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| RESEARCH ARTICLE

Investigating the Dynamics of Single and Dual Infection of Schistosoma Species: A Mathematical Modeling Perspective

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| ABSTRACT
Schistosomiasis is a prevalent parasitic disease that poses significant challenges to effective control measures, particularly in the presence of dual infections. This paper presents a study that aims to investigate the underlying mechanisms of schistosomiasis transmission through mathematical modeling, focusing on the dynamics of both single and dual infections, as well as the interaction between different species or strains of Schistosoma parasites. Sensitivity analysis of the basic reproduction number, R_0, reveals the substantial influence of parameters such as β_{SH} and μ_S on disease transmission. The findings highlight the crucial need for comprehensive management strategies that address the complexities of dual infections and target influential parameters to effectively reduce disease transmission and mitigate the impact of schistosomiasis in endemic areas.

| KEYWORDS
Schistosomiasis, Schistosoma Mansoni, Schistosoma Haematobium, Dual Infection, Single Infection

| ARTICLE INFORMATION
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1. Introduction

Schistosomiasis, commonly referred to as bilharzia, is an infectious disease brought about by parasitic trematode worms belonging to the genus Schistosoma (Ross et al., 2013). These worms reside within the blood vessels of the human body, leading to various health complications. According to the World Health Organization (WHO), schistosomiasis remains a significant global health concern, with nearly 240 million people requiring preventive chemotherapy (World Health Organization, 2013). More than 90 percent of these cases are reported in sub-Saharan Africa (World Health Organization, 2013). Schistosomiasis affects a substantial number of individuals worldwide, with an estimated 290.8 million people infected (Fattori et al., 2023). Approximately 85 percent of these cases are concentrated in underdeveloped regions of Africa, leading to a staggering annual death toll of around 15,000 (Bakare & Nwozo, 2016). Particularly alarming is the impact on children under the age of 14, who are disproportionately affected by schistosomiasis infection in numerous parts of the world (Yang, 2001).

Schistosomes, a type of water-borne flatworm or blood fluke, are the primary culprits behind human schistosomiasis (Chiyaka & Garira, 2009). Among these parasites, three predominant species, namely Schistosoma Japonicum, Schistosoma Haematobium, and Schistosoma Mansoni, are widespread and commonly encountered (Chiyaka & Garira, 2009). Schistosoma Haematobium, in particular, exhibits a predilection for the urinary tract, kidneys, and reproductive systems, with high prevalence rates observed in regions across Africa and the Middle East (Chiyaka & Garira, 2009; Ross et al., 2013). Infections caused by S. Mansoni and S. japonicum often result in chronic liver and intestinal fibrosis (Inobaya et al., 2018).

The life cycle of Schistosoma begins when eggs are released into freshwater bodies through the urine or feces of infected individuals (Castillo-Chavez et al., 2008). These eggs hatch, releasing larvae that infect specific types of snails, which act as intermediate hosts. Within the snails, the larvae undergo development and transformation, eventually emerging as free-swimming cercariae (Coustau et al., 2015). These cercariae actively seek out human hosts by penetrating the skin upon contact with contaminated water. Once inside the body, they migrate through the bloodstream, reaching their target organs and maturing into adult worms. Male and female worms pair up and produce eggs, which are either excreted in urine or feces, perpetuating the cycle of infection. The transmission of schistosomiasis is closely linked to poor sanitation, inadequate access to clean water, and human contact with infested water sources. Activities such as swimming, bathing, and agricultural practices that involve contact with contaminated water contribute to the spread of the disease. Education, improved sanitation, and provision of clean water sources are crucial for effective control and prevention strategies.

2. Literature Review

Extensive research efforts have been dedicated to assessing the infection rates and occurrence of schistosomiasis, particularly in areas where it poses a considerable burden on the population. In the context of the Volta Lake region of Ghana, studies have been conducted to examine the prevalence and impact of both urinary schistosomiasis and female genital schistosomiasis (FGS) among the local communities. Sacolo et al. (2018) conducted a comprehensive study to assess the prevalence of urinary schistosomiasis among individual adults in the Volta Lake region of Ghana. Their findings revealed a concerning prevalence of the disease, indicating that a significant proportion of adults in the region were affected by urinary schistosomiasis. The study by Yirenya et al. (2016) assessed the prevalence of female genital schistosomiasis (FGS) in communities situated in close proximity to the water bodies of the Volta basin in Ghana. Their findings showed a prevalence rate of 10.6 percent for FGS in the region, with women diagnosed with FGS reporting symptoms such as copious discharge, vaginal itch, and lower abdominal pain more frequently compared to those without FGS. In a study by Kukula et al. (2019), knowledge gaps regarding female genital schistosomiasis were identified among communities and local health workers. The study revealed that all sampled groups recognized Volta Lake as a significant source of schistosomiasis, emphasizing the importance of targeted interventions and increased awareness in affected areas.

