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RESEARCH ARTICLE

Uncovering the significance of undone animals' flesh and early treatment on the dynamics of ascariasis using mathematical analysis

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Abstract

Ascariasis is a type of roundworm infection that occurs in the intestines. It is contracted through contact with worm eggs found in tainted food, soil, or water. This work considers both the human and animal populations. The human population has been subdivided into five compartments, including susceptible humans, exposed humans, lightly infected humans, heavily infected humans, and recovered humans. The animal population has been subdivided into three groups: susceptible animals, exposed animals, and infectious animals. The formulated models have been analyzed. To begin the analysis, we verified the existence and uniqueness of the proposed model, followed by the boundedness and positivity of the solutions. Next, we calculated the effective and basic reproduction numbers, and also verified the local stability of the disease-free equilibrium using the Jacobian matrix. This analysis shows that the disease-free equilibrium is locally asymptotically stable if the effective reproduction number is less than unity. The comparison approach has been used to study the global asymptotic stability of the disease-free equilibrium. It demonstrates that if the effective reproduction number is smaller than unity, the disease-free equilibrium is globally asymptotically stable. The Lyapunov function of the Go-Volterra type has been used to study the global asymptotic stability of the endemic equilibrium. It demonstrates that if the effective reproduction number is more than unity, the endemic equilibrium point is globally asymptotically stable. Numerical simulation shows that early treatment plays a vital role in controlling the dynamics of Ascariasis, as well. It has been observed that consuming raw animal flesh can increase or exacerbate diseases in society. This means that reducing the consumption of raw animal flesh can help control the Ascariasis disease in society.

Keywords: Ascariasis, animal flesh, early treatment, mathematical modelling, significance

1. Introduction

Ascariasis is a common helminthic infection caused by the parasitic roundworm Ascaris lumbricoides. It primarily affects the human intestines, with transmission occurring through ingestion

of embryonated eggs in contaminated food or water. After ingestion, the larvae hatch, migrate through the lungs, and mature in the small intestine, where adult worms can reach up to 30 cm in length [1]. Ascariasis has been documented since ancient times. Descriptions of roundworms resembling Ascaris lumbricoides were found in Egyptian medical papyri dating back to 1500 BCE [2]. Hippocrates also mentioned intestinal worms, and Aristotle provided detailed morphological descriptions of them. During the early stage of infection, ingested eggs hatch into larvae that migrate through the lungs. This can cause respiratory symptoms, typically occurring 4–16 days after exposure. Patients may experience: Dry cough, shortness of breath, wheezing, fever, and chest discomfort. These manifestations are often associated with an immune response known as Löffler's syndrome, which includes transient pulmonary infiltrates and eosinophilia [3]. Once the larvae mature into adult worms and settle in the small intestine, the following symptoms may arise: Abdominal pain or discomfort, nausea or vomiting, diarrhoea or constipation, anorexia (loss of appetite), and visible worms in stool or vomit. Severe cases may lead to malnutrition and weight loss, especially in children, due to poor nutrient absorption [4, 5]. Heavy worm burdens can lead to serious complications, such as: Intestinal obstruction, particularly in children, Biliary or pancreatic duct blockage, Appendicitis, Peritonitis due to intestinal perforation. These complications require urgent medical attention and are more common in endemic regions with poor sanitation [6]. In pediatric populations, chronic Ascariasis can contribute to: Growth retardation, Cognitive delays, Anaemia. These effects stem primarily from malnutrition and repeated infections [6, 7].

The scientific classification of the parasite was formalized in the 18th century, and by the 20th century, the life cycle of Alumbricoides had been fully elucidated [3]. Ascariasis is one of the most prevalent neglected tropical diseases (NTDs), especially in regions with inadequate sanitation. An estimated 807 million to 1.2 billion people are infected globally, particularly in sub-Saharan Africa, Latin America, and Southeast Asia [8]. Although many infections are asymptomatic, heavy infestations can cause malnutrition, impaired cognitive development, intestinal obstruction, and respiratory complications during larval migration [9]. In children, chronic infections contribute to stunted growth and poor school performance. While mortality from Ascariasis is relatively low compared to its high prevalence, severe complications such as intestinal blockage and biliary or pancreatic Ascariasis can be fatal if untreated. It is estimated that Ascariasis causes around 60,000 deaths annually, predominantly in children from low-income countries [6]. Preventive chemotherapy and improvements in hygiene and sanitation are critical to reducing morbidity and mortality rates.

In so many decades ago, researchers have worked on the mathematical modelling of Ascariasis. Authors like [10] formulated a mathematical model that considers transmission dynamics of Ascaris lumbricoides – theory and observation, they discuss how it was easier in the age of easier access to medications and new statistical methods to verify these models against reinfection data, present and future models are influencing the design of control strategies. Ref. [11] note that worms coming from pig hosts and those originating from human hosts showed a significant degree of genetic segregation. However, pigs were the source of human Ascaris infections in Europe, and there was proof of human-pig cross-transmission in Africa. There is notable genetic variation across parasite populations from various nations, communities, and hosts. Ref. [12] formulated a mathematical model using the method, which entails establishing a Stoichiometric matrix (S) for the biochemical reactions that take place during the process and computing basis vectors for the null space of S that have biochemical significance. The stoichiometric matrix S in their analysis was of order 26 x 40, and the "basis" for it consisted of 14 column vectors. They discovered that two-column vectors indicate the network's stoichiometry, whereas twelve-column vectors depict the reversible processes involved in the metabolic route.

Ref. [13] specifically studied the dynamics of Ascariasis to determine the typical negative binomial distribution that was commonly observed in human societies. They took into consideration the Anderson–May model of human parasite infections, particularly that for the intestinal worm Ascaris lumbricoides.

Considering all the aforementioned research, especially Ref. [11] discussed how pigs contributed to the spread of Ascariasis, but pig is not the only animal that can transmit Ascaris to humans; other animals transmit too. The researchers did not consider these important points; also, the researchers did not put early treatment and unwell-prepared animal flesh in their studies. In this work, a new mathematical model has been formulated to assess the impact of unprepared animal flesh, early treatment, and personal hygiene.

This is the structure of the research. Section 1 contains the introduction, pertinent literature, and organizational framework of the work; Section 2 contains the model formulation; Section 3 contains the model analysis; Section 4 contains the numerical results and simulation; Section 5 discusses the simulated results; and Section 6 includes the conclusion.

2. Model Description

The susceptible animal compartment is generated at a rate of recruitment λ_a. The entire population of this compartment are at risk of acquiring ascariasis through mingling at a rate $\lambda_a = \frac{\beta_a(I_2+\eta I_1)}{N_h}$, where β_a is the animals' effective contact rate and η is the modification parameter because light-infectious persons are less contagious. The exposed animal compartments are generated through the mingling of animals with the faeces of humans who are infected with light and heavy infectious individuals at the λ_a, as explained above. This class progresses to infectious animals at the rate σ_a. The infectious animals can be generated at the rate σ_a, due to progression. All the animal compartments decrease due to the natural death rate μ_a.

The schematic diagram of Ascariasis, as defined by the assumptions mentioned earlier, is shown in Figure 1, and the relevant model equations obtained from Figure 1 are presented in (1). The variables and parameters are interpreted in Table 1.

