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A Mathematical Model for the Transmission of Giardiasis Incorporating Resistance Strain and Second Line of Treatment

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Abstract

Giardiasis is among the ignored zoonotic illnesses accorded by the World Health Organization that is caused by Giardia lamblia.This study developed a mathematical model for giardiasis transmission that incorporates both sensitive and resistant strains of the parasite. The model, built as a system of nonlinear differential equations, captures host-to-host spread, environmental contamination, and strain interaction. Key thresholds, such as the basic reproduction numbers for each strain, are derived to determine conditions for disease persistence or eradication, local stability of the disease free was determined using the routh hurwitz criterion which shows that the model is locally and asymptotically stable when $R_0 < 1$ and unstable if $R_0 > 1$ while

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the castillo-chavez and song method is used to show the overall adequacy of the global stability of the equilibrium point free from the disease and it satisfied that it is globally and asymptotically stable. In addition, a Lyapunov function was constructed to study the stability of the endemic equilibrium point and the model satisfied both conditions. Sensitivity analysis highlights parameters most influencing transmission, including contact, shedding, and treatment factors. Numerical simulations support the theoretical findings, showing how poor treatment or partial compliance can allow resistant strains to persist even when sensitive strains are controlled. Overall, the study provides a rigorous framework for understanding giardiasis dynamics and offers practical insights for improving treatment and public health interventions. The results offer valuable insights into the thresholds for eradication, the potential risks of drug resistance. We advised that medical treatment programs should be strengthened to increase the recovery rate of infected individuals especially those with the resistant strain, thereby reducing the infectious period. Public health interventions should focus on reducing environmental contamination through improved sanitation, safe water supply, and hygiene promotion.

1 Introduction

Giardia lamblia also called *G. intestinalis* or *G. duodenalis*, is a flagellated protozoan parasite that infects both humans and animals' small intestines. There are two distinct morphological forms of the organism: the motile trophozoite and the infectious cyst. Ingestion of cysts in contaminated food or water usually results in transmission through the fecal-oral route World Health Organization (2017). After ingestion, excystation releases trophozoites which adhere to the intestinal mucosa, leading to malabsorption and watery diarrhea. Clinically, giardiasis presents with foul-smelling stools, abdominal cramps, bloating, and, in chronic cases, nutritional deficiencies. Children under five are particularly vulnerable, with infections contributing to growth stunting and developmental delays Einarsson et al. (2016).The infectious cyst and the trophozoite are the two phases of G. lamblia's life cycle. Ingestion of cysts found in tainted food or water, or direct fecal-oral transfer, can infect humans. The cysts develop into trophozoites inside the host, which then populate the small intestine and divide via binary fission. The cycle is completed when some trophozoites re-encyst and are expelled in feces.. Because cysts are highly resistant to chlorine and can survive for extended periods in cold water, G. lamblia is a major contributor to waterborne outbreaks World Health Organization (2022).
Giardiasis is a widespread parasitic infection affecting nearly 200 million people annually, with the highest burden in developing regions due to poor water sanitation and hygiene infrastructure. According to the WHO, giardiasis is a neglected tropical disease and a significant contributor to diarrheal disease in children World Health Organization (2017). In sub-Saharan Africa, a meta-analysis estimated a pooled prevalence of 18.3 percent among children, with even higher prevalence reported in Nigeria and Ethiopia Moreano & Velasquez Esquivel (2021). In Nigeria, prevalence rates in children vary widely from 5

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percent to over 30 percent, with higher rates in rural areas. Surveillance is limited, but available hospital data suggest giardiasis is a leading cause of pediatric gastrointestinal illness Nwosu et al. (2015). According to Center for Disease Control surveillance data, giardiasis incidence shows a bimodal distribution, affecting both young children and middle-aged adults. Incidence peaks in late summer, coinciding with increased exposure to untreated water during recreational activities.

Standard treatment for giardiasis includes metronidazole and tinidazole, both belonging to the 5-nitroimidazole class. However, treatment failures have been increasingly reported, raising concerns about emerging antimicrobial resistance. Resistant strains do not respond to standard therapies and may require second-line treatments like nitazoxanide, quinacrine, or albendazole. Combination therapy is often used for refractory cases Pasupuleti et al. (2014). The growing number of treatment failures necessitates access to second-line therapy, especially in endemic regions. Clinical studies have demonstrated the efficacy of combining nitroimidazoles with benzimidazoles in resistant infections. Such combinations disrupt multiple metabolic pathways in the parasite, improving treatment outcomes. Access to diverse treatment options is essential for limiting transmission and preventing the spread of resistant strains Ansell et al. (2015). The global health burden of giardiasis, particularly in sub-Saharan Africa, underscores the need for integrated control strategies. The emergence of drug resistance and the importance of second-line therapies demand renewed focus. Mathematical modeling serves as a crucial tool in evaluating control measures and understanding the spread of both sensitive and resistant strains. This thesis aims to develop a comprehensive mathematical model incorporating resistance dynamics and treatment interventions to support evidence-based decision-making in giardiasis control.

Clinical symptoms of giardiasis include gas, nausea, bloating, cramping in the abdomen, profuse diarrhea, exhaustion, and weight loss. Malnutrition and development retardation can be caused by persistent illnesses, particularly in youngsters.. Although the infection can be asymptomatic in some individuals, it is especially severe in malnourished and immunocompromised populations. Reinfections are common in areas where sanitation and water quality are poor Feng & Xiao (2011).

For instance, Liana & Chuma (2023) studied the transmission dynamics of giardiasis with control strategies in the presence of carriers. In their 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

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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, they advise implementing the strategies simultaneously in endemic areas to effectively stop the spread of the giardiasis disease in humans.

Edward & Shaban (2025) developed a deterministic compartmental model that monitors the dynamics of both direct and indirect transmission of Giardiasis. Health education, screening, hospitalization, and sanitation are the four constant controls that are incorporated into the model at the start of the study. An investigation and presentation of the model's analytical results are made.The existence of invariant regions and the positivity of the solutions were proven. There are several endemic equilibria and a distinct disease-free equilibrium in the model. Using the Next-Generation Matrix (NGM) method, the effective reproduction number was determined, and its consequences on the equilibria's stability were investigated. Using the Routh-Hurwitz criterion, the disease-free equilibrium's local stability was verified, and the results of its global stability were also shown. Based on the effective reproduction number, sensitivity analysis was carried out to determine the most important parameters. They provide an optimal control problem to stop Giardiasis from spreading. Using Pontryagin's Maximum Principle, they analytically described optimal control solutions and proved their existence with rigorousness. To assess the efficacy of different control systems, they used numerical simulations. A reassuring solution to the expansion of Giardiasis can be found in the encouraging results, which demonstrate that the simultaneous use of all four control measures—education, screening, treatment, and sanitation—might result in a notable decrease in disease cases.

In "Methods" section, we have established and described the mathematical model. In "Analysis of the model" section, the positivity of the solution was proven to be positive, and the system of nonlinear differential equations was well-posed epidemiologically and mathematically. We were able to determine the fundamental reproduction number by using the next generation method. The balance between the endemic and disease states was established. The disease free is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$. The global stability of the disease free is proved to be globally asymptotically stable when the associated reproduction number $\mathcal{R}_0 < 1$. While the disease will completely disappear in a stable equilibrium, it will persist and spread endemic in an unstable equilibrium. In addition, a Lyapunov function was constructed to study the stability of the endemic equilibrium point and the model satisfied both conditions. Sensitivity analysis highlights parameters most influencing transmission, including contact, shedding, and treatment factors. "Results" Numerical simulations support the theoretical findings, showing how poor treatment or partial compliance can allow resistant strains to persist even when sensitive strains are controlled. Finally, we conclude this research work in "Conclusion" section.

2 Formulation of giardiasis model

This model describes the transmission dynamics of *Giardiasis* in a human population, incorporating strain-specific resistance, asymptomatic transmission, second-line treatment, and environmental contamination. The total human population at time t, denoted by

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$N(t)$, is partitioned into ten epidemiological compartments: susceptible $S(t)$, exposed to resistant strain $E_R(t)$, infectious resistant $I_R(t)$, asymptomatic resistant $A_R(t)$, second-line treated $T_R(t)$, recovered resistant $R_R(t)$, exposed to sensitive strain $E_S(t)$, infectious sensitive $I_S(t)$, asymptomatic sensitive $A_S(t)$, and recovered sensitive $R_S(t)$. Additionally, an environmental compartment $G(t)$ represents the concentration of *Giardia* cysts in the surroundings.

$$N(t) = S + E_R + I_R + A_R + T_R + R_R + E_S + I_S + A_S + R_S. \tag{1}$$

Individuals enter the susceptible class S at a recruitment rate Λ and recovered individuals (R_R, R_S) due to waning immunity at rates ρ_R, and ρ_S respectively. They may leave the class infection by either strain at a force of infection λ, or natural death at rate μ. The change in susceptible individuals is given by:

$$\frac{dS}{dt} = \Lambda + \rho_R R_R + \rho_S R_S - \mu S - \omega_1 \lambda S - (1 - \omega_1)\lambda S,$$

where $\omega_1 \in [0, 1]$ is the proportion of new infections due to the resistant strain, and $1-\omega_1$ for the sensitive strain.

