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Research Article

Mathematical Analysis of Malaria-Schistosomiasis Coinfection Model

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We formulated and analysed a mathematical model to explore the cointeraction between malaria and schistosomiasis. Qualitative and comprehensive mathematical techniques have been applied to analyse the model. The local stability of the disease-free and endemic equilibrium was analysed, respectively. However, the main theorem shows that if $\mathcal{R}_{MS} < 1$, then the disease-free equilibrium is locally asymptotically stable and the phase will vanish out of the host and if $\mathcal{R}_{MS} > 1$, a unique endemic equilibrium is also locally asymptotically stable and the disease persists at the endemic steady state. The impact of schistosomiasis and its treatment on malaria dynamics is also investigated. Numerical simulations using a set of reasonable parameter values show that the two epidemics coexist whenever their reproduction numbers exceed unity. Further, results of the full malaria-schistosomiasis model also suggest that an increase in the number of individuals infected with schistosomiasis in the presence of treatment results in a decrease in malaria cases. Sensitivity analysis was further carried out to investigate the influence of the model parameters on the transmission and spread of malaria-schistosomiasis coinfection. Numerical simulations were carried out to confirm our theoretical findings.

1. Introduction

Malaria is highly endemic in various parts of sub-Saharan Africa in which 85% of global malaria cases and 90% of malaria deaths occur [1]. *Schistosoma mansoni* (the causative agent of intestinal schistosomiasis) is also prevalent in many sub-Saharan African countries [2, 3], accounting for approximately one-third of the total cases of schistosomiasis in the region [4]. The disease is a major contributor to disease burden globally and affects low income countries with climates suitable for transmission seriously. It is a life-threatening disease caused by parasites that are transmitted to people by the bites of infected mosquitoes [4, 5]. The bites by mosquitoes have resulted in the death of a child from malaria every 30 secs according to the report by the World Health Organization (WHO) fact sheet (2009) [4, 5]. *Plasmodium falciparum* and *Plasmodium vivax* are the two common species and *Plasmodium falciparum* is the most deadly. *Plasmodium falciparum* malaria remains a major cause of mortality and morbidity in the tropics and subtropics areas of the globe [4, 6]. According to the 2009 world report, half of the world's population is at risk of malaria, with an estimated 247 million cases that led to about 863,000 death in 2008 mostly among African children, a slight drop from 2006 statistics with the estimation that over 2000 young are lost every day across the globe [4]. This population made malaria the dominant parasitic disease of the tropics and one of the top three killer communicable diseases [4, 5, 7]. Malaria makes development to be very slow in several ways; it affects fertility, population growth, savings and investments, and worker productivity and causes absenteeism and premature mortality [4, 5, 7]. Malaria also affects fetal development during early stage of pregnancy in women due to loss of immunity. However, malaria is preventable and curable when treatment and prevention measures are sought early [4, 5, 7].

The disease, schistosomiasis, also known as bilharziasis or snail fever, is a parasitic disease that was first named *bilharzia* [8, 9] and it is prevalent in several regions of the

developing world, predominantly Africa, South America, and Asia, with about 650 million people living in the endemic areas [10]. It is known that estimated 207 million people are infected, where 85 percent lives in underdeveloped areas of Africa [11], resulting in about 15,000 deaths annually presently [2]. Children below the age of 14 are the major victims of schistosomiasis infection in many parts of the world [10]. The basis of illness in victims is the eggs laid by the parasitic flat worms, that is, blood flukes of the genus *Schistosoma* [8]. The species of the water-borne flatworm or blood flukes known as *schistosomes* is the main type that initiates the human schistosomiasis, but *Schistosoma mansoni*, *Schistosoma japonicum*, and *Schistosoma haematobium* are the three major species that are found everywhere [8]. The urinary tract and kidneys as well as the reproductive systems are affected by the *Schistosoma haematobium*, and they are intense in Africa and the Middle East [8]. The most widely spread species is the *Schistosoma mansoni* while *Schistosoma japonicum* is chiefly found in Asia and these two cause chronic hepatic and intestinal fibrosis [8, 10]. When skin comes in contact with contaminated freshwater in which certain types of snails that carry the parasite are living then the infection can be established [8]. Whenever infected people urinate or defecate in the water, freshwater becomes contaminated by *Schistosoma* eggs [8]. The eggs hatch, and the parasites infect, mature, and reproduce inside the snails when the appropriate species of snails exist in the water [12]. The parasite eventually leaves the snail and go into the water where it can persist for about 48 hours [8, 12]. *Schistosoma* parasites, when wading, swimming, bathing, or washing, can enter the skin of anyone who comes in contact with contaminated freshwater [8, 12]. The parasites migrate through host tissue and develop into adult worms inside the blood vessels of the body for over numerous weeks [8, 12]. The worms mate and females produce eggs after maturity [8, 12]. Several of these eggs eventually travel to the bladder or intestine and are finally passed into the urine or stool [8, 12]. The schistosomiasis symptoms are caused by the body's reaction to the eggs but not by the worms themselves [12, 13]. Eggs shed by the adult worms that do not pass out of the body can become lodged in the intestine or bladder, causing inflammation or scarring [12, 13]. Repeatedly infected children can acquire anemia, malnutrition, and learning difficulties [12, 13]. The parasite can as well damage the liver, intestine, spleen, lungs, and bladder even several years after infection [12, 13]. It is known at present that both malaria and intestinal schistosomiasis contribute to common epidemiological distributions and are currently posing a great task to public health and socioeconomic development throughout the tropical region [14]. The interactive pathology between malaria and *S. mansoni* has received increased investigation in the recent time, as a result of their coendemicities [1, 11, 15, 16]. It has been discovered that considerable *S. mansoni* infections are linked with a major increase in the incidence of malaria among school-age children [11]. In individuals infected with *S. mansoni* the technique responsible for the magnification of malaria is not yet fully understood [1, 9]. Thus, it is observed that the interface between the two diseases is perhaps set in motion by contradicting effects; the parasites possess the immunological cytokines; that is, the balance between *Th1* and *Th2* type immune responses which reduces immunological control of malaria may be altered by *S. mansoni*, while other methods are probable [1, 15, 17–19].

It is our view that this study represents the very first modeling work that presents a mathematical analysis of the qualitative dynamics of malaria-schistosomiasis coinfection. There are few studies done on the malaria-schistosomiasis coinfection model so far. In [20], a coepidemic model of malaria and *S. mansoni* transmission dynamics is established, where the model reports major epidemiological coupling between the two diseases in terms of aggravated malaria incidence among individuals with *S. mansoni* extreme egg output. Their model was factored for *S. mansoni* extreme-risk endemic areas, applying epidemiological and clinical data of the relationship between *S. mansoni* and malaria among children in sub-Saharan Africa. They also assessed the potential influence of the *S. mansoni* malaria interface and mass treatment of schistosomiasis on malaria prevalence in coendemic areas.

In this paper, we develop a mathematical model of the interplay between malaria and *S. mansoni* in which we have modeled the malaria transmission and the *S. mansoni* together as coendemic deterministic model. Our aim here is to study and analyse a mathematical model of malaria-schistosomiasis transmission model. Additionally, there are some important differences between the model in [16] and the one in this paper. This paper is organized as follows: we present a malaria-schistosomiasis coinfection transmission model formulation in Section 2, where the general mathematical framework, notations, and model equations were analysed with the basic properties of the models and their analysis. In Section 3, we present the existence of steady state solution. In Section 4, the basic reproduction number and stability were derived and carried out. Sensitivity analysis of the model was performed to determine the most important parameters that influence R_0 in Section 5. In Section 6, we show our numerical simulation results while, in Section 7, we discussed our conclusions and recommendations.

2. Model Formulation

In this model, we denote the total human population by N_h and subdivide it into the following subclasses of individuals who are susceptible (S_H), individuals with malaria symptoms only (i.e., who are already infected and infective with malaria parasite) (I_m), individuals infected with schistosomiasis only (I_{hs}), individuals infected with both malaria and schistosomiasis (V_{ms}), individuals who recovered from malaria only (R_m), individuals who recovered from schistosomiasis only (R_{hs}), and individuals who recovered from both malaria and schistosomiasis such that $N_h = S_H + I_m + I_{hs} + V_{ms} + R_m + R_{hs} + R_{ms}$. The total snail population is denoted by N_s, which comprises susceptible snails (S_s) and infected as well as infectious snails (I_s). That is, $N_s = S_s + I_s$. The total mosquito population is denoted by N_v, which comprises susceptible mosquitoes (S_v) and infected as well as infectious mosquitoes (I_v). That is, $N_v = S_v + I_v$.