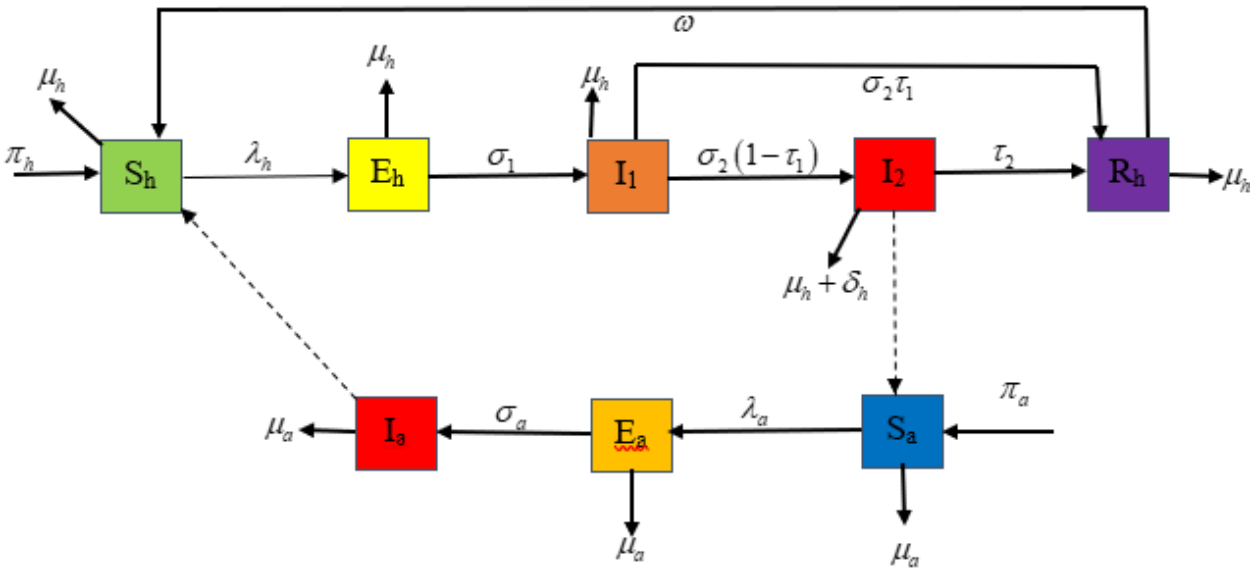


Figure 1. Schematized diagram of Ascariasis

$$
\begin{aligned}
\frac{dS_h}{dt} &= \pi_h + \omega R_h - \lambda_h S_h - \mu_h S_h,\\
\frac{dE_h}{dt} &= \lambda_h S_h - (\sigma_1 + \mu_h) E_h,\\
\frac{dI_1}{dt} &= \sigma_1 E_h - (\mu_h + \sigma_2) I_1,\\
\frac{dI_2}{dt} &= \sigma_2 (1-\tau_1) I_1 - (\mu_h + \delta_h + \tau_2) I_2,\\
\frac{dR_h}{dt} &= \tau_2 I_2 + \sigma_2 \tau_1 I_1 - (\mu_h + \omega) R_h,\\
\frac{dS_a}{dt} &= \pi_a - \lambda_a S_a - \mu_a S_a,\\
\frac{dE_a}{dt} &= \lambda_a S_a - (\sigma_a + \mu_a) E_a,\\
\frac{dI_a}{dt} &= \sigma_a E_a - \mu_a I_a.
\end{aligned} \tag{1}
$$

where

$$
\begin{aligned}
\lambda_h &= \frac{\beta_h q (1-\epsilon)(1-\theta) I_a}{N_a},\\
\lambda_a &= \frac{\beta_a (I_2 + \eta I_1)}{N_h}.
\end{aligned} \tag{2}
$$

Subject to the initial condition;

$$
\begin{aligned}
&S_h(0) > 0, \quad E_h(0) \geq 0, \quad I_1(0) \geq 0, \quad I_2(0) \geq 0, \quad R_h(0) \geq 0,\\
&S_a(0) \geq 0, \quad E_a(0) \geq 0, \quad I_a(0) \geq 0.
\end{aligned}
$$

Table 1. Interpretation of the model's parameters and state variables (1).

Variable	Description
N_h, N_a	Total number of people and animals
S_a, S_h	Animals and people who are susceptible
E_a, E_h	Animals and people who are exposed
I_1, I_2	Light infected humans and heavy infectious humans
I_a	infectious animals
R_h	Recovered humans
Parameter	
π_h, π_a	Recruitment rate of humans and animals
ω	Waning rate of recovered humans
μ_h, μ_a	Natural mortality rate of humans and animals
σ_1, σ_2	Progression rates from E_h to I_1 and from I_1 to I_2
τ_1, τ_2	Early treatment of I_1 and treatment rate of I_2
σ_a	Progression rates from E_a to I_a
β_h	Effective consumption rate of humans
β_a	Effective consumption rate of animals
q	Quantity of not done animal flesh consumed
$(1-\theta)$	Rate of contaminated water
δ_h	Rate of death of humans from disease
η	Modification parameter due to reduced infectiousness of humans
ϵ	Personal hygiene
λ_h, λ_a	Force of infection of humans and animals

3. Fundamental characteristics of the model

The computation of the basic reproduction number, positivity and boundedness of solutions, and all of the classical analysis of model (1) are covered in this section.

3.1. Boundaries and Solutions' Positivity

This paragraph showed that the system (1) is bounded and positive. Let $\mathcal{D}$ be an appropriate physiologically viable region for the model (1) defined by,

$$\mathcal{D} = \{(S_h, E_h, I_1, I_2, R_h, S_a, E_a, I_a) \in R_+^8 : N_h \leq \frac{\pi_h}{\mu_h}, N_a \leq \frac{\pi_a}{\mu_a}\}. \tag{3}$$

Theorem 1. *Let* $\{(S_h(0), E_h(0), I_1(0), I_2(0), R_h(0), S_a(0), E_a(0), I_a(0)) \geq 0\} \in \mathcal{D}$ *be the initial values of the model* (1)*.Thus, for each* $t > 0$*, the set of solutions* $\{S_h(t), E_h(t), I_1(t), I_2(t), R_h(t), S_a(t), E_a(t), I_a(t)\}$ *is positive for model* (1)*.*

Proof. The variables of the solution $(S_h(t), E_h(t), I_1(t), I_2(t), R_h(t), S_a(t), E_a(t), I_a(t))$ of model (1) must be demonstrated to be non-negative in order to correspond to initial conditions such that $S_h(0) > 0, E_h(0) > 0, I_1(0) > 0, I_1(0) > 0, I_2(0) > 0, R_h(0) > 0, S_a(0) > 0$, $E_a(0) > 0$, and $I_a(0) > 0$.
Let $t_1 = sup(t > 0 | \forall u \in [0, 1], S_h(u) > 0$, $E_h(u) > 0$, $I_1(u) > 0$, $I_2(u) > 0$, $R_h(u) > 0$, $S_a(u) > 0, E_a(u) > 0$, $I_a(u) > 0)$.
The existence of t_1 is guaranteed by the aforementioned beginning conditions appended to the continuity of all functions $S_h, E_h, I_1, I_2, R_h, S_a, E_a, I_a$. All of the model (1) solutions are positive if $t_1 = \infty$. There is one or more solution $S_h(t), E_h(t), I_1(t), I_2(t), R_h(t), S_a(t), E_a(t)$ or $I_a(t)$ which are equals to zero at t_1 value, if $t_1 < \infty$, i.e. $t_1 is finite$. By definition, (since$t_1$is a supremum).
Let us assume that $S(t_1) = 0$. Now, let's examine the model's initial equation (1).

$$\frac{dS_h(t)}{dt} = \pi_h + \omega R_h(t) - (\lambda_h + \mu_h) S_h(t), \tag{4}$$

for every $t \in [0, t_1], weknow$, $\omega R_h(t) \geq 0 + \pi_h$. Thus, assuming that $S(t_1) = 0$, let's look at the model's initial equation.

$$\frac{dS_h(t)}{dt} + (\lambda_h + \mu_h) S_h(t) \geq 0, \tag{5}$$

consequently

$$\begin{aligned} &\frac{d}{dt}\left[S_h(t) exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right)\right] \\ &= \frac{dS_h(t)}{dt} exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right) \\ &+ S_h(t)\left(\lambda_h(t) + \mu_h\right) exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right) \\ &exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right) \times \left(\frac{dS_h(t)}{dt} + (\lambda_h + \mu_h) S_h(t)\right) \geq 0, \end{aligned} \tag{6}$$

that is

$$\frac{d}{dt}\left[S_h(t) exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right)\right] \geq 0. \tag{7}$$

When the inequality above is integrated from zero 0 to t_1, it yields

$$\int_0^{t_1} \left[S_h(t) exp\left(\int_0^t \lambda_h(\tau)d\tau + \mu_h t\right)\right] \geq 0, \tag{8}$$

or equivalently

$$S_h(t_1) exp\left(\int_0^{t_1} \lambda_h(\tau)d\tau + \mu_h t_1\right) - S_h(0) \geq 0, \tag{9}$$

this results in

$$S_h(t_1) \geq exp\left(-\mu_h t_1 - \int_0^{t_1} \lambda_h(\tau)d\tau\right) S_h(0) > 0, \tag{10}$$

which go counter to the assertion that $S_h(t_1) = 0$. $E_h(t_1) = 0, I_1(t_1) = 0, I_2(t_1) = 0, R_h(t_1) = 0$, and $S_a(t_1) = 0$ are the other situations. $I_a(t_1) = 0$, $E_a(t_1)$, and produces the same inconsistency.

Therefore, for all $t > 0$, $S_h(t) > 0, E_h(t) > 0, I_1(t) > 0, I_2(t) > 0, R_h(t) > 0, S_a(t) > 0$, $E_a(t) > 0$, and $I_a(t) > 0$. □

Theorem 2. *Let the feasible region* Ω *for system* (1) *be defined as*

$$\mathcal{D} = \left\{ (S_h(t), E_h(t), I_1(t), I_2(t), R_h(t) \in \mathbb{R}_+^5 : N_h(t) \leq \frac{\pi_h}{\mu_h}, (S_a(t), E_a(t)), I_a(t)) \in \mathbb{R}_+^3 : N_a(t) \leq \frac{\pi_a}{\mu_a} \right\}. \tag{11}$$

Then, Ω *is positively invariant under the dynamics of system* (1).

