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A Human-Animal Model of Giardiasis Infection in Contaminated Environment

Research Article

Saul C. Mpeshe[a, *], **Nkuba Nyerere**[b]

[a] *Department of Mathematical Sciences, University of Iringa, P.O. Box 200, Iringa, Tanzania*

[b] *Department Mathematics, Informatics and Computational Science, SUA, P.O. Box 3000, Morogoro, Tanzania.*

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Abstract: In this paper, we presented a modeling approach to investigate the dynamics of *Giardiasis* in humans and domestic animals coupled with a contaminated environment. We computed the basic reproduction number $\mathscr{R}_0$ and employed it in analyzing the effect of initial transmission and the stability of disease when an outbreak occurs. Results show that even when $\eta = 0$, $\mathscr{R}_0 = \mathscr{R}_{0h}$ is greater than 4, showing that person-to-person transmission is the most significant in the dynamics of *Giardiasis*. An increase in η increases the value of $\mathscr{R}_0$ to some extent. Numerical simulations show that whenever there is an outbreak of *Giardiasis* in humans and domestic animals, the disease is likely to persist in the first two months and thereafter it will start to slow down to disease-free-equilibrium.

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Keywords: Giardiasis • Giardia lamblia • basic reproduction number • Metzler matrix • Jacobian matrix • stability analysis

1. Introduction

Giardiasis (also known as *lambliasis*) is the intestinal infection caused by the flagellate protozoan parasite called *Giardia lamblia* (also known as *Giardia intestinalis* or *Giardia duodenalis)*. The parasite is transmitted through ingestion of the infective cysts shed in human or animal faeces and might be present in faecally contaminated food or water [1–4].

An infected individual can shed nearly 10^8 to 10^{10} cysts per day, but as few as 10 cysts have proven to be enough to cause an infection [5–7]. After excretion in faeces, the cyst is immediately infectious to a new host, no period of maturation or latent period is required [8]. Cysts present in faeces can remain viable in a variety of environments, particularly water and/or lower temperatures [9–11]. Sexual transmission of *giardiasis* is also a well-described form of oral-anal transmission and faecal-oral transmission among men who have sex with men [12].

Giardia lamblia was first described by Antonie van Leeuwenhoek in 1681 in his own stool sample [13]. For decades, it was considered of uncertain pathogenicity but now it is regarded as the most common cause of protozoan diarrhea in human, domestic and wild animals worldwide [14]. Infection results in clinical manifestations that range from asymptomatic colonization to acute or chronic diarrhea [15]. Its clinical significance was broadly accepted after

* Corresponding author.
E-mail address(es): saul.mpeshe@uoi.ac.tz (Saul C. Mpeshe), emmankuba@sua.ac.tz (Nkuba Nyerere).

many symptomatic cases of *giardiasis* were diagnosed and reported among visitors to the Soviet Union in the early 1970s. Since then, *giardiasis* has been reported as responsible for many outbreaks throughout the world [16]. The disease is included in the World Health Organization (WHO) neglected diseases initiative owing to its burden and association with poverty [17].

Giardia lamblia has been consistently reported as one of the most common pathogens worldwide [18], and due to high endemicity among humans and animals, it is considered of public and veterinary health importance [19]. Symptomatic infections have been reported by millions in Asia, Africa, and Latin America [20, 21]. In the United States, *giardiasis* has been variably reported since 1992 and was made a nationally notifiable disease in 2002 [1, 5].

Worldwide, the incidence of *giardiasis* has been estimated in 2.8×10^8 cases per year [22]. However, several epidemiological studies have reported that such rates could be significantly underestimated, with *giardiasis* prevalence rates ranging from 10 to 50% in developing countries [17, 23], and from 2 to 5% in developed countries [24, 25]. This could be explained by the large fraction of asymptomatic carriers, which regardless of the absence of symptoms also contribute to the transmission of diseases. *Giardia* infection in animals and humans has been associated to growth retardation [26–28].

Several molecular studies have been developed to help explain the complex epidemiology of *giardiasis*. The studies have shown that *Giardia lamblia* comprises of eight genetic groups (or assemblages), (namely A - H) of which only A and B can cause disease in both humans and animals, while the remaining (C–H) are relatively host-specific [19, 29–32].

Giardiasis is endemic in most countries of the world, with a high prevalence in developing countries. Several factors have been associated with its endemicity worldwide. Hall [6], identified four factors being a) relatively long infection period of *Giardia lamblia*, typically lasting for months; b) to establish the infection a very small number of cysts (< 10 cysts); c) under appropriate conditions, cysts can survive in the environment for several weeks; and d) infections do not render protective immunity to all individuals.

