

Research Article

Mathematical Modeling of Giardiasis Transmission Dynamics with Control Strategies in the Presence of Carriers

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Giardiasis is among the ignored zoonotic illnesses accorded by the World Health Organization that is caused by *Giardia duodenalis*. The disease is ignored regardless of the harm it causes to people and other creatures. In this paper, a mathematical model for *giardiasis* illness transmission is formed, which considers sickness carriers and control measures such as screening, treatment, and sanitation of the environment around people. In the assessment, the basic reproduction number, R_0 , which is used for analyzing the local stability of the equilibria is determined using the state-of-the-art next-generation matrix, while the Metzler constancy speculation is used to show the overall adequacy of the global stability of the equilibrium point free from the disease. In addition, a Lyapunov function has been used to study the stability of the endemic equilibrium point. The assessment of parameters is performed to explore the limits that significantly influence the transmission components of the disease disorders using the normalizing sensitivity index method. The result revealed that the recruitment rate is the most sensitive limit to the reproduction number. The environment-human interaction parameter is the second influential factor in the transmission of *giardiasis* in the community. In the same manner, the outcomes recommend that carriers assume an expected part in the rate of *giardiasis* subsequently; disregarding them could risk endeavors to control the pestilence. Besides, the mathematical recreation of the model shows that a mix of each of the three interventions fundamentally affects the control of *giardiasis*. In this way, we advise implementing the strategies simultaneously in endemic areas to effectively stop the spread of the *giardiasis* disease in humans.

1. Introduction

Giardiasis is a digestive contamination brought about by a parasite called *Giardia lamblia*, *Giardia gastrointestinal*, or *Giardia duodenalis*. The main cause of diarrhea in children below the age of five is *Giardia* protozoan parasites, particularly in poor countries. The life cycle of *Giardia* normally begins with the ingestion of cysts that are located in food and water contaminated by host feces. The transmission of diarrhea increases by using unsafe water and inadequate sanitation and hygiene during food preparation [1]. It is worth noting that only one excretion of the infected person can release as much as 10^9 cysts, while with only 10 cysts, *giardiasis* can begin in the community [2, 3]. At present, there are eight groups of genes from A to H that are recog-

nized for *giardiasis*, but only A and B are species that are unfavorable for humans, and other genotypes C to H mostly infect animals. Symptoms during infections might include severe diarrhea, nutrient malabsorption, cognitive and developmental defects, fever, itchy skin, weight loss, stomach cramps, greasy poop that can float, an upset stomach, or nausea, to mention a few [4, 5].

The parasite infects both developing and developed countries, with widespread in developing countries. According to the World Health Organization, this disease has been designated as an ignored Diseases Initiative in September 2004 for its high burden and association with poverty [6]. The incidence of *giardiasis* worldwide is estimated to be 2.8×10^8 cases per year. Around 200 million people have been detected with *giardiasis* symptoms due to poor

sanitation and access to safe drinking water in developing countries. The high incidence of disease in developing countries is due to the fact that most African countries are facing difficulties with accurately identifying, detecting, and reporting infectious diseases as a result of remote communities, poor transport, and a shortage of qualified health workers and laboratory facilities for accurate diagnosis [5, 7].

The risk of *giardiasis* contamination accelerates with the consumption of raw food. That is why international recommendations provide innocuousness in food preparations, like practicing appropriate hand hygiene for protection against protozoan parasites and maintaining food packed or closed. Also, it is insisted on separating raw from cooked food, using purified or boiled water, and making sure that food is cooked at a temperature of more than 70°C [8, 9].

A substantial contribution has been made in mathematics modeling for a better understanding of the epidemiology and dynamics of diseases, e.g., *giardiasis*. Saul and Nyerere [10] formulate a *giardiasis* mathematical model that includes humans, domestic animals, and contaminated environments in order to assess the dynamics of the disease. The results showed that the transmission from individual to individual is the most significant in the dynamics of *giardiasis*. The *giardiasis* mathematical model describing the *Giardia* transmission dynamics in rural Australia has also been developed by Waters et al. [16]. The study showed that the endemic infection of an animal with zoonotic protozoa can lead to epidemic infections in humans, although there is no human-to-human transmission. These findings demonstrate the importance of transmissible zoonotic species via environmental reservoirs. In addition, Li et al. [11] developed a mouse model of infection to investigate immunity against secondary infections caused by *Giardia duodenalis*. The study suggests that, because of the emergence of robust immunity to reinfection, an effective *giardiasis* vaccine can be developed in adult mouse models. Despite a number of studies looking at the dynamics of this disease, *giardiasis* is still affecting many people in poor countries. To achieve the eradication of this disease, current control interventions need to be assessed. Thus, if we are to eradicate or curb the disease, there is a need to assess the present control interventions. Until now, there are no previous studies that have considered screening, treatment, and sanitation interventions to study the dynamics of *giardiasis*. The purpose of the study is thus to gain insight into the impact of screening, treatment, and sanitation on *giardiasis* transmission dynamics.

The order of the manuscript is as follows: the model is formulated in Section 2, with a dynamical analysis of the mode in Section 3. The model sensitivity analysis with its interpretation has been done in Section 4. Finally, we performed numerical simulation and discussion in Section 5, which leads us to the conclusion of Section 6.

2. Model Description and Formulation

The human population is subdivided into five groups, including susceptible individuals $S(t)$, exposed individuals $E(t)$, infected individuals $I(t)$, carrier or asymptomatic individuals $A(t)$, and the removed population $R(t)$, as described

in the basic model in Figure 1. The susceptible state is the state that involves healthy individuals who are at risk of getting *giardiasis* disease. The exposed population involves individuals who have been infected but have not yet developed clinical symptoms of the disease and are incapable of infecting other humans. The infectious individuals represent individuals who are actively infected and manifest all clinical symptoms of the disease. The infected individuals are capable of infecting other individuals through unhygienic interactions as well as shedding *giardiasis* pathogens into the environment (food and water). The infected individual under this transmission is a key player in the zoonotic aspect. Transmission of *Giardia* normally occurs through the ingestion of infectious cyst stages that are excreted in human or animal feces. *Cysts* may be present in water, food, or utensils contaminated with feces from humans and animals. The infected individual can then pollute the environment (food and water) with the *Giardia* through touching and making it unhygienic if proper controls like washing hands before eating and improper handling of food are not well maintained [12, 13]. The fourth state is the asymptomatic population $A(t)$. This population includes carrier individuals who do not show clinical signs of the disease but transmit the *Giardia* pathogens to other individuals through shedding pathogens into the environment in direct or indirect ways. The recovered population resents individuals who recover after gaining immune-supportive services, treatment, and/or naturally. The *Giardia* population is represented by the letter $G(t)$. The human population is recruited by birth and the loss of immunity at the rates Λ and ρ , respectively. A susceptible human acquires pathogens from both the environment and infected individuals with the force of infections λ that follows the standard mass action principle. Therefore, λ is the combination of three forces of infection which is defined as follows:

$$\lambda = (\beta_1 I + \beta_2 A + \beta_3 G)S. \quad (1)$$

The parameter β_1 represents the direct transmission of the disease pathogens from the infected person to the susceptible person; β_2 represents the direct transmission of the disease pathogens from the carrier person to the susceptible person. The indirect pathway of disease pathogens from the environment to a susceptible person is represented by a parameter β_3 . Once infected, the *Giardia* pathogens incubate for 1–4 weeks in the human intestine and thus progress and become infectious at the incubation rate $(1 - \alpha)\kappa E$, and the remaining portion, $\alpha\kappa E$, becomes asymptomatic to the disease. Also, a portion pI and σI recovers naturally and by treatment, respectively, while ηI become asymptomatic or carriers of the *Giardia* pathogens. In addition, the infected human can die at the rate ψ . The carrier or asymptomatic person can recover naturally at the rate γ and die at the rate δ . The general population is subjected to screening in order to identify any potential carriers; consequently, the identified carriers join the infectious population at the rate ν . The recovered individuals are reduced by the individuals who become susceptible after losing immunity at the waning rate ρ . Moreover, both the infected and asymptomatic