The impact of schistosomiasis on the well-being and overall health of the local populace remains a pressing matter that necessitates urgent attention and effective interventions. Of particular concern is the phenomenon of dual infection, which arises when individuals already infected with one species of Schistosoma, such as Schistosoma Mansoni, become reinfected with another species, such as Schistosoma Haematobium, and vice versa. This co-infection scenario further intensifies the severity of the disease, necessitating a comprehensive examination and analysis of the underlying issues. Chelkeba et al. (2020) conducted a research study aiming to determine the prevalence of helminth infections in schoolchildren, analyzing a sample size of 6,897 individuals. The study identified Schistosoma haematobium, hookworm and Schistosoma Mansoni as the most prevalent helminth species among the schoolchildren in the district. Additionally, the study revealed that hookworm and S. Mansoni were more commonly found in multiple infections with other helminths rather than as single-species infections. In a study by Qi et al. (2019), a schistosomiasis model was developed, taking into account both single and multiple infections. The analysis of the model indicates the possibility of a backward bifurcation when there is a high transmission rate from multiple infected humans to snails. Britton et al. (2015) stated that the infectivity of an infected human host is dependent on the infection history. Therefore, multiple infections may worsen a patient's condition. This means that the patient's previous infections may affect their current infection and make it more severe.

In the context of schistosomiasis treatment, the presence of dual infections poses a significant obstacle despite the availability of drug therapies, hindering effective control measures. Dual infections, characterized by the coexistence of different species or strains of Schistosoma parasites in individuals, complicate treatment regimens, leading to reduced efficacy and an increased risk of treatment failure (Leenstra et al., 2006). This paper seeks to investigate the underlying mechanisms of schistosomiasis transmission through mathematical modeling, specifically focusing on the dynamics of both single and dual infections, as well as the interaction between different species or strains of Schistosoma parasites. By modeling the prevalence and impact of both single and dual infections, the paper aims to shed light on how these infections contribute to the transmission and severity of schistosomiasis. A model can serve as a valuable tool for understanding the underlying mechanisms of schistosomiasis transmission. By addressing these complexities, it becomes imperative to develop comprehensive management strategies that can effectively tackle the multifaceted challenges posed by schistosomiasis.

3. The Model

This section introduces a mathematical model formulation that captures the dynamics of single and dual infection of Schistosoma species. We divide the human population into three separate groups: the susceptible class (S_H), the single Infected Class (with Haematobium, I_{HA} and Mansoni, I_{HM} separately) and dual infected class (with both Haematobium and Mansoni, I_D). The infected class, both the single and dual individuals, after being treated with drugs, will enter the susceptible group. Here, γ_H represents the recovery rate for the three human classes. The transmission rate from a single infected snail to human is β_{SH}.

However, snails are divided into two classes: susceptible snails, (S_S), and infectious snails (that is, snails with S. Mansoni, I_{SM} and snails with S. Haematobium, I_{SA}) infections.
The transmission rate from infectious humans to snails is β_{HS}. Since no single snail can serve as an intermediary host for the two species, we denote ϵ to describe the relative transmission rate from dual infectious humans to snails with $0 < \epsilon < 1$.

The susceptible class S_H of humans are created through birth, with individuals recruited at a rate of Λ_H per year. It is reduced through contact with snails infected by either S. Haematobium (I_{SA}) or S. Mansoni (I_{SM}) at a rate of β_{SH}. Additionally, natural deaths that occur at a rate of μ_S reduce the population. However, the susceptible class of humans (S_H) increases through the recovery of individuals from both the single infected class (I_{HA} and I_{HM}) and the dual infected class, I_D, at a rate of γ_H. Therefore, the rate at which the population in the susceptible human class (S_H) changes can be expressed as follows:

$$\frac{dS_H}{dt} = \Lambda_H - \beta_{SH} I_{SM} S_H + \gamma_H I_{HM} - \mu_H S_H - \beta_{SH} I_{SA} S_H + \gamma_H I_{HA} + \gamma_H I_D \qquad (1)$$

3.1 Single Infected Human Class

The population of the single infected human class increases through the contraction of schistosomiasis via contact between the susceptible human class, S_H, and snails infected by either S. Mansoni (I_{SM}) or S. Haematobium (I_{SA}) at a transmission rate of β_{SH}.