Individuals exposed to the resistant strain enter the compartment E_R and progress at rate κ_R to either symptomatic or asymptomatic stages. where $\alpha_R \in [0, 1]$ represents the proportion becoming exposed for asymptomatic and symptomatic respectively. Exposed individuals to the sensitive strain follow a similar process. Infectious resistant individuals I_R arise from E_R, and may be detected (ν_R), start treatment (ϕ_R^2), become asymptomatic (η_R), die from disease (ψ_R), or shed cysts (ε_1). A proportion d_R^2 undergo treatment while the remainder recover naturally. Asymptomatic resistant individuals A_R arise from E_R or transition from I_R. They may be detected at the rate (ν_R), recover at the rate (ϕ_R^1), receive treatment at the rate (d_R^1), die from the disease at the rate (δ), or shed cysts (ε_2). Treated individuals with the resistant strain are modeled in T_R, recovering at rate γ_R or dying due to complications or naturally. Recovered individuals from the resistant strain are drawn from those completing treatment or recovering directly. They may lose immunity at rate ρ_R or die. For the sensitive strain, infectious individuals I_S come from E_S or detected asymptomatics. They may recover at rate (ϕ_S^2), transition to asymptomatic at rate (η_S), or die due to disease at rate ψ_S. Asymptomatic sensitive individuals A_S arise from I_S, may be screened (ν_S), treated (d_S^1), recover, or die due to disease at rate δ_S. Recovered sensitive individuals are generated from asymptomatic and infectious recovery, with waning immunity at rate ρ_S. The environmental compartment G accumulates cysts from all infectious, asymptomatic, and treated classes, and declines through sanitation and decay. The total force of infection λ experienced by susceptible individuals is a function of human-to-human transmission and environmental exposure, defined as:

$$\lambda = \beta_{1S} I_S + \beta_{2S} A_S + \beta_{1R} I_R + \beta_{2R} A_R + (\beta_{3S} + \beta_{3R})G + \beta_{4R} T_R,$$

where the β parameters represent the transmission coefficients from each compartment: β_{1S} and β_{1R} are from symptomatic individuals, β_{2S} and β_{2R} from asymptomatic carriers, and β_{3S}, β_{3R} from environmental contamination of each strain and β_{4R} from the second

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line of treatment. We therefore present the equations for the model with resistance strain

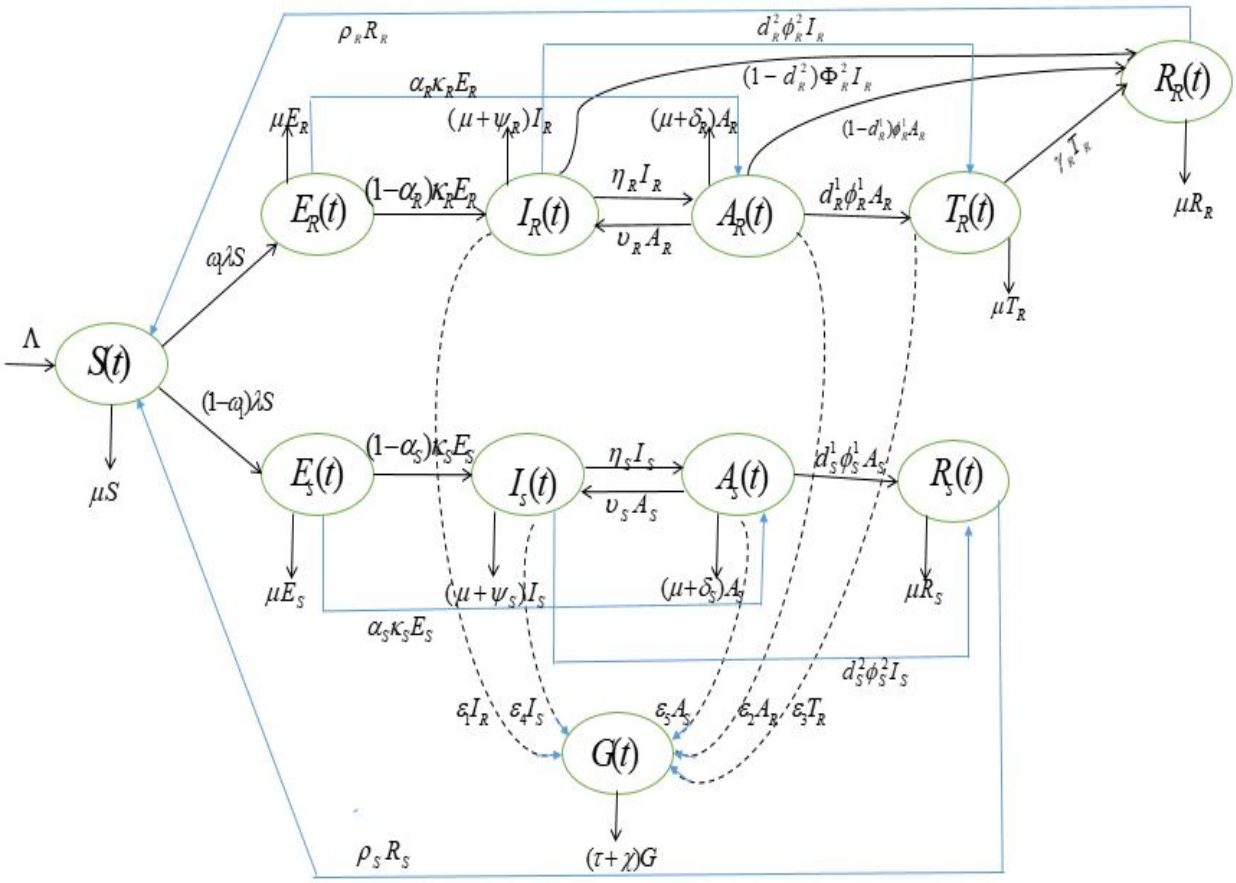

Figure 1. modified schematic model diagram.

and second-line treatment.

$$
\begin{cases}
\frac{dS}{dt} &= \Lambda + \rho_R R_R + \rho_S R_S - \mu S - \omega_1\lambda S - (1-\omega_1)\lambda S \\
\frac{dE_R}{dt} &= \omega_1\lambda S - (\mu+\kappa_R)E_R \\
\frac{dI_R}{dt} &= (1-\alpha_R)\kappa_R E_R + \nu_R A_R - (\mu+\psi_R+\eta_R+\varepsilon_1+\phi_R^2)I_R \\
\frac{dA_R}{dt} &= \eta_R I_R + \alpha_R\kappa_R E_R - (\nu_R+\mu+\delta_R+\varepsilon_2+\phi_R^1)A_R \\
\frac{dT_R}{dt} &= d_R^1\phi_R^1 A_R + d_R^2\phi_R^2 I_R - (\mu+\gamma_R+\varepsilon_3)T_R \\
\frac{dR_R}{dt} &= (1-d_R^1)\phi_R^1 A_R + (1-d_R^2)\phi_R^2 I_R + \gamma_R T_R - (\rho_R+\mu R_R) \\
\frac{dE_S}{dt} &= (1-\omega_1)\lambda S - (\mu+\kappa_S)E_S \\
\frac{dI_S}{dt} &= (1-\alpha_S)\kappa_S E_S + \nu_S A_S - (\mu+\psi_S+\eta_S+\varepsilon_4+d_S^2\phi_S^2)I_S \\
\frac{dA_S}{dt} &= \eta_S I_S + \alpha_S\kappa_S E_S - (\mu+\delta_S+\nu_S+\varepsilon_5+d_S^1\phi_S^1)A_S \\
\frac{dR_S}{dt} &= d_S^1\phi_S^1 A_S + d_S^2\phi_S^2 I_S - (\rho_S+\mu)R_S \\
\frac{dG}{dt} &= \varepsilon_1 I_R + \varepsilon_2 A_R + \varepsilon_3 T_R + \varepsilon_4 I_S + \varepsilon_5 A_S - (\tau+\chi)G
\end{cases}
\tag{2}
$$

where

$$\lambda = \beta_{1S}I_S + \beta_{2S}A_S + \beta_{1R}I_R + \beta_{2R}A_R + (\beta_{3S}+\beta_{3R})G + \beta_{4R}T_R,$$

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with the initial conditions:

$$\begin{cases} S(0) > 0, E_R(0) \geq 0, I_R(0) \geq 0, A_R(0) \geq 0, T_R(0) \geq 0, R_R(0) \geq 0, \\ E_S(0) \geq 0, I_S(0) \geq 0, A_S(0) \geq 0, R_S(0) \geq 0, G(0) \geq 0 \end{cases} \tag{3}$$

Table 1. Giardiasis with resistance model variables.

Variable	Description
$S(t)$	Number of susceptible individuals at time t
$E_R(t)$	Number of exposed individuals to resistant strain at time t
$I_R(t)$	Number of infectious individuals with resistant strain at time t
$A_R(t)$	Number of asymptomatic individuals with resistant strain at time t
$T_R(t)$	Number of individuals undergoing second-line treatment for resistant strain at time t
$R_R(t)$	Number of individuals recovered from resistant strain at time t
$E_S(t)$	Number of exposed individuals to sensitive strain at time t
$I_S(t)$	Number of infectious individuals with sensitive strain at time t
$A_S(t)$	Number of asymptomatic individuals with sensitive strain at time t
$R_S(t)$	Number of individuals recovered from sensitive strain at time t
$G(t)$	Concentration of Giardia lamblia cysts in the environment at time t

Table 2. Parameters values and notation for the giardiasis model.