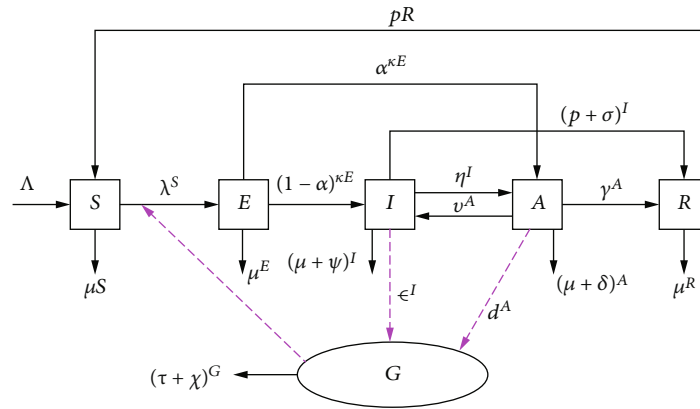


FIGURE 1: A flowchart on the transmission of *giardiasis* disease in human and unclean environment. The solid line shows a transfer of persons from one compartment to another. The dotted lines show cysts being excreted by infected and carrier individuals in the environment. Another dotted arrow shows the force of infections from the unhygienic environment (food and water) to the susceptible human population.

individuals can shed *Giardia* pathogens at the rates ϵ and d , respectively. All human populations are also naturally reduced at the rate μ . The *Giardia* population G grows through deposited pathogens from both the infected and asymptomatic human populations into the environment. The cysts are reduced from the environment by death at a rate of τ .

From the descriptions of the parameters in Table 1 and the model in Figure 1, a system of the nonlinear ordinary differential equations is formulated as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda + \rho R - (\beta_1 I + \beta_2 A + \beta_3 G)S - \mu S, \\
 \frac{dE}{dt} &= (\beta_1 I + \beta_2 A + \beta_3 G)S - (\mu + \kappa)E, \\
 \frac{dI}{dt} &= (1 - \alpha)\kappa E + vA - (\eta + \psi + \mu + p + \sigma)I, \\
 \frac{dA}{dt} &= \alpha\kappa E + \eta I - (\mu + v + \delta + \gamma)A, \\
 \frac{dR}{dt} &= (p + \sigma)I + \gamma A - (\mu + \rho)R, \\
 \frac{dG}{dt} &= \epsilon I + dA - (\tau + \chi)G,
 \end{aligned}
 \tag{2}$$

together with nonnegative initial conditions

$$\begin{aligned}
 S(0) &> 0, \\
 E(0) &\geq 0, \\
 I(0) &\geq 0, \\
 R(0) &\geq 0, \\
 A(0) &> 0, \\
 G(0) &> 0.
 \end{aligned}
 \tag{3}$$

2.1. The Basic Characteristics of the Giardiasis Model

2.1.1. Positivity of the Solution. The positivity and boundedness of the solutions of the model system (2) are tested to see if the model is well-posed and biologically meaningful [17, 18]. Here, it suffices to see that all variables of the model are positive and well-posed in the invariant region.

$\Omega = \{S, E, I, A, R, G \in \mathbb{R}_+^6 : S + E + I + A + R \leq N\}$, where N is the total human population, and, hence the following Lemma:

Lemma 1. Given $S(0) \geq 0, E(0), I(0) > 0, A(0) > 0, R(0) > 0$, and $G(0) \geq 0$, the region $\Omega = (S(t), E(t), I(t), R(t), G(t) \in \mathbb{R}^6)$ of the model (1) positively invariant $\forall t \in \mathbb{R}^+$.

Proof. The model is biologically meaningful if state variables are nonnegative $\forall t > 0$. Now, by using the first equation of the model (2)

$$\frac{dS(t)}{dt} = \Lambda + \rho R(t) - \lambda S(t) - \mu S(t),
 \tag{4}$$

we then have

$$\frac{dS(t)}{dt} \geq -(\lambda + \mu)S(t),
 \tag{5}$$

integrating equation (5) using separation of variables techniques results in

$$S(t) \geq S(0)e^{-(\lambda+\mu)t}.
 \tag{6}$$

Therefore, using equation (6) as $t \rightarrow \infty, S(0) \geq 0$. Similarly, other model variables can be shown and verified non-negative. This concludes that all equations of the model system (2) have nonnegative solutions such that for $\forall t \geq 0$, we have $S(t) \geq 0, E(t) \geq 0, I(t) \geq 0, R(t) \geq 0$, and $G(t) \geq 0$. \square

2.1.2. Invariant Region. According to proposition 4.1 of [18, 19], it immediately follows that \mathbb{R}^6 is positively invariant for the dynamical system (2) which means that any trajectory

TABLE 1: Parameter descriptions of the model system (2).

Parameter	Description
Λ	Recruitment rate of human
ρ	Warning rate of removed individuals
β_1	The rate of transmission between susceptible and infected humans
β_2	The rate of transmission among asymptomatic and susceptible human beings
β_3	Transmission rate between the environment and the susceptible human
K	Half saturation constant in the environment
κ	Incubation period of pathogens in human
μ	Natural death rate of human
α	Probability of becoming infectious to giardiasis
ν	Progression rate of asymptomatic individuals to infectious state/screening rate
γ	Natural recovery rate of asymptomatic human
η	Progression rate of infected to asymptomatic stage in human
ψ	Death of infected human due to giardiasis
ρ	Natural recovery rate of infected human
δ	Death rate of asymptomatic human due to giardiasis
ε	Shedding rate of <i>Giardia</i> by the infected human into the environment
d	Shedding rate of <i>Giardia</i> by the asymptomatic human into the environment
τ	Death rate of the pathogens from the environment
σ	Treatment rate of the infectious individuals
χ	Sanitation rate of the environment

starting from the region \mathbb{R}_+^6 remains inside \mathbb{R}_+^6 for the time $t = [0, T)$.

Proof. Using the dynamical system (2) and initial conditions $x_0 \in \Omega$, the sum of human population $N(t)$ is as follows:

$$N(t) = S(t) + E(t) + I(t) + A(t) + R(t),$$

$$\frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dA(t)}{dt} + \frac{dR(t)}{dt} = \Lambda - \mu N - \psi I - \delta A. \quad (7)$$

Using the positivity of the solution, at disease-free, $\psi = \delta = 0$. Hence,

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t). \quad (8)$$

As a result, the general formula for equation (8) is given as follows in applying the variable separation technique:

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}. \quad (9)$$

As $t \rightarrow \infty$, then $\lim_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$ that is $0 \leq N(t) \leq \Lambda/\mu$. Hence, the initial conditions are bounded in time $t = [0, T)$.