It decreases through disease-related deaths occurring at a rate of μ_H and through the recovery of humans at γ_H, following treatment with drugs. Additionally, the population of the single infected class decreases when an individual infected with S. Mansoni (I_{HM}) becomes reinfected by a snail infected with S. haematobium (I_{SA}), and similarly when an individual infected with S. haematobium (I_{HA}) becomes reinfected by a snail infected with S. Mansoni (I_{SM}). These interrelated factors collectively shape the dynamics of the single infected population. Hence, this is given by

$$\frac{dI_{HM}}{dt} = \beta_{SH} I_{SM} S_H - \beta_{SH} I_{SA} I_{HM} - \gamma_H I_{HM} - \mu_H I_{HM} \qquad (2)$$

$$\frac{dI_{HA}}{dt} = \beta_{SH} I_{SA} S_H - \beta_{SH} I_{SM} I_{HA} - \gamma_H I_{HA} - \mu_H I_{HA} \qquad (3)$$

3.2 Dual Infected Class

The number of individuals afflicted by both strains of schistosomiasis (dual infected class) is increased through the reinfection of the single infected class, $I_{SM} I_{HA}$ and $I_{SA} I_{HM}$, at a rate of β_{SH}. However, it decreases as a result of morbidity-related death occuring at μ_H, where infected individuals succumb to the disease, and through human spontaneous recovery at a rate of γ_H. Hence, this is given as

$$\frac{dI_D}{dt} = \beta_{SH} I_{SA} I_{HM} + \beta_{SH} I_{SM} I_{HA} - \gamma_H I_D - \mu_H I_D \qquad (4)$$

The population of snails are recruited through the birth rate of Λ_S per year to the susceptible class, S_S. It is reduced through contact with a single infected human class, that is I_{HM} and I_{HA}, and dual infected human class, I_D, at a rate of β_{HS}. The susceptible snail class, however, is reduced by death ocurring at μ_S,. Hence, the rate at which the population in the susceptible snail class (S_S) changes is

$$\frac{dS_S}{dt} = \Lambda_S - \beta_{HS} S_S I_{HM} - \beta_{HS} S_S I_D \epsilon - \beta_{HS} S_S I_{HA} - \mu_S S_S \qquad (5)$$

3.3 Snail Infected Class

The infected snail class increases by getting the disease through contact of the susceptible snails with the single and dual infected human class at a rate of β_{HS} and ϵ respectively. It decreases by death occurring at μ_S.

$$\frac{dI_{SM}}{dt} = \beta_{HS}S_S I_{HM} - \beta_{HS}S_S I_D \epsilon - \mu_S I_{SM} \quad (6)$$

$$\frac{I_{SA}}{dt} = \beta_{HS}S_S I_{HA} + \beta_{HS}S_S I_D \epsilon - \mu_S I_{SA} \quad (7)$$

Combining the aforementioned assumptions yields the set of equations which represents the single and dual infection model:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \beta_{SH}I_{SM}S_H + \gamma_H I_{HM} - \mu_H S_H - \beta_{SH}I_{SA}S_H + \gamma_H I_{HA} + \gamma_H I_D \\
\frac{dI_{HM}}{dt} &= \beta_{SH}I_{SM}S_H - \beta_{SH}I_{SA}I_{HM} - \gamma_H I_{HM} - \mu_H I_{HM} \\
\frac{dI_{HA}}{dt} &= \beta_{SH}I_{SA}S_H - \beta_{SH}I_{SM}I_{HA} - \gamma_H I_{HA} - \mu_H I_{HA} \\
\frac{dI_D}{dt} &= \beta_{SH}I_{SA}I_{HM} + \beta_{SH}I_{SM}I_{HA} - \gamma_H I_D - \mu_H I_D \\
\frac{dS_S}{dt} &= \Lambda_S - \beta_{HS}S_S I_{HM} - \beta_{HS}S_S I_D \epsilon - \beta_{HS}S_S I_{HA} - \mu_S S_S \\
\frac{dL_{SM}}{dt} &= \beta_{HS}S_S I_{HM} - \beta_{HS}S_S I_D \epsilon - \mu_S I_{SM} \\
\frac{I_{SA}}{dt} &= \beta_{HS}S_S I_{HA} + \beta_{HS}S_S I_D \epsilon - \mu_S I_{SA}
\end{aligned} \quad (8)$$

with initial conditions, $S_H(0) = S_{H,0}, I_{HM}(0) = I_{HM,0}, I_{HA}(0) = I_{HA,0}, I_D(0) = I_{D,0}, S_S(0) = S_{S,0}, I_{SM}(0) = I_{SM,0}, I_{SA}(0) = I_{SA,0}$

3.4 Model Dynamics

We perform a qualitative analysis of the model to investigate the existence and stability of equilibrium points to gain insights into the long-term behaviour of the disease. Our objective is to ascertain whether the disease will be eliminated (disease-free equilibrium) or if it will persist and become endemic.