2. The Giardiasis Model couple with environment

The potential transmission of *giardiasis* to human beings is evident. While Giardiasis is endemic worldwide, little is known by many people in developed and developing countries. Mathematical modeling has played a major role to help our understanding of population dynamics and epidemiology of the infectious disease, but, hardly a few models exist for *Giardia* infection. The model by Waters et al. [33] can be considered as the first time model for *giardiasis*. Therefore, modeling the dynamics of *giardisis* is very important to have a better understanding of its transmission dynamics, the risk associated with the disease, and measures to control the disease.

The study of the dynamics of *giardiasis* including human and domestic animal host coupled with the contaminated environment is important in its own right. In this article, we analyze a mathematical model of *giardiasis* transmission, including aspects of human and domestic animals as well as the environment. The proposed model is of the classical SEIR-type, which is a simplified representation of the complex biology of *giardiasis*. As a related work, we mention Walters et al.[33] who consider an SIS model for humans, animals and contaminated water. We build upon their model and include food contamination in the environment, and that both human and animals can contaminate the environment. We also consider the recruitment in susceptible population as well as the natural death in both humans and animals.

2.1. Model formulation

The model considers two populations, namely: humans and domestic animals coupled with contaminated water and food in the environment. There is a natural death rate in each stage because the infection may take a long time, and therefore individual may die naturally. The mode of transmission of *giardiasis* is the environment to host, host to host, and host to the environment is shown in Fig. 1. To simplify the model, age structure in hosts and spatial effects are not included in the model. The domestic animal population contains four compartments: susceptible S_a, exposed E_a, infectious I_a and recovered R_a. The size of the domestic animal population is therefore given by $N_a = S_a + E_a + I_a + R_a$. The human population contain also four compartments, namely susceptible S_h, exposed E_h, infectious I_h and recovered R_h. The total population for humans is therefore given by $N_h = S_h + E_h + I_h + R_h$.

The number of cyst in the environment is represented by only one compartment W since after excretion in faeces, the cyst is immediately infectious to a new host. We also assume temporary immunity, though there is a possibility of an individual to be infected just after recovery [8]. Humans and animals can deposit cyst to the environment

at a rate α_h and α_a respectively. All humans can ingest cyst at a rate η, and the ingested cyst by susceptible humans will be converted to infection at a rate ρ. Model parameters and their description as they have been used in the model are given in Table 1. Fig. 1 shows the transmission dynamics of *giardiasis* with variables and parameters as described

Table 1. Parameters and their description

Parameter	Description
Λ_a	recruitment rate in domestic animals
Λ_h	recruitment rate in humans
β_a	disease transmission rate in domestic animals
β_h	disease transmission rate in humans
ϵ_a	rate of progression from latency to infectious in domestic animals
ϵ_h	rate of progression from latency to infectious in humans
μ_a	natural death rate of animals
μ_h	natural death rate of humans
μ	natural decay rate of cysts in the environment
η	rate at which human ingest cysts from the environment
ρ	rate of convention of ingested cysts to infection
α_a	rate at which the environment is contaminated by domestic animals
α_h	rate at which the environment is contaminated by humans
γ_a	disease recovery rate in domestic animals
γ_h	disease recovery rate in humans

in Table 1.

Using the parameters in Table 1 and Fig. 1, an SEIR model is derived using first order nonlinear ordinary dif-

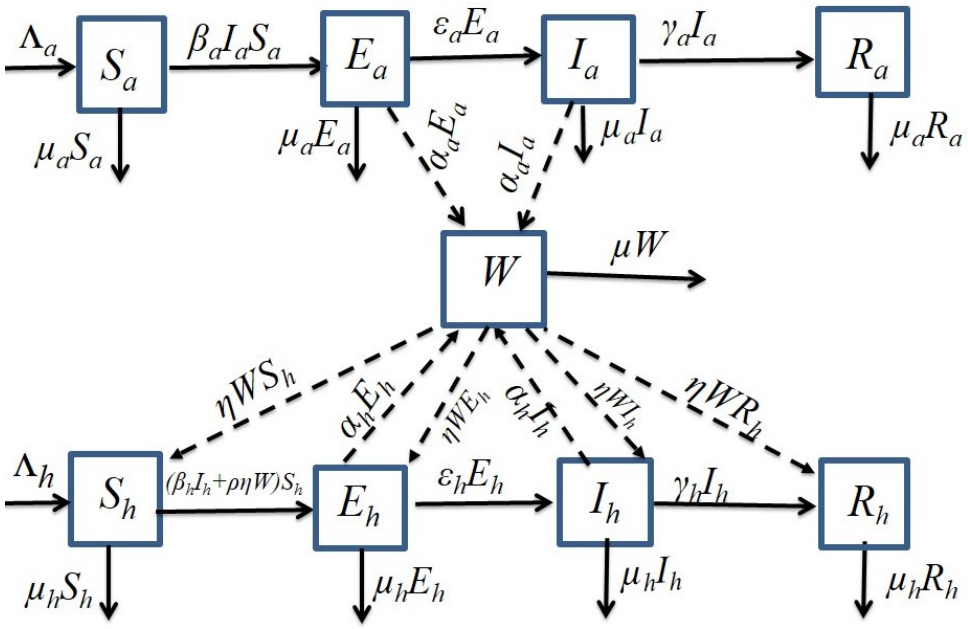


Fig. 1. Transmission diagram for Giardiasis

ferential equations as follows:
The dynamics of infection in the domestic animal population is given by