Proof. We must demonstrate that the entire human population at time t, $N(t)$, satisfies the boundedness property. In the case where $0 \leq N_h(0) \leq \frac{\pi_h}{\mu_h}$ and $0 \leq N_a(0) \leq \frac{\pi_a}{\mu_a}$, $0 \leq N_h(t) \leq \frac{\pi_h}{\mu_h}$ and $0 \leq N_a(t) \leq \frac{\pi_a}{\mu_a}$. We note that in the ideal scenario, where there are no active instances of Ascariasis, this bound represents the unique equilibrium of the entire population's dynamics. The conservation law Yaleu *et al.,* (2017) is obtained by inserting the model's equation (1). Our main goal was to demonstrate that any solution in $\mathcal{D}$ stays in $\mathcal{D}$. Consequently, the population's overall change rate of humans is determined by

$$\frac{dN_h}{dt} = \pi_h - \mu_h N_h - \delta_1 A_p - \delta_h I_2 \leq \pi_h - \mu_h N_h, \tag{12}$$

equation (12) may be solved using the Groomwall inequality to obtain

$$N_h(t) \leq \frac{\pi_h}{\mu_h} + \left[N_h(0) - \frac{\pi_h}{\mu_h} \right] e^{-\mu_h t}, \forall t \geq 0, \tag{13}$$

which implies that $0 \leq N_h(t) \leq \frac{\pi_h}{\mu_h}$ whenever $0 \leq N_h(0) \leq \frac{\pi_h}{\mu_h}$ $\forall t \geq 0$ in another words, from (13), as $t \to 0$, $N(t) \to N(0) \Rightarrow N_h(t) \leq \frac{\pi_h}{\mu_h}$, and as $t \to \infty$ $N_h(t) \to \frac{\pi_h}{\mu_h}$. Therefore, $\mathcal{D}$ is positively invariant and a global attractor of all positive solutions of the system (1) for all positive initial conditions. Consequently, the population's overall change rate of animals is determined by

$$\frac{dN_a}{dt} = \pi_a - \mu_a N_a = \pi_a - \mu_a N_a. \tag{14}$$

Equation (14) may be solved using the Groomwall inequality to obtain

$$N_a(t) \leq \frac{\pi_a}{\mu_a} + \left[N_a(0) - \frac{\pi_a}{\mu_a} \right] e^{-\mu_a t}, \forall t \geq 0, \tag{15}$$

which implies that $0 \leq N_a(t) \leq \frac{\pi_a}{\mu_a}$ whenever $0 \leq N_a(0) \leq \frac{\pi_a}{\mu_a}$ $\forall t \geq 0$ in another words, from (15), as $t \to 0$, $N(t) \to N(0) \Rightarrow Na - N_a(t) \leq \frac{\pi_a}{\mu_a}$, and as $t \to \infty$ $N_a(t) \to \frac{\pi_a}{\mu_a}$. Therefore, $\mathcal{D}$ is positively invariant and a global attractor of all positive solutions of the system (1) for all positive initial conditions. □

3.2. Existence of solutions and their uniqueness

The existence and uniqueness of the model (1)'s solution will be tested using the surety test. If the solution is found to exist, it must be demonstrated to be unique. It is determined using the Lipschitz criteria in this way:

$$
\begin{aligned}
v_1 &= \pi_h + \omega R_h - \left(\frac{\beta_h q(1-\epsilon)(1-\theta)I_a}{N_a} + \mu_h\right) S_h, \\
v_2 &= \frac{\beta_h q(1-\epsilon)(1-\theta)I_a}{N_a} S_h - (\sigma_1 + \mu) E_h, \\
v_3 &= \sigma_h E_h S - (\sigma_2 + \mu_h) I_1, \\
v_4 &= \sigma_2(1-\tau_1) I_1 - (\mu_h + \delta_h + \tau_2) I_2, \\
v_5 &= \tau_2 I_2 + \sigma_2 \tau_1 I_1 - (\mu_h + \omega) R_h, \\
v_6 &= \pi_a - (\frac{\beta_a(I_2 + \eta I_1}{N_h} + \mu_a) S_a, \\
v_7 &= \frac{\beta_a(I_2 + \eta I_1}{N_h} S_a - (\sigma_a + \mu_a) E_a, \\
v_8 &= \sigma_a E_a - \mu_a I_a.
\end{aligned} \tag{16}
$$

In the area $\mathcal{D}$ where its partial derivative is inside $\delta_t \in \mathcal{D}$, where δ_t is a constant greater than zero, we will look at the boundedness of the model (1)'s solution.

Theorem 3. *In the region $\mathcal{D}$, the model* (1) *has a unique solution, given that,*

$$
\frac{dv_i}{dy_j}, i, j = 1, 2, ..., 8, : y_1 = S_h, y_2 = E_h, y_3 = I_1, \ldots, y_8 = I_a \tag{17}
$$

it is bounded and continuous in $\mathcal{D}$.

Proof. For each state variable in (1), we partially differentiate v_1 to get

$$
\begin{aligned}
\left|\frac{\partial v_1}{\partial S_h}\right| &= \left|-\left(\frac{\beta_h q(1-\epsilon)(1-\theta)I_a}{N_a} + \mu_h\right)\right| < \infty, \\
\left|\frac{\partial v_1}{\partial E_h}\right| &= \left|\frac{\partial v_1}{\partial I_1}\right| = \left|\frac{\partial v_1}{\partial I_2}\right| = 0 < \infty, \\
\left|\frac{\partial v_1}{\partial R_h}\right| &= |\omega| < \infty, \\
\left|\frac{\partial v_1}{\partial S_a}\right| &= \left|\frac{\partial v_1}{\partial E_a}\right| = \left|\frac{\partial v_1}{\partial I_a}\right| = 0 < \infty,
\end{aligned} \tag{18}
$$

the same is true for $v_2, v_3, v_4, v_5, v_6, v_7$, and v_8. As a result, model (1) exists in $\mathcal{D}$ and has a unique solution, as all of the partial derivatives are less than infinity. □

3.3. Disease-free equilibrium

The disease-free equilibrium (DFE) of system (1) is as given in (19).

$$
\Omega^0 = (S_h^0, E_h^0, I_1^0, I_2^0, R_h^0, S_a^0, E_a^0, I_a^0) = \left(\frac{\pi_h}{\mu_h}, 0, 0, 0, 0, \frac{\pi_a}{\mu_a}, 0, 0\right). \tag{19}
$$

3.4. Basic Reproduction Number

The effective reproduction number, denoted by $R_c = \rho(FV^{-1})$ in the model 1, where ρ represents the spectral radius of the next generation matrix FV^{-1}, is the number of new infections that Ascariasis-infected individuals cause when they interact with susceptible populations.

$$\mathcal{F} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_h q(1-\epsilon)(1-\theta)\, S_h}{N_a} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_a S_a}{N_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{20}$$

and

$$\mathcal{G} = \begin{bmatrix} k_1 & 0 & 0 & 0 & 0 \\ -\sigma_1 & k_2 & 0 & 0 & 0 \\ 0 & -\sigma_2\,(1-\tau_1) & k_3 & 0 & 0 \\ 0 & 0 & 0 & k_5 & 0 \\ 0 & 0 & 0 & -\sigma_a & \mu_a \end{bmatrix}. \tag{21}$$

where

$$k_1 = \sigma_1 + \mu_h, k_2 = \sigma_2 + \mu_h, k_3 = \delta_h + \mu_h + \tau_2, k_4 = \omega + \mu_h, k_5 = \sigma_a + \mu_a, \tag{22}$$

The inverse of (21) is given below

$$\mathcal{G}^{-1} = \begin{bmatrix} {k_1}^{-1} & 0 & 0 & 0 & 0 \\ \frac{\sigma_1}{k_2 k_1} & {k_2}^{-1} & 0 & 0 & 0 \\ -\frac{\sigma_2(-1+\tau_1)\sigma_1}{k_2 k_1 k_3} & -\frac{\sigma_2(-1+\tau_1)}{k_2 k_3} & {k_3}^{-1} & 0 & 0 \\ 0 & 0 & 0 & {k_5}^{-1} & 0 \\ 0 & 0 & 0 & \frac{\sigma_a}{k_5 \mu_a} & {\mu_a}^{-1} \end{bmatrix}, \tag{23}$$

then, the $\mathcal{F}\mathcal{G}^{-1}$ is given by

$$\mathcal{F}\mathcal{G}^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_h q(1-\epsilon)(1-\theta)\, S_h \sigma_a}{N_a k_5 \mu_a} & \frac{\beta_h q(1-\epsilon)(1-\theta)\, S_h}{N_a \mu_a} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ -\frac{\beta_a S_a \sigma_2(-1+\tau_1)\sigma_1}{N_h k_2 k_1 k_3} & -\frac{\beta_a S_a \sigma_2(-1+\tau_1)}{N_h k_2 k_3} & \frac{\beta_a S_a}{N_h k_3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{24}$$