The system of equations for a diseases free state gives;

$$\begin{aligned}
S'_H &= \Lambda_H - \mu_H S_H \\
I'_{HM} &= 0 \\
I'_{HA} &= 0 \\
I'_D &= 0 \\
S'_S &= \Lambda_S - \mu_S S_S \\
I'_{SM} &= 0 \\
I'_{SA} &= 0
\end{aligned} \quad (9)$$

This equilibrium is present for all parameter values considered in the analysis.

As the disease naturally dies out, the solution converges asymptotically to a disease-free equilibrium, denoted as E_0, characterized by the following form: $E_0 = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_S}{\mu_S}, 0, 0\right)$.

Thus, the basic reproduction number, R_0 serves as the threshold that determines the stability of this equilibrium.

When the basic reproduction number (R_0) is less than 1, it signifies that, on average, each infected individual produces fewer than one new infection leading to the eventual extinction of the disease. Conversely, when $R_0 > 1$, it shows that, on average, each infected person transmits the disease to more than one new individual, allowing the spread and invasion of the disease within the susceptible human class. By evaluating the value of R_0, we can determine the potential for disease eradication or its ability to persist and propagate within the population.

To calculate the R_0, we employ the concept of the next generation matrix, $K = FV^{-1}$ (Castillo-Garsow & Castillo-Chavez, 2020). The matrix F characterizes the rate at which new infections occur in different compartments:

$$\begin{aligned}
F_1 &= \beta_{SH}I_{SM}S_H \\
F_2 &= \beta_{SH}I_{SA}S_H
\end{aligned}$$

$$\begin{aligned} F_3 &= 0 \\ F_4 &= \beta_{HS} S_S I_{HM} + \beta_{HS} S_S I_D \epsilon \\ F_5 &= \beta_{HS} S_S I_{HA} + \beta_{HS} S_S I_D \epsilon \end{aligned} \tag{10}$$

Where V represents the rate at which infectives are transferred from one compartment to another.

$$\begin{aligned} V_1 &= \gamma_H I_{HM} + \mu_H I_{HM} \\ V_2 &= \gamma_H I_{HA} + \mu_H I_{HA} \\ V_3 &= \gamma_H I_D + \mu_H I_D \\ V_4 &= \mu_S I_{SM} \\ V_5 &= \mu_S I_{SA} \end{aligned} \tag{11}$$

The matrices for F and V are formed as below;

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_{SH}\frac{\Lambda_H}{\mu_H} & 0 \\ 0 & 0 & 0 & 0 & \beta_{SH}\frac{\Lambda_H}{\mu_H} \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{HS}\frac{\Lambda_S}{\mu_S} & 0 & \beta_{HS}\frac{\Lambda_S}{\mu_S}\epsilon & 0 & 0 \\ 0 & \beta_{HS}\frac{\Lambda_S}{\mu_S} & \beta_{HS}\frac{\Lambda_S}{\mu_S}\epsilon & 0 & 0 \end{pmatrix} \tag{12}$$

$$V = \begin{pmatrix} \gamma_H + \mu_H & 0 & 0 & 0 & 0 \\ 0 & \gamma_H + \mu_H & 0 & 0 & 0 \\ 0 & 0 & \gamma_H + \mu_H & 0 & 0 \\ 0 & 0 & 0 & \mu_S & 0 \\ 0 & 0 & 0 & 0 & \mu_S \end{pmatrix} \tag{13}$$

The matrix FV^{-1} gives,

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_{SH}\Lambda_H}{\mu_H\mu_S} & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_{SH}\Lambda_H}{\mu_H\mu_S} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{HS}\Lambda_S}{(\gamma_H+\mu_H)\mu_S} & 0 & \frac{\beta_{HS}\Lambda_S\epsilon}{(\gamma_H+\mu_H)\mu_S} & 0 & 0 \\ 0 & \frac{\beta_{HS}\Lambda_S}{(\gamma_H+\mu_H)\mu_S} & 0 & 0 & 0 \end{pmatrix} \tag{14}$$

R_0 is given by the largest eigenvalue (ρ) of $K = FV^{-1}$.