The population of susceptible humans is generated through birth (at a constant per capita rate b_H), by the loss of immunity to the malaria disease only (at a constant per capita rate γ), loss of immunity to the schistosomiasis disease only at a rate k, and loss of immunity to malaria and schistosomiasis disease at a rate φ. It is reduced by natural death (at a rate d_H) and through the rate of acquiring malaria through contact with infectious mosquitoes (at a rate $\beta_1\epsilon_h\sigma$), where β_1 is the transmission probability per bite, ϵ_h is the per capita biting rate of mosquitoes, and σ is the contact rate of mosquito per human per unit time. It is also reduced by rate of acquiring schistosomiasis through contact with infected snails (at a rate β_2). Hence, the rate of change of population of susceptible humans is given by

$$\frac{dS_H}{dt} = b_H + \gamma R_m + kR_{hs} + \varphi R_{ms} - \beta_1\epsilon_h\sigma S_H I_v - \beta_2 S_H I_s - d_H S_H. \quad (1)$$

The rate of change of the population of individuals with malaria only is increased by the rate of acquiring malaria through contact with infectious mosquitoes (at a rate $\beta_1\epsilon_h\sigma$) and by the rate of acquiring schistosomiasis through contact with infectious snail (at a rate β_2) It is also reduced by human spontaneous recovery (at a rate ω). It is also reduced by the disease induced death rate (at per capita rate θ) and by the natural death rate (at per capita rate d_H). Hence it is given by

$$\frac{dI_m}{dt} = \beta_1\epsilon_h\sigma S_H I_v - \beta_2 I_s I_m - (\omega + \theta + d_H) I_m. \quad (2)$$

The rate of change of the population of individuals infected with schistosomiasis only is increased by the rate of acquiring schistosomiasis through contact with infectious snail (at a rate β_2) and decreased by infected mosquitoes (at a rate $\beta_1\epsilon_h\sigma$) and by human spontaneous recovery from schistosomiasis only (at a rate q_H). It is also reduced by the disease induced death rate (at per capita rate ρ_H) and by the natural death rate (at per capita rate d_H). Hence it is given by

$$\frac{dI_{hs}}{dt} = \beta_2 S_H I_s - \beta_1\epsilon_h\sigma I_v I_{hs} - (q_H + \rho_H + d_H) I_{hs}. \quad (3)$$

The rate of change of the population of individuals infected with schistosomiasis and malaria is increased by the rate of acquiring malaria by infected mosquitoes (at a rate $\beta_1\epsilon_h\sigma$) and schistosomiasis through contact with infectious snails (at a rate β_2) and reduced by human spontaneous recovery from schistosomiasis only (at a rate α). It is also reduced by the malaria disease induced death rate (at per capita rate m) and schistosomiasis induced death rate (at per capita rate r) and by the natural death rate (at per capita rate d_H). Hence it is given by

$$\frac{dV_{ms}}{dt} = \beta_1\epsilon_h\sigma I_v I_{hs} + \beta_2 I_s I_m - (\alpha + r + m + d_H) V_{ms}. \quad (4)$$

The individuals who recovered from malaria only is generated following a human spontaneous recovery (at a rate ω) and by the dually infected individuals who recovered from malaria only at a rate ($\phi(1-\alpha)$) decreased by loss of immunity (at a rate γ) and by natural death (at a rate d_H). Then

$$\frac{dR_m}{dt} = \omega I_m - (\gamma + d_H) R_m + \phi(1-\alpha) V_{ms}. \quad (5)$$

The individuals who recovered from schistosomiasis only are generated following a human spontaneous recovery (at a rate q_H) and by the dually infected individuals who recovered from schistosomiasis only at a rate ($(1-\phi)(1-\alpha)$) decreased by loss of immunity (at a rate γ) and by natural death (at a rate d_H). Then

$$\frac{dR_{hs}}{dt} = q_H I_{hs} - (k + d_H) R_{hs} + (1-\phi)(1-\alpha) V_{ms}. \quad (6)$$

The individuals who recovered from malaria and schistosomiasis are generated following a human spontaneous recovery (at a rate α) decreased by loss of immunity (at a rate φ) and by natural death (at a rate d_H). Then

$$\frac{dR_{ms}}{dt} = \alpha V_{ms} - (\varphi + d_H) R_{ms}. \quad (7)$$

Susceptible snail population is generated by the birth of snails (at a per capita rate of b_s). It is reduced by rate of acquiring schistosomiasis through contacts with infected humans at a rate β_3, where η_1 is a modification parameter. It is also reduced by natural death (at a rate d_s). Thus,

$$\frac{dS_s}{dt} = b_s - \beta_3 (I_{hs} + \eta_1 V_{ms}) S_s - d_s S_s. \quad (8)$$

The population of infected snail is increased by rate of acquiring schistosomiasis through contacts with infected humans at a rate β_3 and decreased by the natural death rate (at a rate d_s), where η_1 is a modification parameter. Hence, it is given by

$$\frac{dI_s}{dt} = \beta_3 (I_{hs} + \eta_1 V_{ms}) S_s - (d_s + \rho_s) I_s. \quad (9)$$

Susceptible mosquito population is generated by the birth of mosquitoes (at a per capita rate of b_v). It is reduced by rate of acquiring malaria through contacts with infected humans at a rate $\beta_4\epsilon_v\sigma$, where β_4 is probability for a vector (mosquito) to get infected by an infectious human, where η_2 is a modification parameter. It is also reduced by natural death (at a rate d_v). Thus,

$$\frac{dS_v}{dt} = b_v - \beta_4\epsilon_v\sigma (I_m + \eta_2 V_{ms}) S_v - d_v S_v. \quad (10)$$

The population of infected mosquito is increased by rate of acquiring malaria through contacts with infected humans at a rate $\beta_2\epsilon_v\sigma$ and decreased by the natural death rate (at a rate d_v), where η_2 is a modification parameter. Hence, it is given by

$$\frac{dI_v}{dt} = \beta_4\epsilon_v\sigma (I_m + \eta_2 V_{ms}) S_v - d_v I_v. \quad (11)$$

2.1. The Full Schistosomiasis-Malaria Coinfection Model. Bringing the above formulation and assumptions together leads to the following set of ordinary differential equations which may be a new malaria-schistosomiasis coinfection model:

$$\begin{aligned}
\frac{dS_H}{dt} &= b_H + \gamma R_m + kR_{hs} + \varphi R_{ms} - \beta_1 \epsilon_h \sigma S_H I_v \\
&\quad - \beta_2 S_H I_s - d_H S_H \\
\frac{dI_m}{dt} &= \beta_1 \epsilon_h \sigma S_H I_v - \beta_2 I_s I_m - (\omega + \theta + d_H) I_m \\
\frac{dI_{hs}}{dt} &= \beta_2 S_H I_s - \beta_1 \epsilon_h \sigma I_v I_{hs} - (q_H + \rho_H + d_H) I_{hs} \\
\frac{dV_{ms}}{dt} &= \beta_1 \epsilon_h \sigma I_v I_{hs} + \beta_2 I_s I_m - (\alpha + r + m + d_H) V_{ms} \\
\frac{dR_m}{dt} &= \theta I_h - (\gamma + d_H) R_m + \phi (1 - \alpha) V_{ms} \\
\frac{dR_{hs}}{dt} &= q_H I_{hs} - (g + d_H) R_{hs} + (1 - \phi)(1 - \alpha) V_{ms} \\
\frac{dR_{ms}}{dt} &= \alpha V_{ms} - (\varphi + d_H) R_{ms} \\
\frac{dS_s}{dt} &= b_s - \beta_3 (I_{hs} + \eta_1 V_{ms}) S_s - d_s S_s \\
\frac{dI_s}{dt} &= \beta_3 (I_{hs} + \eta_1 V_{ms}) S_s - (d_s + \rho_s) I_s \\
\frac{dS_v}{dt} &= b_v - \beta_4 \epsilon_v \sigma (I_m + \eta_2 V_{ms}) S_v - d_v S_v \\
\frac{dI_v}{dt} &= \beta_4 \epsilon_v \sigma (I_m + \eta_2 V_{ms}) S_v - d_v I_v
\end{aligned} \tag{12}$$

subject to the initial conditions $S_H(0) = S_{H,0}$, $I_m(0) = I_{m,0}$, $I_{hs}(0) = I_{hs,0}$, $V_{ms}(0) = V_{ms,0}$, $R_m(0) = R_{m,0}$, $R_{hs}(0) = R_{hs,0}$, $R_{ms}(0) = R_{ms,0}$, $S_s(0) = S_{s,0}$, $I_s(0) = I_{s,0}$, $S_v(0) = S_{v,0}$, and $I_v(0) = I_{v,0}$.