$$\frac{dS_a}{dt} = \Lambda_a - \mu_a S_a - \beta_a S_a I_a \tag{1a}$$

$$\frac{dE_a}{dt} = \beta_a S_a I_a - (\varepsilon_a + \mu_a) E_a \tag{1b}$$

$$\frac{dI_a}{dt} = \varepsilon_a E_a - (\mu_a + \gamma_a) I_a \tag{1c}$$

$$\frac{dR_a}{dt} = \gamma_a I_a - \mu_a R_a \tag{1d}$$

The dynamics of live infective cysts in the environment W is given by

$$\frac{dW}{dt} = \alpha_a(E_a + I_a) + \alpha_h(E_h + I_h) - \eta W N_h - \mu W \tag{2}$$

where N_h is the total human population.
The dynamics of infection in the human population is modeled as

$$\frac{dS_h}{dt} = \Lambda_h - \mu_h S_h - \beta_h S_h I_h - \rho\eta W S_h \tag{3a}$$
$$\frac{dE_h}{dt} = \beta_h S_h I_h + \rho\eta W S_h - (\varepsilon_h + \mu_h) E_h \tag{3b}$$
$$\frac{dI_h}{dt} = \varepsilon_h E_h - (\mu_h + \gamma_h) I_h \tag{3c}$$
$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \tag{3d}$$

2.2. Feasibility of the Model Solution

The feasibility of the model solution helps us to determine whether the model is well-posed epidemiologically as well as mathematically. Since R_a and R_h does not appear in other equations, then the equation for R_a and R_h can be omitted from the analysis for its value can be obtained when the values for S_a, E_a, I_a, S_h, E_h and I_h are known. The remaining system becomes

$$\frac{dS_a}{dt} = \Lambda_a - \mu_a S_a - \beta_a S_a I_a \tag{4a}$$
$$\frac{dE_a}{dt} = \beta_a S_a I_a - (\varepsilon_a + \mu_a) E_a \tag{4b}$$
$$\frac{dI_a}{dt} = \varepsilon_a E_a - (\mu_a + \gamma_a) I_a \tag{4c}$$
$$\frac{dW}{dt} = \alpha_a(E_a + I_a) + \alpha_h(E_h + I_h) - \eta W N_h - \mu W \tag{4d}$$
$$\frac{dS_h}{dt} = \Lambda_h - \mu_h S_h - \beta_h S_h I_h - \rho\eta W S_h \tag{4e}$$
$$\frac{dE_h}{dt} = \beta_h S_h I_h + \rho\eta W S_h - (\varepsilon_a + \mu_h) E_h \tag{4f}$$
$$\frac{dI_h}{dt} = \varepsilon_h E_h - (\mu_h + \gamma_h) I_h \tag{4g}$$

The model system (4) can be written in compact form as

$$\frac{dX}{dt} = M(x)X + F \tag{5}$$

where $X = (S_a, E_a, I_a, W, S_h, E_h, I_h)^T$, $M(x)$ is a 7×7 matrix, and F is constant column matrix.

Further computation shows that

$$M(x) = \begin{bmatrix} -(\mu_a + \beta I_a) & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_a I_a & -(\varepsilon_a + \mu_a) & 0 & 0 & 0 & 0 & 0 \\ 0 & \varepsilon_a & -(\mu_a + \gamma_a) & 0 & 0 & 0 & 0 \\ 0 & \alpha_a & \alpha_a & -(\eta S_h + \mu) & 0 & \alpha_h & \alpha_h \\ 0 & 0 & 0 & 0 & -(\beta_h I_h + \rho\eta W + \mu_h) & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_h I_h + \rho\eta W & -(\varepsilon_h + \mu_h) & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_h & -(\mu_h + \gamma_h) \end{bmatrix} \tag{6}$$

and

$$F = (\Lambda_a, 0, 0, 0, \Lambda_h, 0, 0)^T \geq 0. \tag{7}$$

Since all non-diagonal entries are non-negative, then $M(x)$ is a Metzler stable matrix for all entries in $\mathbb{R}^7_+$ and F is Lipschitz continuous [34, 35]. Therefore, the model system is feasible in the region $\Omega = (S_a, E_a, I_a, W, S_h, E_h, I_h) \geq 0 \in \mathbb{R}^7_+$ and the model solution is positively invariant in $\mathbb{R}^7_+$. That is to say, the model solution remain in the feasible region Ω if it starts in this region.