Also, by considering the *Giardia* pathogens population, we have

$$\frac{dG(t)}{dt} = \varepsilon I(t) + dA(t) - \tau G(t) \leq \frac{\varepsilon \Lambda}{\mu} - \tau G(t). \quad (10)$$

Hence, the solution $0 \leq \liminf_{t \rightarrow \infty} G(t) \leq \limsup_{t \rightarrow \infty} G(t) \leq \varepsilon \Lambda/\mu$. This shows that all solutions $(S(t), E(t), I(t), A(t), R(t), \text{ and } G(t))$ of the model system (2) are attracted in the invariant region $\Omega = \{(S, E, I, A, R \in \mathbb{R}_+^5) : 0 \leq N(t) \leq \Lambda/\mu ; G(t) \in \mathbb{R}_+^1 : 0 \leq G \leq \varepsilon \Lambda/\mu\}$. Therefore, model system (2) is biologically meaningful. \square

3. Dynamical Analysis of the Model

3.1. Equilibrium Point Free from Disease. The model system (2) has a unique equilibrium point free from disease which is obtained by putting all of the infected classes equal to zero, that is, at an equilibrium point free from disease $E = I = A = 0$ and $G = 0$. Thus, solving the system is given by

$$E_0 = (S^0, E^0, I^0, A^0, R^0, G^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0 \right). \quad (11)$$

3.2. *Basic Reproductive Number.* The basic reproductive number R_0 referred to the number of secondary cases caused by one infectious person during the entire [20] in a totally susceptible population. Using the next-generation operator method, the basic reproduction number is calculated. It shall be obtained by taking a dominant eigenvalue from the matrix. It is achieved by taking a lead eigenvalue from the matrix

$$\left[\frac{\partial \mathcal{F}_i(E_0)}{\partial x_j} \right] \cdot \left[\frac{\partial \mathcal{V}_i(E_0)}{\partial x_j} \right]^{-1} = FV^{-1}, \quad (12)$$

where \mathcal{F}_i is the rate of appearance of new infection in compartment i , $\mathcal{V}_i = \mathcal{V}_i^- - \mathcal{V}_i^+$ is the transfer rate of individuals from one compartment i to another, with \mathcal{V}_i^- denoting the rate of transfer of individuals out of the compartment i , \mathcal{V}_i^+ is the rate of transfer of individuals into compartment i , x_j is the infected classes (E , I , A , and G) of model system (2), and E_0 is the disease-free equilibrium point. For the model system (2), the infectious classes are as follows:

$$\begin{aligned} \frac{dE}{dt} &= \beta_1 IS + \beta_2 AS + \beta_3 GS - \phi_1 E, \\ \frac{dI}{dt} &= \phi_5 \kappa E + \nu A - \phi_2 I, \\ \frac{dA}{dt} &= \alpha \kappa E + \eta I - \phi_3 A, \\ \frac{dG}{dt} &= \varepsilon I + dA - \phi_6 G, \end{aligned} \quad (13)$$

where

$$\begin{aligned} \phi_1 &= \mu + \kappa, \\ \phi_2 &= \eta + \psi + \mu + \rho + \sigma, \\ \phi_3 &= \mu + \nu + \delta + \gamma, \\ \phi_4 &= \mu + \rho, \\ \phi_5 &= 1 - \alpha, \\ \phi_6 &= \tau + \chi, \\ \phi_7 &= \rho + \sigma. \end{aligned} \quad (14)$$

From the system (13), we obtain

$$\mathcal{F}_i = \begin{bmatrix} \beta_1 IS + \beta_2 AS + \beta_3 GS \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad (15)$$

$$\mathcal{V}_i = \begin{bmatrix} \phi_1 E \\ \phi_2 I - \phi_5 \kappa E - \gamma A \\ \phi_3 A - \alpha \kappa E - \eta I \\ \phi_6 G - \varepsilon I - dA \end{bmatrix}.$$

Then, the partial derivative of \mathcal{F}_i and \mathcal{V}_i with respect to E , I , A , and G evaluated at E_0 gives

$$F = \begin{pmatrix} 0 & \frac{\Lambda \beta_1}{\mu} & \frac{\Lambda \beta_2}{\mu} & \frac{\Lambda \beta_3}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad (16)$$

$$V = \begin{pmatrix} \phi_1 & 0 & 0 & 0 \\ -\kappa \phi_5 & \phi_2 & -\gamma & 0 \\ -\alpha \kappa & -\eta & \phi_3 & 0 \\ 0 & -\varepsilon & -d & \phi_6 \end{pmatrix},$$

$$V^{-1} = \begin{pmatrix} \frac{\phi_2 \phi_3 \phi_6 - \gamma \eta \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & 0 & 0 & 0 \\ \frac{\alpha \gamma \kappa \phi_6 + \kappa \phi_3 \phi_5 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\phi_1 \phi_3 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\gamma \phi_1 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & 0 \\ \frac{\alpha \kappa \phi_2 \phi_6 + \eta \kappa \phi_5 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\eta \phi_1 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\phi_1 \phi_2 \phi_6}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & 0 \\ \frac{\alpha \gamma \varepsilon \kappa + d \alpha \phi_2 \kappa + d \eta \phi_5 \kappa + \varepsilon \phi_3 \phi_5 \kappa}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{d \eta \phi_1 + \varepsilon \phi_3 \phi_1}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\gamma \varepsilon \phi_1 + d \phi_2 \phi_1}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} & \frac{\phi_1 \phi_2 \phi_3 - \gamma \eta \phi_1}{\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6} \end{pmatrix}. \quad (17)$$

It follows that the reproduction number R_0 is the spectral radius of the next-generation matrix $\rho(FV^{-1})$ given as

$$R_0 = R_{e1} + R_{e2} + R_{e3}, \tag{18}$$

given that

$$\begin{aligned} R_{e1} &= \frac{\beta_1 \Lambda (\alpha \gamma \kappa \phi_6 + \kappa \phi_3 \phi_5 \phi_6)}{\mu (\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6)}, \\ R_{e2} &= \frac{\beta_2 \Lambda (\alpha \kappa \phi_2 \phi_6 + \eta \kappa \phi_5 \phi_6)}{\mu (\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6)}, \\ R_{e3} &= \frac{\beta_3 \Lambda (\alpha \gamma \kappa \varepsilon + \alpha d \kappa \phi_2 + d \eta \kappa \phi_5 + \kappa \varepsilon \phi_3 \phi_5)}{\mu (\phi_1 \phi_2 \phi_3 \phi_6 - \gamma \eta \phi_1 \phi_6)}, \end{aligned} \tag{19}$$

where $R_{ei} (i=1, 2, 3)$ are partial reproduction numbers induced by being susceptible to infectious transmission, susceptible to asymptomatic carrier transmission, and susceptible to environmental transmission, respectively.

3.3. Local Stability of an Equilibrium Point Free from Disease. Local stability of an equilibrium point free from the disease E_0 is determined by first finding the Jacobian matrix of the model system (2) concerning each state variable (i.e., $S, E, I, A, R,$ and G). Based on the sign of the real parts of the eigenvalues evaluated at E_0 of the Jacobian matrix, the stability of the model system (2) will be evaluated. The partial differentiation of the model system (2) with respect to $S, E, I, A, R,$ and G at E_0 gives the Jacobian matrix $J(E_0)$ as

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\frac{\Lambda \beta_1}{\mu} & -\frac{\Lambda \beta_2}{\mu} & \rho & -\frac{\Lambda \beta_3}{\mu} \\ 0 & -\phi_1 & \frac{\Lambda \beta_1}{\mu} & \frac{\Lambda \beta_2}{\mu} & 0 & \frac{\Lambda \beta_3}{\mu} \\ 0 & \kappa \phi_5 & -\phi_2 & \nu & 0 & 0 \\ 0 & \alpha \kappa & \eta & -\phi_3 & 0 & 0 \\ 0 & 0 & \phi_7 & \gamma & -\phi_4 & 0 \\ 0 & 0 & \varepsilon & d & 0 & -\phi_6 \end{pmatrix}, \tag{20}$$

where $\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6,$ and ϕ_7 have the same meaning as in (14).

