29]. Thus, every solution of the Age Structured Hookworm model with initial conditions in $\mathcal{D}$ remains in $\mathcal{D}$ for all time $t > 0$. Therefore, the ω-limit sets of the Age-Structured Hookworm model are contained in $\mathcal{D}$.

3.2 Local Stability of the Disease-Free Equilibrium

The Disease-Free Equilibrium (DFE) for the Age-Structured Hookworm model is obtained by setting the diseased classes to zero and solving the resulting system. The DFE of Model 2 is given by:

$$\mathcal{E}_0 = (S_c^0, S_a^0, E_c^0, E_a^0, I_c^0, I_a^0, R_c^0, R_a^0, V^0) = \left(\frac{\Lambda}{\eta + \mu}, \frac{\Lambda\eta}{\mu(\mu + \eta)}, 0,0,0,0,0,0,0\right). \tag{10}$$

The local asymptotic stability (LAS) of the DFE is investigated using the next generation matrix operator method [31]. Using notations similar to the ones used in [31], the matrices F and V, of new infection terms as well as the remaining transfer terms are respectively given by:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \dfrac{A\beta_c\Lambda}{\kappa(\eta + \mu)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \dfrac{A\beta_a\Lambda\eta}{\kappa\mu(\eta + \mu)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{11}$$

and

$$V = \begin{pmatrix} T_2 & 0 & 0 & 0 & 0 \\ -\gamma_c & T_3 & 0 & 0 & 0 \\ -\eta & 0 & T_5 & 0 & 0 \\ 0 & -\eta & -\gamma_a & T_6 & 0 \\ 0 & -\pi_c & 0 & -\pi_a & \chi \end{pmatrix} \tag{12}$$

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

$$\mathcal{R}_0 = \frac{A\Lambda[\beta_a\gamma_a\pi_a\eta T_2T_3 + \beta_c\mu(\eta\gamma_a\pi_aT_3 + \eta\gamma_c\pi_aT_5 + \gamma_c\pi_cT_5T_6)]}{\kappa\chi\mu T_1T_2T_3T_5T_6} \tag{13}$$

where $A = 1 - \phi\xi, T_1 = \eta + \mu, T_2 = \gamma_c + \eta + \mu, T_3 = \alpha_c + \delta_c + \eta + \mu, T_4 = \sigma_c + \eta + \phi_c + \mu, T_5 = \gamma_a + \mu, T_6 = \alpha_a + \delta_a + \mu, T_7 = \sigma_a + \mu$.

Using Theorem 2 in [31] we claim the following result:

Lemma 2: The DFE of the system 2 is Locally Asymptomatically Stable (LAS) in $\mathcal{D}$ if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

Epidemiologically, Lemma 2 implies that Hookworm can be eradicated from the population when $\mathcal{R}_0 < 1$, if the initial sizes of the sub-populations of the system (2) lie in the basin of attraction of the DFE. This also implies that if a small number of infectious hookworm patients enter such a population, it will not lead to a large outbreak where control is available.

3.2.1 Analysis of the Reproduction Number ($\mathcal{R}_0$)

Analysis of the threshold quantity ($\mathcal{R}_0$) with respect to some key parameters ($\alpha_c, \alpha_a, \phi\xi$) is investigated by considering the partial derivatives and limits of $\mathcal{R}_0$ with respect to these parameters.

$$\frac{\partial\mathcal{R}_0}{\partial\alpha_c} = -\frac{(1-p)\Lambda\beta_c\gamma_c(\eta\pi_a + \pi_cT_6)}{\kappa\chi T_1T_2T_4^2T_6} \tag{14}$$

$$\frac{\partial\mathcal{R}_0}{\partial\phi\xi} = -\frac{\Lambda[\eta\pi_a\beta_a\gamma_aT_2T_3 + \mu\beta_c(\eta\gamma_c\pi_aT_5 + \pi_c\gamma_cT_5T_3T_6)]}{\kappa\chi\mu T_1T_2T_3T_5T_6} \tag{14}$$

Clearly, it follows from (14) that $\frac{\partial\mathcal{R}_0}{\partial\alpha_c} < 0$ and $\frac{\partial\mathcal{R}_0}{\partial\phi\xi} < 0$ unconditionally.

$$\frac{\partial\mathcal{R}_0}{\partial\alpha_a} = -\frac{\left[(1-p)\eta\Lambda\pi_a\left(\beta_a\gamma_aT_2T_3 - \mu\beta_c\left(\mu\gamma_c + \gamma_a(\eta+\mu+\alpha_c+\gamma_c+\delta_c)\right)\right)\right]}{\kappa\chi\mu T_1T_2T_3T_5T_6^2} \tag{15}$$

From (15) it follows that $\frac{\mathcal{R}_0}{\partial\alpha_a} < 0$ if

$$\triangle_1 > \triangle_2 \text{ , where } \triangle_1 = \beta_a\gamma_aT_2T_3, \text{ and } \triangle_2 = \mu\beta_c\left(\mu\gamma_c + \gamma_a(\eta+\mu+\alpha_c+\gamma_c+\delta_c)\right). \tag{16}$$

Epidemiologically, increasing the treatment rates in both children (α_c) and adult (α_a) subpopulations will have a positive impact on the control of hookworm. However, increasing the treatment rate (α_a) in the adult subpopulation will only impact the control of hookworm positively in the population if $\triangle_1 > \triangle_2$. Also, making the control available and ensuring that the controls are effective ($\phi\xi$) in the population will have a positive impact in the control of hookworm.