2.3. The Basic Reproduction Number

The basic reproduction number $\mathscr{R}_0$ is computed using the next generation method as described by van den Driessche and Watmough [36]. From this approach, we find that

$$F = \begin{bmatrix} 0 & \beta_a S_a^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \rho\eta S_h^* & 0 & \beta_h S_h^* \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{8}$$

$$V = \begin{bmatrix} \varepsilon_a + \mu_a & 0 & 0 & 0 & 0 \\ -\varepsilon_a & \mu_a + \gamma_a & 0 & 0 & 0 \\ -\alpha_a & -\alpha_a & \eta N_h^* + \mu & -\alpha_h & -\alpha_h \\ 0 & 0 & 0 & \varepsilon_h + \mu_h & 0 \\ 0 & 0 & 0 & -\varepsilon_h & \mu_h + \gamma_h \end{bmatrix} \tag{9}$$

and

$$FV^{-1} = \begin{bmatrix} \frac{\beta_a S_a^* \varepsilon_a}{\varepsilon_a + \mu_a} & \frac{\beta_a S_a^*}{\mu_a + \gamma_a} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ A & \frac{\rho\eta S_h^* \alpha_a}{(\mu_a + \gamma_a)(\eta N_h^* + \mu)} & \frac{\rho\eta S_h^*}{\eta N_h^* + \mu} & B & C \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{10}$$

where

$$\begin{aligned} A &= \frac{\rho\eta S_h^* \alpha_a (\varepsilon_a + \mu_a + \gamma_a)}{(\mu_a + \gamma_a)(\varepsilon_a + \mu_a)(\eta N_h^* + \mu)} \\ B &= \frac{\rho\eta S_h^* \alpha_h (\varepsilon_h + \mu_h + \gamma_h)}{(\mu_h + \gamma_h)(\varepsilon_h + \mu_h)(\eta N_h^* + \mu)} + \frac{\beta_h S_h^* \varepsilon_h}{(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)} \\ C &= \frac{\rho\eta S_h^* \alpha_h}{(\mu_h + \gamma_h)(\eta N_h^* + \mu)} + \frac{\beta_h S_h^*}{\mu_h + \gamma_h} \end{aligned}$$

The basic reproduction number $\mathscr{R}_0$ is the largest eigenvalue of the FV^{-1} matrix. Now solving for the eigenvalues of FV^{-1} and substitute $S_a^* = \frac{\Lambda_a}{\mu_a}$ and $N_h^* = S_h^* = \frac{\Lambda_h}{\mu_h}$ at disease-free equilibrium, gives

$$\mathscr{R}_0 = \max\{\frac{\beta_a \Lambda_a \varepsilon_a}{\mu_a(\varepsilon_a + \mu_a)(\mu_a + \gamma_a)}, \frac{\rho\eta\Lambda_h \alpha_h (\varepsilon_h + \mu_h + \gamma_h)}{(\eta\Lambda_h + \mu_h\mu)(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)} + \frac{\beta_h \Lambda_h \varepsilon_h}{\mu_h(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)}\}. \tag{11}$$

It can be observed that

$$\frac{\beta_a \Lambda_a \varepsilon_a}{\mu_a(\varepsilon_a + \mu_a)(\mu_a + \gamma_a)} = \mathscr{R}_{0a}, \tag{12}$$

is the basic reproduction number for the SEIR model of domestic animals, and

$$\frac{\rho\eta\Lambda_h \alpha_h (\varepsilon_h + \mu_h + \gamma_h)}{(\eta\Lambda_h + \mu_h\mu)(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)} + \frac{\beta_h \Lambda_h \varepsilon_h}{\mu_h(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)} = \mathscr{R}_{0h}, \tag{13}$$

is the basic reproduction number of the SEIR model of human coupled with environment. When the rate at which human ingest cyst from environment η is zero, then we have

$$\frac{\beta_h \Lambda_h \varepsilon_h}{\mu_h(\varepsilon_h + \mu_h)(\mu_h + \gamma_h)} = \mathscr{R}_{0h}^*, \tag{14}$$

which is the basic reproduction number of the human-to-human transmission SEIR model. Furthermore,

$$\frac{\partial \mathscr{R}_{0h}}{\partial \rho} > 0 \tag{15}$$

and

$$\frac{\partial \mathscr{R}_{0h}}{\partial \eta} > 0. \tag{16}$$

Thus, $\mathscr{R}_{0h}$ increases with increase in η and ρ.