Variable	**Description**	**Value**	**Source**
$S(t)$	Number of susceptible individuals at time t	4000	Liana & Chuma (2023)
$E_R(t)$	Number of exposed individuals with resistant strain at time t	1000	Assumed
$I_R(t)$	Number of infectious individuals with resistant strain at time t	800	Assumed
$A_R(t)$	Number of asymptomatic individuals with resistant strain at time t	400	Assumed
$T_R(t)$	Number of individuals undergoing second-line treatment for resistant strain at time t	300	Assumed
$R_R(t)$	Number of individuals recovered from resistant strain at time t	300	Assumed
$E_S(t)$	Number of exposed individuals to sensitive strain at time t	2000	Liana & Chuma (2023)
$I_S(t)$	Number of infectious individuals with sensitive strain at time t	1200	Liana & Chuma (2023)
$A_S(t)$	Number of asymptomatic individuals with sensitive strain at time t	600	Liana & Chuma (2023)
$R_S(t)$	Number of individuals recovered from sensitive strain at time t	500	Liana & Chuma (2023)
$G(t)$	Concentration of *Giardia lamblia* cysts in the environment at time t	50000	Liana & Chuma (2023)

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2.1 Basic properties

Theorem 2.1. *Let the initial data* $S(0), E_S(0), I_S(0), A_S(0), R_S(0), E_R(0), I_R(0), A_R(0), T_R(0), R_R(0), G(0) > 0$. *Then all the solution sets* $S(t), E_S(t), I_S(t), A_S(t), R_S(t), E_R(t), I_R(t), A_R(t), T_R(t), R_R(t), G(t)$ *of the model equations (3.5–3.15) are non-negative for all* $t > 0$.

Moreover, $\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$. *In addition, if* $N(0) \leq \frac{\Lambda}{\mu}$, *then* $N(t) \leq \frac{\Lambda}{\mu}$ *if the feasible region for system (2)*

$$\Omega^* = \left\{ (S, E_R, I_R, A_R, T_R, R_R, E_S, I_S, A_S, R_S, G) \in \mathbb{R}_+^{11} : N(t) \leq \frac{\Lambda}{\mu}, G(t) \leq \tau + \chi \right\} \quad (4)$$

is attractive and positively invariant with regard to the system (2).

Proof. From the first equation of system (2), we have the following

$$\frac{dN_{(t)}}{dt} \leq \Lambda - \mu N_{(t)} \quad (5)$$

From time $t = 0$, we integrating (5) to get

$$N(t) \leq \frac{\Lambda}{\mu} + \left(dN_{(t)}0 - \frac{\Lambda}{\mu} \right) e^{-\mu t}. \quad (6)$$

As $t \to \infty$, we have:

$$\lim_{t\to\infty} N(t) = \frac{\Lambda}{\mu}. \quad (7)$$

We used a similar approach to prove that $L(t), I_A(t), I_C(t), R(t), V(t), V(t) > 0$ remain non-negative for all $t > 0$. The second portion of the theorem, which states that model system (2) is positively invariant, is proved using equation (**??**) so that $N(t) \leq \frac{\nu}{\mu} + (N(0) - \frac{\nu}{\mu}) \exp^{-\mu t}$. It follows that as $t \longrightarrow \infty \quad N(t) \leq \frac{\nu}{\mu}$. Furthermore, If $N(0) \leq \frac{\nu}{\mu}$, then $N(t) \leq \frac{\nu}{\mu}$. This establishes that Ω is the manifold on which the population has non-zero size.

Thus, proves the boundedness of the solutions inside Ω. Hence, the solutions to system (2) are attractive in a region and positively invariant Ω. We notice that system (2) is feasible biologically and mathematically well posed in Ω from theorem (2.1). □

3 The giardiasis model analysis

This section establishes the giardiasis-free equilibrium state, giardiasis-present equilibrium state, the basic reproduction number is obtained and carryout stability analysis.

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3.1 Equilibrium points

The system (2) equilibrium points are established by the setting of the system (2) to zero to get

$$\begin{cases} \frac{dS}{dt} = \Lambda + \rho_R R_R + \rho_S R_S - \mu S - \omega_1 \lambda S - (1-\omega_1)\lambda S & = 0 \\ \frac{dE_R}{dt} = \omega_1 \lambda S - (\mu + \kappa_R) E_R & = 0 \\ \frac{dI_R}{dt} = (1-\omega_1)\lambda S - (\mu + \kappa_S) E_S & = 0 \\ \frac{dA_R}{dt} = (1-\alpha_R)\kappa_R E_R + \nu_R A_R - (\mu + \psi_R + \eta_R + \varepsilon_1 + \phi_R^2) I_R & = 0 \\ \frac{dT_R}{dt} = (1-\alpha_S)\kappa_S E_S + \nu_S A_S - (\mu + \psi_S + \eta_S + \varepsilon_4 + d_S^2 \phi_S^2) I_S & = 0 \\ \frac{dR_R}{dt} = \eta_R I_R + \alpha_R \kappa_R E_R - (\nu_R + \mu + \delta_R + \varepsilon_2 + \phi_R^1) A_R & = 0 \\ \frac{dE_S}{dt} = \eta_S I_S + \alpha_S \kappa_S E_S - (\mu + \delta_S + \nu_S + \varepsilon_5 + d_S^1 \phi_S^1) A_S & = 0 \\ \frac{dI_S}{dt} = d_R^1 \phi_R^1 A_R + d_R^2 \phi_R^2 I_R - (\mu + \gamma_R + \varepsilon_3) T_R & = 0 \\ \frac{dA_S}{dt} = (1-d_R^1)\phi_R^1 A_R + (1-d_R^2)\phi_R^2 I_R + \gamma_R T_R - (\rho_R + \mu R_R) & = 0 \\ \frac{dR_S}{dt} = d_S^1 \phi_S^1 A_S + d_S^2 \phi_S^2 I_S - (\rho_S + \mu) R_S & = 0 \\ \frac{dG}{dt} = \varepsilon_1 I_R + \varepsilon_2 A_R + \varepsilon_3 T_R + \varepsilon_4 I_S + \varepsilon_5 A_S - (\tau + \chi) G & = 0 \end{cases} \tag{8}$$

In the absence of giardiasis infection, the system (2) has a steady-state, this steady state is termed giardiasis-free equilibrium. We calculated and evaluated the Jacobian of the system (2) at the giardiasis-free equilibrium in order to determine the type of stability of the giardiasis-free equilibrium. The signs of the Jacobian's eigenvalues are used in order to determine the local stability of the giardiasis-free equilibrium. The system's giardiasis-free equilibrium (2) is

$$(S^0, E_R^0, I_R^0, A_R^0, T_R^0, R_R^0, E_S^0, I_S^0, A_S^0, R_S^0,) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0\right) \tag{9}$$

This only indicates that the susceptible individuals' recruitment and mortality rates change proportionally in the absence of giardiasis disease.
For the second equilibrium point, we let

$$E^{**} = (S^{**}, {E_R}^{**}, {I_R}^{**}, {A_R}^{**}, {T_R}^{**}, {R_R}^{**}, {E_S}^{**}, {I_S}^{**}, {A_S}^{**}, {R_S}^{**})$$

be the giardiasis present equilibrium of the model (2). At equilibrium state, we let $\lambda^{**} = \beta_{1S} I_S + \beta_{2S} A_S + \beta_{1R} I_R + \beta_{2R} A_R + (\beta_{3S} + \beta_{3R}) G + \beta_{4R} T_R$, be the forces of infection and $N^{**} = S^{**} + {E_R}^{**} + {I_R}^{**} + {A_R}^{**} + {T_R}^{**} + {R_R}^{**} + {E_S}^{**} + {I_S}^{**} + {A_S}^{**} + {R_S}^{**}$, solving

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system (2) at steady state yields

$$\begin{cases} S^{**} = & \frac{\Lambda+\rho_R R_R^*+\rho_S R_S^*}{m_1} \\ E_R^{**} = & \lambda^* \frac{\omega_1}{m_1 m_2}\left(\Lambda+\rho_R R_R^*+\rho_R R_S^*\right) \\ E_S^{**} = & -\frac{\lambda^*}{m_1 m_{12}}\left(\omega_1-1\right)\left(\Lambda+\rho_R R_R^*+\rho_R R_S^*\right) \\ I_R^{**} = & \frac{(1-\alpha_R)\kappa_R E_R^*+\upsilon_R A_R^*}{m_3} \\ I_S^{**} = & \frac{(1-\alpha_S)\kappa_S E_S^*+\upsilon_S A_S^*}{m_7} \\ A_R^{**} = & \frac{\alpha_R\kappa_R E_R^*+\eta_R I_R^*}{m_4} \\ A_S^{**} = & \frac{\alpha_S\kappa_S E_S^*+\eta_S I_S^*}{m_8} \\ T_R^{**} = & \frac{d_R^1\phi_R^1 A_R^*+d_R^2\phi_R^2 I_R^*}{m_5} \\ R_R^{**} = & \frac{\gamma_R T_R^*+(1-d_R^1)\phi_R^1 A_R^*+(1-d_R^2)\phi_R^2 I_R^*}{\mu+\rho_R} \\ R_S^{**} = & \frac{d_S^1\phi_S^1 A_S^*+d_S^2\phi_S^2 I_S^*}{m_9} \end{cases} \tag{10}$$

where
$m_1 = \lambda^*+\mu, m_2 = (\kappa_R+\mu), m_3 = (\mu+\psi_R+\eta_R+\varepsilon_1+\phi_R^2), m_4 = (\upsilon_R+\mu+\delta_R+\varepsilon_2+\phi_R^1), m_5 = (\mu+\gamma_R+\varepsilon_3), m_7 = (\mu+\psi_S+\eta_s+\varepsilon_4+d_S^2\phi_s^2), m_8 = (\upsilon_S+\mu+\delta_S+\varepsilon_5+d_S^1\phi_S^1), m_9 = (\mu+\rho_s)$