3.3 Analysis of the Endemic Equilibrium

Let

$$\mathcal{E}_1 = (S_c^{**}, S_a^{**}, E_c^{**}, E_a^{**}, I_c^{**}, I_a^{**}, R_c^{**}, R_a^{**}, V^{**}) \tag{17}$$

be an Endemic Equilibrium Point (EEP) for the system (2). Solving the right-hand side of the equations in (2) in terms of the force of infection at the EEP, gives:

$$S_c^{**} = \frac{T_2T_3T_4\Lambda}{G_1\lambda_1^{**}+T_1T_2T_3T_4}, \quad S_a^{**} = \frac{\Lambda(G_3+G_4\lambda_1^{**})}{(G_1\lambda_1^{**}+T_1T_2T_3T_4)(G_2\lambda_2^{**}+\mu T_5T_6T_7)},$$
$$E_c^{**} = \frac{\Lambda T_3T_4\lambda_1^{**}}{G_1\lambda_1^{**}+T_1T_2T_3T_4}, \quad E_a^{**} = \frac{\Lambda(G_5\lambda_1^{**}\lambda_2^{**}+G_6\lambda_1^{**}+G_3\lambda_2^{**})}{T_5(G_1\lambda_1^{**}+T_1T_2T_3T_4)(G_2\lambda_2^{**}+\mu T_5T_6T_7)},$$
$$I_c^{**} = \frac{T_4\Lambda\gamma_c\lambda_1^{**}}{G_1\lambda_1^{**}+T_1T_2T_3T_4}, \quad I_a^{**} = \frac{\Lambda(G_7\lambda_1^{**}\lambda_2^{**}+G_8\lambda_1^{**}+\gamma_aG_3\lambda_2^{**})}{T_5T_6(G_1\lambda_1^{**}+T_1T_2T_3T_4)(G_2\lambda_2^{**}+\mu T_5T_6T_7)}, \quad (18)$$
$$R_c^{**} = \frac{\alpha_c\gamma_c\lambda_1^{**}\Lambda}{G_1\lambda_1^{**}+T_1T_2T_3T_4}, \quad R_a^{**} = \frac{\Lambda(G_9\lambda_1^{**}\lambda_2^{**}+G_{10}\lambda_1^{**}+\alpha_a\gamma_aG_3\lambda_2^{**})}{T_5T_6T_7(G_1\lambda_1^{**}+T_1T_2T_3T_4)(G_2\lambda_2^{**}+\mu T_5T_6T_7)},$$
$$V^{**} = \frac{\Lambda(G_{11}\lambda_1^{**}\lambda_2^{**}+G_{12}\lambda_1^{**}+\pi_a\gamma_aG_3\lambda_2^{**})}{\chi T_5T_6(G_1G_2\lambda_1^{**}\lambda_2^{**}+\mu T_5T_6T_7G_1\lambda_1^{**}+T_1T_2T_3T_4G_2\lambda_2^{**}+T_1T_2T_3T_4T_5T_6T_7\mu)}$$

Where:

$$G_1 = T_2T_3T_4 - \gamma_c\alpha_c\sigma_c > 0, G_2 = T_5T_6T_7 - \gamma_a\alpha_a\sigma_a > 0,$$
$$G_3 = \eta T_2T_3T_4T_5T_6T_7,$$
$$G_4 = \eta\alpha_c\gamma_c\sigma_aT_5T_6 + \eta\gamma_c\sigma_a\alpha_aT_4T_5 + \eta\sigma_a\alpha_a\gamma_aT_3T_4 + \phi_c\sigma_c\gamma_cT_5T_6T_7,$$
$$G_5 = \eta T_3T_4G_2 + G_4, G_6 = \eta\mu T_3T_4T_5T_6T_7, \quad (19)$$
$$G_7 = \eta\gamma_cT_4T_5G_2 + \gamma_aG_5, G_8 = \eta\gamma_c\mu T_4T_5^2T_6T_7 + \gamma_aG_6,$$
$$G_9 = \eta\alpha_c\gamma_cT_5T_6G_2 + \alpha_aG_7, G_{10} = \eta\alpha_c\gamma_c\mu T_5^2T_6^2T_7 + \alpha_aG_8,$$
$$G_{11} = \pi_c\gamma_cT_4T_5T_6G_2 + \pi_aG_7, G_{12} = \pi_c\gamma_cT_4T_5^2T_6^2T_7\mu + \pi_aG_8,$$