3. Stability analysis of Equilibrium Points

Setting the left-hand-side of the model system (4) equal to zero and that $I_a = I_h = W = 0$ we have the disease-free equilibrium E_0 given by

$$\Omega_0 = \left(\frac{\Lambda_a}{\mu_a}, 0, 0, 0, \frac{\Lambda_h}{\mu_h}, 0, 0\right) \tag{17}$$

The endemic equilibrium is $\Omega^* = (S_a^*, E_a^*, I_a^*, W^*, S_h^*, E_h^*, I_h^*)$ where

$$S_a^* = \frac{1}{\beta_a \varepsilon_a}(\varepsilon_a + \mu_a)(\mu_a + \gamma_a) = \frac{\Lambda_a}{\mu_a}\frac{1}{\mathscr{R}_{0a}}, \tag{18a}$$

$$E_a^* = \frac{\mu_a + \gamma_a}{\varepsilon_a} I_a^* = \frac{\mu_a(\mu_a + \gamma_a)}{\beta_a \varepsilon_a}(\mathscr{R}_{0a} - 1) \tag{18b}$$

$$I_a^* = \frac{\Lambda_a \varepsilon_a}{(\varepsilon_a + \mu_a)(\mu_a + \gamma_a)} - \frac{\mu_a}{\beta_a} = \frac{\mu_a}{\beta_a}(\mathscr{R}_{0a} - 1), \tag{18c}$$

$$W^* = \frac{\alpha_a(E_a^* + I_a^*) + \alpha_h(E_h^* + I_h^*)}{\eta N_h^* + \mu} \tag{18d}$$

$$S_h^* = \frac{\Lambda_h}{\mu_h + \beta_h I_h^* + \rho\eta W^*} \tag{18e}$$

$$E_h^* = \frac{\Lambda_a - \mu_h S_h^*}{\varepsilon_h + \mu_h} \tag{18f}$$

$$I_h^* = \frac{\varepsilon_h(\Lambda_h - \mu_h S_h^*)}{(\mu_h + \gamma_h)(\varepsilon_h + \mu_h)} \tag{18g}$$

3.1. Local Stability of the Disease-Free Equilibrium

Theorem 3.1.
The disease-free equilibrium of the Giardiasis model (4) is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$.

Proof. To prove this theorem, we need to show that the Jacobian matrix $J(\Omega_0)$ of the *Giardiasis* model (4) at the disease-free equilibrium has negative eigenvalues. Further computations show that

$$J(\Omega_0) = \begin{bmatrix} -\mu_a & 0 & -\beta_a S_a^* & 0 & 0 & 0 & 0 \\ 0 & -(\varepsilon_a + \mu_a) & \beta_a S_a^* & 0 & 0 & 0 & 0 \\ 0 & \varepsilon_a & -(\mu_a + \gamma_a) & 0 & 0 & 0 & 0 \\ 0 & \alpha_a & \alpha_a & -(\eta N_h^* + \mu) & 0 & \alpha_h & \alpha_h \\ 0 & 0 & 0 & -\rho\eta S_h^* & -\mu_h & 0 & -\beta_h S_h^* \\ 0 & 0 & 0 & \rho\eta S_h^* & 0 & -(\varepsilon_h + \mu_h) & \beta_h S_h^* \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_h & -(\mu_h + \gamma_h) \end{bmatrix} \tag{19}$$

From $J(\Omega_0)$ we find that the diagonal entries $-\mu_a$ and $-\mu_h$ are two eigenvalues of the Jacobian. Excluding these columns and their corresponding rows, we remain with a 5×5 matrix

$$J^*(\Omega_0) = \begin{bmatrix} -(\varepsilon_a + \mu_a) & \beta_a S_a^* & 0 & 0 & 0 \\ \varepsilon_a & -(\mu_a + \gamma_a) & 0 & 0 & 0 \\ \alpha_a & \alpha_a & -(\eta N_h^* + \mu) & \alpha_h & \alpha_h \\ 0 & 0 & \rho\eta S_h^* & -(\varepsilon_h + \mu_h) & \beta_h S_h^* \\ 0 & 0 & 0 & \varepsilon_h & -(\mu_h + \gamma_h) \end{bmatrix} \tag{20}$$

Since the remaining matrix $J^*(\Omega_0)$ is a Metzler matrix, then it has negative eigenvalues. Hence, $J(\Omega_0)$ has negative eigenvalues and we conclude that the disease-free equilibrium is locally asymptotically stable. □

3.2. Global Stability of the Disease-Free Equilibrium

Theorem 3.2.
The disease-free equilibrium of the Giardiasis model (4) is globally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$.

Proof. To prove the global stability of DFE we apply the procedure outlined by Castillo-Chavez et al [37]. With this procedure, we write the system in the form

$$\begin{cases} \frac{dX_n}{dt} = A_1(X_n - X_{DFE,n}) + A_{12}X_e, \\ \frac{dX_e}{dt} = A_2 X_e \end{cases} \tag{21}$$

where X_n is the vector representing the non-transmitting class, and X_e is the vector representing the transmitting class. The disease-free equilibrium is globally asymptotically stable if A_1 has negative real eigenvalues and A_2 is a Metzler matrix.