3.2 Basic reproduction number

The term "basic reproduction number" refers to the typical number of secondary infections induced by one infectious person when the entire population is susceptible, $\mathcal{R}_0$. The giardiasis epidemiological threshold is represented by the symbol $\mathcal{R}_0 = \rho(FV^{-1})$, ρ denotes the dominant eigenvalue. For simplicity of the model analysis, we consider a case for the resistant strain and another case for the sensitive strain, then we combine the results from the two cases to make up our basic reproduction number for our Giardiasis model. To determine the basic reproduction number of the system (2), We implemented the strategies in Van den Driessche & Watmough (2002) to get

$$F_R = \begin{pmatrix} \omega_1\big(\beta_{1R}I_R+\beta_{2R}A_R+\beta_{1S}I_S+\beta_{2S}A_S+\beta_{4R}T_R+(\beta_{3S}+\beta_{3R})G\big)S \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

and

$$V_R = \begin{pmatrix} (\kappa_R+\mu)E_R \\ -(1-\alpha_R)\kappa_R E_R-\upsilon_R A_R+(\varepsilon_1+\psi_R+\eta_R+\phi_R^2+\mu)I_R \\ -\alpha_R\kappa_R E_R-\eta_R I_R+(\delta_R+\varepsilon_2+\upsilon_R+\phi_R^1+\mu)A_R \\ -d_R^1\phi_R^1 A_R-d_R^2\phi_R^2 I_R+(\varepsilon_R+\gamma_R+\mu) \\ -\varepsilon_1 I_R\ -\varepsilon_2 A_R\ -\varepsilon_3 T_R\ +(\tau+\chi)G \end{pmatrix}.$$

$$F_S = \begin{bmatrix} (1-\omega_1)\lambda S \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

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$$V_s = \begin{bmatrix} (\kappa_s + \mu)E_s \\ -(1-\alpha_S)\kappa_s E_s - \upsilon_s A_s + (\psi_s + \varepsilon_4 + d_S^2\phi_s^2 + \eta_s + \mu)I_s \\ -\alpha_s\kappa_s E_s - \eta_s I_s + (\delta_S + \varepsilon_5 + d_S^1\phi_s^1 + \nu_s + \mu)A_s \\ (\tau + \chi) \end{bmatrix} \tag{11}$$

New infection terms and transition terms are respectively contained in the matrices F and V in the system (2). At giardiasis-free equilibrium, the Jacobian matrices of F and V are evaluated, producing the following results.

$$F_R = \begin{pmatrix} 0 & \omega_1\beta_{1R}S_0 & \omega_1\beta_{2R}S_0 & \omega_1\beta_{4R}S_0 & \omega_1\beta_{3R}S_0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & 0 \end{pmatrix}$$

and

$$V_R = \begin{pmatrix} z_1 & 0 & 0 & 0 & 0 \\ -k_1 & z_2 & -\nu_R & 0 & 0 \\ -k_2 & -\eta_R & z_3 & 0 & 0 \\ 0 & -k_3 & -k_4 & z_4 & 0 \\ 0 & 0 & 0 & 0 & z_5 \end{pmatrix}$$

Also

$$F_s = \begin{bmatrix} 0 & (1-\omega_1)\beta_{1S}S_0 & (1-\omega_1)\beta_{2S}S_0 & (1-\omega_1)\beta_{3S}S_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \varepsilon_4 & \varepsilon_5 & 0 \end{bmatrix} \tag{12}$$

$$V_s = \begin{bmatrix} z_5 & 0 & 0 & 0 \\ -k_5 & z_6 & -\nu_s & 0 \\ -k_6 & -\eta_s & z_7 & 0 \\ 0 & 0 & 0 & z_8 \end{bmatrix} \tag{13}$$

Therefore, system (2) has a basic reproduction number provided by

$$\mathcal{R}_0 = \mathcal{R}_{0R}^S + \mathcal{R}_{0R}^A \tag{14}$$

where

$$\mathcal{R}_{0R}^S = \frac{(1-\omega_1)\beta_{1S}S_0(k_5z_7 + k_6\upsilon_S)}{(z_6z_7 - \eta_S\upsilon_S)z_5}, \qquad \mathcal{R}_{0R}^A = \frac{(1-\omega_1)\beta_{2S}S_0(\eta_S k_5 + k_6z_6)}{(z_6z_7 - \eta_S\upsilon_S)z_5}$$

3.3 Local stability of the giardiasis disease-free equilibrium

Theorem 3.1. *The giardiasis disease-free equilibrium of system (2) is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and unstable if otherwise.*

Proof. The Jacobian matrix of the system (2) at giardiasis-free equilibrium $J(E^0)$ is be

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given by

$$\begin{pmatrix} -m_1 & 0 & -a_1 & -a_2 & -a_3 & \rho_R & 0 & -a_{12} & -a_{13} & \rho_s & a_{14} \\ 0 & -m_2 & a_4 & a_5 & a_6 & 0 & 0 & a_{15} & a_{16} & 0 & a_{17} \\ 0 & m_{15} & -m_3 & \upsilon_R & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_R k_R & \eta_R & -m_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_7 & a_8 & -m_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & m_{14} & m_{13} & \gamma_R & -m_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_9 & a_{10} & a_{11} & 0 & -m_{12} & a_{18} & a_{19} & 0 & a_{20} \\ 0 & 0 & 0 & 0 & 0 & 0 & m_{15} & -m_7 & -\upsilon_s & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_S k_S & \eta_S & -m_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{21} & a_{22} & -m_9 & 0 \\ 0 & 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & 0 & 0 & \epsilon_4 & \epsilon_5 & 0 & -m_{10} \end{pmatrix} \tag{15}$$

where

$m_1 = \lambda + \mu, \quad m_2 = (\kappa_R + \mu), \quad m_3 = (\mu + \psi_R + \eta_R + \varepsilon_1 + \phi_R^2), \quad m_4 = (\upsilon_R + \mu + \delta_R + \varepsilon_2 + \phi_R^1),$

$m_5 = (\mu + \gamma_R + \varepsilon_3), \quad m_6 = (\mu + \rho_R), \quad m_7 = (\mu + \psi_S + \eta_s + \varepsilon_4 + d_S^2\phi_s^2),$

$m_8 = (\upsilon_S + \mu + \delta_S + \varepsilon_5 + d_S^1\phi_S^1), \quad m_9 = (\mu + \rho_s), \quad m_{10} = (\tau + \chi),$

$m_{11} = \lambda = [\beta_{1s} I_s + \beta_{2s} A_s + \beta_{1R} I_R + \beta_{2R} A_R + \beta_{4R} T_R + (\beta_{3s} + \beta_{3R}) G],$

$m_{12} = (\kappa_s + \mu), \quad m_{13} = (1 - d_R^1)\phi_R^1, \quad m_{14} = (1 - d_R^2)\phi_R^2, \quad m_{15} = (1 - \alpha_R)\kappa_R,$

$a_1 = \beta_{1R} S_0, \quad a_2 = \beta_{2R} S_0, \quad a_3 = \beta_{4R} S_0, \quad a_4 = \omega_1 \beta_{1R} S_0, \quad a_5 = \omega_2 \beta_{2R} S_0,$

$a_6 = \omega_1 \beta_{4R} S_0, \quad a_7 = d_R^2 \phi_R^2, \quad a_8 = d_R^1 \phi_R^1, \quad a_9 = (1 - \omega_1)\beta_{1R} S_0,$

$a_{10} = (1 - \omega_1)\beta_{2R} S_0, \quad a_{11} = (1 - \alpha_R)\beta_{4R} S_0, \quad a_{12} = \beta_{1S} S_0, \quad a_{13} = \beta_{2S} S_0, \quad a_{14} = (\beta_{3R} + \beta_{3S}) S_0,$

$a_{15} = \omega_1 \beta_{1S} S_0, \quad a_{16} = \omega_1 \beta_{2S} S_0, \quad a_{17} = \omega_1 (\beta_{3R} + \beta_{3S}) S_0,$

$a_{18} = (1 - \omega_1)\beta_{1S} S_0, \quad a_{19} = (1 - \omega_1)\beta_{2S} S_0, \quad a_{20} = (1 - \omega_1)\beta_{3S} S_0,$

$a_{21} = d_S^2 \phi_S^2, \quad a_{22} = d_S^1 \phi_S^1.$

The characteristics polynomial $J(E^0)$ for (11) is hereby defined as follows:

$$(\lambda^6 + B_1\lambda^5 + B_2\lambda^4 + B_3\lambda^3 + B_4\lambda^2 + B_5\lambda + B_6)(\lambda^5 + P_1\lambda^4 + P_2\lambda^3 + P_3\lambda^2 + P_4\lambda + P_5) = 0, \tag{16}$$

where the coefficients of (11) are given as

$B_0 = 1,$

$B_1 = m_5 + m_4 + m_2 + m_3 + m_6 + m_1,$

$B_2 = (\alpha_R\kappa_R a_5 - a_4 m_{15} - \eta_R\nu_R + m_2 m_3 + m_4 m_2 + m_5 m_2 + m_4 m_3 + m_5 m_3 + m_5 m_4$
$+ (m_5 + m_4 + m_2 + m_3) m_6 + (m_5 + m_4 + m_2 + m_3 + m_6) m_1,$