We describe the associated model variables and parameters in the following list and Table 1.

Variables in the Model

S_H: susceptible human

I_m: human infected with malaria only

I_{hs}: human infected with schistosomiasis only

V_{ms}: human infected with malaria and schistosomiasis only

R_m: human recovered from malaria only

R_{hs}: human recovered from schistosomiasis only

Table 1: Table showing numerical values of sensitivity Indices.

Parameter	Parameter values	Sensitivity to $\mathscr{R}_{MS}$
θ	0.05	−0.00000000000186
β_1	0.8333	+1.0
β_2	0.406	+1.0
β_3	0.004	+1.0
β_4	0.09	+0.99
b_v	1000	+0.74
d_H	0.00004	−2.0
ϵ_v	0.2	+1.0
σ	0.502	+2.0
ϵ_h	0.2	+1.0
q_H	0.56	−0.99
ρ_S	0.0004012	−0.876
ρ_H	0.0039	−0.0069
d_v	0.1429	−0.23
b_s	200	+1.0
b_H	100	+2.0
d_s	0.0000569	−0.11
ω	0.05	−0.91

R_{ms}: human recovered from malaria and schistosomiasis only

S_s: susceptible snail

I_s: infected snail

S_v: susceptible mosquito

I_v: infected mosquito

2.2. Qualitative Analysis of the Full Schistosomiasis-Malaria Coinfection Model. The schistosomiasis-malaria coinfection model (12) will be analysed in a biologically feasible region for both humans, snail, and mosquito populations. Hence, for it to be epidemiologically well posed, it is necessary to show that all its state variables are nonnegative for all time $t > 0$.

2.3. Positivity of the Solution

Theorem 1. *If the initial value for* $S_H \geq 0$, $I_m \geq 0$, $I_{hs} \geq 0$, $V_{ms} \geq 0$, $R_m \geq 0$, $R_{hs} \geq 0$, $R_{ms} \geq 0$, $S_s \geq 0$, $I_s \geq 0$, $S_v \geq 0$, *and* $I_v \geq 0$ *then the solutions* $(S_H(t), I_m(t), I_{hs}(t), R_{hs}(t), V_{ms}(t), R_m(t), R_{hs}(t), R_{ms}(t), S_s(t), I_s(t), S_v(t), I_v(t))$ *of the schistosomiasis only model (16) are nonnegative for all* $t > 0$.

Proof. We let $\Gamma = \sup\{t > 0 : S_H(t) > 0, I_m(t) > 0, I_{hs}(t) > 0, V_{ms}(t) > 0, R_m(t) > 0, R_{hs}(t) > 0, R_{ms}(t) > 0, S_s(t) > 0, I_s(t) > 0, S_v(t) > 0, I_v(t) > 0\}$.

Since the variables $S_H(0) > 0$, $I_m(0) > 0$, $I_{hs}(0) > 0$, $V_{ms}(0) > 0$, $R_m(0) > 0$, $R_{hs}(0) > 0$, $R_{ms}(0) > 0$, $S_s(0) > 0$, $I_s(0) > 0$, $S_v(0) > 0$, and $I_v(0) > 0$ then, $\Gamma > 0$. If $\Gamma < \infty$, then

$S_H, I_m, I_{hs}, V_{ms}, R_m, R_{hs}, R_{ms}, S_s, I_s, S_v, I_v$ are equal to zero at Γ. It follows from the first equation of the system (12) that

$$\frac{dS_H}{dt} = b_H + \gamma R_m + kR_{hs} + \varphi R_{ms} - \beta_1 \epsilon_h \sigma S_H I_v - \beta_2 S_H I_s - d_H S_H. \tag{13}$$

Therefore,

$$\frac{d}{dt}\left\{S_H(t)\exp\left[\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)t\right]\right\} = \left(b_H + kR_{hs} + \gamma R_m + \varphi R_{ms}\right) \cdot \exp\left[\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)t\right]. \tag{14}$$

Hence

$$S_H(\Gamma)\exp\left[\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)t\right] - S_H(0) = \int_0^\Gamma \left(b_H + kR_{hs} + \gamma R_m + \varphi R_{ms}\right) \cdot \exp\left[\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)z\right]dz, \tag{15}$$

$$S_H(\Gamma) = S_H(0)\exp\left[-\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)\Gamma\right] + \exp\left[-\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)\Gamma\right] \cdot \int_0^\Gamma \left(b_H + kR_{hs} + \gamma R_m + \varphi R_{ms}\right) \cdot \exp\left[\left(\beta_1\epsilon_h\sigma I_v + \beta_2 I_s + d_H\right)y\right]dy \geq 0. \tag{16}$$

Also

$$\frac{dI_m}{dt} = \beta_1\epsilon_h\sigma S_H I_v - \beta_2 I_s I_m - \left(\omega + \theta + d_H\right)I_m. \tag{17}$$

Now,

$$\frac{d}{dt}\left\{I_m(t)\exp\left[\left(\beta_2 I_s + \omega + \theta + d_H\right)t\right]\right\} = \int_0^\Gamma \left(\beta_1\epsilon_h\sigma S_H I_v\right)\exp\left[\left(\beta_2 I_s + \omega + \theta + d_H\right)y\right]dy,$$

$$I_m(\Gamma_1) = I_m(0)\exp\left[\left(-\left(\beta_2 I_s + \omega + \theta + d_H\right)\right)\Gamma\right] + \exp\left[\left(-\left(\beta_2 I_s + \omega + \theta + d_H\right)\right)\Gamma\right] \cdot \int_0^\Gamma \beta_1\epsilon_h\sigma S_H I_v \exp\left[\left(\beta_2 I_s + \omega + \theta + d_H\right)y\right]dy \geq 0. \tag{18}$$

And then

$$\frac{dI_{hs}}{dt} = \beta_2 S_H I_s - \beta_1\epsilon_h\sigma I_v I_{hs} - \left(q_H + \rho_H + d_H\right)I_{hs}. \tag{19}$$

Now,

$$\frac{d}{dt}\left\{I_{hs}(t)\exp\left[\left(q_H + \rho_H + d_H\right)t\right]\right\} = \int_0^\Gamma \left(\beta_2 S_H I_s\right) \cdot \exp\left[\left(\beta_1\epsilon_h\sigma I_v + q_H + \rho_H + d_H\right)y\right]dy,$$

$$I_{hs}(\Gamma) = I_{hs}(0)\exp\left[\left(-\left(\beta_1\epsilon_h\sigma I_v + q_H + \rho_H + d_H\right)\right)\Gamma\right] + \exp\left[\left(-\left(\beta_1\epsilon_h\sigma I_v + q_H + \rho_H + d_H\right)\right)\Gamma\right]\int_0^\Gamma \beta_2 S_H I_s \cdot \exp\left[\left(\beta_1\epsilon_h\sigma I_v + q_H + \rho_H + d_H\right)y\right]dy \geq 0. \tag{20}$$

Furthermore,

$$\frac{dV_{ms}}{dt} = \beta_1\epsilon_h\sigma I_v I_{hs} + \beta_2 I_s I_m - \left(\alpha + r + m + d_H\right)V_{ms}. \tag{21}$$

Now,

$$\frac{d}{dt}\left\{V_{ms}(t)\exp\left[\left(\alpha + r + m + d_H\right)t\right]\right\} = \int_0^\Gamma \left(\beta_1\epsilon_h\sigma I_v I_{hs} + \beta_2 I_s I_m\right) \cdot \exp\left[\left(\alpha + r + m + d_H\right)y\right]dy,$$

$$V_{ms}(\Gamma) = V_{ms}(0)\exp\left[\left(-\left(\alpha + r + m + d_H\right)\right)\Gamma\right] + \exp\left[\left(-\left(\alpha + r + m + d_H\right)\right)\Gamma\right] \cdot \int_0^\Gamma \left(\beta_1\epsilon_h\sigma I_v I_{hs} + \beta_5 I_{hs} I_m\right) \cdot \exp\left[\left(\alpha + r + m + d_H\right)y\right]dy \geq 0. \tag{22}$$

Also

$$\frac{dR_m}{dt} = \theta I_h - \left(\gamma + d_H\right)R_m + \phi\left(1 - \alpha\right)V_{ms}. \tag{23}$$