From matrix (20), the first columns have diagonal entries. Therefore, the diagonal $-\mu$ is the first eigenvalue of the Jacobian matrix (20). Thus, excluding that column and row containing $\lambda_1 = -\mu$, the rest eigenvalues are calculated. Then, the reduced 5×5 matrix from (20) becomes

$$\xi = \begin{pmatrix} -\phi_1 & \frac{\Lambda \beta_1}{\mu} & \frac{\Lambda \beta_2}{\mu} & 0 & \frac{\Lambda \beta_3}{\mu} \\ \kappa \phi_5 & -\phi_2 & \nu & 0 & 0 \\ \alpha \kappa & \eta & -\phi_3 & 0 & 0 \\ 0 & \phi_7 & \gamma & -(\mu + \rho) & 0 \\ 0 & \varepsilon & d & 0 & -\phi_6 \end{pmatrix}. \tag{21}$$

Again, it is easily seen that one eigenvalue of matrix ξ is $\lambda_2 = \mu + \rho$. After omitting the diagonal entry of matrix ξ , the matrix (20) is reduced to 4×4 matrix and becomes

$$\Psi = \begin{pmatrix} -\phi_1 & \frac{\Lambda \beta_1}{\mu} & \frac{\Lambda \beta_2}{\mu} & 0 - \frac{\Lambda \beta_3}{\mu} \\ \kappa \phi_5 & -\phi_2 & \nu & 0 \\ \alpha \kappa & \eta & -\phi_3 & 0 \\ 0 & \varepsilon & d & 0 - \phi_6 \end{pmatrix}, \tag{22}$$

and the characteristic polynomial for the remaining matrix (22) is

$$\mathcal{D}(\lambda) = \lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4. \tag{23}$$

The corresponding Routh-Hurwitz matrix of the polynomial (23) is

$$\mathcal{D}_4 = \begin{pmatrix} b_1 & b_3 & 0 & 0 \\ 1 & b_2 & b_4 & 0 \\ 0 & b_1 & b_3 & 0 \\ 0 & 1 & b_2 & b_4 \end{pmatrix}, \tag{24}$$

where

$$\begin{aligned} b_1 &= \phi_1 + \phi_2 + \phi_3 + \phi_6, \\ b_2 &= -\frac{\alpha \beta_2 \kappa \Lambda}{\mu} - \frac{\beta_1 \kappa \Lambda \phi_5}{\mu} - \eta \nu + \phi_1 \phi_2 + \phi_1 \phi_3 + \phi_2 \phi_3 + \phi_1 \phi_6 + \phi_2 \phi_6 + \phi_3 \phi_6, \\ b_3 &= -\frac{\alpha \beta_1 \kappa \Lambda \nu}{\mu} - \frac{\alpha \beta_2 \kappa \Lambda \phi_2}{\mu} - \frac{\beta_2 \eta \kappa \Lambda \phi_5}{\mu} - \frac{\beta_1 \kappa \Lambda \phi_3 \phi_5}{\mu} - \frac{\beta_3 \kappa \Lambda \varepsilon \phi_5}{\mu} - \frac{\alpha \beta_3 d \kappa \Lambda}{\mu} - \eta \nu \phi_1 + \phi_1 \phi_2 \phi_3 - \frac{\alpha \beta_2 \kappa \Lambda \phi_6}{\mu} - \frac{\beta_1 \kappa \Lambda \phi_5 \phi_6}{\mu} - \eta \nu \phi_6 + \phi_1 \phi_2 \phi_6 + \phi_1 \phi_3 \phi_6 + \phi_2 \phi_3 \phi_6, \\ b_4 &= \phi_1 \phi_2 \phi_3 \phi_6 - \eta \nu \phi_1 \phi_6 (1 - R_0). \end{aligned} \tag{25}$$

The equilibrium point free from the disease is locally asymptotically stable if the principal leading minors of \mathcal{D}_n are all positive for $n = 1, 2, \dots, 4$. Thus,

$$\begin{aligned} \Delta\mathcal{D}_1 &= b_1 = \phi_1 + \phi_2 + \phi_3 + \phi_6, 0, \\ \Delta\mathcal{D}_2 &= \begin{vmatrix} b_1 & b_3 \\ 1 & b_2 \end{vmatrix} = b_1 b_2 - b_3, \\ \Delta\mathcal{D}_3 &= \begin{vmatrix} b_1 & b_3 & 0 \\ 1 & b_2 & b_4 \\ 0 & b_1 & b_3 \end{vmatrix} = -b_4 b_1^2 + b_2 b_3 b_1 - b_3^2, \\ \Delta\mathcal{D}_4 &= \begin{vmatrix} b_1 & b_3 & 0 & 0 \\ 1 & b_2 & b_4 & 0 \\ 0 & b_1 & b_3 & 0 \\ 0 & 1 & b_2 & b_4 \end{vmatrix} = -b_4 b_3^2 + b_1 b_2 b_4 b_3 - b_1^2 b_4^2. \end{aligned} \tag{26}$$

Then, the remaining four eigenvalues of the Jacobian matrix (22) have negative real parts if they satisfy the Routh-Hurwitz criteria [21, 22], that is, $\Delta\mathcal{D}_i > 0$, $i = 1, 2, \dots, 4$. It can be noted that $\Delta\mathcal{D}_1 > 0$ since $\phi_1 + \phi_2 + \phi_3 + \phi_6 > 0$, $\Delta\mathcal{D}_2 > 0$ if $b_1 b_2 b_3 > 0$, $\Delta\mathcal{D}_3 > 0$ if $b_1 b_2 b_3 b_1^2 b_4 + b_3^2 > 0$ and $\Delta\mathcal{D}_4 > 0$ if $R_0 > 1$ and $b_1 b_2 b_3 b_4 b_3^2 + b_1^2 b_4^2 > 0$.

The following theorem is therefore established.

Theorem 2. *If the leading minors of $\Delta\mathcal{D}_n$ are all positive, then the equilibrium point free from the disease E_0 of the model system (2) is locally asymptotically stable when $R_0 > 1$.*

Biologically, it indicates that *giardiasis* can be eliminated from the population provided that the size of the populations of the model (2) is on the basis of the attraction of an equilibrium point free from the disease E_0 when $R_0 > 1$, that is to say, an outbreak does not arise when infectious human beings are introduced into a community of susceptible individuals. By contrast, if $R_0 < 1$ it is surely that *giardiasis* in the population, then it means that this disease is persisting.