Substituting the forces of infection, $\lambda_1 = \frac{A\beta_cV}{\kappa+V}$ and $\lambda_2 = \frac{A\beta_aV}{\kappa+V}$ into V^{**} of the expressions for the EEP in (18), it follows (after several algebraic manipulations) that the EEP of the system (2) satisfies the following polynomial at the steady state:

$$P_2V^{**^2} + P_1V^{**} + P_0 = 0. \quad (20)$$

where

$$P_2 = \chi T_5T_6G_1G_2A_2\beta_c\beta_a + \chi T_5^2T_6^2T_7G_1A\beta_c + \chi T_1T_2T_3T_4T_5T_6G_2A\beta_a + \chi T_1T_2T_3T_4T_5^2T_6^2T_7\mu$$

$$P_1 = A\chi T_5^2T_6^2T_7G_1\beta_c\kappa + A\chi T_1T_2T_3T_4T_5T_6\beta_a\kappa + 2\chi T_1T_2T_3T_4T_5T_6T_7\mu\kappa - A^2\Lambda G_{11}\beta_c\beta_a - A\Lambda G_{11}\beta_c - A\Lambda\pi_a\gamma_aG_3\beta_a$$

$$P_0 = \chi T_1T_2T_3T_4T_5T_6T_7\mu\kappa^2(1-\mathcal{R}_0), \quad (21)$$

The components of the EEP are then obtained by solving for V^{**} from the polynomial (20), and substituting the positive values of V^{**} into the expressions for λ_1^{**} and λ_2^{**} and substituting again into the expressions in (18).

Moreover, the number of positive roots of the polynomial (20) depends on the sign change between the coefficients P_1 and P_0.

The following results can be deduced.

Theorem 2: The model (2) has

1. two endemic equilibria if $P_1 < 0$ and $\mathcal{R}_0 < 1$,
2. unique endemic equilibria if $P_1 > 0$ and $\mathcal{R}_0 > 1$,
3. no endemic equilibrium otherwise, when $\mathcal{R}_0 < 1$.

Item 1 of Theorem 2 suggests the possibility of backward bifurcation (BB) in the system (2).

The phenomenon of the BB is characterized by the co-existence of a stable DFE and a stable EEP when the corresponding reproduction number of the model is less than unity.

The epidemiological Implication in an age-structured population (where there is some level of control): the classical requirement of having the reproduction number less than unity is necessary but no longer sufficient for effective control of hookworm.

3.3.1 Bifurcation Analysis

Theorem 3: The system (2) exhibits backward bifurcation at $\mathcal{R}_0 = 1$ whenever a bifurcation coefficient, denoted by **a** is positive.

Proof: Let

$$\mathcal{E}_a = (S_c^{**}, S_a^{**}, E_c^{**}, E_a^{**}, I_c^{**}, I_a^{**}, R_c^{**}, R_a^{**}, V^{**}) \tag{22}$$

represent an arbitrary EEP of the system (2). The existence of backward bifurcation is explored using the Center Manifold Theory.
Consider the case with $\beta_c = \beta_c^*$, a bifurcation parameter. Solving for $\beta_H = \beta_c^*$ from $\mathcal{R}_0 = 1$ yields

$$\beta_c^* = \frac{\kappa\chi\mu T_1T_2T_3T_5T_6 - A\Lambda\beta_a\gamma_a\pi_a\eta T_2T_3}{A\Lambda\mu(\eta\gamma_a\pi_aT_3 + \eta\gamma_c\pi_aT_5 + \gamma_c\pi_cT_5T_6)}. \tag{23}$$

Let $S_c = x_1, E_c = x_2, I_c = x_3, R_c = x_4, S_a = x_5, E_a = x_6, I_a = x_7$, and $R_a = x_8$, $V = x_9$. It follows that the system (2) can be rewritten as:

$$\begin{aligned}
\dot{x}_1 &\equiv f_1 = \Lambda - \lambda_c x_1 + \sigma_c x_4 - T_1 x_1,\\
\dot{x}_2 &\equiv f_2 = \lambda_c x_1 - T_2 x_2,\\
\dot{x}_3 &\equiv f_3 = \gamma_c x_2 - T_3 x_3,\\
\dot{x}_4 &\equiv f_4 = \alpha_c x_3 - T_4 x_4,\\
\dot{x}_5 &\equiv f_5 = \eta x_1 - \lambda_a x_5 + \sigma_a x_8 + \phi_c x_4 - \mu x_5,\\
\dot{x}_6 &\equiv f_6 = \eta x_2 + \lambda_a x_5 - T_5 x_6,\\
\dot{x}_7 &\equiv f_7 = \eta x_3 + \gamma_a x_6 - T_6 x_7,\\
\dot{x}_8 &\equiv f_8 = \eta x_4 + \alpha_a x_7 - T_7 x_8,\\
\dot{x}_9 &\equiv f_9 = \pi_c x_3 + \pi_a x_7 - \chi x_9,
\end{aligned} \tag{24}$$