Therefore, the eigenvalues are given by

$$\begin{bmatrix} 0 \\ 0 \\ 0 \\ \frac{\sqrt{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a \beta_a S_a^0 \sigma_2 \sigma_1 \beta_h q(1-\theta)\, S_h^0 \sigma_a (1-\epsilon-\tau_1+\tau_1\epsilon)}}{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a} \\ -\frac{\sqrt{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a \beta_a S_a^0 \sigma_2 \sigma_1 \beta_h q(1-\theta)\, S_h^0 \sigma_a (1-\epsilon-\tau_1+\tau_1\epsilon)}}{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a} \end{bmatrix}, \tag{25}$$

putting all the denominators of equation (25) under the roots, we obtain

$$\begin{bmatrix} 0 \\ 0 \\ 0 \\ \sqrt{\frac{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a \beta_a S_a^0 \sigma_2 \sigma_1 \beta_h q(1-\theta)\, S_h^0 \sigma_a (1-\epsilon-\tau_1+\tau_1\epsilon)}{\left(N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a\right)^2}} \\ -\sqrt{\frac{N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a \beta_a S_a^0 \sigma_2 \sigma_1 \beta_h q(1-\theta)\, S_h^0 \sigma_a (1-\epsilon-\tau_1+\tau_1\epsilon)}{\left(N_h^0 k_2 k_1 k_3 N_a^0 k_5 \mu_a\right)^2}} \end{bmatrix}, \tag{26}$$

at disease-free equilibrium $S_h^0 \leq N_h^0$ and $S_a^0 \leq N_a^0$, now, after simplification, the spectral radius can be given by

$$\rho(\mathcal{F}\mathcal{G}^{-1}) = \sqrt{\frac{\beta_a \sigma_1 \sigma_2 \beta_h q(1-\theta)\sigma_a(1-\epsilon)(1-\tau_1)}{k_1 k_2 k_3 k_5 \mu_a}}. \tag{27}$$

Therefore, the basic reproduction number $\mathcal{R}_c$ of system (1) is given by the spectral radius of the matrix $\mathcal{F}\mathcal{G}^{-1}$,

$$\mathcal{R}_c = \sqrt{\frac{\beta_a \sigma_1 \sigma_2 \beta_h q(1-\theta)\sigma_a(1-\epsilon)(1-\tau_1)}{k_1 k_2 k_3 k_5 \mu_a}}. \tag{28}$$

3.5. Stability of Disease-Free Equilibrium Globally

Theorem 4. *For the model* (1)*, the disease-free equilibrium (DFE) is globally-asymptotically stable (GAS) in* $\mathcal{D}$ *when* $\mathcal{R}_c < 1$*; when* $\mathcal{R}_c > 1$*, it is unstable.*

Proof. For the DFE's GAS to work, $\mathcal{R}_c < 1$ [14] must satisfy the two axioms $[G_1]$ and $[G_2]$. In the form, we rewrite the system.

$$\begin{aligned} \frac{dX_a}{dt} &= F(X_a, X_b), \\ \frac{dX_b}{dt} &= G(X_a, X_b) : G(X_a, 0) = 0, \end{aligned} \tag{29}$$

where $X_b = (E_h^0, I_1^0, I_2^0, E_a^0, I_a^0)$ and $X_a = (S_h^0, R_h^0, S_a^0)$, where the uninfected population is represented by the elements of $X_a \in R_+^3$ and the infected population is represented by the elements of $X_b \in R_+^5$.
The DFE's current symbol is $M^0 = (X_a^*, 0)$,
where,
$X_a^* = (N_h^0/N_a^0, 0)$. Now let's discuss the first scenario. X_a^*'s globally asymptotic stability yields

$$\frac{dX_a}{dt} = F(X_1, 0) = \begin{bmatrix} \pi_h + \omega R_h - \mu_h S_h^0 \\ -(\mu_h + \omega) R_h^0 \\ \pi_a - \mu_a S_a^0 \end{bmatrix}. \tag{30}$$

A linear ODE solving gives

$$\begin{aligned} \frac{\pi_h + \omega R_h}{\mu_h} - \frac{\pi_h + \omega R_h}{\mu_h} e^{-\mu_h t} + S_h^0(0) e^{-\mu_h t} &= S_h^0(t), \\ -\frac{1}{(\omega + \mu_h)} e^{-(\omega+\mu_h)t} + R_h^0(0) e^{-(\omega+\mu_h)t} &= R_h^0(t), \\ \frac{\pi_h}{\mu_a} - \frac{\pi_a}{\mu_a} e^{-\mu_a t} + S_a^0(0) e^{-\mu_a t} &= S_a^0(t). \end{aligned}$$

Now, clearly from system (1) we have, $S_h^0(t) + R_h^0(t) \to N_h^0(t)$ and $S_a^0(t) \to N_a^0(t)$ as $t \to \infty$ regardless of the value of $S_h^0(t), R_h^0(t)$, and $S_a^0(t)$. Thus , $X_a^* = (N_h^0/N_a^0, 0)$ is stable asymptotically on a global scale.

Afterwards, regarding the second requirement, which is $\tilde{G}(X_a, X_a) = AX_b - G(X_a, X_b) \geq 0$

$$A = \begin{pmatrix} -k_1 & 0 & 0 & 0 & \frac{\beta_h q(1-\epsilon)(1-\theta)S_h}{N_a} \\ \sigma_1 & -k_2 & 0 & 0 & 0 \\ 0 & \sigma_2(1-\tau_1) & 0 & 0 & 0 \\ 0 & \frac{\beta_a \eta S_a}{N_h} & \frac{\beta_a S_a}{N_h} & -k_5 & 0 \\ 0 & 0 & 0 & \sigma_a & -\mu_a \end{pmatrix}. \tag{31}$$

As the matrix A has non-negative off-diagonal components, it is a Metziller matrix.

$$G(X_a, X_b) = \begin{pmatrix} \frac{\beta_h q(1-\epsilon)(1-\theta)I_a S_h}{N_a} - k_1 E_h, \\ \sigma_1 E_h - k_2 I_1, \\ \sigma_2(1-\tau_1)I_1 - k_3 I_2, \\ \frac{\beta_a (I_2 + \eta I_1) S_a}{N_h} - k_5 E_a, \\ \sigma_a E_a - \mu_a I_a \end{pmatrix}. \tag{32}$$

Then,

$$\tilde{G}(X_a, X_a) = AX_b - G(X_a, X_b) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

That is

$$\tilde{G}(X_a, X_b) = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \end{bmatrix}^T.$$

Clearly, $\tilde{G}(X_a, X_b) = 0$. □

According to the epidemiological implications of the theorem 4, as long as the control reproduction number is less than one, the disease can be controlled in society. It is important to emphasize that one of the primary methods for reducing the number of reproductions is to control Ascariasis in society.

3.6. Endemic equilibrium point

Let

$$\Phi^{**} = (S_h^{**}, E_h^{**}, I_1^{**}, I_2^{**}, R_h^{**}, S_a^{**}, E_a^{**}, I_a^{**}) \tag{33}$$

be the endemic equilibrium, to achieve this, we let the right-hand side of equation (1) to zero and solve simultaneously at the endemic equilibrium, we have.

$$\begin{aligned}
S_h^{**} &= \frac{k_1 k_2 k_3 k_4 \pi_h}{(-\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \omega \sigma_1 + k_1 k_2 k_3 k_4) \lambda_h^{**} + k_3 k_4 k_2 k_1 \mu_h}, \\
E_h^{**} &= \frac{k_2 k_3 k_4 \pi_h \lambda_h^{**}}{(-\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \omega \sigma_1 + k_1 k_2 k_3 k_4) \lambda_h^{**} + k_3 k_4 k_2 k_1 \mu_h}, \\
I_1^{**} &= \frac{k_3 k_4 \sigma_1 \lambda_h^{**} \pi_h}{(-\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \omega \sigma_1 + k_1 k_2 k_3 k_4) \lambda_h^{**} + k_3 k_4 k_2 k_1 \mu_h}, \\
I_2^{**} &= \frac{\sigma_2 (1-\tau_1) k_4 \sigma_1 \pi_h \lambda_h^{**}}{(-\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \omega \sigma_1 + k_1 k_2 k_3 k_4) \lambda_h^{**} + k_3 k_4 k_2 k_1 \mu_h}, \\
R_h^{**} &= \frac{\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \sigma_1 \pi_h \lambda_h^{**}}{(-\sigma_2 (k_3\tau_1 + \tau_2 (1-\tau_1)) \omega \sigma_1 + k_1 k_2 k_3 k_4) \lambda_h^{**} + k_3 k_4 k_2 k_1 \mu_h}, \\
S_a^{**} &= \frac{\pi_a}{\lambda_a^{**} + \mu_a}, E_a^{**} = \frac{\pi_a \lambda_a^{**}}{k_5(\lambda_a^{**} + \mu_a)}, I_a^{**} = \frac{\sigma_a \pi_a \lambda_a^{**}}{\mu_a k_5 (\lambda_a^{**} + \mu_a)}
\end{aligned} \tag{34}$$

3.7. Stability of the Endemic Equilibrium Point Globally

Theorem 5. *In the model* (1)*, the endemic equilibrium (EE),* ϵ^**, is globally asymptotically stable (GAS) when* $\mathcal{R}_c > 1$ *and unstable when* $\mathcal{R}_c < 1$*.*

Proof. Let $\mathcal{V}$ be a Lyapunov function of the Goh-Volterra type, as shown below.