3.4. Global Stability of the Equilibrium Point Free from the Disease. The approach of Castillo-Chavez is applied to analyze the global stability of the disease-free equilibrium solution of the model system (2) (Castillo et al. [23]). Using this approach, the model system (2) can be written as follows:

$$\begin{cases} \frac{dH_n}{dt} = D_1(H_n - H_{E_0}) + D_{12}H_i, \\ \frac{dH_i}{dt} = D_2H_i, \end{cases} \tag{27}$$

where H_n stands for classes of at-risk individuals such as $H_n = (S, R)^T$. H_i stands for carrier and infected individuals, that is, $H_i = (E, I, A, G)^T$, where T stands for transpose of H_n and H_i while H_{E_0} is H_n at equilibrium point free from the disease E_0 . The matrices D_1 and D_{12} are obtained by differentiating the nontransmitting equations of the model system (2) to nontransmitting and transmitting state variables, respectively. The equilibrium point free from the disease E_0 is globally asymptotically stable if the real part of the eigenvalues of D_1 is negative and D_2 is a Metzler matrix (that is, the off-diagonal elements of D_2 are nonnegative). Thus, from the model system (2)

$$\begin{aligned} D_1 &= \begin{pmatrix} -\mu & \rho \\ 0 & -\phi_4 \end{pmatrix}, \\ D_{12} &= \begin{pmatrix} 0 & -\frac{\Lambda\beta_1}{\mu} & -\frac{\Lambda\beta_2}{\mu} & -\frac{\Lambda\beta_3}{\mu} \\ 0 & \phi_7 & \gamma & 0 \end{pmatrix}. \end{aligned} \tag{28}$$

The eigenvalues of D_1 are $-\mu$ and $-(\mu + \rho)$. Furthermore, the matrix D_2 is obtained by differentiating the transmitting equations of the model system (2) with respect to transmitting variables at the equilibrium point free from the disease, and it is given as follows:

$$D_2 = \begin{pmatrix} -\phi_1 & \frac{\Lambda\beta_1}{\mu} & \frac{\Lambda\beta_2}{\mu} & \frac{\Lambda\beta_3}{\mu} \\ \phi_5\kappa & -\phi_2 & \nu & 0 \\ \alpha\kappa & \eta & -\phi & 0 \\ 0 & \varepsilon & d & -\phi_6 \end{pmatrix}. \tag{29}$$

It can be noted, that the eigenvalues of D_1 are all negative and real. In addition, the matrix D_2 is a Metzler matrix since all of its off-diagonal elements are positive. This shows that at the equilibrium point free from the disease, $dH_n/dt = D_1(H_n - H_{E_0}) + D_{12}H_i$ is globally asymptotically stable.

3.5. The Endemic Equilibrium Point. The *giardiasis* present equilibrium point is a point $\mathcal{E}^* = \{S^*, E^*, I^*, R^*, A^*\}$ that found when the model system (2) is set to zero and solved simultaneously [24] in conditions that $I^* \neq 0$, $E \neq 0$, and $R^* \neq 0$, where

$$\begin{aligned} S^* &= \frac{\Lambda\phi_1\phi_3 + \rho\phi_1\phi_3\phi_7I^* + \rho\gamma\eta\phi_1I^* + \rho\gamma\alpha\kappa\phi_1\lambda^*}{\phi_1\phi_3\phi_4(\lambda^* + \mu)}, \\ E^* &= \frac{\lambda^*}{\phi_1}, \\ I^* &= \frac{\phi_1\phi_2\phi_3\nu\alpha\kappa\lambda^*}{\phi_1\phi_2\phi_3 - \phi_3\phi_5\kappa\lambda^*}, \end{aligned}$$

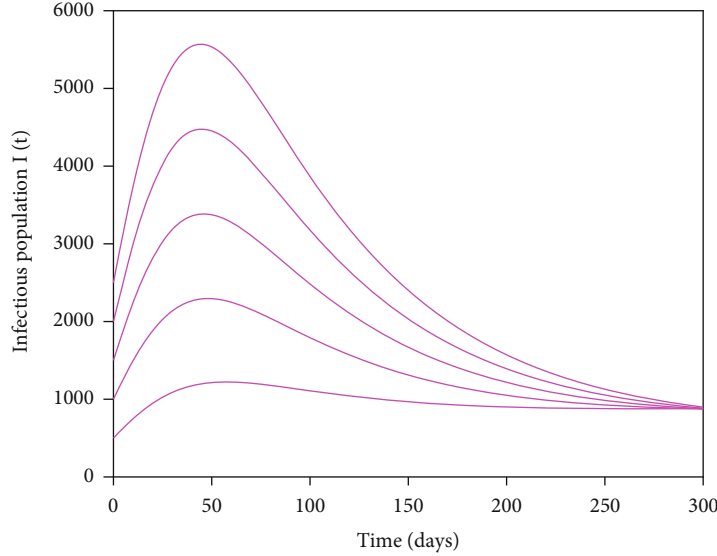


FIGURE 2: Dynamics of the infectious population for $R_0 > 1$. Using different initial values of $I(t)$, the phase portraits converge to the endemic equilibrium point.

$$\begin{aligned}
 R^* &= \frac{\phi_1 \phi_7 \phi_3 I^* + \gamma \eta \phi_1 I^* + \gamma \kappa \lambda^*}{\phi_1 \phi_3 \phi_4}, \\
 G^* &= \frac{\varepsilon \phi_1 \phi_3 I^* + d \eta \phi_1 I^* + d \alpha \kappa \phi_6 \lambda^*}{\phi_1 \phi_3 \phi_6}, \\
 A^* &= \frac{\eta \phi_1 I^* + \alpha \kappa \lambda^*}{\phi_1 \phi_3}, \\
 \lambda^* &= (\beta_1 I^* + \beta_2 A^* + \beta_3 G^*) S^*.
 \end{aligned} \tag{30}$$

3.6. Stability of Endemic Equilibrium Point

Theorem 3. The endemic equilibrium point \mathcal{E}^* of the model system (2) is globally asymptotically stable if $R_0 > 1$.

Proof. Consider the following Lyapunov function as used in [18, 24] with state variables x^* and nonnegative Lyapunov constants $w_i (i = 1, 2, \dots, 6)$,

$$\begin{aligned}
 L &= w_1 \left(S - S^* \ln \frac{S}{S^*} \right) + w_2 \left(E - E^* \ln \frac{E}{E^*} \right) \\
 &+ w_3 \left(I - I^* \ln \frac{I}{I^*} \right) + w_4 \left(A - A^* \ln \frac{A}{A^*} \right) \\
 &+ w_5 \left(R - R^* \ln \frac{R}{R^*} \right) + w_6 \left(G - G^* \ln \frac{G}{G^*} \right),
 \end{aligned} \tag{31}$$

then the first derivative of function L is

$$\begin{aligned}
 \frac{dL}{dt} &= w_1 \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + w_2 \left(1 - \frac{E^*}{E} \right) \frac{dE}{dt} \\
 &+ w_3 \left(1 - \frac{I^*}{I} \right) \frac{dI}{dt} + w_4 \left(1 - \frac{A^*}{A} \right) \frac{dA}{dt} \\
 &+ w_5 \left(1 - \frac{R^*}{R} \right) \frac{dR}{dt} + w_6 \left(1 - \frac{G^*}{G} \right) \frac{dG}{dt}.
 \end{aligned} \tag{32}$$

By using the model system (2), equation (32) is expressed as follows:

$$\begin{aligned}
 \frac{dL}{dt} &= w_1 \left(1 - \frac{S^*}{S} \right) (\Lambda + \rho R - (\beta_1 I + \beta_2 A + \beta_3 G) S - \mu S) \\
 &+ w_2 \left(1 - \frac{E^*}{E} \right) ((\beta_1 I + \beta_2 A + \beta_3 G) S - \phi_1 E) \\
 &+ w_3 \left(1 - \frac{I^*}{I} \right) (\phi_5 \kappa E + \nu A - \phi_2 I) \\
 &+ w_4 \left(1 - \frac{A^*}{A} \right) (\alpha \kappa E + \eta I - \phi_3 A) \\
 &+ w_5 \left(1 - \frac{R^*}{R} \right) (\phi_7 I + \gamma A - \phi_7 R) \\
 &+ w_6 \left(1 - \frac{G^*}{G} \right) (\varepsilon I + dA - \phi_6 G).
 \end{aligned} \tag{33}$$

□

At the equilibrium point, we have the following constants:

$$\begin{aligned}
 \Lambda &= (\beta_1 I^* + \beta_2 A^* + \beta_3 G) S^* + \mu S^* - \rho R^*, \\
 (\mu + \kappa) E^* &= \beta_1 I^* + \beta_2 A^* + \beta_3 G^*, \\
 \phi_2 I^* &= \phi_5 \kappa E + \nu A, \\
 \phi_3 A^* &= \alpha \kappa E + \eta I, \\
 \phi_7 R^* &= \phi_7 I + \gamma A, \\
 \phi_6 G &= \varepsilon I + dA.
 \end{aligned} \tag{34}$$

TABLE 2: Sensitivity index values of R_0 using parameter values in Table 3.