$$R_0 = \rho(FV^{-1}) = \sqrt{\frac{\Lambda_H \Lambda_S \beta_{SH} \beta_{HS}}{\mu_S^2 \mu_H (\gamma_H + \mu_H)}} \tag{15}$$

Omitting the square root sign gives the same threshold for stability at 1, but for R_0^2, in this case, we have:

$$R_0^2 = \frac{\Lambda_H \Lambda_S \beta_{SH} \beta_{HS}}{\mu_S^2 \mu_H (\gamma_H + \mu_H)} \tag{16}$$

Considering the values of the parameters presented in Table 1, the calculated value of the basic reproduction number, $R_0 \approx 2.79$. This indicates that the disease-free equilibrium is unstable, suggesting the presence of an endemic equilibrium.

Table 1: Parameters

Parameters	Values (per year)	References
β_{SH}	0.0406	(Bakare & Nwozo, 2016)
β_{HS}	0.0032	(Qi et al., 2019)
γ_H	0.9	(Riley et al., 2008; Qi et al., 2019)
μ_H	0.014	(Qi et al., 2019)
Λ_H	25	(Feng et al., 2004; Qi et al., 2019)
Λ_S	8	(Feng et al., 2004; Qi et al., 2019)
μ_S	0.51	(Feng et al., 2004; Qi et al., 2019)
ϵ	0.7	(Qi et al., 2019)

4. Sensitivity Analysis of the Model

This is a valuable scientific tool that allows us to investigate the influence of independent variables on a dependent variable under specific assumptions. In the context of disease modeling, the estimation of parameter values often involves uncertainties. Therefore, it becomes imperative to conduct sensitivity analysis in order to assess the influence of these parameters on the basic reproduction number, R_0. Through this analysis, we can identify the parameters that wield significant influence over R_0, enabling us to determine the most efficacious control measures for disease management.

To compute the sensitivity of R_0 to the model parameters, we employ the normalized forward sensitivity index (Castillo-Garsow & Castillo-Chavez, 2020).

The normalized forward sensitivity index of R_0, denoted as $K_z^{R_0}$ is given by $K_z^{R_0} = \frac{\partial R_0}{\partial z} \times \frac{z}{R_0}$, where z represents each parameter in R_0.

The sensitivity indices of the reproduction number R_0 is given below;

$$
\begin{aligned}
K_{\Lambda_H}^{R_0} &= \frac{\partial R_0}{\partial \Lambda_H} \times \frac{\Lambda_H}{R_0} = +1.0 \\
K_{\Lambda_S}^{R_0} &= \frac{\partial R_0}{\partial \Lambda_S} \times \frac{\Lambda_S}{R_0} = +1.0 \\
K_{\beta_{SH}}^{R_0} &= \frac{\partial R_0}{\partial \beta_{SH}} \times \frac{\beta_{SH}}{R_0} = +1.0 \\
K_{\beta_{HS}}^{R_0} &= \frac{\partial R_0}{\partial \beta_{HS}} \times \frac{\beta_{HS}}{R_0} = +1.0 \\
K_{\mu_S}^{R_0} &= \frac{\partial R_0}{\partial \mu_S} \times \frac{\mu_S}{R_0} = -2.0 \\
K_{\mu_H}^{R_0} &= \frac{\partial R_0}{\partial \mu_H} \times \frac{\mu_H}{R_0} = -1.02 \\
K_{\gamma_H}^{R_0} &= \frac{\partial R_0}{\partial \gamma_H} \times \frac{\gamma_H}{R_0} = -0.5
\end{aligned}
\tag{17}
$$

4.1 Interpretation of Sensitivity Indices

A positive sensitivity index indicates that an increase in the value of that parameter corresponds to an increase in the value of R_0, while a decrease in each parameter value leads to a decline in the value of R_0 (Olaniyi & Obabiyi. 2014). Notably, the parameters $\Lambda_H, \Lambda_S, \beta_{SH}$ and β_{HS} all exhibit sensitivity indices of $+1$. This suggests that a 10 percent decrease or increase in the parameter values results in a proportional decrease or increase of 10 percent in the value of R_0.

The sensitivity indices with negative signs, however, indicate an inverse relationship between parameter values and the magnitude of the disease's basic reproduction number (Olaniyi & Obabiyi. 2014). Based on the calculated sensitivity indices, it is evident that, for example, a 10 percent increase or decrease in the parameter μ_S results in a significant 20 percent decrease or increase, respectively, in the value of R_0. Moreover, an increase of 10 percent in the recovery rate of humans, denoted as γ_H, corresponds to a notable 5 percent reduction in the R_0.