Therefore

$$\frac{d}{dt}\left\{R_m(t)\exp\left[\left(\gamma + d_H\right)t\right]\right\} = \int_0^\Gamma \left(\theta I_h + \phi\left(1 - \alpha\right)V_{ms}\right)\exp\left[\left(\gamma + d_H\right)y\right]dy. \tag{24}$$

Hence

$$R_m(\Gamma)\exp\left[\left(\gamma + d_H\right)t\right] - R_{hs}(0) = \int_0^{\Gamma_1} \left(\theta I_h + \phi\left(1 - \alpha\right)V_{ms}\right)\exp\left[\left(\gamma + d_H\right)y\right]dy \tag{25}$$

so that

$$\begin{aligned} R_m(\Gamma) &= R_{hs}(0)\exp\left[-(\gamma+d_H)\Gamma\right] \\ &\quad + \exp\left[-(\gamma+d_H)\Gamma\right] \\ &\quad \cdot \int_0^\Gamma (\theta I_h + \phi(1-\alpha)V_{ms})\exp\left[(\gamma+d_H)y\right]dy \\ &\quad \geq 0. \end{aligned} \tag{26}$$

Also,

$$\frac{dR_{hs}}{dt} = q_H I_{hs} - (k+d_H)R_{hs} + (1-\phi)(1-\alpha)V_{ms}. \tag{27}$$

Therefore

$$\begin{aligned} &\frac{d}{dt}\left\{R_{hs}(t)\exp\left[(k+d_H)t\right]\right\} \\ &\quad = \int_0^\Gamma \left(q_H I_{hs} + (1-\phi)(1-\alpha)V_{ms}\right) \\ &\quad \cdot \exp\left[(k+d_H)y\right]dy. \end{aligned} \tag{28}$$

Hence

$$\begin{aligned} &R_{hs}(\Gamma)\exp\left[(k+d_H)t\right] - R_{hs}(0) \\ &\quad = \int_0^\Gamma \left(q_H I_{hs} + (1-\phi)(1-\alpha)V_{ms}\right) \\ &\quad \cdot \exp\left[(k+d_H)y\right]dy \end{aligned} \tag{29}$$

so that

$$\begin{aligned} R_{hs}(\Gamma) &= R_{hs}(0)\exp\left[-(k+d_H)\Gamma\right] \\ &\quad + \exp\left[-(k+d_H)\Gamma_1\right] \\ &\quad \cdot \int_0^\Gamma \left(q_H I_{hs} + (1-\phi)(1-\alpha)V_{ms}\right) \\ &\quad \cdot \exp\left[(k+d_H)y\right]dy \geq 0. \end{aligned} \tag{30}$$

Also,

$$\frac{dR_{ms}}{dt} = \alpha V_{ms} - (\varphi+d_H)R_{ms}. \tag{31}$$

Then,

$$\begin{aligned} &\frac{d}{dt}\left\{R_{ms}(t)\exp\left[(\varphi+d_H)t\right]\right\} \\ &\quad = \int_0^\Gamma (\alpha V_{ms})\exp\left[(\varphi+d_H)y\right]dy. \end{aligned} \tag{32}$$

Hence

$$\begin{aligned} &R_{hs}(\Gamma)\exp\left[(\varphi+d_H)t\right] - R_{ms}(0) \\ &\quad = \int_0^\Gamma (\alpha V_{ms})\exp\left[(\varphi+d_H)y\right]dy \end{aligned} \tag{33}$$

so that

$$\begin{aligned} R_{ms}(\Gamma) &= R_{ms}(0)\exp\left[-(\varphi+d_H)\Gamma\right] \\ &\quad + \exp\left[-(\varphi+d_H)\Gamma\right] \\ &\quad \cdot \int_0^\Gamma (\alpha V_{ms})\exp\left[(\varphi+d_H)y\right]dy \geq 0. \end{aligned} \tag{34}$$

Also,

$$\frac{dS_s}{dt} = b_s - \beta_3(I_{hs} + \eta_1 V_{ms})S_s - d_s S_s. \tag{35}$$

Therefore,

$$\begin{aligned} &S_s(t)\exp\left[(\beta_3(I_{hs}+\eta_1 V_{ms}) + d_s)t\right] \\ &\quad = \int_0^\Gamma b_v \exp\left[(\beta_3(I_{hs}+\eta_1 V_{ms}) - d_s)y\right]dy. \end{aligned} \tag{36}$$

Then, we have

$$\begin{aligned} S_s(\Gamma) &= S_s(0)\exp\left[-(\beta_3(I_{hs}+\eta_1 V_{ms}) + d_s)\Gamma\right] \\ &\quad + \exp\left[-(\beta_3(I_{hs}+\eta_1 V_{ms}) + d_s)\Gamma\right] \\ &\quad \cdot \int_0^\Gamma b_s \exp\left[(\beta_3(I_{hs}+\eta_1 V_{ms}) + d_s)y\right]dy \geq 0. \end{aligned} \tag{37}$$

Similarly,

$$\frac{dI_s}{dt} = \beta_3(I_{hs} + \eta_1 V_{ms})S_s - (d_s+\rho_s)I_s. \tag{38}$$

Therefore,

$$\begin{aligned} &\frac{d}{dt}\left\{I_s(t)\exp\left[(d_s+\rho_s)t\right]\right\} \\ &\quad = (\beta_3(I_{hs}+\eta_1 V_{ms}))\exp\left[(d_s+\rho_s)y\right]dy \end{aligned} \tag{39}$$

so that

$$\begin{aligned} I_s(\Gamma) &= I_s(0)\exp\left[-(d_s+\rho_s)\Gamma\right] + \exp\left[-(d_s+\rho_s)\Gamma\right] \\ &\quad \cdot \int_0^\Gamma (\beta_3(I_{hs}+\eta_1 V_{ms}))\exp\left[(d_s+\rho_s)y\right]dy \geq 0 \end{aligned} \tag{40}$$

for all $t > 0$.

Also,

$$\frac{dS_v}{dt} = b_v - \beta_4 \epsilon_v \sigma(I_m + \eta_2 V_{ms})S_v - d_v S_v. \tag{41}$$

Therefore,

$$S_v(t)\exp\left[\left(\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)+d_v\right)t\right] = \int_0^{\Gamma} b_v\exp\left[\left(\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)+d_v\right)z\right]dz. \quad (42)$$

Then, we have

$$S_v(\Gamma) = S_v(0)\exp\left[-\left(\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)+d_v\right)\Gamma\right] + \exp\left[-\left(\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)+d_v\right)\Gamma\right] \cdot \int_0^{\Gamma} b_v\exp\left[\left(\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)+d_v\right)z\right]dz \geq 0. \quad (43)$$

Similarly,

$$\frac{dI_v}{dt} = \beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)S_v - d_vI_v. \quad (44)$$

Therefore,

$$\frac{d}{dt}\left\{I_v(t)\exp\left[\left(d_v\right)t\right]\right\} = \beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)\exp\left[(dv)\,z\right]dz \quad (45)$$

so that

$$I_v(\Gamma) = I_v(0)\exp\left[-\left(d_v\right)\Gamma\right] + \exp\left[-\left(d_v\right)\Gamma\right] \cdot \int_0^{\Gamma}\beta_4\epsilon_v\sigma\left(I_m+\eta_2V_{ms}\right)\exp\left[\left(d_v\right)z\right]dz \geq 0 \quad (46)$$

for all $t > 0$. □

2.4. Boundedness of the Solution

Theorem 2. *All solutions* $(S_H(t), I_m(t), I_{hs}(t), V_{ms}(t), R_m(t), R_{hs}(t), R_{ms}(t), S_s(t), I_s(t), S_v(t), I_v(t))$ *of the malaria-schistosomiasis coinfection model (12) are bounded. Therefore, from (12) if*

$$\limsup_{t\to\infty} N_h(t) \leq \frac{b_H}{d_H},$$
$$\limsup_{t\to\infty} N_s(t) \leq \frac{b_s}{d_s}, \quad (47)$$
$$\limsup_{t\to\infty} N_v(t) \leq \frac{b_v}{d_v}$$

then $N_s = S_s + I_s$, $N_v = S_v + I_v$, *and* $N_h = S_H + I_m + I_{hs} + V_{ms} + R_{hs} + R_m + R_{hs} + R_{ms}$.