Parameter	Sensitivity index value	Parameter	Sensitivity index value
Λ	+1	η	+0.0963
β_1	+0.36	ψ	-0.0098
β_2	+0.6692	ρ	-0.0703
β_3	+0.9996	δ	-0.0179
κ	-0.2047	ε	+0.1482
μ	-0.9999	d	+0.5162
α	-0.0297	τ	-0.4942
ν	+0.0928	σ	-0.5982
γ	-0.1721	χ	-0.3295

By inserting the constants (34) into equation (33) we then have

$$\begin{aligned} \frac{dL}{dt} = & w_1\mu S \left(1 - \frac{S^*}{S}\right) \left[\left(\frac{S^*}{S} - 1\right) - \left(\frac{\lambda - \lambda^*}{\mu S}\right) \right] \\ & - \phi_2 w_2 E \left(1 - \frac{E^*}{E}\right)^2 - \phi_2 w_3 I \left(1 - \frac{I^*}{I}\right)^2 \\ & - \phi_3 w_4 A \left(1 - \frac{A^*}{A}\right)^2 - \phi_7 w_5 R \left(1 - \frac{R^*}{R}\right)^2 \\ & - \phi_6 w_6 G \left(1 - \frac{G^*}{G}\right)^2. \end{aligned} \tag{35}$$

The function (35) became less than or equal to zero when $S = S^*$, $E = E^*$, $I = I^*$, $A = A^*$, $R = R^*$, and $G = G^*$, following the methodology employed in [18, 24, 25]. As a result, the endemic equilibrium point, (\mathcal{E}^*) , is the singleton with the largest compact invariant set in the domain for which $dL/dt = 0$. We demonstrate that the endemic equilibrium point \mathcal{E}^* is globally asymptotically stable in the invariant set Ω for $R_0 > 1$ as noted in the work of Mukandavire et al. [25] and LaSalle [26]. Numerically, Figure 2 shows the phase portraits that are asymptotically converging to the endemic point.

4. Sensitivity Analysis

In this section, we study the robustness of the model parameters through sensitivity analysis using the basic reproduction number R_0 . The analysis helps identify which parameters require greater attention to successfully manage the disease at the appropriate moment. The sensitivity index of each parameter of the model (2) is analytically calculated through the normalized forward sensitivity index [24, 27–29]. For example, the sensitivity index of R_0 for Λ is obtained using the formula $\Gamma_{\Lambda}^{R_0} = \partial R_0 / \partial \Lambda \times \Lambda / R_0 = +1$. Sensitivity indices for other parameters of R_0 can be computed using a similar method. It can be noticed from Table 2 that the parameters Λ , β_1 , β_2 , β_3 , η , ε , and d have positive sensitivity indices stipulating that the increasing of these parameters leads to the increase of R_0

TABLE 3: Parameter values of the model system (2).

Parameter	Parameter value per day	Source
Λ	0.036-0.06	[10, 14]
ρ	0.0001	Assumed
β_1	0.00035	[10, 15]
β_2	0.00034	Assumed
β_3	0.00034	[10]
κ	0.0001	Assumed
μ	0.00004215	[16]
α	0.001	Assumed
ν	0.0025	Assumed
γ	0.5-0.04	[10]
η	0.1	Assumed
ψ	0.00001	Assumed
ρ	0.0714	[10]
δ	0.001	Assumed
ε	0.25	[10]
d	0.0025	Assumed
τ	0.03	[15]
σ	0.01	Assumed
χ	0.5	Assumed

and thus brings a chance of disease outbreak. On the other hand, the parameters κ , μ , α , ν , γ , ψ , ρ , δ , τ , σ , and χ have negative indices. This indicates that increasing any parameter in this group while maintaining other parameters constant lowers the basic reproduction number, therefore lowering the burden of disease in the population. The R_0 decreases with an increase in the parameters catering for control measures (ν , σ , and χ). This suggests that a suitable combination of sanitation, screening for asymptomatic carriers, and treatment of symptomatic patients as an optimal control measure is required to address the problem of *giardiasis* in the population.

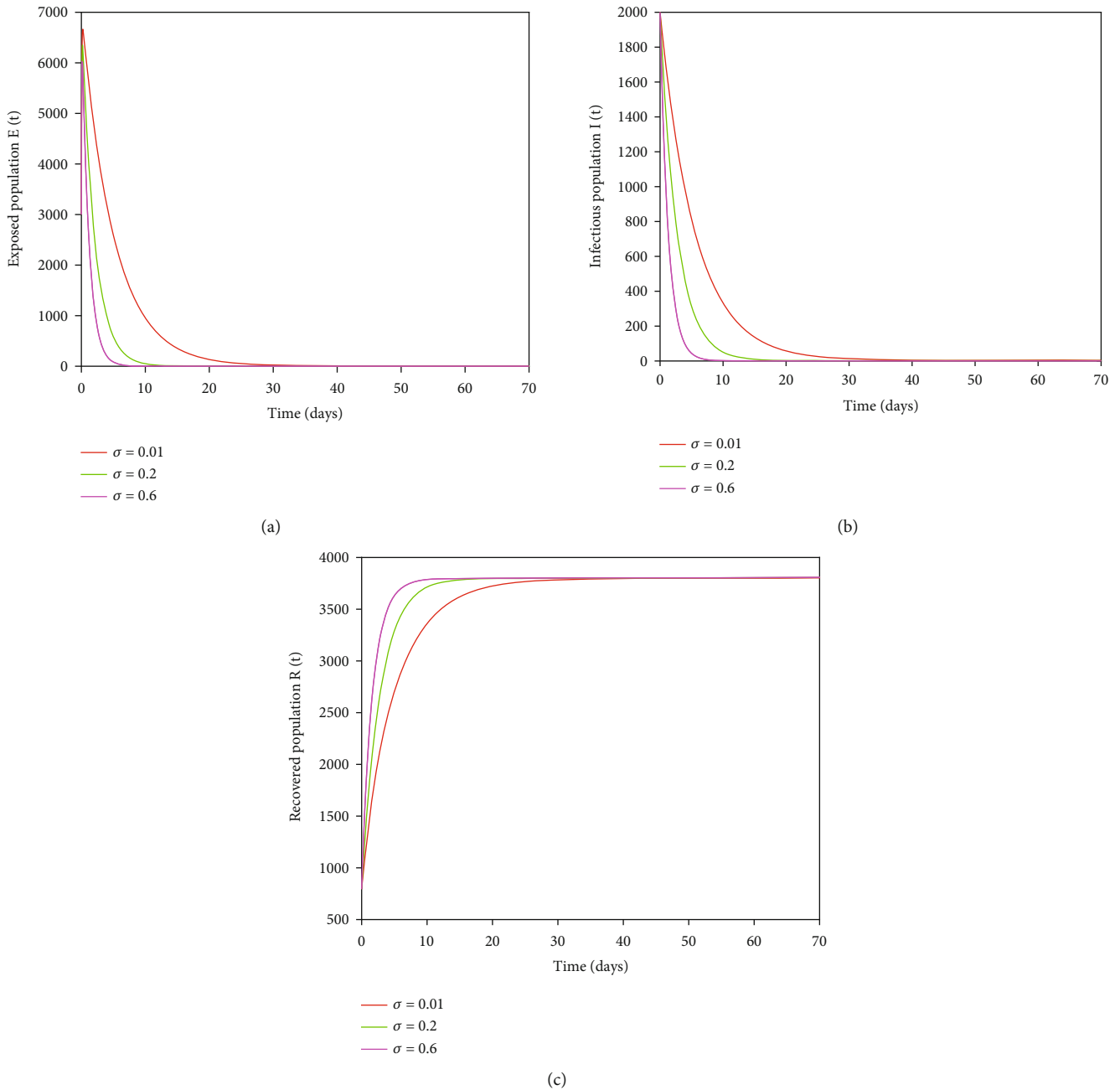


FIGURE 3: (a–c) The effects of treatment on different epidemiological classes of *giardiasis* transmission dynamics.