$$\begin{aligned}\mathcal{V} &= \left(S_h - S_h^{**} - S_h^{**} ln\frac{S_h}{S_h^{**}}\right) + \left(E_h - E_h^{**} - E_h^{**} ln\frac{E_h}{E_h^{**}}\right) + \frac{\sigma_1+\mu_h}{\sigma_1}\left(I_1 - I_1^{**} - I_1^{**} ln\frac{I_1}{I_1^{**}}\right)\\ &+ \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)}{\sigma_1\sigma_2}\left(I_2 - I_2^{**} - I_2^{**} ln\frac{I_2}{I_2^{**}}\right)\\ &+ \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)(\mu_h+\delta_h+\tau_2)}{\sigma_1\sigma_2\tau_2}\left(R_h - R_h^{**} - R_h^{**} ln\frac{R_h}{R_h^{**}}\right)\\ &+ \left(S_a - S_a^{**} - S_a^{**} ln\frac{S_a}{S_a^{**}}\right) + \left(E_a - E_a^{**} - E_a^{**} ln\frac{E_a}{E_a}\right) + \frac{(\mu_a+\sigma_a)}{\sigma_a}\left(I_a - I_a^{**} - I_a^{**} ln\frac{I_a}{I_a^{**}}\right),\end{aligned} \tag{35}$$

when we differentiate (35) in relation to time, we obtain

$$\begin{aligned}\dot{\mathcal{V}} &= \left(1 - \frac{S_h^{**}}{S_h}\right)\dot{S}_h + \left(1 - \frac{E_h^{**}}{E_h}\right)\dot{E}_h + \frac{\sigma_1+\mu_h}{\sigma_1}\left(1 - \frac{I_1^{**}}{I_1}\right)\dot{I}_1 + \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)}{\sigma_1\sigma_2}\left(1 - \frac{I_2^{**}}{I_2}\right)\dot{I}_2\\ &+ \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)(\mu_h+\delta_h+\tau_2)}{\sigma_1\sigma_2\tau_2}\left(1 - \frac{R_h^{**}}{R_h}\right)\dot{R}_h + \left(1 - \frac{S_a^{**}}{S_a}\right)\dot{S}_a + \left(1 - \frac{E_a^{**}}{E_a}\right)\dot{E}_a\\ &+ \frac{(\mu_a+\sigma_a)}{\sigma_a}\left(1 - \frac{I_a^{**}}{I_a}\right)\dot{I}_a,\end{aligned} \tag{36}$$

with

$$\begin{aligned}\pi_h &= \lambda_h^{**} S_h^{**} + \mu_h S_h^{**},\\ \pi_a &= \lambda_a^{**} S_a^{**} + \mu_a S_a^{**}.\end{aligned} \tag{37}$$

where

$$\begin{aligned}\lambda_h^{**} &= \frac{\beta_h q(1-\epsilon)(1-\theta)I_a^{**}}{N_a^{**}},\\ \lambda_a^{**} &= \frac{\beta_a(I_2^{**} + \eta I_1^{**}}{N_h^{**}}\end{aligned} \tag{38}$$

Substituting (1) into (36), we have

$$\begin{aligned}\dot{\mathcal{V}} &= \left(1 - \frac{S_h^{**}}{S_h}\right)(\lambda_h S_h^{**} + \mu_h S_h^{**} - \lambda_h S_h - \mu_h S_h)\\ &+ \left(1 - \frac{E_h^{**}}{E_h}\right)(\lambda_h S_h - (\sigma_1+\mu_h)E_h) + \frac{\sigma_1+\mu_h}{\sigma_1}\left(1 - \frac{I_1^{**}}{I_1}\right)(\sigma_h E_h - (\mu_h+\sigma_2)I_1)\\ &+ \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)}{\sigma_1\sigma_2}\left(1 - \frac{I_2^{**}}{I_2}\right)(\sigma_2 I_1 - (\mu_h+\delta_h+\tau_2)I_2)\\ &+ \frac{(\mu_h+\sigma_1)(\mu_h+\sigma_2)(\mu_h+\delta_h+\tau_2)}{\sigma_1\sigma_2\tau_2}\left(1 - \frac{R_h^{**}}{R_h}\right)(\tau_2 I_2 - (\mu_h+\omega)R_h)\\ &+ \left(1 - \frac{S_a^{**}}{S_a}\right)(\lambda_a S_a^{**} + \mu_a S_a^{**} - \lambda_a S_a - \mu_a S_a) + \left(1 - \frac{E_a^{**}}{E_a}\right)(\lambda_a S_a - (\sigma_a+\mu_a)E_a)\\ &+ \frac{(\mu_a+\sigma_a)}{\sigma_a}\left(1 - \frac{I_a^{**}}{I_a}\right)(\sigma_a E_a - \mu_a I_a),\end{aligned} \tag{39}$$

with relations

$$\begin{aligned}(\sigma_1+\mu_h) &= \frac{\lambda_h S_h^{**}}{E_h^{**}},\\ (\sigma_2+\mu_h) &= \frac{\sigma_1 E_h^{**}}{I_1^{**}},\\ \mu_h &= \frac{\tau_2 I_2^{**}}{R_h^{**}},\\ (\sigma_a+\mu_a) &= \frac{\lambda_a S_a^{**}}{E_a^{**}},\\ \mu_a &= \frac{\sigma_a E_a^{**}}{I_a^{**}},\end{aligned} \tag{40}$$

substituting the relations in (40) into (39), we have

$$\begin{aligned}\dot{\mathcal{V}} \le{}& \mu_h S_h^{**}\left(2-\frac{S_h}{S_h^{**}}-\frac{S_h^{**}}{S_h}\right)+\lambda_h^{**}S_h^{**}\left(6-\frac{S_h^{**}}{S_h}-\frac{S_h E_h^{**}}{S_h^{**}E_h}-\frac{E_h I_1^{**}}{E_h^{**}I_1}-\frac{I_1 I_2^{**}}{I_1^{**}I_2}-\frac{R_h}{R_h^{**}}-\frac{I_2 R_h^{**}}{I_2^{**}R_h}\right)\\ &+\mu_a S_a^{**}\left(2-\frac{S_a}{S_a^{**}}-\frac{S_a^{**}}{S_a}\right)+\lambda_a S_a^{**}\left(4-\frac{S_a^{**}}{S_a}-\frac{S_a E_a^{**}}{S_a^{**}E_a}-\frac{I_a}{I_a^{**}}-\frac{E_a I_a^{**}}{E_a^{**}I_a}\right).\end{aligned} \tag{41}$$

Then, using the relationship between the arithmetic and geometric means, we have

$$\begin{aligned}&\left(2-\frac{S_h}{S_h^{**}}-\frac{S_h^{**}}{S_h}\right)\le 0,\left(6-\frac{S_h^{**}}{S_h}-\frac{S_h E_h^{**}}{S_h^{**}E_h}-\frac{E_h I_1^{**}}{E_h^{**}I_1}-\frac{I_1 I_2^{**}}{I_1^{**}I_2}-\frac{R_h}{R_h^{**}}-\frac{I_2 R_h^{**}}{I_2^{**}R_h}\right)\le 0,\\ &\left(2-\frac{S_a}{S_a^{**}}-\frac{S_a^{**}}{S_a}\right)\le 0,\left(4-\frac{S_a^{**}}{S_a}-\frac{S_a E_a^{**}}{S_a^{**}E_a}-\frac{I_a}{I_a^{**}}-\frac{E_a I_a^{**}}{E_a^{**}I_a}\right)\le 0.\end{aligned} \tag{42}$$

With the following criteria, we have $\dot{\mathcal{V}}\le 0$: $\omega=0$ and $\mathcal{R}_c>1$. Since all of the variables in the model that are involved, including $S_h, E_h, I_1, I_2, R_h, S_a, E_a, I_a$, are at steady state, this can be replaced into the concerned variable of (1) to create

$$\lim_{t\to\infty}\left(S_h(t),E_h(t),I_1(t),I_2(t),R_h(t),S_a(t),E_a(t),I_a(t)\right)\to\left(S_h,E_h,I_1,I_2,R_h,S_a,E_a,I_a\right). \tag{43}$$

Accordingly, using Lassalle's invariant principle, the endemic equilibrium point is globally asymptotically stable (GAS) [15–19, 27–32]. □

4. Numerical simulations

This section displays the state of the model as simulated numerically using the parameter values in Table 2. An analysis of numerical simulations has been conducted to comprehend the transmission dynamics of the model (1) fully, we used the ODE45 solver in MATLAB 2014. Time-series diagrams were used to illustrate the behaviour of the compartments and how essential factors influence state variables. All the parameter values used are as given in Table 2.

Table 2. Ranges and baseline values of parameters of model (1).