where

$$\lambda_c = \frac{(1-\phi\xi)\beta_c^* x_9}{\kappa + x_9} \text{ and } \lambda_a = \frac{(1-\phi\xi)\beta_a x_9}{\kappa + x_9} \tag{25}$$

are the forces of infection corresponding to children and adult populations, respectively.
The Jacobian of the transformed system at the DFE with $\beta_c = \beta_c^*$, is given by:

$$J_{\beta_c^*} = \begin{pmatrix}
-T_1 & 0 & 0 & \sigma_c & 0 & 0 & 0 & 0 & -\frac{A\beta_c^* x_1^*}{\kappa}\\
0 & -T_2 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{A\beta_c^* x_1^*}{\kappa}\\
0 & \gamma_c & -T_3 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \alpha_c & -T_4 & 0 & 0 & 0 & 0 & 0\\
\eta & 0 & 0 & \phi_c & -\mu & 0 & 0 & \sigma_a & -\frac{A\beta_a x_5^*}{\kappa}\\
0 & \eta & 0 & 0 & 0 & -T_5 & 0 & 0 & \frac{A\beta_a x_5^*}{\kappa}\\
0 & 0 & \eta & 0 & 0 & \gamma_a & -T_6 & 0 & 0\\
0 & 0 & 0 & \eta & 0 & 0 & \alpha_a & -T_7 & 0\\
0 & 0 & \pi_c & 0 & 0 & 0 & \pi_a & 0 & -\chi
\end{pmatrix}. \tag{26}$$

The matrix $J_{\beta_c^*}$ has a simple zero eigenvalue and all other eigenvalues have negative real part.

Eigenvectors of $J_{\beta_c^*}$
The Jacobian $J_{\beta_c^*}$ has a right eigenvector $\mathbf{w} = (w_1, w_2, \ldots, w_9)^T$, satisfying where

$w_1 = \frac{A\beta_c^* x_1^*(\sigma_c\alpha_c\gamma_c - T_2T_3T_4)}{\kappa T_1T_2T_3T_4}w_9, \quad w_2 = \frac{A\beta_c^* x_1^*}{\kappa T_2}w_9, \quad w_3 = \frac{A\gamma_c\beta_c^* x_1^*}{\kappa T_2T_3}w_9,$

$w_4 = \frac{A\alpha_c\gamma_c\beta_c^* x_1^*}{\kappa T_2T_3T_4}w_9$

$w_5 = \frac{Aw_9}{\kappa\mu T_1T_2T_3T_4T_5T_6T_7}[\beta_c^* x_1^*(T_5T_6T_6\gamma_c\alpha_c\mu\phi_c - T_1\eta\mu(T_5T_6\gamma_c\alpha_c + T_4T_5T_7\gamma_c + T_4T_5\gamma_c\alpha_a + T_3T_4\gamma_a\alpha_a + T_3T_4T_7\gamma_a + T_3T_4T_6T$
$-T_4T_7\eta(T_5T_6\gamma_c\delta_c + T_1T_5\gamma_c\delta_a + T_1T_3\gamma_a\delta_a)) - \beta_a x_5^* T_1T_2T_3T_4(T_6T_7\mu + T_7\gamma_a(\delta_a+\mu) + \gamma_a\alpha_a\mu)],$

$w_6 = \frac{A(\eta\beta_c x_1^* + T_2\beta_a x_5^*)}{\kappa T_2T_5}w_9, w_7 = \frac{A[\beta_c^* x_1^*(T_5\eta\gamma_c + T_3\eta\gamma_a) + T_2T_3\gamma_a\beta_a x_5^*]}{\kappa T_2T_3T_5T_6}w_9$
$w_8 = \frac{A[\beta_c^* x_1^*(T_5T_6\eta\alpha_c\gamma_c + T_4T_5\eta\alpha_a\gamma_a + T_3T_4\eta\gamma_a\alpha_a) + T_2T_3T_4\alpha_a\gamma_a\beta_a x_5^*]}{\kappa T_2T_3T_4T_5T_6T_7}w_9$ (29)
$w_9 = w_9 > 0$

Similarly, $J_{\beta_c^*}$ has a left eigenvector $\mathbf{v} = (v_1, v_2, \dots, v_9)$, satisfying $\mathbf{v}\cdot\mathbf{w} = 1$, where