From the model system (4) we have $X_n = (S_a, S_h)^T$, and $X_e = (E_a, I_a, W, E_h, I_h)^T$, with

$$A_1 = \begin{bmatrix} -\mu_a) & 0 \\ 0 & -\mu_h \end{bmatrix} \tag{22}$$

$$A_{12} = \begin{bmatrix} 0 & -\beta_a S_a^* & 0 & 0 & 0 \\ 0 & 0 & -\rho\eta S_h^* & 0 & -\beta_h S_h^* \end{bmatrix} \tag{23}$$

and

$$A_2 = \begin{bmatrix} -(\varepsilon_a+\mu_a) & \beta_a S_a^* & 0 & 0 & 0 \\ \varepsilon_a & -(\mu_a+\gamma_a) & 0 & 0 & 0 \\ \alpha_a & \alpha_a & -(\eta N_h^*+\mu) & \alpha_h & \alpha_h \\ 0 & 0 & \rho\eta S_h^* & -(\varepsilon_h+\mu_h) & \beta_h S_h^* \\ 0 & 0 & 0 & \varepsilon_h & -(\mu_h+\gamma_h) \end{bmatrix}, \tag{24}$$

A direct computation shows that the eigenvalues of A_1 are real and negative. Thus, the system

$$\frac{dX_n}{dt} = A_1(X_n - X_{DFE,n}) + A_{12}X_e \tag{25}$$

is locally and globally stable at the disease-free equilibrium point. Furthermore, A_2 is a Metzler matrix. Hence, the disease-free equilibrium is globally asymptotically stable. □

3.3. Global Stability of the Endemic Equilibrium

The local stability of the disease-free equilibrium suggests local stability of the endemic equilibrium for the reverse condition [36]. Hence, we only investigate the global stability of the endemic equilibrium via the construction of a suitable Lyapunov function using Korobeinikov approach [38, 39], which is useful for compartmental epidemic models with any number of compartments. In this approach, we construct Lyapunov functions of the form

$$V = \sum \omega_i (x_i - x_i^* \ln x_i) \tag{26}$$

where $\omega_i > 0$ is constant to be chosen, x_i is the population of the i^{th} compartment, and x_i^* is the equilibrium point.

Thus, consider the Lyapunov function

$$\begin{aligned} V = {} & \omega_1(S_a - S_a^* \ln S_a) + \omega_2(E_a - E_a^* \ln E_a) + \omega_3(I_a - I_a^* \ln I_a) + \omega_4(W - W^* \ln W) \\ & + \omega_5(S_h - S_h^* \ln S_h) + \omega_6(E_h - E_h^* \ln E_h) + \omega_7(I_h - I_h^* \ln I_h) \end{aligned} \tag{27}$$

The time derivative of V is then given by

$$\begin{aligned} \frac{dV}{dt} = {} & \omega_1(1 - \frac{S_a^*}{S_a})\frac{dS_a}{dt} + \omega_2(1 - \frac{E_a^*}{E_a})\frac{dE_a}{dt} + \omega_3(1 - \frac{I_a^*}{I_a})\frac{dI_a}{dt} + \omega_4(1 - \frac{W^*}{W})\frac{dW}{dt} \\ & + \omega_5(1 - \frac{S_h^*}{S_h})\frac{dS_h}{dt} + \omega_6(1 - \frac{E_h^*}{E_h})\frac{dE_h}{dt} + \omega_7(1 - \frac{I_h^*}{I_h})\frac{dI_h}{dt} \end{aligned} \tag{28}$$

From the model system (4) we have

$$\begin{aligned} \frac{dV}{dt} = {} & \omega_1(1 - \frac{S_a^*}{S_a})[\Lambda_a - \mu_a S_a - \beta_a S_a I_a] + \omega_2(1 - \frac{E_a^*}{E_a})[\beta_a S_a I_a - (\varepsilon_a + \mu_a)E_a] \\ & + \omega_3(1 - \frac{I_a^*}{I_a})[\varepsilon_a E_a - (\mu_a + \gamma_a)I_a] + \omega_4(1 - \frac{W^*}{W})[\alpha_a(E_a + I_a) + \alpha_h(E_h + I_h) - \eta W N_h - \mu W] \\ & + \omega_5(1 - \frac{S_h^*}{S_h})[\Lambda_h - \mu_h S_h - \beta_h S_h I_h - \rho\eta W S_h] + \omega_6(1 - \frac{E_h^*}{E_h})[\beta_h S_h I_h + \rho\eta W S_h - (\varepsilon_a + \mu_h)E_h] \\ & + \omega_7(1 - \frac{I_h^*}{I_h})[\varepsilon_h E_h - (\mu_h + \gamma_h)I_h] \end{aligned} \tag{29}$$