Parameter	Ranges (Baseline)	Unit	Reference
π_h, π_a	$100, 2000$	$year^{-1}$	[21, 22, 25, 26]
μ_h, μ_a	$0.0004, 0.020$	$year^{-1}$	[20]
β_h, β_a	$0.0005, 0.800$	$year^{-1}$	[20]
$\sigma_1, \sigma_2, \sigma_a$	$0.0004, 0.020$	$year^{-1}$	[20]
τ_1, τ_2	$(0,1), (0,1)$	$year^{-1}$	Control parameter
q	0.05	$year^{-1}$	Assume
η	0.17	$year^{-1}$	Assumed
ω	0.034	$year^{-1}$	Assume
ϵ	$(0,1)$	$year^{-1}$	Control parameter
δ_h	0.02	$year^{-1}$	[24]

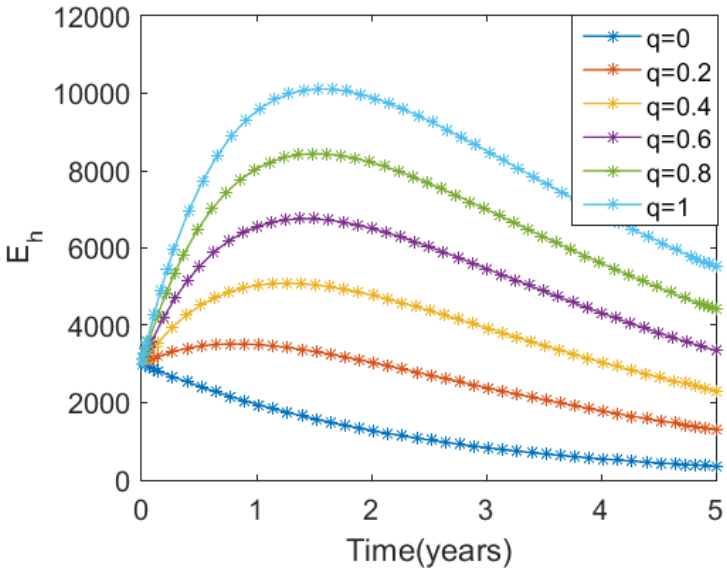


Figure 2. Quantity of not done animal flesh consumed influence on the Exposed humans compartment

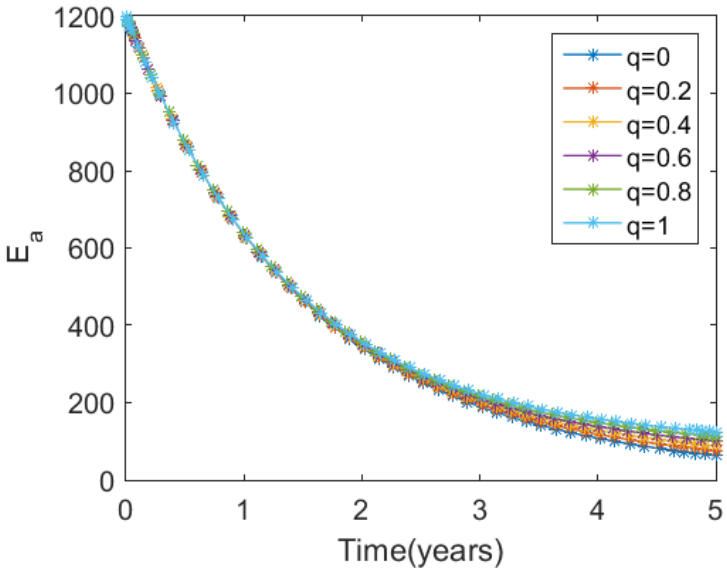


Figure 3. Quantity of not done animal flesh consumed influence on the Exposed animals compartment

5. Examining simulated figures

Figure 2 describes how the quantity of not done animal flesh consumed affects the dynamics of exposed humans. As consumption increases, the population of exposed humans also increases; this indicates that the consumption of improperly prepared animal flesh is directly affecting the dynamics of exposed humans. Figure 3 describes how the quantity of not done animals' flesh consumed affects the dynamics of exposed animals. As consumption increases, the population of exposed animals also increases slightly; this suggests that the consumption of not yet exposed animals' flesh has no direct effect on the dynamics of exposed animals. Figure 4 describes how the quantity of not done animal flesh consumed affects the dynamics of the Light-infected humans compartment. As the consumption increases, the population of the Light-infected humans compartment also increases; this shows that the consumption of not done animal flesh is directly

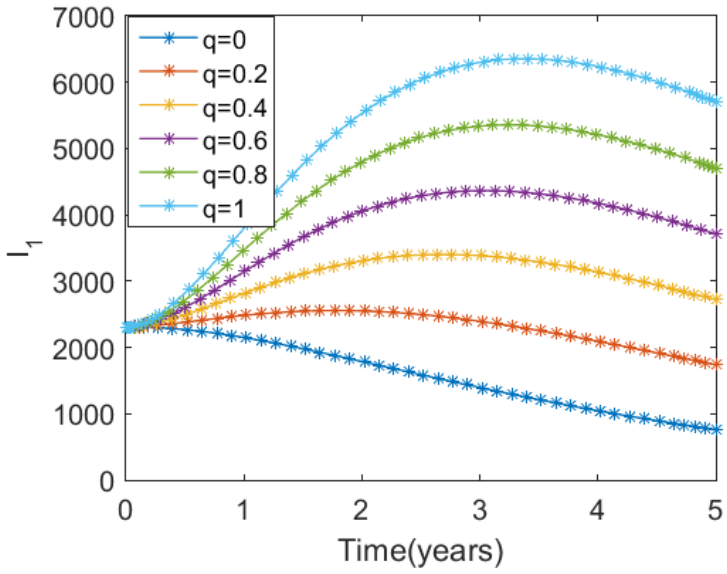


Figure 4. Quantity of not done animal flesh consumed influence on the Light infected humans compartment

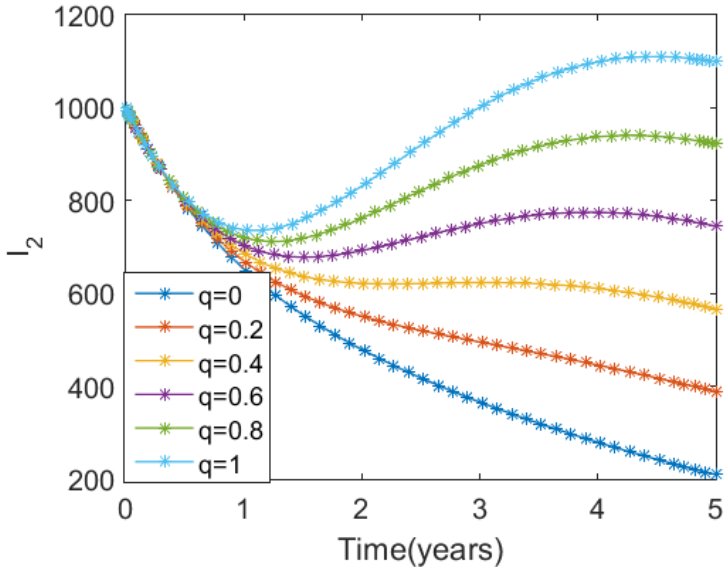


Figure 5. Quantity of not done animal flesh consumed influence on the Heavy infectious human compartment

affecting the dynamics of the Light-infected humans compartment. Figure 5 describes how the quantity of not done animal flesh consumed affects the dynamics of the heavy infectious human compartment. As the consumption increases, the population of the heavy infectious humans compartment also increases; this shows that the consumption of not done animal flesh is directly affecting the dynamics of the heavy infectious humans compartment. Figure 6 describes how the quantity of not done animal flesh consumed affects the dynamics of the contagious animal compartment. As the consumption increases, the population of the infectious animal compartment remains unchanged. This suggests that the consumption of improperly prepared animal flesh has no connection to the dynamics of the contagious animal compartment.

Figure 7 illustrates how early treatment impacts the dynamics of exposed individuals. As the early treatment increases, the population of exposed humans decreases slightly; this indicates that the early treatment has a minimal effect on the dynamics of exposed humans. Figure 8 describes

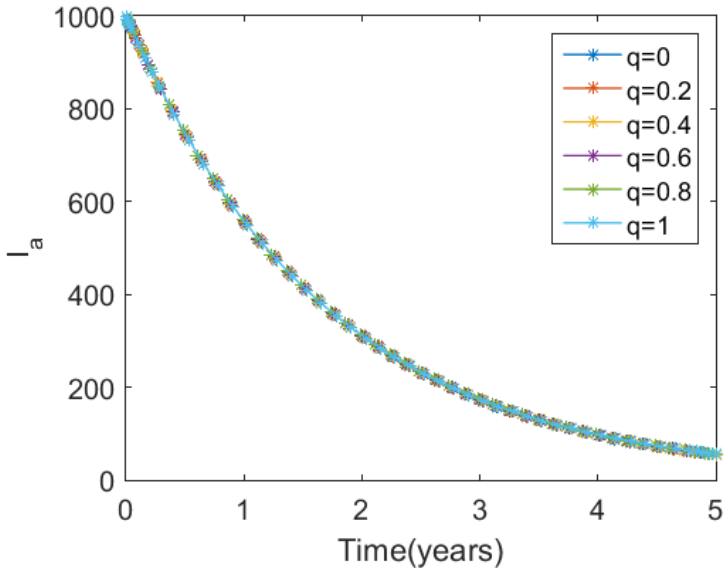


Figure 6. Quantity of not done animal flesh consumed influence on the infectious animal compartment