$B_3 = \Big(\alpha_R\kappa_R a_4\nu_R - \alpha_s\kappa_s a_5 m_3 - \alpha_s\kappa_s a_5 m_5 - a_8\alpha_R\kappa_R a_6 - a_4 m_4 m_{15} - a_4 m_5 m_{15} - a_5\eta_R m_{15}$
$- a_7 m_{15} a_6 - \eta_R m_2\nu_R - \eta_R m_5\nu_R + m_2 m_3 m_4 + m_2 m_3 m_5 + m_2 m_4 m_5$
$+ m_3 m_4 m_5 + (-\alpha_R\kappa_R a_5 - a_4 m_{15} - \eta_R\nu_R + m_2 m_3 + m_2 m_4 + m_2 m_5 + m_3 m_4 + m_3 m_5 + m_4 m_5)\, m_6$
$+ (-\alpha_R\kappa_R a_5 - a_4 m_{15} - \eta_R\nu_R + m_2 m_3 + m_4 m_2 + m_5 m_2 + m_4 m_3 + m_5 m_3 + m_5 m_4)\, m_1\Big),$

$B_4 = \Big(- \alpha_R\kappa_R a_4 m_5\nu_R - \alpha_s\kappa_s a_5 m_3 m_5 - \alpha_R\kappa_R a_6 a_7\nu_R - \alpha_s\kappa_s a_6 a_8 m_3 - a_4 m_4 m_5 m_{15} -$

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$a_5\eta_R m_5 m_{15}$
$- a_6a_7m_4m_{15} - a_6a_8\eta_R m_{15} - \eta_R m_2 m_5\nu_R + m_2m_3m_4m_5$
$+(-\alpha_R\kappa_R a_4\nu_R - \alpha_s\kappa_s a_5 m_3 - \alpha_s\kappa_s a_6a_8 - a_4m_4m_{15} - a_4m_5m_{15} - a_5\eta_R m_{15} - a_6a_7m_{15} - \eta_R m_2\nu_R - \eta_R m_$
$+(-\alpha_R\kappa_R a_4\nu_R - \alpha_R\kappa_R a_5 m_3 - \alpha_s\kappa_s a_6a_8 - a_4m_4m_{15} - a_4m_5m_{15} - a_5\eta_R m_{15} - a_7m_{15}a_6 - \eta_R m_2\nu_R - \eta_R m$
$B_5 = \Big(\Big(\Big((m_4+m_2)m_3 - \alpha_R\kappa_R a_5 + m_4m_2 - a_4m_{15} - \eta_R\nu_R\Big)m_5 + \Big(-a_4m_4 - a_5\eta_R - a_6a_7\Big)m_{15}$
$- \kappa_R\big(a_4\nu_R + a_5m_3 + a_6a_8\big)\alpha_R + m_2\big(-\eta_R\nu_R + m_3m_4\big)\Big)m_6 + \Big(\big(-a_4m_4 - a_5\eta_R\big)m_{15}$
$- \kappa_R\big(a_4\nu_R + a_5m_3\big)\alpha_R + m_2\big(-\eta_R\nu_R + m_3m_4\big)\Big)m_5 - a_6\Big(\big(a_7m_4 + a_8\eta_R\big)m_{15} + \alpha_R\kappa_R\big(a_7\nu_R +$
$a_8m_3\big)\Big)\Big)m_1$
$- \Big(\Big(\big(a_4m_4 + a_5\eta_R\big)m_{15} + \kappa_R\big(a_4\nu_R + a_5m_3\big)\alpha_R - m_2\big(-\eta_R\nu_R + m_3m_4\big)\Big)m_5$
$+ a_6\Big(\big(a_7m_4 + a_8\eta_R\big)m_{15} + \alpha_R\kappa_R\big(a_7\nu_R + a_8m_3\big)\Big)\Big)\Big)m_6,$
$B_6 = \Big(-\alpha_R\kappa_R a_4m_5\nu_R - \alpha_s\kappa_s a_3m_3m_5 - \alpha_R\kappa_R a_6a_7\nu_R - \alpha_s\kappa_s a_6a_8m_3 - a_4m_4m_5m_{15}$
$- a_5\eta_R m_5m_{15} - a_6a_7m_4m_{15} - a_6a_8\eta_R m_{15} - \eta_R m_2m_5\nu_R + m_2m_3m_4m_5\Big)m_6m_1$ and

$$
\begin{aligned}
P_0 &= 1,\\
P_1 &= m_{10} + m_8 + m_{12} + m_7 + m_9,\\
P_2 &= -\alpha\kappa a_{19} - a_{18}m_{15} + \eta_s\nu_s + m_8m_7 + m_{10}m_7 + m_{12}m_7 + m_{10}m_8 + m_8m_{12} + m_{10}m_{12}\\
&\quad + (m_{10} + m_8 + m_{12} + m_7)m_9,\\
P_3 &= \Big[(m_8 + m_{12} + m_7)m_9 + (m_7 + m_8)m_{12} - \alpha\kappa a_{19} - a_{18}m_{15} + \eta_s\nu_s + m_8m_7\Big]m_{10}\\
&\quad + \Big[(m_7 + m_8)m_{12} - \alpha\kappa a_{19} - a_{18}m_{15} + \eta_s\nu_s + m_8m_7\Big]m_9\\
&\quad + (\eta_s\nu_s + m_7m_8)m_{12} - \alpha\kappa a_{19}m_7 - a_{18}m_8m_{15} + (-a_{19}\eta_s - a_{20})m_{15}\\
&\quad + \alpha\kappa(a_{18}\nu_s - a_{20}),\\
P_4 &= \Big[-a_{18}m_{15} - \alpha\kappa a_{19} + (m_{12} + m_8)m_7 + m_8m_{12} + \eta_s\nu_s\Big]m_{10}\\
&\quad + \big(-a_{18}m_8 - a_{19}\eta_s - a_{20}\big)m_{15} + \kappa(a_{18}\nu_s - a_{19}m_7 - a_{20})\alpha + (\eta_s\nu_s + m_7m_8)m_{12}\\
&\quad \Big]m_9 + \Big[(-a_{18}m_8 - a_{19}\eta_s)m_{15} + \kappa(a_{18}\nu_s - a_{19}m_7)\alpha + (\eta_s\nu_s + m_7m_8)m_{12}\Big]m_{10}\\
&\quad + a_{20}\Big[(-4m_8 - \eta_s)m_{15} + \alpha\kappa(\nu_s - m_7)\Big],\\
P_5 &= m_9\Big\{\Big[(-a_{18}m_8 - a_{19}\eta_s)m_{15} + \kappa(a_{18}\nu_s - a_{19}m_7)\alpha + (\eta_s\nu_s + m_7m_8)m_{12}\Big]m_{10}\\
&\quad + a_{20}\Big[(m_8 - \eta_s)m_{15} + \alpha\kappa(\nu_s - m_7)\Big]\Big\}.
\end{aligned}
\tag{17}
$$

Using the Routh-Hurwitz criterion, which stipulates that all polynomial equation (16) roots have a negative real component if and only if the coefficients are positive and the determinant of the matrices $H_i > 1$ for $i = 1, ...6$. It is clear that $D_1 > 0$. Therefore, if $D_j > 0$ for $j = 2...7$ and Since the giardiasis free equilibrium is locally asymptotically stable, the Routh-Hurwitz conditions for the 7th-order characteristics polynomial in (16) are satisfied, we conclude the giardiasis free equilibrium is locally asymptotically stable (LAS).

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□

3.4 Global stability for giardiasis-free equilibrium

The strategy used in Castillo-Chavez et al. (2002) to examine the global asymptotic stability (GAS) of the giardiasis-free equilibrium for the model (2).