Further simplification gives

$$\frac{dV}{dt} = -\omega_1\mu_a S_a(1 - \frac{S_a^*}{S_a})^2 - \omega_5\mu_h S_h(1 - \frac{S_h^*}{S_h})^2 + F(\Omega) \tag{30}$$

where $\Omega = (S_a, E_a, I_a, W, S_h, E_h, I_h) \geq 0$ and

$$
\begin{aligned}
F(\Omega) = &-\omega_1(1-\frac{S_a^*}{S_a})(1-\frac{S_a^*I_a^*}{S_aI_a}) + \omega_2\beta_aS_aI_a(1-\frac{E_a^*}{E_a})(1-\frac{I_a^*E_a}{I_aE_a^*}) + \omega_3\varepsilon_aE_a(1-\frac{I_a^*}{I_a})(1-\frac{E_a^*I_a}{E_aI_a^*}) \\
&+\omega_4(1-\frac{W^*}{W})[\alpha_aE_a(1-\frac{E_a^*W}{E_aW^*}) + \alpha_aI_a(1-\frac{I_a^*W}{I_aW^*}) + \alpha_hE_h(1-\frac{E_h^*W}{E_hW^*}) + \alpha_hI_h(1-\frac{I_h^*W}{I_hW^*})] \\
&-\omega_4\eta WN_h(1-\frac{W^*}{W})(1-\frac{N_h^*}{N_h}) - \omega_5\beta_hS_hI_h(1-\frac{S_h^*}{S_h})(1-\frac{S_h^*I_h^*}{S_hI_h}) - \omega_5\rho\eta W(1-\frac{S_h^*}{S_h})(1-\frac{S_h^*W^*}{S_hW}) \\
&+\omega_6\beta_hS_hI_h(1-\frac{E_h^*}{E_h})(1-\frac{S_h^*I_h^*E_h}{S_hI_hE_h^*}) + \omega_6\rho\eta WS_h(1-\frac{E_h^*}{E_h})(1-\frac{S_h^*E_hW^*}{S_hE_h^*W}) \\
&+\omega_7\varepsilon_hE_h(1-\frac{I_h^*}{I_h})(1-\frac{E_h^*I_h}{E_hI_h^*})
\end{aligned} \tag{31}
$$

Following the approach by Korobeinikov [38, 39] and [40], we find that $F(\Omega)$ is non-positive in Ω. Hence, $\frac{dV}{dt} \leq 0$ in Ω and is zero when $\omega = \Omega^*$. Therefore, the largest compact invariant set in Ω such that $\frac{dV}{dt} = 0$ is the singleton $\{\Omega^*\}$ which is the endemic equilibrium point. LaSalle's invariant principle then implies that Ω^* is globally asymptotically stable in the interior of Ω.

4. Numerical Simulations

In this section, we perform numerical simulation for the model system (4) and the basic reproduction number. Numerical simulation help to study the persistence of the disease when introduced in a closed or isolated system. The initial values used in simulations are $S_a = 1000$, $E_a = I_a = 1$, $W = 1000$, $S_h = 1000$, $E_h = I_h = 1, N_h = 1000$. The recruitment rate in the human and domestic animal population is approximated from the number of births for each population. The natural death rate for each population is approximated from the life span of each population. In some experimentally infected animals, clinical signs were reported to occur around the time cyst excretion begins. The incubation period is found to be 5 to 16 days in dogs and cats, and 3 to 10 days in ruminants [41]. For analysis, we use the incubation period of 10 days in domestic animals. In humans, the incubation period is generally 7 to 10 days although it may take 3 to 25 days for longer periods [42]. For analysis, we use the incubation period of 10 days. Other parameter values and their source are as given in Table 2.

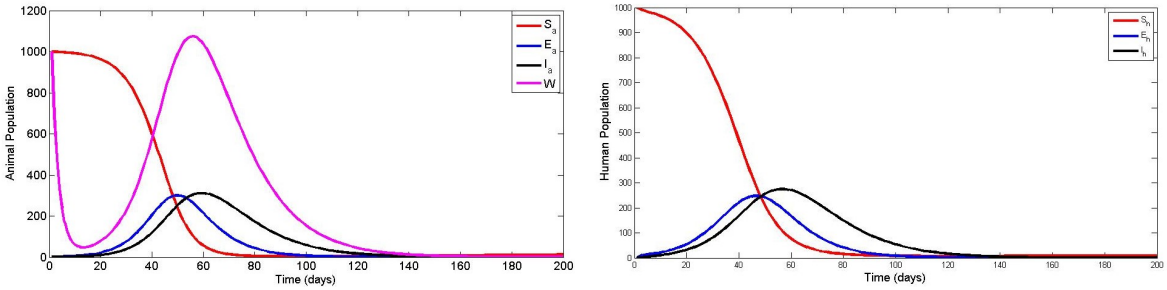
Fig. 2 shows the variations of domestic animal population (Fig. 2(a)) and the variations of human population