$v_1 = 0, \quad v_2 = \frac{\eta\pi_a(T_3\gamma_a + T_5\gamma_c) + T_5T_6\gamma_c\pi_c}{T_2T_3T_5T_6}v_9, \quad v_3 = \frac{\pi_a\eta + T_6\pi_c}{T_3T_6}v_9$

$v_4 = 0, \quad v_5 = 0, \quad v_6 = \frac{\gamma_a\pi_a}{T_5T_6}v_9, \quad v_7 = \frac{\pi_a}{T_6}v_9, \quad v_8 = 0, \ v_9 = v_9 > 0.$ (30)

Applying the Center Manifold Theory in [26], we compute the associated non-zero partial derivatives of the right-hand sides of the transformed system of (24), evaluated at the DFE with $\beta_c = \beta_c^*$ and the bifurcation coefficients, a and b :

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \text{ and } b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_H^*}(0,0), \quad (31)$$

Computation of a and b

For the transformed system (24), the associated non-zero partial derivatives are given by

$\frac{\partial^2 f_2}{\partial x_1\partial x_9} = \frac{\partial^2 f_2}{\partial x_9\partial x_1} = -\frac{A\beta_c^*}{\kappa}, \quad \frac{\partial^2 f_2}{\partial x_9\partial x_9} = -\frac{2A\beta_c^* x_1^*}{\kappa^2}, \quad \frac{\partial^2 f_6}{\partial x_5\partial x_9} = \frac{\partial^2 f_6}{\partial x_9\partial x_5} = -\frac{A\beta_a}{\kappa},$
$\frac{\partial^2 f_6}{\partial x_9\partial x_9} = -\frac{2A\beta_a^* x_5^*}{\kappa^2}, \quad \frac{\partial^2 f_6}{\partial x_9\partial\beta_a} = -\frac{Ax_5^*}{\kappa}$ (32)

It follows from the above expressions (after several algebraic manipulations) that

$a = -\frac{2Av_9w_9}{\kappa\mu T_1T_2^2T_3^2T_4T_5^2T_6^2T_7}[\beta_c^*\mu T_5T_6T_7(\eta\pi_a(T_3\gamma_a + T_5\gamma_c) + T_5T_6\gamma_c\pi_c) +$
$\beta_a T_2T_3\gamma_a\pi_a w_9(x_5^*\mu T_1T_2T_3T_4T_5T_6T_7 + A\beta_c^* x_1^*(\eta\mu(T_5T_6\gamma_c\alpha_c + T_4T_5T_7\gamma_c +$
$T_4T_5\gamma_c\alpha_a + T_3T_4\gamma_a\alpha_a + T_3T_4T_7\gamma_a + T_3T_4T_6T_7) + T_4T_7\eta(T_5T_6\gamma_c\delta_c + T_1T_5\gamma_c\delta_a$
$+T_1T_3\gamma_a\delta_a) - T_5T_6T_7\gamma_c\alpha_c\mu\phi_c) + A\beta_a x_5^* T_1T_2T_3T_4(T_6T_7\mu + T_7\gamma_a(\delta_a+\mu) + \gamma_a\alpha_a\mu)]$ (33)

Hence if $\phi_c = 0$ it implies that $a < 0$. Also, for the computation of b, we have that,

$b = \frac{Aw_9v_9\gamma_a\pi_a x_5^*}{\kappa T_5T_6} > 0.$ (34)

3.3.2 Non-existence of backward bifurcation

Consider the special case of the transformed system 2 with the assumption that recovered children are immune for life after recovery (ie $\phi_c = 0$). Then the backward bifurcation coefficient, a, given by (33) reduces to:

$$a = -\frac{2Av_9w_9}{\kappa\mu T_1T_2^2T_3^2T_4T_5^2T_6^2T_7}\left[\beta_c^*\mu T_5T_6T_7(\eta\pi_a(T_3\gamma_a + T_5\gamma_c) + T_5T_6\gamma_c\pi_c) + \beta_aT_2T_3\gamma_a\pi_aw_9(x_5^*\mu T_1T_2T_3T_4T_5T_6T_7 + A\beta_c^*x_1^*(\eta\mu(T_5T_6\gamma_c\alpha_c + T_4T_5T_7\gamma_c + T_4T_5\gamma_c\alpha_a + T_3T_4\gamma_a\alpha_a + T_3T_4T_7\gamma_a + T_3T_4T_6T_7) + T_4T_7\eta(T_5T_6\gamma_c\delta_c + T_1T_5\gamma_c\delta_a + T_1T_3\gamma_a\delta_a)) + A\beta_ax_5^*T_1T_2T_3T_4(T_6T_7\mu + T_7\gamma_a(\delta_a + \mu) + \gamma_a\alpha_a\mu)\right] < 0. \quad (35)$$

Hence, this study has confirmed that the waning of treatment in children will cause a backward bifurcation in the transmission dynamics of hookworm.