Lemma 3.2. *Let system (2) be in the form*

$$\begin{cases} \frac{dX}{dt} & = F(X,Z) \\ \frac{dZ}{dt} & = G(X,Z,),(X,0)=0 \end{cases} \tag{18}$$

where $X=(S,R_R,R_S)$ *and* $Z=(E_R,I_R,A_R,T_R,E_S,I_S,A_S,G)$ *and components of* $X\in\mathbb{R}^3$ *represent the population that are not infected and components of* $Z\in\mathbb{R}^8$ *represent the population that are infected Castillo-Chavez et al. (2002). Considering the giardiasis-free equilibrium* $E^0=(X^0,0)$*, where*

$$X^0=\left(\frac{\Lambda}{\mu},0,0\right) \tag{19}$$

The following requirements must be satisfied to provide global asymptotic stability:
$H_1:\frac{dX}{dt}=F(X^0,0)$, X^0 *is (GAS.*
$H_2:G(X,Z)=PZ-\hat{G}(X,Z),\hat{G}(X,Z)\geq 0$ *for* $(X,Z)\in\Omega$*, where* $P=D_zG(X^0,0)$ *is an M-matrix and* Ω *is the biological feasible region. Hence,* E^0 *is (GAS) if* $\mathcal{R}_0<1$*.*

Theorem 3.3. *The giardiasis-free equilibrium of system (2) is (GAS) if* $\mathcal{R}_0<1$ *and unstable if otherwise.*

Proof. We have to establish that the conditions (H_1) and (H_2) hold when $\mathcal{R}_0<1$. For the uninfected population we have

$$F(X,0)=\begin{pmatrix}\frac{\Lambda}{\mu}\\0\\0\end{pmatrix} \tag{20}$$

and $X\in\mathbb{R}^3$ denotes the uninfected compartments in the model (2), we have
$G(X,Z)=CZ-\hat{G}(X,Z)$
where

$$C=\begin{pmatrix}
-m_2 & \omega_1\beta_{1R}S_0 & \omega_1\beta_{2R}S_0 & \omega_1\beta_{4R}S_0 & 0 & \omega_1\beta_{1S}S_0 & \omega_1\beta_{2S}S_0 & \omega(\beta_{3R}+\beta_{3S})S_0\\
m_{15} & -m_3 & \upsilon_R & 0 & 0 & 0 & 0 & 0\\
\alpha_R\kappa_R & \eta_R & -m_4 & 0 & 0 & 0 & 0 & 0\\
0 & d_R^2\phi_R^2 & d_R^1\phi_R^1 & -m_5 & 0 & 0 & 0 & 0\\
0 & (1-\omega)\beta_{1R}S_0 & (1-\omega)\beta_{2R}S_0 & (1-\omega)\beta_{4R}S_0 & -m_{12} & (1-\omega)\beta_{1S}S_0 & (1-\omega)\beta_{2S}S_0 & (1-\omega)(\beta_3S+\beta_3R)S_0\\
0 & 0 & 0 & 0 & m_{15} & -m_7 & \upsilon_S & 0\\
0 & 0 & 0 & 0 & \alpha_S\kappa_S & \eta_S & -m_8 & 0\\
0 & \varepsilon_1 & \varepsilon_2 & \varepsilon_3 & 0 & \varepsilon_4 & \varepsilon_5 & -m_{10}
\end{pmatrix}$$

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Thus,

$$\hat{G}(X,Z) = \begin{pmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \\ \hat{G}_3(X,Z) \\ \hat{G}_4(X,Z) \end{pmatrix} \begin{pmatrix} \omega_1\lambda S \\ 0 \\ 0 \\ 0 \\ (1-\omega_1)\lambda S \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{21}$$

Therefore, Since $\frac{S}{1+\alpha C} \leq S^0$, we have $\hat{G}(X,Z) \geq 0$. The global stability of $X^0 = (\omega_1\lambda S, 0, 0, 0, (1-\omega_1)\lambda S, 0, 0, 0)$ of the system $\frac{dX}{dt} = F(X^0, 0)$ is easy to verify. Therefore, X^0 is globally asymptotically stable if $\mathcal{R}_0 < 1$. This completes the proof. □

3.5 Global stability for giardiasis-endemic equilibrium

Theorem 4.5 : The endemic equilibrium is globally asymptotically stable if $R_0^G > 1$ and unstable otherwise.

Proof: Consider the following Lyapunov function

$L : (S, E_R, I_R, A_R, T_R, R_R, E_S, I_S, A_S, R_S, G) \in \Omega \subset \mathbb{R}^{11}_+$

$S, E_R, I_R, A_R, T_R, R_R, E_S, I_S, A_S, R_S, G > 0$

$$\begin{cases} L &= (S - S^* - S^* \ln \frac{S}{S^*}) + (E_R - E_R^* - E_R^* \ln \frac{E_R}{E_R^*}) + (I_R - I_R^* - I_R^* \ln \frac{I_R}{I_R^*}) + (A_R - A_R^* - A_R^* \ln \frac{A_R}{A_R^*}) \\ &\quad + (T_R - T_R^* - T_R^* \ln \frac{T_R}{T_R^*}) + (R_R - R_R^* - R_R^* \ln \frac{R_R}{R_R^*}) + (E_S - E_S^* - E_S^* \ln \frac{E_S}{E_S^*}) \\ &\quad + (I_S - I_S^* - I_S^* \ln \frac{I_S}{I_S^*}) + (A_S - A_S^* - A_S^* \ln \frac{A_S}{A_S^*}) + (R_S - R_S^* - R_S^* \ln \frac{R_S}{R_S^*}) \\ &\quad + (G - G^* - G^* \ln \frac{G}{G^*}) \end{cases} \tag{22}$$

Where L is C^1 within the region Ω. P^* is the global minimum of L on Ω and

$L : (S, E_R, I_R, A_R, T_R, R_R, E_S, I_S, A_S, R_S, G) = 0$

Thus, the time derivative of is given by:

$$\begin{cases} \frac{dL}{dt} = \left(1 - \frac{S}{S^*}\right) \frac{dS}{dt} + \left(1 - \frac{E_R}{E_R^*}\right) \frac{dE_R}{dt} + \left(1 - \frac{I_R}{I_R^*}\right) \frac{dI_R}{dt} + \left(1 - \frac{A_R}{A_R^*}\right) \frac{dA_R}{dt} \\ + \left(1 - \frac{T_R}{T_R^*}\right) \frac{dT_R}{dt} + \left(1 - \frac{R_R}{R_R^*}\right) \frac{dR_R}{dt} + \left(1 - \frac{E_S}{E_S^*}\right) \frac{dE_S}{dt} + \left(1 - \frac{I_S}{I_S^*}\right) \frac{dI_S}{dt} \\ + \left(1 - \frac{A_S}{A_S^*}\right) \frac{dA_S}{dt} + \left(1 - \frac{R_S}{R_S^*}\right) \frac{dR_S}{dt} + \left(1 - \frac{G}{G^*}\right) \frac{dG}{dt} \end{cases} Z \tag{23}$$

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Hence,

$$L = \begin{cases} -\left(\frac{S-S^*}{S}\right)\left[(\rho_R(R_R+R_R^*)-\mu(S-S^*)+\rho_S(R_S+R_S^*)+\omega_1\lambda(S-S^*)-(1-\omega_1)\lambda(S-S^*))\right] \\ -\left(\frac{E_R-E_R^*}{E_R}\right)\left[(\mu+\kappa_R)(E_R-E_R^*)\right] \\ -\left(\frac{I_R-I_R^*}{I_R}\right)\left[\nu_R(A_R-A_R^*)-(\psi_R+\eta_R+\epsilon_1+\phi_R^2+\mu)(I_R-I_R^*)\right] \\ -\left(\frac{A_R-A_R^*}{A_R}\right)\left[\eta_R(I_R-I_R^*)-(\nu_R+\mu+\delta_R+\epsilon_2+\phi_R^1)(A_R-A_R^*)\right] \\ -\left(\frac{T_R-T_R^*}{T_R}\right)\left[d_R^1\phi_R^1(I_R-I_R^*)-(\gamma_R+\epsilon_3+\mu)(T_R-T_R^*)\right] \\ -\left(\frac{R_R-R_R^*}{R_R}\right)\left[(1-d_R^1)\phi_R^1(A_R-A_R^*)-(\rho_R+\mu)(R_R-R_R^*)+(1-d_R^2)\phi_R^2(I_R-I_R^*)\right] \\ -\left(\frac{E_S-E_S^*}{E_S}\right)\left[(\mu+\kappa_s)(E_S-E_S^*)\right] \\ -\left(\frac{I_S-I_S^*}{I_S}\right)\left[\nu_S(A_S-A_S^*)-(\psi_s+\eta_S+\epsilon_4+\phi_S^2+\mu)(I_S-I_S^*)\right] \\ -\left(\frac{A_S-A_S^*}{A_S}\right)\left[\eta_S(I_S-I_S^*)-(\nu_S+\mu+\delta_s+\epsilon_5+\phi_S^1)(A_S-A_S^*)\right] \\ -\left(\frac{R_S-R_S^*}{R_S}\right)\left[d_S^2\phi_S^2(I_S-I_S^*)-(\rho_S+\mu)(R_S-R_S^*)\right] \\ -\left(\frac{G-G^*}{G}\right)\left[\epsilon_1(A_R-A_R^*)+\epsilon_3(T_R-T_R^*)+\epsilon_4(I_S-I_S^*)+\epsilon_5(A_S-A_S^*)-(\tau+\chi)(G-G^*)\right] \end{cases} \tag{24}$$

Therefore, $L < 0$, hence L = 0 *iff*

$$S = S^*, \quad E_R = E_R^*, \quad I_R = I_R^*, \quad A_R = A_R^*, \quad T_R = T_R^*, \quad R_R = R_R^*,$$

$$E_S = E_S^*, \quad I_S = I_S^*, \quad A_S = A_S^*, \quad R_S = R_S^*, \quad G = G^*$$

The biggest set, where the solution remain within Ω where the expression L = 0 holds, for the variables $(S, E_R, I_R, A_R, T_R, R_R, E_S, I_S, A_S, R_S, G)$, is singleton P^*, representing the endemic equilibrium state. According to Lassalle's principle, (1986), the interior of Ω is Globally Asymptotically Stable for P^*.