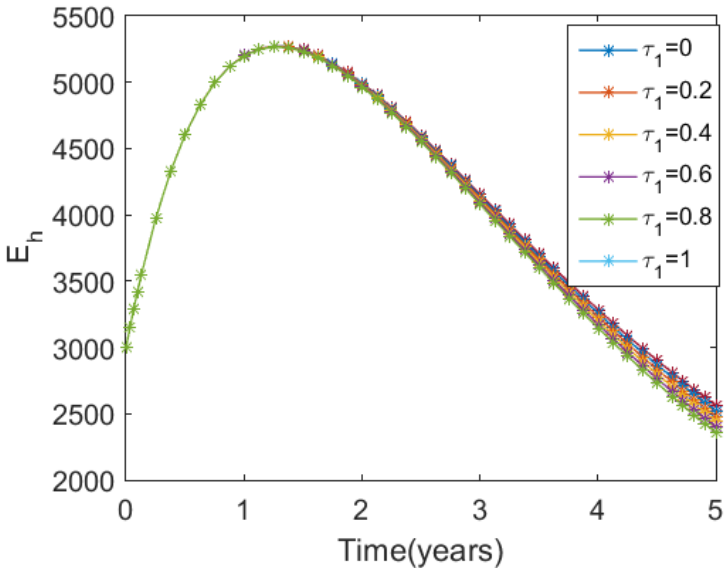


Figure 7. Early treatment influence on the Exposed humans compartment

how the quantity of early treatment affects the dynamics of exposed animals. As the early treatment increases, the population of exposed animals also decreases slightly; this indicates that the early treatment has a minimal effect on the dynamics of exposed animals. Figure 9 describes how early treatment affects the dynamics of the Light-infected humans compartment. As the early treatment increases, the population of the Light-infected humans compartment decreases slightly; this indicates that the early treatment has a minimal effect on the dynamics of the Light-infected humans compartment. Figure 10 describes how early treatment affects the dynamics of the heavy infectious human compartment. As the early treatment increases, the population of the heavy infectious humans compartment decreases drastically, which shows that the early treatment is directly affecting the dynamics of the heavy infectious humans compartment. Figure 11 illustrates how early treatment impacts the dynamics of the contagious animal compartment. As the early treatment increases the population of the infectious animals compartment, it remains

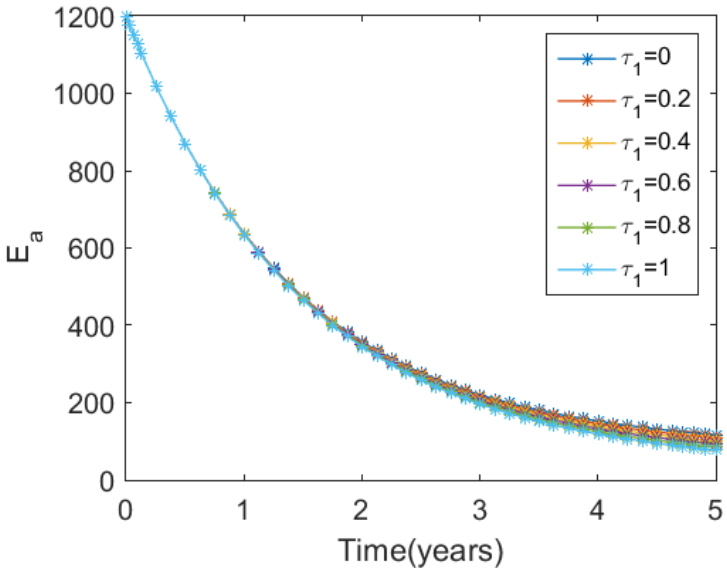


Figure 8. Early treatment influence on Exposed animals compartment

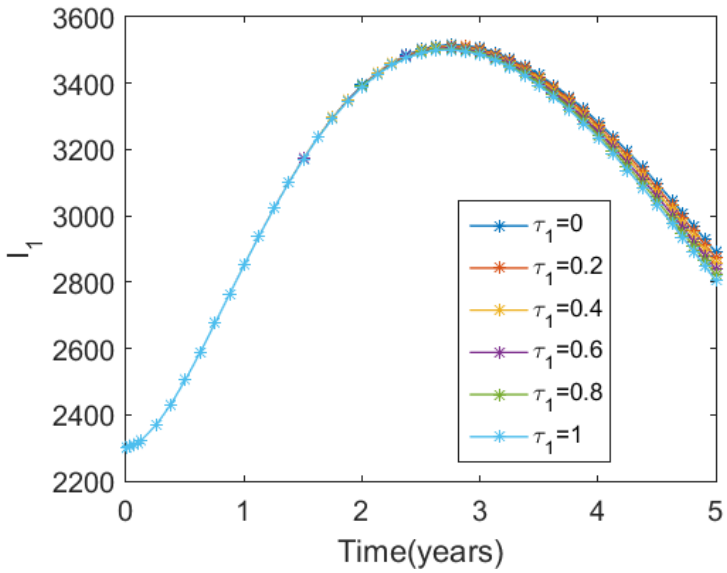


Figure 9. Early treatment influence on Light-infected human compartment

unchanged, which shows that the early treatment has no connection with the dynamics of the contagious animals compartment.

Figure 12 illustrates how the treatment of the heavily infected human compartment impacts the dynamics of exposed individuals. As the treatment of the heavily infectious human compartment increases, the population of exposed humans decreases slightly, indicating that the treatment of the heavily infectious human compartment has a minimal effect on the dynamics of exposed humans. Figure 13 illustrates how the treatment of the heavily infected human compartment affects the dynamics of exposed animals. As the treatment of heavily infected human compartments increases, the population of exposed animals also decreases slightly, indicating that the treatment of heavily infected human compartments has a minimal effect on the dynamics of exposed animals. Figure 14 describes how treatment of the Heavy infectious humans compartment affects the dynamics of the Light infected humans compartment, as the treatment of the Heavy infectious humans compartment increases, the population of Light infected humans compartment decreases a little, this shows that the treatment of the Heavy infectious humans compartment has

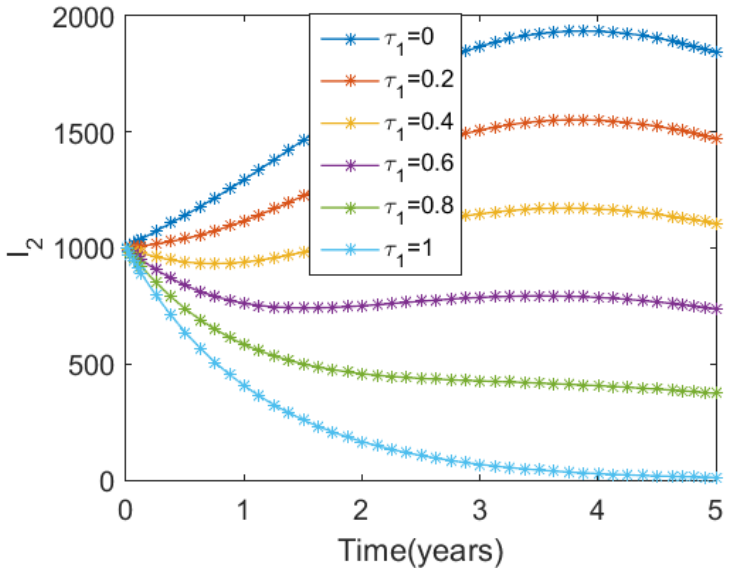


Figure 10. Early treatment influence on the heavily infectious human compartment

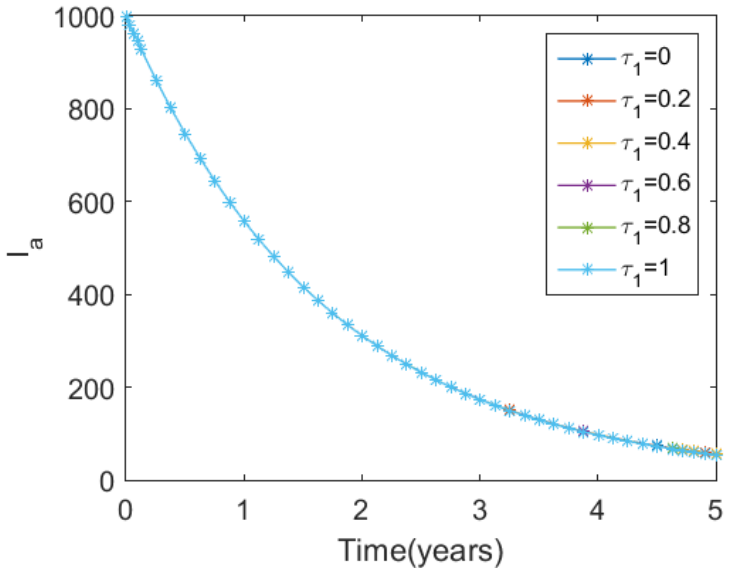


Figure 11. Early treatment influence on infectious animal compartments

a little effect on the dynamics of Light infected humans compartment. Figure 15 describes how the treatment of the Heavy infectious humans compartment affects the dynamics of the heavy infectious humans compartment. As the treatment of the heavily infectious human compartment increases, the population of the heavily infectious human compartment decreases drastically. This indicates that early treatment has a direct impact on the dynamics of the heavily infected human compartment. Figure 16 describes how the treatment of the heavily infected human compartment affects the dynamics of the infectious animal compartment. As the treatment of the heavily infected human compartment increases, the population of the infectious animals compartment remains unchanged, indicating that the treatment of the heavily infected human compartment has no connection with the dynamics of the infectious animals compartment.