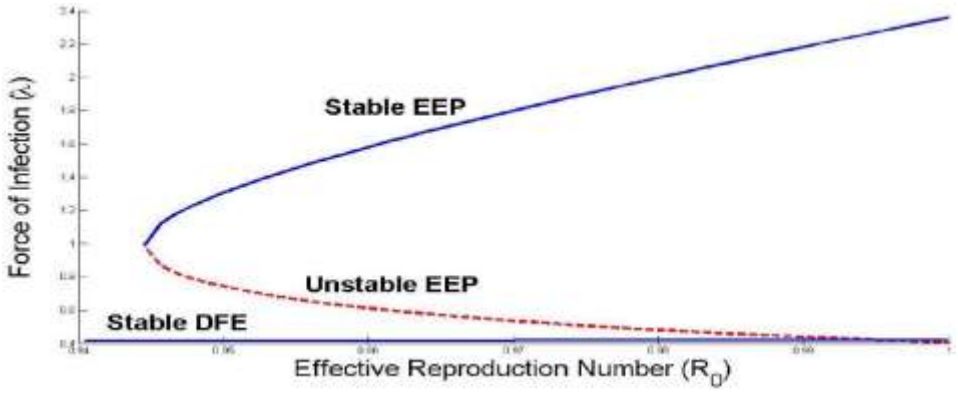

Fig. 7: Backward bifurcation diagram for the hookworm model (2), showing force of infection against the reproduction number $\mathcal{R}_0$.

3.4 Global Asymptotic Stability of the DFE

Theorem 4 The DFE of the model (2), is GAS in $\mathcal{D}$ if $\mathcal{R}_0 \leq 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof:

Consider the following Lyapunov function

$$\mathcal{T} = A_1E_c + A_2I_c + A_3E_a + A_4I_a + A_5V, \quad (36)$$

Where

$$A_1 = \frac{T_3\eta\gamma_a\pi_a + T_5\eta\gamma_c\pi_a + T_5T_6\gamma_c\pi_c}{\chi T_2T_3T_5T_6},\ A_2 = \frac{\eta\pi_a + T_6\pi_c}{\chi T_3T_6},\ A_3 = \frac{\gamma_a\pi_a}{\chi T_5T_6},\ A_4 = \frac{\pi_a}{\chi T_6},\ A_5 = \frac{1}{\chi}. \quad (37)$$

with Lyapunov derivatives (where a dot represents a time derivative)

$$\dot{\mathcal{T}} = A_1\dot{E}_c + A_2\dot{I}_c + A_3\dot{E}_a + A_4\dot{I}_a + A_5\dot{V}. \quad (38)$$

Substituting the right-hand side of system (2) into (38) and simplifying gives

$$\begin{aligned}
\dot{\mathcal{T}} = & A_1\lambda_1 S_c + A_3\lambda_2 S_a - [A_1T_2 - A_2\gamma_c - A_3\eta]E_c - [A_3T_5 - A_4\gamma_a]E_a \\
& -[A_2T_3 - A_4\eta + A_5\pi_c]I_c - [A_4T_6 - A_5\pi_a]I_a - A_5\chi V, \\
= & \frac{T_3\eta\gamma_a\pi_a + T_5\eta\gamma_c\pi_a + T_5T_6\gamma_c\pi_c}{\chi T_2T_3T_5T_6}\lambda_1 S_c + \frac{\gamma_a\pi_a}{\chi T_5T_6}\lambda_2 S_a - \left[\frac{\eta\gamma_a\pi_a}{\chi T_5T_6} + \frac{\eta\gamma_c\pi_a}{\chi T_3T_6} + \right. \\
& \left. \frac{\gamma_c\pi_c}{\chi T_3} - \frac{\eta\gamma_c\pi_a}{\chi T_3T_6} - \frac{\gamma_c\pi_c}{\chi T_3} - \frac{\eta\gamma_a\pi_a}{\chi T_5T_6}\right]E_c - \left[\frac{\gamma_a\pi_a}{\chi T_6} - \frac{\gamma_a\pi_a}{\chi T_6}\right]E_a - \left[\frac{\eta\pi_a}{\chi T_6} + \frac{\pi_c}{\chi} - \frac{\eta\pi_a}{\chi T_6}\right. \\
& \left. - \frac{\pi_c}{\chi}\right]I_c - \left[\frac{\pi_a}{\chi} - \frac{\pi_a}{\chi}\right]I_a - V, \\
= & \frac{(T_3\eta\gamma_a\pi_a + T_5\eta\gamma_c\pi_a + T_5T_6\gamma_c\pi_c)A\beta_c V}{\chi T_2T_3T_5T_6(\kappa+V)}S_c + \frac{\gamma_a\pi_a A\beta_a V}{\chi T_5T_6(\kappa+V)}S_a - V \\
= & V\left[\frac{A}{\chi T_2T_3T_5T_6(\kappa+V)}(\beta_c S_c(T_3\eta\gamma_a\pi_a + T_5\eta\gamma_c\pi_a + T_5T_6\gamma_c\pi_c) + \beta_a S_a T_2T_3\gamma_a\pi_a) - 1\right] \\
\leq & V\left[\frac{A}{\chi T_2T_3T_5T_6\kappa}(\beta_c S_c(T_3\eta\gamma_a\pi_a + T_5\eta\gamma_c\pi_a + T_5T_6\gamma_c\pi_c) + \beta_a S_a T_2T_3\gamma_a\pi_a) - 1\right] \\
\leq & V[\mathcal{R}_0 - 1]
\end{aligned} \tag{39}$$