3.6 Giardiasis model sensitivity analysis

In order to determine how these parameters affect the spread of giardiasis, this subsection investigates on how the system (2) parameters affect the basic reproduction number $\mathcal{R}_0$. The sensitivity index was obtained by partially differentiating $\mathcal{R}_0$ with regard to the model (2) parameters. For example, we develop a formula to obtain the sensitivity index

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of all the basic parameters in this work, which is defined as ψ is $\frac{\partial \mathcal{R}_0}{\partial \psi} \times \frac{\partial \psi}{\partial \mathcal{R}_0}$, The same is applied to all model (2) parameters. The result is presented in the Table 3.

Table 3. Sensitivity indices for the giardiasis model $\mathcal{R}_0$

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Parameter	Baseline values	References	Sensitivity Index	Remarks
Λ	0.0036	Liana & Chuma (2023)	-2.9557 × 10	Negative
α	0.5000	Conners (2021)	0.9977	Positive
κ_R	1/10	Kitowska & Sadkowska-Todys (2021)	-0.9996	Negative
κ_S	1/10	Kitowska & Sadkowska-Todys (2021)	-0.9996	Negative
δ_R	0.1430	Liana & Chuma (2023)	-0.2616	Negative
δ_S	0.1430	Liana & Chuma (2023)	-0.2616	Negative
η_S	0.1000	Liana & Chuma (2023)	0.00029	Positive
η_R	0.0050	Assumed	-0.0683	Negative
υ_S	0.0030	Liana & Chuma (2023)	-0.0054	Negative
β_{1S}	0.00035	Mpeshe & Nyerere (2021)	0.0011	Positive
β_{2S}	0.4500	Mpeshe & Nyerere (2021)	0.9989	Positive
β_{3S}	0.1000	Mpeshe & Nyerere (2021)	0.6667	Positive
β_{1R}	0.8000	Assumed	0.6836	Positive
β_{2R}	0.4000	Assumed	0.2871	Positive
β_{4R}	0.2000	Assumed	0.0293	Positive
ϕ_{1R}	0.2000	Mpeshe & Nyerere (2021)	-0.0818	Negative
ϕ_{2R}	0.2000	Assumed	-0.2587	Negative
d_{1R}	0.3	Assumed	0.0147	Positive
d_{2R}	0.3	Assumed	0.0147	Negative
ϕ_{1S}	0.3	Assumed	-0.05488	Negative
ϕ_{2S}	0.13	Assumed	-0.00038	Negative
γ_R	0.2	Mpeshe & Nyerere (2021)	-0.00014	Negative
τ	0.03	Waters et al. (2016)	-0.0566	Negative
ϵ_1	0.25	Mpeshe & Nyerere (2021)	-0.3417	Negative
ϵ_2	0.25	Mpeshe & Nyerere (2021)	-0.1205	Negative
ϵ_3	0.15	Assumed	-0.0126	Negative

We observe that the parameters β_{1R}, β_{2R}, β_{3R}, β_{1S}, β_{2S}, β_{3S} have positive sensitive indices, this is means that as the parameter is increased, the basic reproduction numbers $\mathcal{R}_0^R$, and $\mathcal{R}_0^S$ rise. The remaining parameters, δ_R, α_R, ϵ_2, κ_S, ϕ_S^1, and ν_S have negative values which implies that $\mathcal{R}_0^R$, and $\mathcal{R}_0^S$ decreases for higher values of the parameters.

4 Numerical simulations

In this section, the state variables were varied against time using the initial conditions

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Table 4. Initial values for the variables.

Variables	Description	Values	Sources
$S(t)$	Number of susceptible individuals at time t	4000	Liana & Chuma (2023)
$E_R(t)$	Number of exposed individuals with resistant strain at time t	1000	Assumed
$I_R(t)$	Number of infectious individuals with resistant strain at time t	800	Assumed
$A_R(t)$	Number of asymptomatic individuals with resistant strain at time t	400	Assumed
$T_R(t)$	Number of individuals undergoing second-line treatment for resistant strain at time t	300	Assumed
$R_R(t)$	Number of individuals recovered from resistant strain at time t	300	Assumed
$E_S(t)$	Number of exposed individuals to sensitive strain at time t	2000	Liana & Chuma (2023)
$I_S(t)$	Number of infectious individuals with sensitive strain at time t	1200	Liana & Chuma (2023)
$A_S(t)$	Number of asymptomatic individuals with sensitive strain at time t	600	Liana & Chuma (2023)
$R_S(t)$	Number of individuals recovered from sensitive strain at time t	500	Liana & Chuma (2023)
$G(t)$	Concentration of *Giardia lamblia* cysts in the environment at time t	50000	Liana & Chuma (2023)

The variation of susceptible population against time

The graph shows that the susceptible population starts at about 4,000 but drops to zero almost immediately within the first day and stays there for the entire 100-day period. This indicates that all individuals are instantly exposed or infected at the outset, likely due to extremely high infection pressure or specific initial conditions in the model. Unlike typical real-world scenarios, this outcome reflects a modeling choice where the susceptible class is fully depleted from the beginning, with the population distributed entirely among exposed, infected, or recovered compartments.

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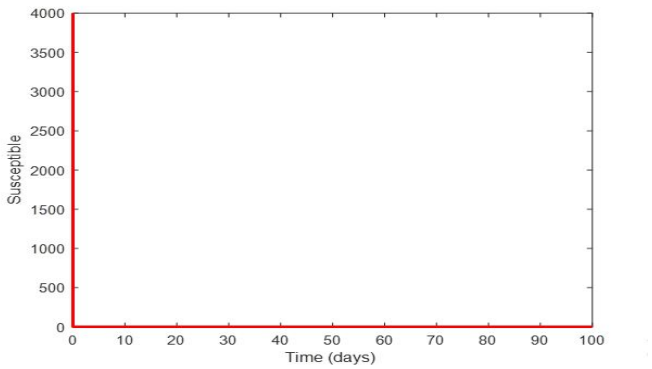

Figure 2. Dynamics of susceptible population against time

The variation of the exposed drug-resistant population against time

The graph shows that the exposed drug-resistant population starts very high (approximately 1,800) but drops sharply within the first 20 days to below 200, indicating rapid transition out of exposure. Afterward, the decline slows, forming a long-tail decay, and by about day 50, cases approach zero and stay negligible through day 100. This suggests most exposures are short-lived, with the system quickly eliminating new cases.

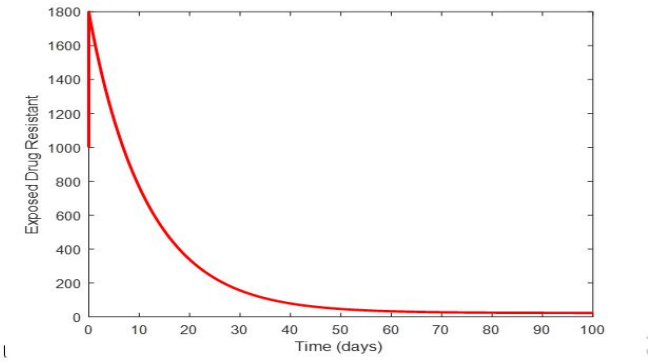

Figure 3. Dynamics of exposed with resistant strain population against time

The variation of the symptomatic drug-resistant infected population against time

The symptomatic drug-resistant infected population starts near 800 cases but falls sharply to under 100 within the first 10 days. By day 30–40, cases approach zero and remain minimal, indicating near elimination. The steep early decline suggests effective early interventions, natural recovery, reduced transmission, or a mix of these factors, preventing long-term persistence.

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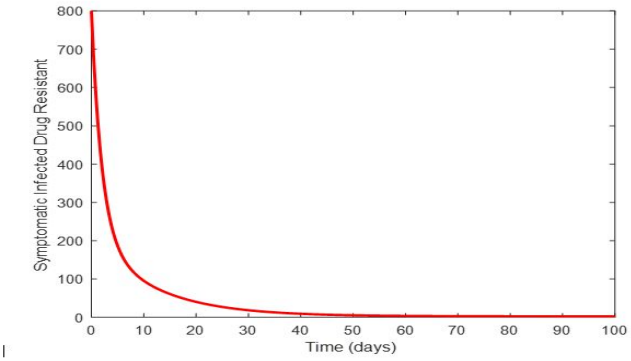

Figure 4. Dynamics of symptomatic with resistant strain population against time

The variation of the asymptomatic drug-resistant infected population against time

The asymptomatic drug-resistant infected population begins near 400 cases and drops rapidly in the first few days, showing a high initial removal rate. By 20–30 days, numbers are minimal, and after 40 days they approach zero. The decline is fast and sustained, with most cases disappearing within a month, though final eradication is slower and involves very few remaining infections.

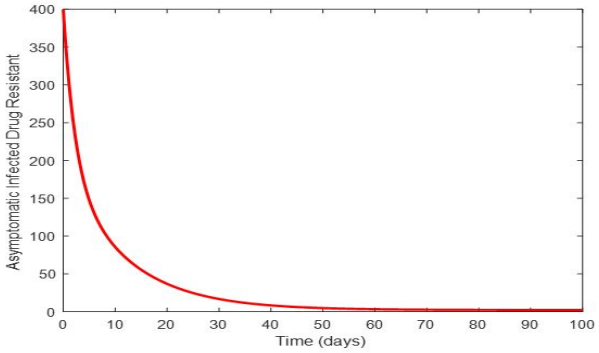

Figure 5. Dynamics of asymptomatic infected with resistant strain population against time

The variation of the second-line treated drug-resistant infected population against time

The second-line treated drug-resistant population starts near 310 and drops sharply in the first 10–15 days. By day 30, fewer than 20 remain, and after day 50 the number stays near zero. This rapid decline suggests effective removal through treatment, death, or transition consistent with an exponential decay pattern.