5. Numerical Simulation and Discussion

In this section, the numerical simulations of the model system (2) were carried out using the MATLAB ODE45 solver. In order to illustrate the specific behavior of the model, the initial conditions of the state variables have been arbitrarily chosen and are as follows: $S(0) = 4000$, $E(0) = 3000$, $I(0) = 2000$, $A(0) = 1000$, $R(0) = 800$, and $G(0) = 50000$. To support some of the analytical results previously presented, various graphical representations are presented and discussed. Since many of the parameters were not readily available, we used those we found in the literature review while making assumptions about others for

the sake of illustration. Parameter values in Table 3 were used to perform the simulations.

5.1. Effect of Treatment Intervention. Figures 3(a)–3(c) demonstrate that with an increased treatment rate, σ , there is a corresponding decrease in exposed individuals, $E(t)$. It can also be witnessed from Figure 3(b) that an increase in treatment rates σ tends to significantly lower the infection related to this disease in infectious individuals, $I(t)$. On the other hand, it can be observed that adequate treatment indicates that more individuals can recover; see Figure 3(c).

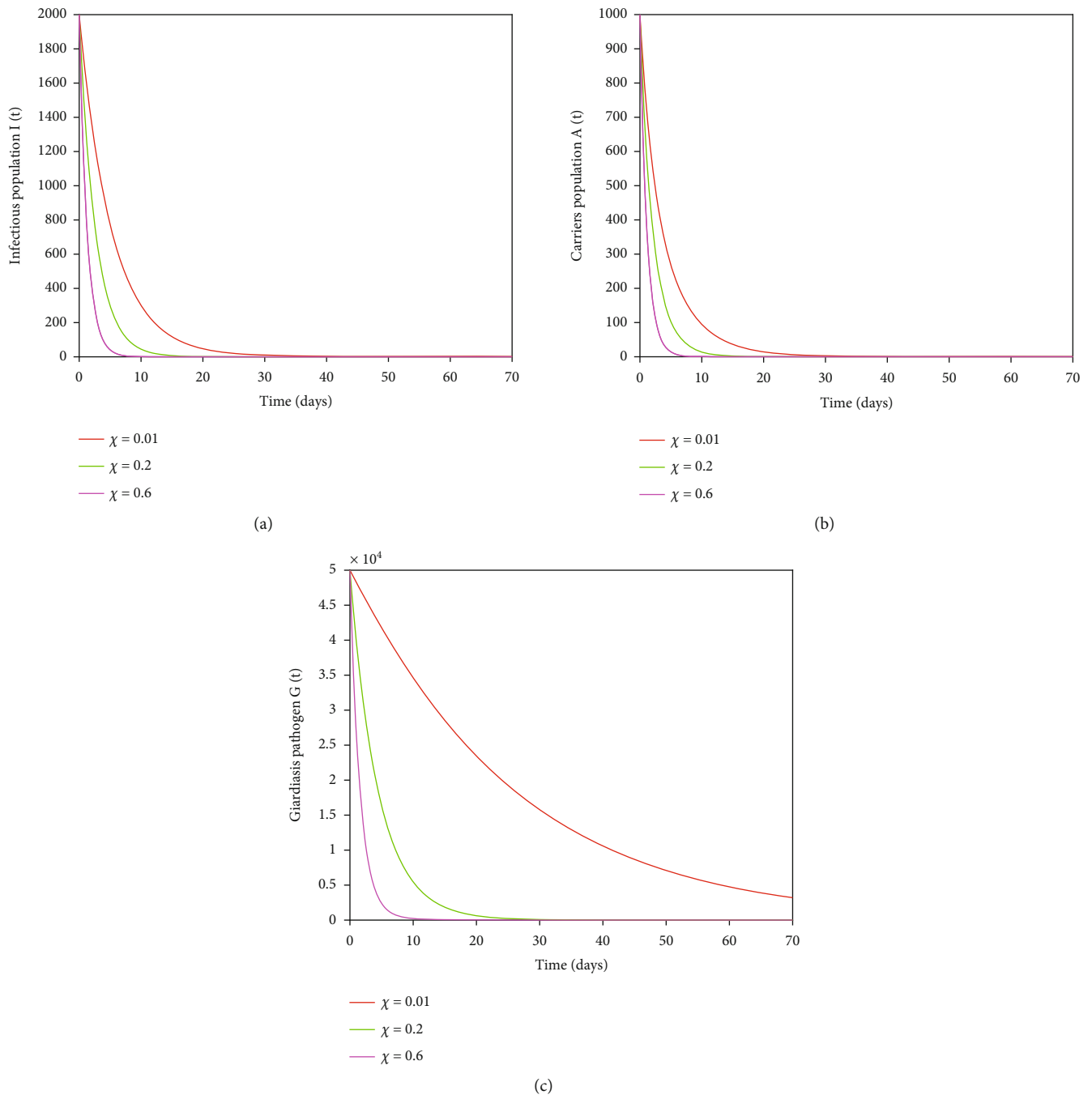


FIGURE 4: (a–c) The effects of sanitation on different epidemiological classes of *giardiasis* transmission dynamics.

5.2. *Effects of Sanitation Intervention.* Figures 4(a) and 4(b) show that as the sanitation rate increases from 1% to 60%, there is a significant decline in the number of both infectious and carrier individuals. Also, it can be noticed that as the sanitation rate increases, the number of *giardiasis* pathogens in the surroundings decreases, as depicted in Figure 4(c). The results suggest the significance of undertaking sanitation if we are to end this epidemic.

5.3. *Effect of Screening Intervention.* Here, we go over how screening interventions can help prevent the *giardiasis* disease. Figure 5(a) demonstrates that an increase in screen rate

ν results in a drop in carriers, as those who are being screened eventually enroll in the infectious (see Figure 5(b)) class and receive treatment. This finding suggests that if *giardiasis* carriers are not detected and treated, the disease will spread more quickly. Similarly, it can be observed from Figure 5(c) that, as a result of more screening, *giardiasis* is greatly minimized as screened individuals receive medical attention, which also reduces environmental contamination.