These insightful observations provide valuable insights into the significant role that these parameters play in shaping disease transmission dynamics. This knowledge is crucial for devising effective strategies to mitigate disease transmission. By comprehending how variations in parameters impact the value of R_0, it becomes possible to tailor public health interventions to

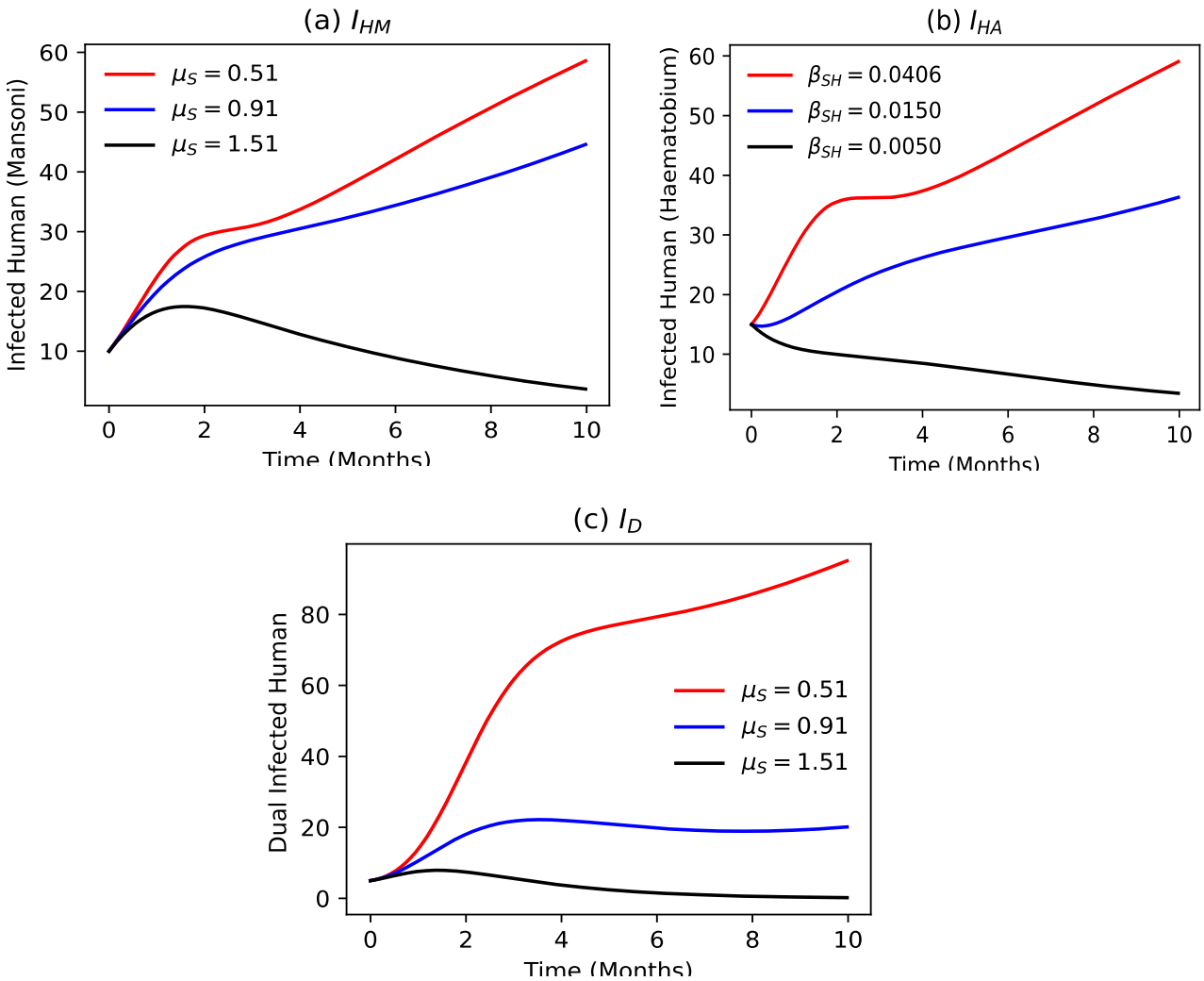
specifically target the parameters that have the greatest influence on reducing R_0. This targeted approach aids in containing and managing the disease. For instance, considering the substantial impact of μ_S on R_0, interventions aimed at increasing this parameter, such as the application of molluscicide spray, can effectively lower R_0 and potentially reduce the transmission rate.