Hence, $\dot{\mathcal{T}} \leq 0$ whenever $\mathcal{R}_0 \leq 1$ with $\dot{\mathcal{T}} = 0$ if and only if $V = 0$. Hence, $\mathcal{T}$ is a Lyapunov function in $\mathcal{D}$. Thus, it follows from [30] that:

$$(E_c(t), E_a(t), I_c(t), I_a(t), R_c(t), R_a(t), V(t)) \to (0,0,0,0,0,0,0) \text{ as } t \to \infty. \tag{40}$$

Hence, every orbit of the equations of the model (??), approaches the DFE of the model (??), as $t \to \infty$ for $\mathcal{R}_0 \leq 1$.

The epidemiological Implication of the result shows that in an age structured population, if $\phi_c = 0$, the DFE will be GAS whenever $\mathcal{R}_0 \leq 1$ hence, Hookworm will be eliminated from the population irrespective of the initial sizes of the sub-population whenever $\mathcal{R}_0 \leq 1$.

Consider the special case of the model (2) (with $\phi_c = \sigma_c = \sigma_a = 0$). Furthermore, let the stable manifold of the DFE of the model (2) (with $\phi_c = \sigma_c = \sigma_a = 0$) be

$$\mathcal{D}_0 = \{(S_c, E_c, I_c, R_c, S_a, E_a, I_a, R_a, V) \in \mathcal{D}: E_c = I_c = E_a = I_a = V = 0\}. \tag{41}$$

We claim the following result.

Theorem 5 The EEP, $\mathcal{E}_1$ (with $\phi_c = \sigma_c = \sigma_a = 0$) is GAS in $\mathcal{D} \setminus \mathcal{D}_0$ whenever $\mathcal{R}_0 > 1$.

Proof:

Consider also, the following non-linear Lyapunov function

$$\begin{aligned}
\mathcal{Q} = & S_c - S_c^{**}\ln\left(\frac{S_c}{S_c^{**}}\right) + E_c - E_c^{**}\ln\left(\frac{E_c}{E_c^{**}}\right) + B_1\left(I_c - I_H^{**}\ln\frac{I_c}{I_c^{**}}\right) + S_a - S_a^{**}\ln\left(\frac{S_a}{S_a^{**}}\right) + E_a - E_a^{**}\ln\left(\frac{E_a}{E_a^{**}}\right) \\
& + B_2\left(I_a - I_a^{**}\ln\frac{I_a}{I_a^{**}}\right) + \left(V - V^{**}\ln\frac{V}{V^{**}}\right)
\end{aligned} \tag{42}$$

where: $B_1 = \frac{T_1}{\gamma_c}, B_2 = \frac{T_5}{\gamma_a}$.

$\mathcal{Q}$ has Lyapunov derivatives, given as:

$$\dot{Q} = \left(1-\frac{S_c^{**}}{S_c}\right)\dot{S}_H + \left(1-\frac{E_c^{**}}{E_c}\right)\dot{E}_c + B_1\left(1-\frac{I_c^{**}}{I_c}\right)\dot{I}_c + \left(1-\frac{S_a^{**}}{S_a}\right)\dot{S}_a + \left(1-\frac{E_a^{**}}{E_a}\right)\dot{E}_a + B_2\left(1-\frac{I_a^{**}}{I_a}\right)\dot{I}_a + \left(1-\frac{V}{V^{**}}\right)\dot{V}. \quad (43)$$