Proof. For the proof of boundedness, we note that $0 < I_m(t) \leq N_h(t)$, $0 < I_{hs}(t) \leq N_h(t)$, $0 < V_{ms}(t) \leq N_h(t)$, $0 < I_s(t) \leq N_s(t)$, and $0 < I_v(t) \leq N_v(t)$. We add the first eight equations and the last two equations of the malaria-schistosomiasis coinfection model (12) and yield

$$\frac{dN_h}{dt} = b_h - d_HN_h - \omega I_m - \rho_HI_{hs} - (r+m)V_{ms}$$
$$\frac{dN_s}{dt} = b_s - d_sN_s - \rho_sI_s \quad (48)$$
$$\frac{dN_v}{dt} = b_v - d_vN_v.$$

All solutions of model (12) are bounded. The feasible region for the human population is given by $\Gamma_h = \{S_H, I_m, I_{hs}, V_{ms}, R_{hs}, R_m, R_{hs}, R_{ms} \mid S_H + I_m + I_{hs} + V_{ms} + R_{hs} + R_m + R_{hs} + R_{ms} \leq b_H/d_H, 0 \leq S_H \leq S_H(t) \leq b_H/d_H, I_{hs} \geq 0, R_{hs} \geq 0\}$. And the feasible region for the snail population is given by

$$\Gamma_s = \left\{S_s, I_s \mid S_s + I_s \leq \frac{b_s}{d_s}, 0 \leq S_s \leq S_s(t) \leq \frac{b_s}{d_s}, I_s \geq 0\right\}. \quad (49)$$

And the feasible region for the mosquito population is given by

$$\Gamma_v = \left\{S_v, I_v \mid S_v + I_v \leq \frac{b_v}{d_v}, 0 \leq S_v \leq S_v(t) \leq \frac{b_v}{d_v}, I_v \geq 0\right\}. \quad (50)$$

Therefore,

$$b_H - d_HN_h - \left(\omega + \rho_H + (r+m)\right)N_h(t) \leq \frac{dN_H(t)}{dt} \leq b_H - d_HN_h(t)$$
$$b_s - \left(d_s + \rho_s\right)N_s(t) \leq \frac{dN_s(t)}{dt} \leq b_s - d_sN_s(t) \quad (51)$$
$$b_v - \left(d_v + \rho_v\right)N_v(t) \leq \frac{dN_v(t)}{dt} \leq b_v - d_vN_v(t).$$

Hence,

$$\frac{b_H}{d_H+\rho_H} \leq \liminf_{t\to\infty} N_h(t) \leq \limsup_{t\to\infty} N_h(t) \leq \frac{b_H}{d_H},$$
$$\frac{b_s}{d_s+\rho_s} \leq \liminf_{t\to\infty} N_s(t) \leq \limsup_{t\to\infty} N_s(t) \leq \frac{b_s}{d_s} \quad (52)$$
$$\frac{b_v}{d_v} \leq \liminf_{t\to\infty} N_v(t) \leq \limsup_{t\to\infty} N_v(t) \leq \frac{b_v}{d_v}.$$

□

2.5. Invariant Region. Here, we analysed the malaria-schistosomiasis only model (12) in a biologically feasible

region. Hence the system of (12) is split into three parts, namely. We have the human population (N_h: with $N_h = S_H + I_m + I_{hs} + V_{ms} + R_{hs} + R_m + R_{hs} + R_{ms}$), the snail (vector) population (N_s: with $N_s = S_s + I_s$), and the mosquito (vector) population (N_v: with $N_v = S_v + I_v$). Let us consider the feasible region as

$$\Gamma = \Gamma_h \cup \Gamma_s \cup \Gamma_v \subset \mathbb{R}_+^7 \times \mathbb{R}_+^2 \times \mathbb{R}_+^2 \tag{53}$$

with

$$\begin{aligned}
\Gamma_h &= \left\{ \left(S_H, I_m, I_{hs}, V_{ms}, R_{hs}, R_m, R_{hs}, R_{ms}\right) \in \mathbb{R}_+^7 : S_H \right. \\
&\quad \left. + I_m + I_{hs} + V_{ms} + R_{hs} + R_m + R_{hs} + R_{ms} \le \frac{b_H}{d_H} \right\} \\
\Gamma_s &= \left\{ \left(S_s, I_s\right) \in \mathbb{R}_+^2 : S_s + I_s \le \frac{b_s}{d_s} \right\} \\
\Gamma_v &= \left\{ \left(S_v, I_v\right) \in \mathbb{R}_+^2 : S_v + I_v \le \frac{b_v}{d_v} \right\}.
\end{aligned} \tag{54}$$

We take the following steps to reaffirm the positive invariance of Γ (i.e., solution in Γ remains in Γ for all $t > 0$). The rate of change of humans, snails, and mosquitoes population is given in (12) and it follows that

$$\begin{aligned}
\frac{dN_h(t)}{dt} &\le b_H - d_H N_h \\
\frac{dN_s(t)}{dt} &\le b_s - d_s N_s \\
\frac{dN_v(\mathrm{t})}{dt} &\le b_v - d_v N_v.
\end{aligned} \tag{55}$$

By the standard comparison theorem we obtain

$$\begin{aligned}
N_h(t) &\le N_h(0)\, e^{-d_H t} + \frac{b_H}{d_H}\left(1 - e^{-d_H t}\right) \\
N_s(t) &\le N_s(0)\, e^{-d_s t} + \frac{b_s}{(d_s)}\left(1 - e^{-d_s t}\right) \\
N_v(t) &\le N_v(0)\, e^{-d_v t} + \frac{b_v}{(d_v)}\left(1 - e^{-d_v t}\right).
\end{aligned} \tag{56}$$

In particular, $N_h(t) \le b_H/d_H$ if $N_h(0) \le b_H/d_H$, $N_s(t) \le b_s/d_s$ if $N_s(0) \le b_s/d_s$, and $N_v(t) \le b_v/d_v$ if $N_v(0) \le b_v/d_v$. Therefore, the region Γ is positively invariant. Hence, it is enough to consider the dynamics of the flow generated by (12) in Γ. Thus, the model can be considered to be epidemiologically and mathematically well posed in this region. Therefore every solution of the malaria-schistosomiasis coinfection model (12) with initial data (conditions) in Γ remains in Γ. The summary of this result is given below.

Theorem 3. *The region $\Gamma = \Gamma_h \cup \Gamma_s \cup \Gamma_v \subset \mathbb{R}_+^7 \times \mathbb{R}_+^2 \times \mathbb{R}_+^2$ is positively invariant for the malaria-schistosomiasis coinfection model (12) with nonnegative initial conditions in $\mathbb{R}_+^{11}$.*

3. Existence of Steady States Solution

We analysed the model equation (12) in this section qualitatively to investigate the condition of existence of equilibrium points. We would like to know what will eventually happen to the disease in the long run. The question that will arise is (i) will the disease (malaria-schistosomiasis disease) die out? Or (ii) will it be established in the population and become endemic? In order to answer these questions we have to investigate the long-term behaviour of the solutions. This behaviour depends largely on the equilibrium points, that is, time-independent solutions of the system. Since these solutions do not depend on time, we set

$$\begin{aligned}
&\frac{dS_H}{dt} = \frac{dI_m}{dt} = \frac{dI_{hs}}{dt} = \frac{dV_{ms}}{dt} = \frac{dR_m}{dt} = \frac{dR_{hs}}{dt} \\
&\quad = \frac{dR_{ms}}{dt} = 0 \\
&\frac{dS_s}{dt} = \frac{dI_s}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0.
\end{aligned} \tag{57}$$

Therefore, we have the equilibrium solution

$$\begin{aligned}
D_{MS} &= \left(S_H = \frac{b_H}{d_H}, I_m = 0, I_{hs} = 0, V_{ms} = 0, R_{hs} \right. \\
&\quad = 0, R_m = 0, R_{hs} = 0, R_{ms} = 0, S_s = \frac{b_s}{d_s}, I_s = 0, S_v \\
&\quad \left. = \frac{b_v}{d_v}, I_v = 0 \right).
\end{aligned} \tag{58}$$

This equilibrium exists for all values of the parameters. Since $I_{hs} = R_{hs} = I_s = 0$ it implies that the disease will disappear from the population. Thus, this equilibrium is referred to as the disease-free equilibrium (DFE).

3.1. Existence of Disease-Free Equilibrium Point (DFE). We analysed the system qualitatively by studying the system of equation in closed set

$$\begin{aligned}
\Omega &= \left\{ \left(S_H, I_m, I_{hs}, V_{ms}, R_{hs}, R_m, R_{hs}, R_{ms}, S_s, I_s, S_v, I_v\right) \right. \\
&\quad \in \mathbb{R}_+^{11} \mid 0 \le S_H \le N_h, 0 \le I_{hs} \le N_h, 0 \le R_{hs} \le N_h, 0 \\
&\quad \left. \le S_s \le N_s, 0 \le I_s \le N_s, 0 \le S_v \le N_v, 0 \le I_v \le N_v \right\}.
\end{aligned} \tag{59}$$

A qualitative study of the system exists in two forms, namely,

(i) the disease-free (dies out),

(ii) endemic.