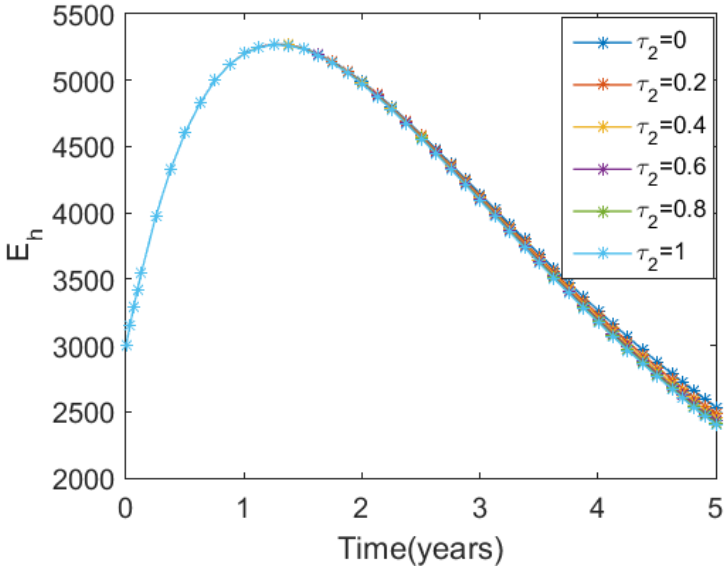


Figure 12. Treatment rate of heavily infectious humans influence on the Exposed humans compartment

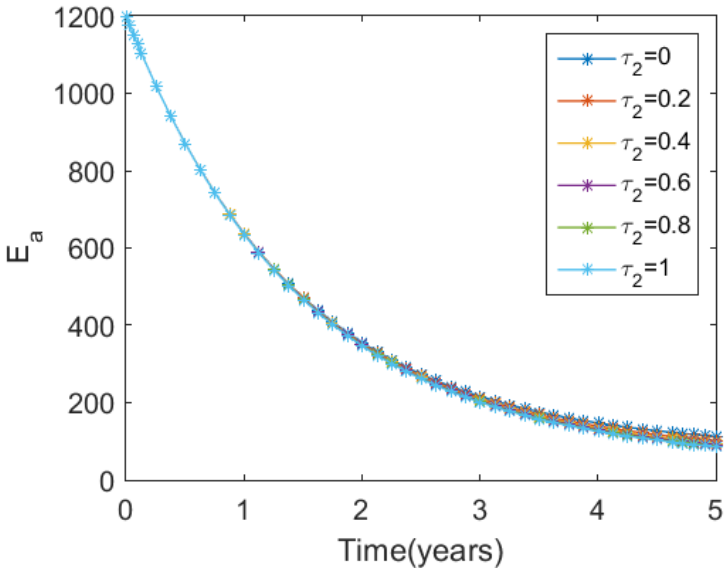


Figure 13. Treatment rate of heavily infectious humans influence on the Exposed animals compartment

6. Conclusion

This work considers both the human and animal populations. The human population has been subdivided into five compartments, including susceptible humans, exposed humans, lightly infected humans, heavily infected humans, and recovered humans. The animal population has been subdivided into three groups: susceptible animals, exposed animals, and infectious animals. The formulated models have been analyzed. To begin the analysis, we verified the existence and uniqueness of the proposed model, followed by the boundedness and positivity of the solutions. Next, we computed the practical and basic reproduction numbers, and we also checked the local stability of the disease-free equilibrium using a Jacobian matrix. This analysis shows that the disease-free equilibrium is locally asymptotically stable if the effective reproduction number is

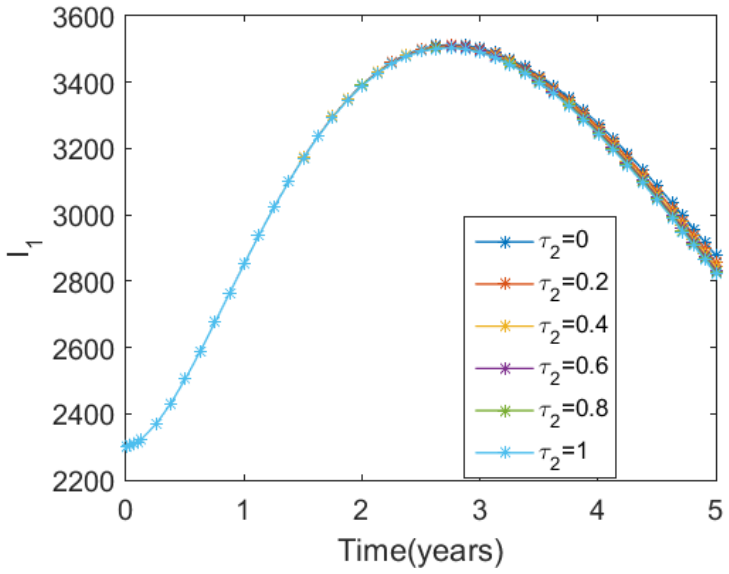


Figure 14. Treatment rate of heavily infectious humans influence on the Light-infected humans compartment

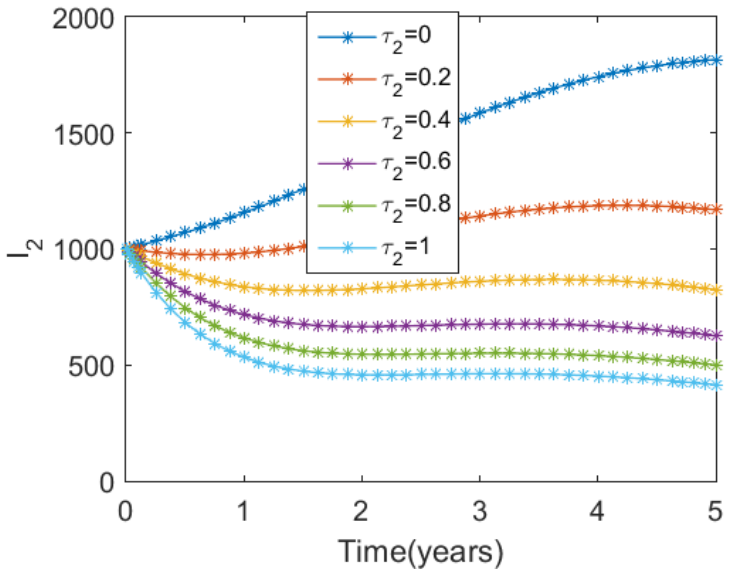


Figure 15. Treatment rate of heavily infectious humans influence on the Heavy infectious humans compartment

less than unity. The global asymptotic stability of the disease-free equilibrium has been investigated using the comparison method, which demonstrates that the disease-free equilibrium is globally asymptotically stable if the effective reproduction number is less than one. The global asymptotic stability of the endemic equilibrium has been studied using the Lyapunov function of Go-Volterra type, which shows that the endemic equilibrium point is globally asymptotically stable if the effective reproduction number is greater than unity. Numerical simulation shows that early treatment plays a vital role in controlling the dynamics of Ascariasis, as well. It has been observed that consuming raw animal flesh can increase or exacerbate diseases in society. This means that reducing the consumption of raw animal flesh can help control the Ascariasis disease in society. It is therefore recommended that early treatment should be prioritized, and eating the flesh of uncooked animals should be reduced in order to control the disease in society.

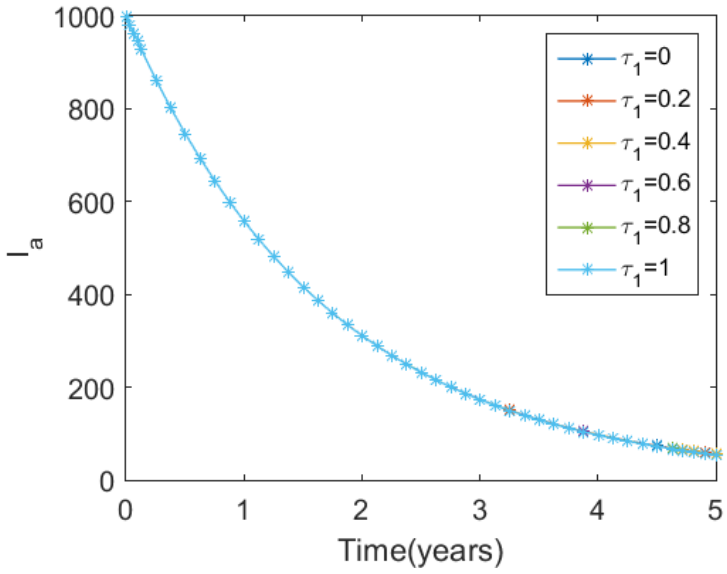


Figure 16. Treatment rate of heavily infectious humans influence on the infectious animals compartment

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Conflict of interest

There is no conflict of interest to disclose.

Author contributions

James Andrawus: Methodology, Conceptualization, Formal analysis, Writing - Original draft. **Ibrahim Ibrahim Muhammad:** Methodology, Conceptualization, Formal analysis, Writing - Original draft. **Felix Yakubu Eguda:** Methodology, Conceptualization, Formal analysis, Writing - Original draft. **Abdullahi Yusuf:** Validation, Investigation, Formal analysis, Supervision

Declaration of using AI tools

The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

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