Substituting the right-hand sides of the equations in model (2) corresponding to $\dot{S}_c, \dot{E}_c, \dot{I}_c, \dot{S}_a, \dot{E}_a, \dot{I}_a, \dot{V}$ into (43), after several algebraic calculations gives:

$$\dot{Q} \leq T_1 S_c^{**}\left(2-\frac{S_c^{**}}{S_c}-\frac{S_c}{S_c^{**}}\right)+\mu S_a^{**}\left(2-\frac{S_a^{**}}{S_a}-\frac{S_a}{S_a^{**}}\right)+\bar{\beta}_c V^{**}S_c^{**}\left(4-\frac{S_c^{**}}{S_c}-\frac{E_c^{**}}{E_c}-\frac{I_c^{*}*E_c}{I_c E_c^{**}}-\frac{I_c}{I_c^{**}}\right)$$

$$+\bar{\beta}_a V^{**}S_a^{**}\left(4-\frac{S_a^{**}}{S_a}-\frac{E_a^{**}}{E_a}-\frac{I_a^{*}*E_a}{I_a E_a^{**}}-\frac{I_a}{I_a^{**}}\right)+\eta E_c^{**}\left(3-\frac{E_a^{**}}{E_a}-\frac{I_a^{**}E_a}{I_a E_a^{**}}-\frac{I_a}{I_a^{**}}\right)+\frac{\eta^2 E_c^{**}I_c^{**}}{\gamma_a E_a^{**}}\left(2-\frac{I_a^{**}}{I_a}-\frac{I_A}{I_a^{**}}\right)$$

$$+\frac{\eta I_c^{**}\bar{\beta}_a V^{**}S_a^{**}}{\gamma_a E_a^{**}}\left(2-\frac{I_a^{**}}{I_a}-\frac{I_A}{I_a^{**}}\right) \quad (44)$$

Since the arithmetic mean exceeds the geometric mean, the following inequalities holds

$$2-\frac{S_c^{**}}{S_c}-\frac{S_c}{S_c^{**}} \leq 0, 2-\frac{S_a^{**}}{S_a}-\frac{S_a}{S_a^{**}} \leq 0,$$
$$2-\frac{I_a^{**}}{I_a}-\frac{I_a}{I_a^{**}} \leq 0, 3-\frac{E_a^{**}}{E_a}-\frac{E_a I_a{**}}{E_a^{*} I_a}-\frac{I_a}{I_a^{**}} \leq 0,$$
$$4-\frac{S_c^{**}}{S_c}-\frac{E_c^{**}}{E_c}-\frac{I_c^{*}*E_c}{I_c E_c^{**}}-\frac{I_c}{I_c^{**}} \leq 0, 4-\frac{S_a^{**}}{S_a}-\frac{E_a^{**}}{E_a}-\frac{I_a^{*}*E_a}{I_a E_a^{**}}-\frac{I_a}{I_a^{**}} \quad (45)$$

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

Since the relevant variables in the equations for R_c and R_a are at the endemic steady state, these can be substituted into the equations for R_c and R_a in the model (2) (with $\phi_c = \sigma_c = \sigma_a = 0$), so that $R_c(t) \to R_c^{**}$ and $R_a(t) \to R_a^{**}$ as $t \to \infty$. (46)

Hence, Q is a Lyapunov function in $\mathcal{D} \setminus \mathcal{D}_0$.

The epidemiological Implication of this result shows that in an age structured population, if $\phi_c = \sigma_c = \sigma_a = 0$, the EEP will be GAS whenever $\mathcal{R}_0 > 1$ and hence, Hookworm will persist in the population regardless of the initial sizes of the sub-population whenever $\mathcal{R}_0 > 1$.

4 Conclusion

The formulated model has been rigorously analysed and it was found to be bounded, mathematically and epidemiologically well posed. Also, the Human sub-population is shown to be $N_h \leq \frac{\Lambda}{\mu}$ and the Aquatic environment is shown to be $V \leq \frac{\Lambda(\pi_c+\pi_a)}{\mu\chi}$. The computed Reproduction Number, $\mathcal{R}_0$ shows that the disease will die out in this Age-Structured Hookworm model whenever $\mathcal{R}_0 < 1$ and will be endemic whenever $\mathcal{R}_0 > 1$. Analysing the $\mathcal{R}_0$ shows that increasing the treatment rates in both children (α_c) and adult (α_a) subpopulations will have a positive impact in the control of hookworm. However, increasing the treatment rate (α_a) in the adult subpopulation will only impact positively in the control of hookworm in the population if $\triangle_1 = \beta_a\gamma_a T_2 T_3 > \triangle_2 = \mu\beta_c(\mu\gamma_c + \gamma_a(\eta+\mu+\alpha_c+\gamma_c+\delta_c))$. Also, making the control available and ensuring that the controls are effective $(\phi\xi)$ in the population will have a positive impact in the control of hookworm. It is shown that the Age-Structured Hookworm model exhibited a backward bifurcation phenomenon, which is induced by the waning of treatment (ϕ_c) in the children sub-population: this suggests that the treatment should be repeated as the children become adults. The Disease-Free Equilibrium was shown to be Stable globally and the Endemic Equilibrium was shown to stable locally and globally for a special case when the waning of treatment in children and loss of immunity within each age group is negligible $(\phi_c = \sigma_c = \sigma_a = 0)$.

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