When the malaria-schistosomiasis disease dies out naturally, the solution asymptotically tends to a disease-free equilibrium D_{MS} of the form $D_{MS} = (S_H = b_H/d_H, I_m = 0, I_{hs} = 0, V_{ms} = 0, R_{hs} = 0, R_m = 0, R_{hs} = 0, R_{ms} = 0, S_s = b_s/d_s, I_s = 0, S_v = b_v/d_v, I_v = 0)$. Hence, the threshold that determines the stability of this equilibrium is the reproduction number (which is the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime).

4. The Basic Reproduction Number $\mathscr{R}_{MS}$

Whenever $\mathscr{R}_{MS} < 1$, each individual releases on average less than one infected individual and therefore the disease (malaria-schistosomiasis only) dies out. Whenever $\mathscr{R}_{MS} < 1$, each individual release more than one new infected individual and therefore the disease is able to invade the susceptible population. This allows us to determine the effectiveness of control measures. Epidemiologically, the reproductive number tells us how many secondary cases will one infected individual or vector produce in an entirely susceptible population of hosts and vectors, in other words, to determine the number of infected people that are generated by the introduction of a single infected person into a susceptible population. We need the computation of the basic reproduction number $\mathscr{R}_{MS}$ to assess the stability of the disease-free equilibrium (DFE) and the endemic equilibrium point (EEP). The basic reproduction number for particular infections is dependent on the biological characteristics of the disease and on the behavioural pattern of the population. When there is a high transmission of the disease per unit time and durations of the infectious period, the basic reproduction number increases.

Proposition 4. *The value of the basic reproduction number is* $\mathscr{R}_{MS} = \sqrt{\beta_2\beta_3 b_H b_s / d_H d_s (d_H + \rho_H + q_H)(d_s + \rho_s)}$.

Proof. The argument uses the approach of the next generation matrix (see [21]). Suppose

$$F = \begin{pmatrix} \frac{\partial f_1(D_{MS})}{\partial I_m} & \frac{\partial f_1(D_{MS})}{\partial I_{hs}} & \frac{\partial f_1(D_{MS})}{\partial V_{ms}} & \frac{\partial f_1(D_{MS})}{\partial I_s} & \frac{\partial f_1(D_{MS})}{\partial I_v} \\ \frac{\partial f_2(D_{MS})}{\partial I_m} & \frac{\partial f_2(D_{MS})}{\partial I_{hs}} & \frac{\partial f_2(D_{MS})}{\partial V_{ms}} & \frac{\partial f_2(D_{MS})}{\partial I_s} & \frac{\partial f_2(D_{MS})}{\partial I_v} \\ \frac{\partial f_3(D_{MS})}{\partial I_m} & \frac{\partial f_3(D_{MS})}{\partial I_{hs}} & \frac{\partial f_3(D_{MS})}{\partial V_{ms}} & \frac{\partial f_3(D_{MS})}{\partial I_s} & \frac{\partial f_3(D_{MS})}{\partial I_v} \\ \frac{\partial f_4(D_{MS})}{\partial I_m} & \frac{\partial f_4(D_{MS})}{\partial I_{hs}} & \frac{\partial f_4(D_{MS})}{\partial V_{ms}} & \frac{\partial f_4(D_{MS})}{\partial I_s} & \frac{\partial f_4(D_{MS})}{\partial I_v} \\ \frac{\partial f_5(D_{MS})}{\partial I_m} & \frac{\partial f_5(D_{MS})}{\partial I_{hs}} & \frac{\partial f_5(D_{MS})}{\partial V_{ms}} & \frac{\partial f_5(D_{MS})}{\partial I_s} & \frac{\partial f_5(D_{MS})}{\partial I_v} \end{pmatrix} \tag{60}$$

which results into

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\beta_1 \epsilon_H \sigma b_H}{d_H} \\ 0 & 0 & 0 & \frac{\beta_2 b_H}{d_H} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_3 b_s}{d_s} & \frac{\beta_3 \eta_1 b_s}{d_s} & 0 & 0 \\ \frac{\beta_4 \epsilon_v \sigma b_v}{d_v} & 0 & \frac{\beta_4 \epsilon_v \sigma b_v}{d_v} & 0 & 0 \end{pmatrix}, \tag{61}$$

where F is the rate of appearance of new infections in one compartment and V is the transfer of individuals and mosquitoes into one compartment which is a Jacobian matrix evaluated at D_{0s} and the Jacobian matrix of V is evaluated at D_{MS} to give

$$V = \begin{pmatrix} (\omega+\theta+d_H) & 0 & 0 & 0 & 0 \\ 0 & (\rho_H+q_H+d_H) & 0 & 0 & 0 \\ 0 & 0 & (\alpha+r+m+d_H) & 0 & 0 \\ 0 & 0 & 0 & d_s+\rho_s & 0 \\ 0 & 0 & 0 & 0 & d_v \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\omega+\theta+d_H)} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{(\rho_H+q_H+d_H)} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\alpha+r+m+d_H)} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{(d_s+\rho_s)} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{(d_v)} \end{pmatrix}. \tag{62}$$

The product of matrix F and V^{-1} gives

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\beta_1\epsilon_h\sigma b_H}{d_H d_v} \\ 0 & 0 & 0 & \frac{\beta_2 b_H}{d_H(d_s+\rho_s)} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_3 b_s}{d_s(\rho_H+q_H+d_H)} & \frac{\beta_3 b_s\eta_1}{(\alpha+r+m+d_H)} & 0 & 0 \\ \frac{\beta_4\epsilon_v\sigma b_v}{d_v(\omega+\theta+d_H)} & 0 & \frac{\beta_4\epsilon_v\sigma b_v\eta_2}{d_v(\alpha+r+m+d_H)} & 0 & 0 \end{pmatrix}. \tag{63}$$

Finding the eigenvalue of FV^{-1}, we obtain $\mathcal{R}_{MS}$ for the malaria-schistosomiasis coinfection model (12) is given by

$$R_{M1}^2 = \frac{b_H^2\beta_4\epsilon_v\epsilon_h\beta_1\sigma^2 b_v}{d_H(\omega+d_H+\theta)d_v^2}$$

$$R_{S1}^2 = \frac{\beta_3 b_s\beta_2 b_H}{d_s d_H(d_s+\rho_s)(d_H+\rho_H+q_H)}$$

$$\mathcal{R}_{MS} = \sqrt{\frac{\beta_3 b_s\beta_2 b_H^2\beta_4\epsilon_v\epsilon_h\beta_1\sigma^2 b_v}{d_s d_H^2(d_s+\rho_s)(d_H+\rho_H+q_H)(\omega+d_H+\theta)d_v^2}} \tag{64}$$

$$\mathcal{R}_{MS}^2 = \frac{\beta_3 b_s\beta_2 b_H^2\beta_4\epsilon_v\epsilon_h\beta_1\sigma^2 b_v}{d_s d_H^2(d_s+\rho_s)(d_H+\rho_H+q_H)(\omega+d_H+\theta)d_v^2}.$$

Considering the parameters for our model from Table 1, the value of $\mathcal{R}_{MS}$ appears to be far greater than 1. This implies that the disease (malaria-schistosomiasis coinfection model) will not die out but remains endemic in the population until some control strategies are applied properly. The disease-free equilibrium is locally stable if $\mathcal{R}_{MS} < 1$. We can achieve the global stability for the disease-free equilibrium by following the Lyapunov argument for $\mathcal{R}_{MS} < 1$. Hence, when the disease-free equilibrium is unstable, there exists an endemic equilibrium. Next we discuss the local stability of the disease-free equilibrium. □

4.1. Local Stability of the Disease-Free Equilibrium of Malaria-Schistosomiasis Coinfection Model. The disease-free equilibrium for the malaria-schistosomiasis coinfection model given by $D_{MS} = (S_H^*, I_m^*, I_{hs}^*, V_{ms}^*, R_m^*, R_{hs}^*, R_{ms}^*, S_s^*, I_s^*, S_v^*, I_v^*) = (b_H/d_H, 0, 0, 0, 0, 0, 0, 0, b_s/d_s, 0, b_v/d_v, 0)$ exists for all values of $d_H > 0$, $d_s > 0$, and $d_v > 0$. If the threshold value is $\mathcal{R}_{MS}$, then the disease-free equilibrium is locally asymptotically stable and the disease cannot invade or spread

in the population or community. The summary of the result is given by Theorem 5.