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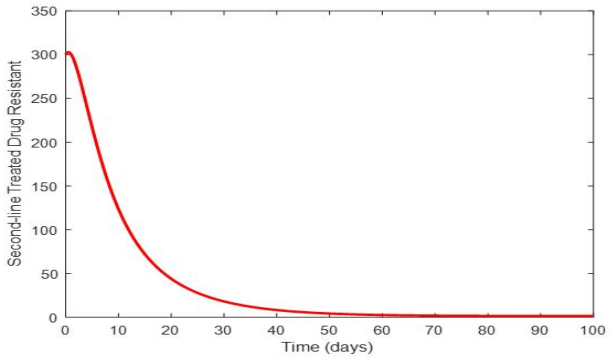

Figure 6. Dynamics of second-line treated with resistant strain population against time

The variation of the recovered drug-resistant infected population against time

The recovered drug-resistant population starts near 300, rises sharply to over 430 within the first week, then declines rapidly. By day 30 it drops below 50, and by days 70–80 it nears zero, remaining stable afterward. The pattern suggests an early recovery surge followed by decline due to lack of new cases, waning immunity, or other removal processes.

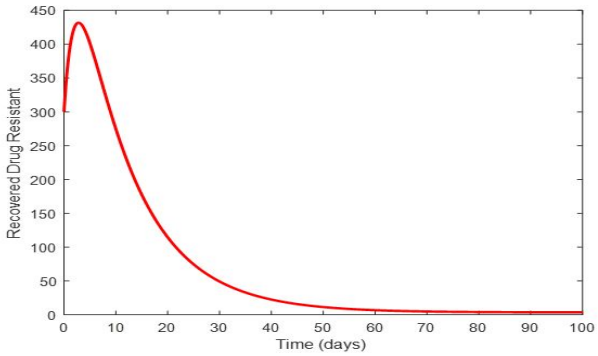

Figure 7. Dynamics of recovered with resistant strain population against time

The variation of the exposed drug-sensitive population against time

The exposed drug-sensitive population starts just above 5,000 and drops sharply in the first 20–25 days to under 1,000. Afterward, the decline slows, and by day 50 the number is nearly zero, remaining minimal through day 100. This indicates exposure is short-lived for most individuals, with infection risk rapidly diminishing.

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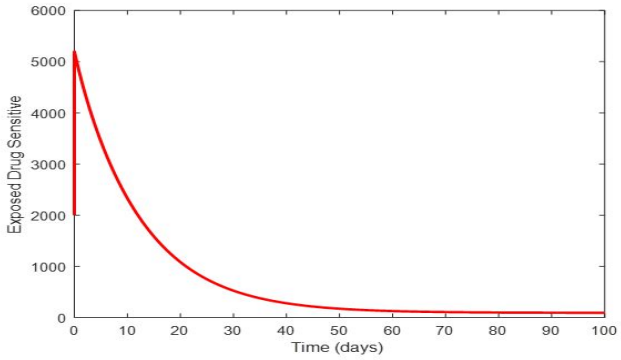

Figure 8. Dynamics of exposed with sensitive strain population against time

The variation of the symptomatic drug-sensitive population against time

The symptomatic drug-sensitive population begins near 1,200 cases and drops sharply to under 200 within 15–20 days. The decline then slows, and by day 50 cases are nearly zero, staying minimal thereafter. This rapid reduction is likely due to effective treatment, high recovery rates, and control measures, making the infection clear faster than drug-resistant cases.

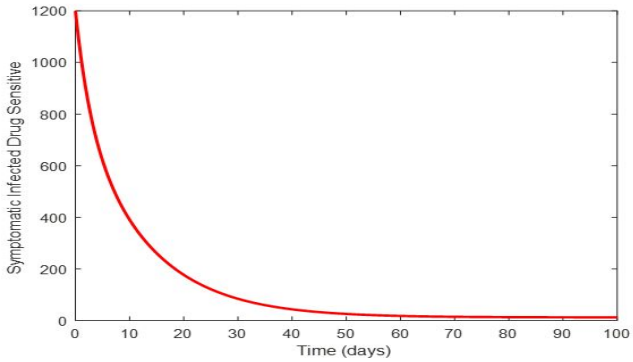

Figure 9. Dynamics of symptomatic with sensitive strain population against time

The variation of the asymptomatic drug-sensitive population against time

The asymptomatic drug-sensitive population starts near 600, rises slightly in the first few days, then drops steeply over 20–30 days to under 100. After day 30, the decline slows, and by day 50 cases are near zero, staying minimal through day 100. This pattern, typical of exponential decay, suggests rapid clearance due to treatment or natural recovery.

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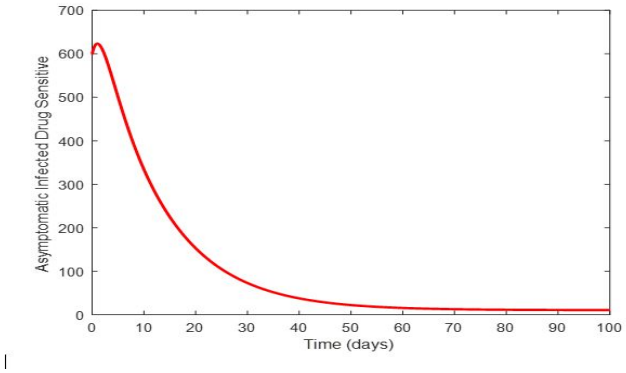

Figure 10. Dynamics of asymptomatic with sensitive strain population against time

The variation of the recovered drug-sensitive population against time

The recovered drug-sensitive population starts near 500 and rises sharply in the first 20–25 days, reaching about 1,950 by day 50. It then slows and plateaus just over 2,000 by day 100. This pattern shows rapid early recovery due to effective treatment and immunity, with recovery rates slowing as infections decline contrasting with the drug-resistant recovery trend, which peaks early then falls.

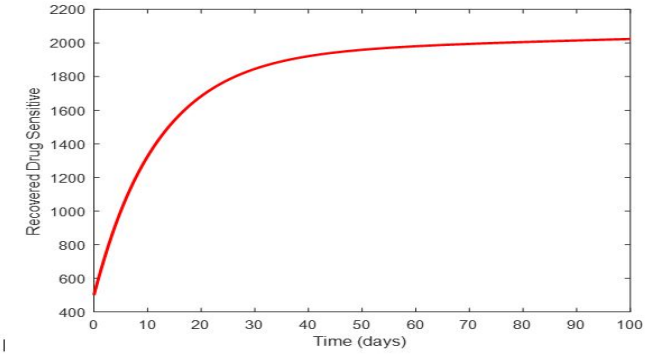

Figure 11. Dynamics of recovered with sensitive strain population against time

The variation of the Giardia cyst concentration against time

The environmental Giardia lamblia cyst concentration starts around 5×10^4 and drops sharply to near zero within the first 10 days. Afterward, only negligible amounts remain, declining slowly to near elimination by day 100. This rapid loss suggests short environmental survival due to degradation factors, making transmission risk minimal after the initial decay phase.

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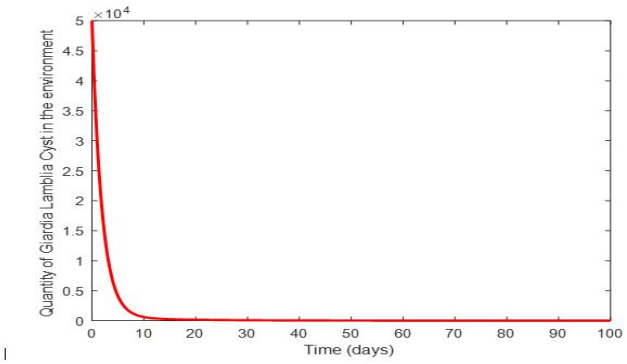

Figure 12. Dynamics of giardia lamblia cysts population against time

5 Conclusion

This paper developed and analyzed a deterministic compartmental model for the transmission dynamics of Giardiasis, incorporating both drug-sensitive and drug-resistant strains. The model, expressed as a nonlinear system of ordinary differential equations, stratified the host population into epidemiologically relevant classes and included environmental contamination as a key driver of persistence. The basic reproduction numbers for both strains were derived using the next-generation matrix method and identified as the fundamental thresholds governing the system. Analytical results established that the disease-free equilibrium is stable when both reproduction numbers are below unity, while endemic equilibria arise when either threshold exceeds one. The interplay between the two strains revealed scenarios of coexistence and competitive exclusion, determined by the relative sizes of the reproduction numbers. Stability analysis confirmed the asymptotic behavior of equilibria, and sensitivity analysis highlighted treatment efficacy, contact rates, and environmental parameters as the most influential factors. Numerical simulations validated the theoretical results and showed that resistant strains may persist under incomplete treatment, underscoring the importance of environmental interventions. Overall, this work provides a rigorous mathematical framework for understanding multi-strain giardiasis dynamics.

Availability of data and material

Not Applicable.

Competing Interest

No competing interest is hereby declared by the authors.

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Authors' contributions

The development and simulation of the model was done by Abdulfatai, Atte Momoh. Model analysis, discussion of results and typesetting were implemented by Bello Barakat.

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All authors approved the final manuscript.

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