5. Numerical Simulation

In order to explore different scenarios, numerical simulations were carried out using different initial conditions as specified in Table 1. The graphical representations of these simulations can be seen in Figure 1 and Figure 2.

Figure 1 and 2 illustrates the temporal dynamics of the single and dual infected human populations. In Figure 1, we observe that the population of single infected individuals increases over time when lower values of μ_S are considered (e.g., $\mu_S = 0.51$). This increase occurs at a faster rate in the initial two months, which can be attributed to the higher transmission rate from infectious snails to humans as more individuals become infected. However, the rate of increase gradually decreases, suggesting that some of the single infected individuals may have been reinfected and consequently transitioned to the dual infected class. In other words, individuals who were initially infected with only one type of infection may have contracted the second infection, resulting in their classification as dual infected individuals. On the other hand, as the values of μ_S increase over time (e.g., $\mu_S = 1.51$), the population of single infected individuals actually decreases below the initial values. This indicates that higher values of μ_S lead to a reduction in the number of single infected individuals over time. Similarly, the population of individuals in the dual infected class also exhibits an upward trend over time, characterized by an increasing rate (as seen in Figure 1c for $\mu_S = 0.51$). This phenomenon can be attributed to the combined effect of an increase in the number of single infectious individuals for both types of infections. As more individuals become infected with either Mansoni or Haematobium, the likelihood of contracting the other infection also increases, leading to an increase in the dual infected population.

However, as the values of μ_S increase, which signifies a higher rate of death of the infectious snails, the population of the dual infected class begins to decrease (as observed in Figure 1c). The decrease in the population of the dual infected class can be attributed to the reduced availability of snails capable of transmitting both Mansoni and Haematobium infections, as higher snail mortality rates limit the opportunities for individuals to acquire both infections simultaneously.



different values of μ_S for (a) infected human (Mansoni) (b) infected human (Haematobium), and (c) dual infected human.

Figure 2 also depicts the temporal dynamics of the single infected human population. Similar to Figure 1, we observe a trend where the population of single infected individuals decreases over time. However, in Figure 2, this trend occurs for lower values of β_{SH}). As the values of β_{SH} decrease (e.g., $\beta_{SH} = 0.0050$), representing a lower transmission rate from snails to humans, the population of single infected individuals also decreases over time. Thus, lower values of β_{SH} lead to a reduction in the number of single infected individuals. Moreover, in Figure 2c, as the values of β_{SH} decrease, the population of the dual infected class shows a corresponding decrease over time. This decline suggests that with a lower transmission rate, there are fewer opportunities for individuals to acquire both Mansoni and Haematobium infections concurrently, resulting in a reduction in the population of the dual infected class.