Table 2. Parameters and their description

Parameter	Description	Value	Source
Λ_a	recruitment rate in domestic animals	0.11	[43]
Λ_h	recruitment rate in humans	0.036	[44]
β_a	transmission rate in domestic animals	0.0005	Estimated
β_h	disease transmission rate in humans	0.00035	Estimated
$1/\varepsilon_a$	incubation period in domestic animals	10	[41]
$1/\epsilon_h$	incubation period in humans	10	[42]
μ_a	natural death rate of domestic animals	1/10/365	[43]
μ_h	natural death rate of humans	1/65/365	[44]
μ	natural decay rate of cysts in the environment	0.03	[33]
η	rate at which human ingest cysts from the environment	0.00034	[33]
ρ	rate of convention of ingested cysts to infection	0.02	[33]
α_a	rate at environmental contamination by domestic animals	0.49	[33]
α_h	rate of environmental contamination by humans	0.25	Estimated
γ_a	disease recovery rate in domestic animals	1/14	Estimated
γ_h	disease recovery rate in humans	1/14	[42]

(Fig. 2(b)) with respect to to time. The curves for S_a and S_h are the normal S-curves while the curve for W oscillates in the first two months before slowing down to equilibrium, and the curves for E_a, I_a, E_h and I_h are log-normal curves.

Using the parameter values in Table 2, we find that $\mathscr{R}_{0a} = 2.792$ and $\mathscr{R}_{0h} = 4.244$, therefore $\mathscr{R}_0 = max\{2.792, 4.244\} = 4.244$. When $\eta = 0$, $\mathscr{R}_{0h} = 4.181$, showing that the environment has little contribution to the transmission of *giardiasis* than person-to-person transmission. Fig. 3 shows the variation of $\mathscr{R}_{0h}$ with respect to $\eta = [0.0 - 0.00034]$ and $\rho = [0.0 - 0.02.]$ In the whole range, $\mathscr{R}_{0h}$ is between 4 and 4.5 which is greater than unity.



(a) Variations in domestic animal population (b) Variation in human population

Fig. 2. Variations in the domestic animal and human population

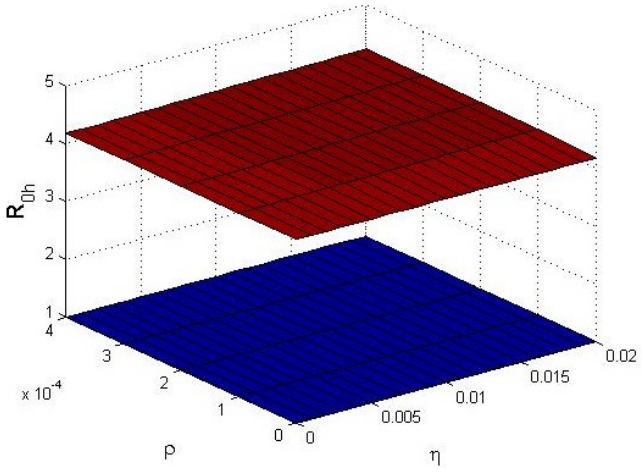


Fig. 3. Variations of $\mathscr{R}_{0h}$ with η and ρ

5. Discussion

In this paper, we used a modeling approach to investigate the dynamics of *giardiasis* coupled with a contaminated environment. To study the effect of initial transmission of the disease we computed the basic reproduction number $\mathscr{R}_0$ of the model and analysed the stability of the disease-free equilibrium and endemic equilibrium. The analysis of the stability of equilibrium points indicates that both the disease-free equilibrium and endemic equilibrium of the model system are locally and globally asymptotically stable. The stability of the disease-free equilibrium implies that the outbreak can be controlled provided that $\mathscr{R}_0 < 1$.

The impact of the rate at which humans ingest cyst from the environment, η and the rate of conversion of the ingested cyst into infection, ρ to $\mathscr{R}_0$ were also examined. It was observed that when $\eta = 0$, the $\mathscr{R}_0 = \mathscr{R}_{0h} = 4.181$ which is the basic reproduction number of person-to-person transmission. Increase in η and ρ cause an increase in $\mathscr{R}_0$.

To analyse the variation of each population in the model with respect to time we performed numerical simulations. The result from the numerical simulation shows that whenever there is an outbreak coupled with a contaminated environment, the disease is likely to persist in the first two months and then slow down to a disease-free equilibrium.

6. Conclusion

Giardiasis is the most common enteric protozoan infection worldwide, affecting both humans and animals. Its ability to remain endemic in most countries of the world is its nature of transmission. The rate at which humans ingest cyst η and the rate of conversion of the cyst into infection ρ have been seen to increase $\mathscr{R}_0$ to some extent. However, person-to-person transmission has remained a potential way of *giardiasis* transmission in human. Therefore, it is important to look into mechanisms that will reduce the person-to-person transmission at the same time reduce η and ρ to reduce $\mathscr{R}_0$. Effective educational campaign about the nature of the disease itself and its dynamics, and proper

hygiene may help to reduce.

Data availability statement

The set of parameter values are mainly from articles similar to the work, while the unavailable data especially values of parameter were estimated for the purpose of verifying results of the mathematical analysis of the model developed.

Conflict of Interest

The authors declares no conflict of interest regarding the publication of this manuscript.

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