Theorem 5. *The disease-free equilibrium point D_{MS} is locally asymptotically stable if $\mathcal{R}_{MS} < 1$ and unstable if $\mathcal{R}_{MS} > 1$.*

It means that the malaria-schistosomiasis coinfection can be eliminated from the population whenever $\mathcal{R}_{MS} < 1$. $\mathcal{R}_{MS} < 1$; then, averagely an infected person produces less than one newly infected person over the entire period of his infectiousness and malaria-schistosomiasis coinfection dies out. For $\mathcal{R}_{MS}$ to be less than one, the β_1, β_2, β_3, and β_4 (rates of transmission) must decrease without bounds. Otherwise, if $\mathcal{R}_{MS} > 1$, then each infected person produces less, averagely more than one new infection, and the malaria-schistosomiasis coinfection can spread or invade the population. Consider the model equation (12). At the equilibrium state D_{MS}, the Jacobian is given by

$$\left|J_{D_{MS}}\right| = \begin{bmatrix} -d_H & 0 & 0 & 0 & \gamma & k & \varphi & 0 & -m_1 & 0 & -m_2 \\ 0 & -m_7 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & m_2 \\ 0 & 0 & -m_8 & 0 & 0 & 0 & 0 & 0 & m_1 & 0 & 0 \\ 0 & 0 & 0 & -m_9 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \omega & 0 & \phi(1-\alpha) & -(\gamma + d_H) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & q_H & 0 & 0 & -m_{10} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha & 0 & 0 & -(\varphi + d_H) & 0 & 0 & 0 & 0 \\ 0 & 0 & -m_3 & -m_4 & 0 & 0 & 0 & -d_s & 0 & 0 & 0 \\ 0 & 0 & m_3 & m_4 & 0 & 0 & 0 & 0 & -(d_s + \rho_s) & 0 & 0 \\ 0 & -m_5 & 0 & -m_6 & 0 & 0 & 0 & 0 & 0 & -d_v & 0 \\ 0 & m_5 & 0 & m_6 & 0 & 0 & 0 & 0 & 0 & 0 & -d_v \end{bmatrix}, \tag{65}$$

where $m_1 = \beta_2 b_H/d_H$, $m_2 = \beta_1 \epsilon_h \sigma b_H/d_H$, $m_3 = \beta_3 b_s/d_s$, $m_4 = \beta_3 \eta_1 b_s/d_s$, $m_5 = \beta_4 \epsilon_v \sigma b_v/d_v$, $m_6 = \beta_4 \epsilon_v \sigma \eta_2 b_v/d_v$, $m_7 = (\omega + d_H + \theta)$, $m_8 = (d_H + \rho_H + q_H)$, $m_9 = (\alpha + r + m + d_H)$, and $m_{10} = (g + d_H) + (1 - \phi)(1 - \alpha)$.

The local stability of D_{MS} is determined by the signs of the eigenvalues of the Jacobian matrix (65). The disease-free equilibrium point, D_{MS}, is said to be locally asymptotically stable if the real parts of the eigenvalues of the Jacobian matrix (65) are all negative; otherwise it is said to be unstable. By considering the Jacobian matrix (65), we obtain

$$\left|J_{D_{MS}}\right| = \begin{vmatrix} -d_H & 0 & 0 & 0 & \gamma & k & \varphi & 0 & -m_1 & 0 & -m_2 \\ 0 & -m_7 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & m_2 \\ 0 & 0 & -m_8 & 0 & 0 & 0 & 0 & 0 & m_1 & 0 & 0 \\ 0 & 0 & 0 & -m_9 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \omega & 0 & \phi(1-\alpha) & -(\gamma + d_H) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & q_H & 0 & 0 & -m_{10} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha & 0 & 0 & -(\varphi + d_H) & 0 & 0 & 0 & 0 \\ 0 & 0 & -m_3 & -m_4 & 0 & 0 & 0 & -d_s & 0 & 0 & 0 \\ 0 & 0 & m_3 & m_4 & 0 & 0 & 0 & 0 & -(d_s + \rho_s) & 0 & 0 \\ 0 & -m_5 & 0 & -m_6 & 0 & 0 & 0 & 0 & 0 & -d_v & 0 \\ 0 & m_5 & 0 & m_6 & 0 & 0 & 0 & 0 & 0 & 0 & -d_v \end{vmatrix} = 0, \tag{66}$$

where $m_1 = \beta_2 b_H/d_H$, $m_2 = \beta_1 \epsilon_h \sigma b_H/d_H$, $m_3 = \beta_3 b_s/d_s$, $m_4 = \beta_3 \eta_1 b_s/d_s$, $m_5 = \beta_4 \epsilon_v \sigma b_v/d_v$, $m_6 = \beta_4 \epsilon_v \sigma \eta_2 b_v/d_v$, $m_7 = (\omega + d_H + \theta)$, $m_8 = (d_H + \rho_H + q_H)$, $m_9 = (\alpha + r + m + d_H)$, and $m_{10} = (g + d_H) + (1 - \phi)(1 - \alpha)$, such that

$$\lambda_1 = -d_H < 0$$

$$\lambda_2 = -(\alpha + d_H + r + m) < 0$$

$$\lambda_3 = -(\gamma + d_H) < 0$$

$$\lambda_4 = -(g - d_H - 1 + \alpha + \phi - \phi\alpha) < 0$$

$$\lambda_5 = -(\varphi + d_H) < 0$$

$$\lambda_6 = -d_s < 0$$

$$\lambda_7 = -d_v < 0. \tag{67}$$

The remaining characteristic polynomial corresponding to $J_{D_{MS}}$ is

$$F_1(\lambda) = \lambda^2 + (d_v + \omega + d_H + \theta)\lambda - \left(d_v(\omega + d_H + \theta) - \frac{\beta_v \epsilon_v \sigma^2 b_v \beta_1 \epsilon_h b_H}{d_v d_H} + d_v(\omega + d_H + \theta)\right) = 0, \tag{68}$$

where

$$A_1 = 1$$

$$A_2 = (d_v + \omega + d_H + \theta)$$

$$A_3 = d_v(\omega + d_H + \theta) - \frac{\beta_v \epsilon_v \sigma^2 b_v \beta_1 \epsilon_h b_H}{d_v d_H} + d_v(\omega + d_H + \theta). \tag{69}$$

The Routh-Hurwitz conditions to establish that all roots of (F_1) have negative real parts are $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, and $A_1 A_2 > A_3$. Note that all model parameters are positive. For A_3 to be positive set

$$d_v(\omega + d_H + \theta) - \frac{\beta_v \epsilon_v \sigma^2 b_v \beta_1 \epsilon_h b_H}{d_v d_H} + d_v(\omega + d_H + \theta) > 0 \tag{70}$$

which implies that

$$d_v(\omega + d_H + \theta) \cdot \left(1 - \frac{\beta_v \epsilon_v \sigma^2 b_v \beta_1 \epsilon_h b_H}{d_v^2 d_H} + d_v(\omega + d_H + \theta)\right) > 0. \tag{71}$$

This leads to $(1 - R_{M1}^2) > 0 \Rightarrow R_{M1} < 1$.

$$d_v(\omega + d_H + \theta) \cdot \left(1 - \frac{\beta_v \epsilon_v \sigma^2 b_v \beta_1 \epsilon_h b_H}{d_v^2 d_H} + d_v(\omega + d_H + \theta)\right) > 0 \tag{72}$$

which is true. Thus, by the Routh-Hurwitz condition, all the eigenvalues have negative real parts whenever $R_{M1} < 1$, such that D_{MS} is locally asymptotically stable. $F_2(\lambda) = \lambda^2 + (d_s + \rho_s + d_H + \rho_H + q_H)\lambda - \beta_3 b_s \beta_2 b_H / d_s d_H + (d_s + \rho_s)(d_H + \rho_H + q_H)$, where

$$E_1 = 1$$

$$E_2 = (d_s + \rho_s + d_H + \rho_H + q_H)$$

$$E_3 = (d_s + \rho_s)(d_H + \rho_H + q_H) - \frac{\beta_3 b_s \beta_2 b_H}{d_s d_H}. \tag{73}$$

The Routh-Hurwitz conditions to make certain that all roots of (F_2) have negative real parts are $E_1 > 0$, $E_2 > 0$, $E_3 > 0$, and $E_1 E_2 > E_3$. Note that all model parameters are positive. For E_3 to be positive set

$$(d_s + \rho_s)(d_H + \rho_H + q_H) - \frac{\beta_3 b_s \beta_2 b_H}{d_s d_H} > 0 \tag{74}$$

which implies that

$$1 - \frac{\beta_3 b_s \beta_2 b_H}{d_s d_H (d_s + \rho_s)(d_H + \rho_H + q_H)} > 0. \tag{75}$$

This leads to $(1 - R_{S1}^2) > 0 \Rightarrow R_{S1} < 1$. It can be verified that $E_1 E_2 - E_3 > 0$; that is,

$$(d_s + \rho_s + d_H + \rho_H + q_H) - (d_s + \rho_s)(d_H + \rho_H + q_H)\left(1 - R_{S1}^2\right) > 0; \tag{76}$$

hence it is true. Accordingly, by the Routh-Hurwitz criteria, all the eigenvalues have negative real parts whenever $R_{S1} < 1$, such that D_{MS} is locally asymptotically stable.