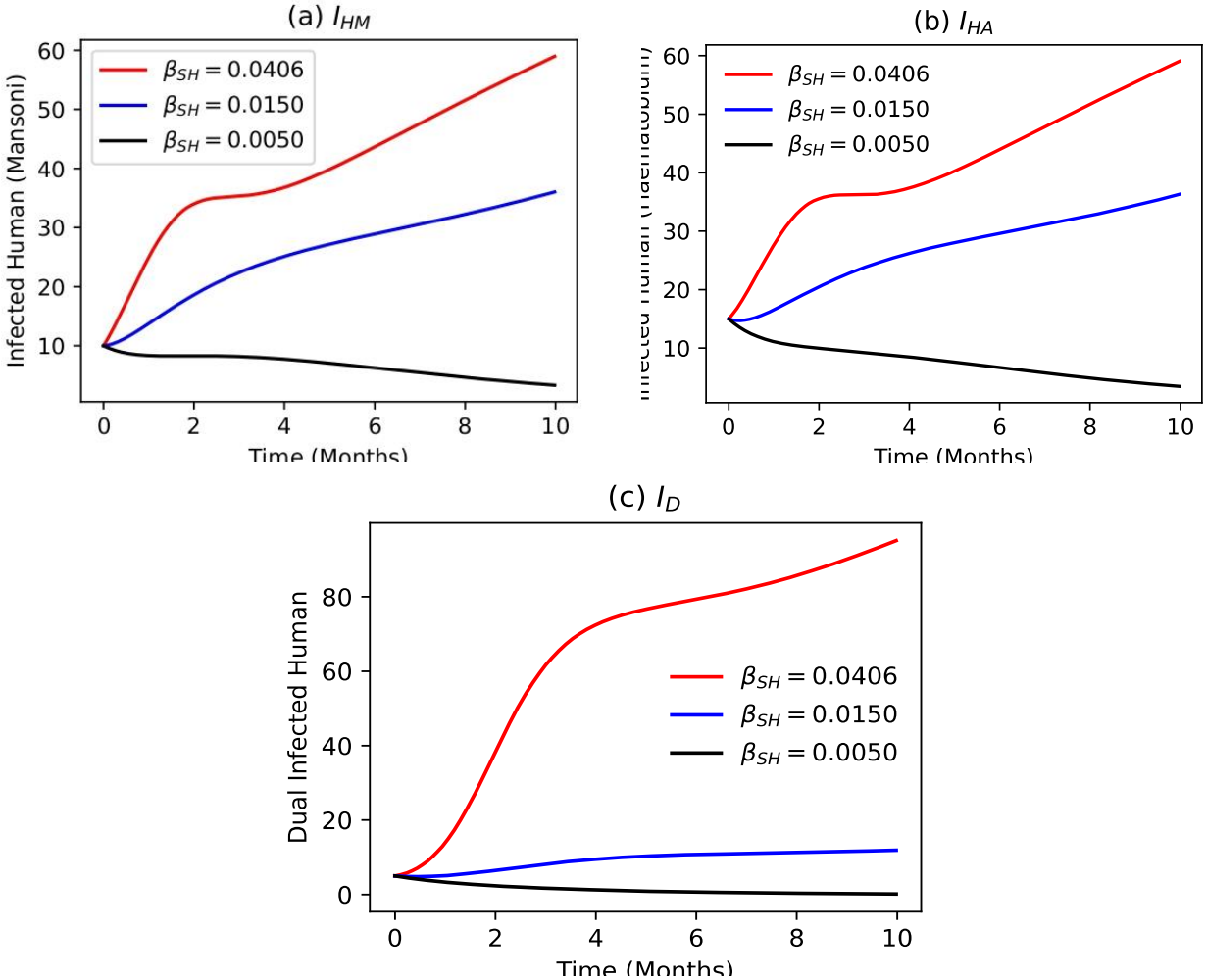


Figure 1: Figure 1:Temporal variations at different values of β_{SH} for (a) infected human (Mansoni) (b) infected human (Haematobium), and (c) dual infected human.Figure 2: Temporal variations at

It is important to note that both the single and dual infected populations decrease below their initial values due to the increasing nature of μ_S in Figure 1 and the decreasing value of β_{SH} in Figure 2. These findings emphasize the significant impact of parameter alterations on the prevalence and spread of infections among the human population.

Ultimately, understanding and acknowledging the rising occurrence of dual infections are crucial, as they directly influence the expanding population affected by single infections. These observations shed light on the intricate interplay between single and dual infections, underscoring the necessity to consider both facets when comprehending the progression and consequences of the disease.

6. Conclusion

In conclusion, the findings presented in this paper shed light on the complexities of schistosomiasis transmission and the challenges posed by dual infections. The coexistence of different species or strains of Schistosoma parasites in individuals complicates treatment regimens and increases the risk of treatment failure. By investigating the dynamics of both single and dual infections, as well as the interaction between different parasite species or strains, this study provides valuable insights into the prevalence and impact of these infections in relation to the transmission and severity of schistosomiasis.

The mathematical models used in this research serve as powerful tools for comprehending the underlying mechanisms of schistosomiasis transmission. The sensitivity analysis conducted on the basic reproduction number, R_0, highlights the influential

role played by various parameters in shaping the magnitude of disease transmission. The analysis reveals that the transmission rate from infectious snails to humans (β_{SH}) and the death rate of snails (μ_S), among other parameters, significantly contribute to the transmission and spread of schistosomiasis. Thus, to effectively eliminate schistosomiasis, efforts should focus on reducing the basic reproduction number, R_0, by decreasing β_{SH} or increasing μ_S. Control measures such as molluscicide spraying can help increase the mortality rate of snails, thereby reducing the transmission rate from infectious snails to humans.

Overall, the insights gained from this study emphasize the importance of comprehensive management strategies that address the multifaceted challenges posed by schistosomiasis. By considering the complexities of dual infections, understanding the influential parameters, and recognizing the ongoing cycle of transmission between infected snails and humans, effective control measures can be developed to mitigate the spread and impact of schistosomiasis.

The study is, however, limited by the scarcity of data on certain parameters, including Λ_S and Λ_H, which may potentially impact the accuracy of the model's predictions.

Future research endeavors to conduct thorough evaluations of control measures like molluscicide spraying and mass drug administration. The goal is to ascertain their long-term effectiveness in mitigating disease transmission and further enhance our understanding of the most efficient strategies for combating schistosomiasis.

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