5.3.1. *Effect of Combined Interventions.* The impact on the transmission dynamics of *giardiasis* disease of using

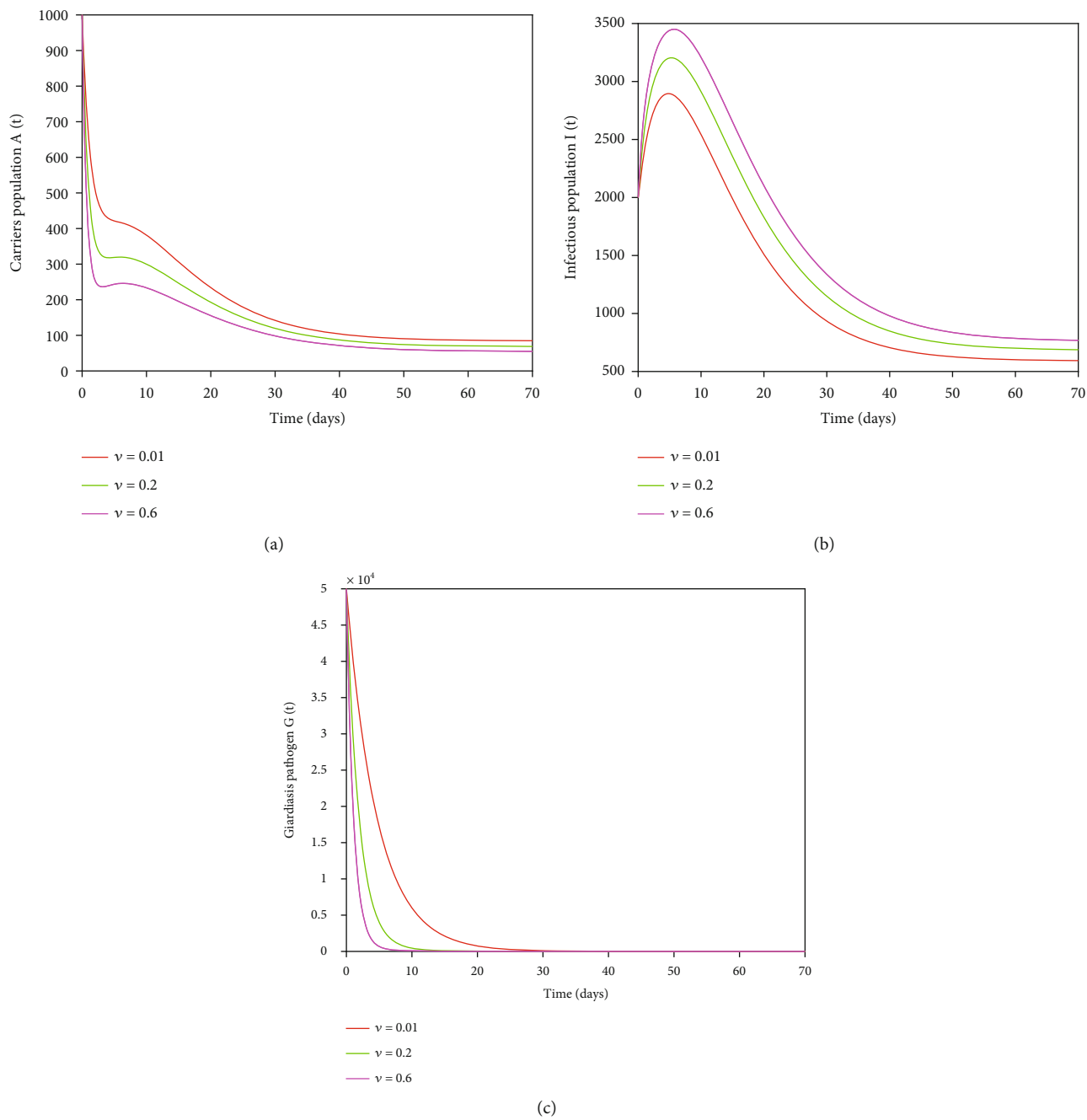


FIGURE 5: (a–c) The effects of screening on different epidemiological classes of *giardiasis* transmission dynamics.

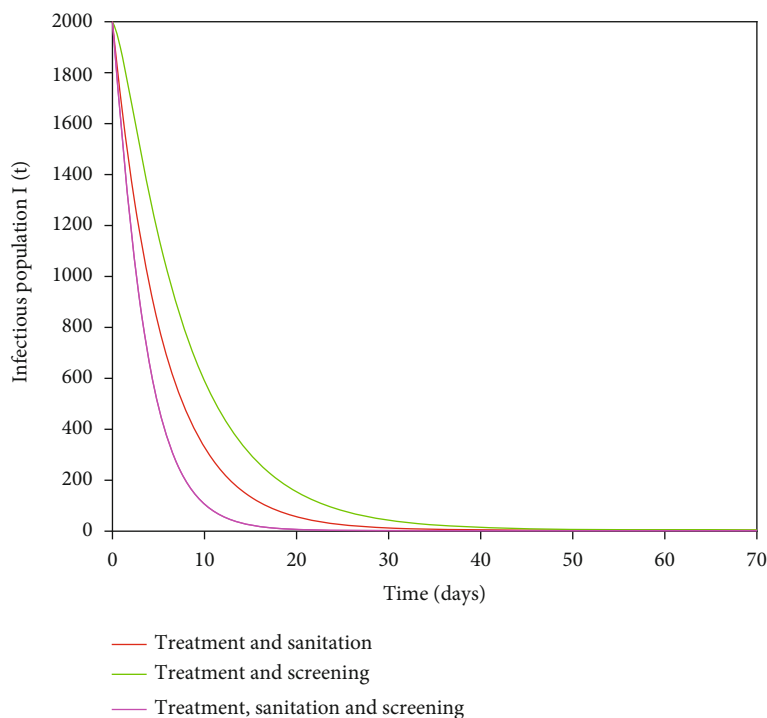


FIGURE 6: Effect of combined treatment and sanitation; treatment and screening; and treatment, sanitation, and screening on *giardiasis* transmission dynamics.

sanitation and screening as complementary to treatments is shown in Figure 6. The data showed that there was a larger reduction in the number of isolated individuals when all three interventions were used at the same time, as opposed to applying two interventions simultaneously.

6. Conclusion

A deterministic mathematical model for the transmission dynamics of *giardiasis* has been developed and analyzed in the present work. The model, which considers both direct and indirect transmission, consists of five compartments: susceptible human, exposed human, infectious human, recovered human, and the *Giardia* population. The model is a system of ordinary differential equations that include multiple interventions in the presence of carriers. We calculated the reproduction number using the next-generation, R_0 , and utilized it to ascertain the stability of the DFE point. It was determined that the DFE point was unstable when $R_0 > 1$ and locally asymptotically stable for $R_0 < 1$. The DFE point was globally asymptotically stable when $R_0 < 1$, according to the Metzler stability theory. The developed model includes intervention strategies such as sanitation, treatment, and carrier screening. Our results demonstrate that including these control measures in the model provides more major benefits for the eradication of the *Giardia* epidemic. The analysis of the model parameters reveals that hygiene, screening, and treatment control strategies have a negative value as their increase reduces the risk of transmission of disease in the community. As control interventions increase, model simulations demonstrate that the number of infectious individuals decreases more rapidly over time. In addition,

numerical simulations demonstrate that when all three interventions are combined (treatment, screening, and sanitation), they significantly reduce the prevalence of disease in the population faster than with just two interventions. Since the model has shown that treatment, screening, and sanitation facilities have a major influence in reducing the number of *giardiasis* cases in the community, we thus recommend that these areas be enhanced in endemic regions. Since the model presented in this work is not all-inclusive, its underlying assumptions can be modified to take into consideration additional treatments, including public health education, which is essential in increasing illness awareness. In addition, the assumptions might be loosened to accommodate the cost-effectiveness tactics of the control.

Data Availability

The authors certify that the data used to support the conclusion of the study are included in the manuscript and its references.

Conflicts of Interest

There are no competing interests among the writers.

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