4.2. Impact of Schistosomiasis on Malaria and Vice Versa. The impact of schistosomiasis on malaria and vice versa can be analysed by expressing R_{0m} in terms of R_{0s}. We solve d_H to obtain the following.

Suppose

$$P_1 = d_s(q_H + \rho_H)(d_s + \rho_s)$$

$$P_2 = \beta_3 b_s \beta_2 b_H d_s (d_s + \rho_s) \tag{77}$$

$\therefore$

$$d_H = \frac{-P_1 R_{0s} + \sqrt{P_1^2 R_{0s}^2 + 4P_2}}{2d_s(d_s + \rho_s) R_{0s}}. \tag{78}$$

Let

$$\sqrt{P_1^2 R_{0s}^2 + 4P_2} = P_3 R_{0s} + P_4. \tag{79}$$

Therefore

$$d_H = \frac{-P_1 2R_{0s} + P_3 R_{0s} + P_4}{2d_s(d_s + \rho_s) R_{0s}}. \tag{80}$$

Hence

$$d_H = \frac{(P_3 - P_1) R_{0s} + P_4}{2d_s(d_s + \rho_s) R_{0s}}. \tag{81}$$

We substitute d_H in R_{0m} to obtain

$$R_{0m}^2 = \frac{\beta_4 \epsilon_v b_v \beta_1 \epsilon_h \sigma^2 d_s^2 (d_s + \rho_s)^2}{(R_{0s}P_3 - R_{0s}P_1 + P_4)(2\omega d_s^2 + 2\omega d_s \rho_s + P_3 - P_1 + P_4 + 2\theta d_s^2 + 2\theta d_s \rho_s) R_{0s} d_v^2} \tag{82}$$

$$\frac{\partial R_{0m}^2}{\partial R_s} = \frac{-\beta_4 \epsilon_v b_v \beta_1 \epsilon_h \sigma^2 d_s^2 (d_s + \rho_s)^2 \left(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s\right)(P_3 - P_1 + 1)}{(P_3 R_{0s} - P_1 R_{0s} + P_4)(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s)^2} \tag{83}$$

$$\frac{\partial R_{0m}^2}{\partial R_s} = \frac{-\beta_4 \epsilon_v b_v \beta_1 \epsilon_h \sigma^2 d_s^2 (d_s + \rho_s)^2 \left(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s\right)(P_1 - (P_3 + 1))}{(P_3 R_{0s} - P_1 R_{0s} + P_4)(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s)^2} \tag{84}$$

$$\frac{\partial R_{0m}^2}{\partial R_s} = \frac{-\beta_4 \epsilon_v b_v \beta_1 \epsilon_h \sigma^2 d_s^2 (d_s + \rho_s)^2 (P_3 - P_1)\left(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s\right) - (mP_3 - mP_1 + P_4)}{d_v^2 (P_3 R_{0s} - P_1 R_{0s} + P_4)(2\omega d_s^2 R_{0s} + 2\omega d_s R_{0s}\rho_s + R_{0s}P_3 - R_{0s}P_1 + P_4 + 2\theta d_s^2 R_{0s} + 2\theta d_s R_{0s}\rho_s)^2}. \tag{85}$$

If $(\omega + \theta)d_s^2 + \rho_s(\omega + \theta)d_s + (1/2)P_3 - (1/2)P_1(P_1 - P_3) \geq (1/1)((\omega + \theta)d_s^2 + \rho_s(\omega + \theta)d_s - P_1 + P_3)P_4$ is strictly positive, then the malaria enhances schistosomiasis infection. This means that whenever (85) is greater than zero, an increase in schistosomiasis will result in an increase of malaria cases in the community. If (85) is equal to zero, it implies that schistosomiasis cases will have no significant impact on the transmission dynamics of malaria. Accordingly, by expressing d_H in terms of R_{0M} (impact of malaria on schistosomiasis)

$$d_H = \frac{-\left((1/2)\, d_v\omega + (1/2)\, d_v\theta\right) R_{0M} + (1/2)\sqrt{d_v^2 R_{0M}^2 \omega^2 + 2d_v^2 R_v^2 \omega\theta + d_v^2 R_{0M}^2 \theta^2 + 4\beta_4 b_v \beta_1 \epsilon_h b_H \sigma^2}}{2d_v R_{0M}}, \tag{86}$$

where

$$\begin{aligned} Q_1 &= d_v\omega + d_v\theta \\ Q_2 &= \beta_4 b_v \beta_1 \epsilon_h b_H \sigma^2 \\ d_H &= \frac{-Q_1 R_{0M} + \sqrt{Q_1^2 R_{0M}^2 + 4Q_2}}{4d_v R_{0M}}. \end{aligned} \tag{87}$$

By assigning

$$\sqrt{Q_1^2 R_{0M}^2 + 4Q_2} = Q_3 R_{0M} + Q_4 \tag{88}$$

therefore

$$\begin{aligned} d_H &= \frac{-Q_1 R_{0M} + Q_3 R_{0M} + Q_4}{4d_v R_{0M}} \\ R_{0S} &= \frac{4\beta_3 b_s b_H d_v^2 R_{0M}^2}{d_s (R_{0M}Q_1 - R_{0M}Q_3 - Q_4)(d_s + \rho_s)(R_{0M}Q_1 - Q_4 - R_{0M}Q_3 - 2\rho_H d_v R_{0M} - 2q_H d_v R_{0M})}. \end{aligned} \tag{89}$$

By differentiating R_{0S} partially with respect to R_{0M} we obtain

$$\frac{\partial R_{0S}^2}{R_{0M}} = \frac{8\beta_3 b_s \beta_2 b_H d_v^2 R_{0M} Q_4 (R_{0M}(\rho_H d_v + q_H d_v - Q_1 + Q_3) + Q_4)}{d_s (R_{0M}Q_1 - R_{0M}Q_3 - Q_4)^2 (d_s + \rho_s)(R_{0M}Q_1 - Q_4 - R_{0M}Q_3 - 2\rho_H d_v R_{0M} - 2q_H d_v R_{0M})^2}. \tag{90}$$

If (90) is greater than zero, it means that an increase in the malaria accounts for an increase of schistosomiasis cases in the population. The impact of malaria treatment on schistosomiasis is evaluated by partially differentiating R_{0M}

with respect to ω. We obtain $\partial R_{0M}/\partial\omega = -1/(\theta + \omega + d_H)$. Since R_{0M} is a decreasing function of ω, the treatment of schistosomiasis will have a positive impact on the dynamics of malaria.

5. Sensitivity Analysis of the Malaria-Schistosomiasis Coinfection Model

Sensitivity analysis is the study of how the uncertainty in the output of a mathematical model or system can be distributed to different sources of uncertainty in its outputs. It is a scientific procedure used in determining how different values of an independent variable will affect a particular dependent variable under a given set of hypotheses. This method is applied within certain limits that will depend on more input variables. The sensitivity index is a measure of the relative change in a variable with respect to the relative change in its parameters. It is known that there are uncertainties which are associated with the estimation of certain parameter values. So, it will be useful to study the effect of these parameters on the basic reproduction number $\mathscr{R}_{MS}$. The understanding from this study will help us to identify those parameters that cause the most increase/decrease in $\mathscr{R}_{MS}$ and by this we will be able to recognize the type of control measure(s) that is most suitable and effective in controlling the disease. Hence, we compute the normalized forward sensitivity index of $\mathscr{R}_{MS}$ with respect to the parameters in the model equation (12).

The normalized forward sensitivity index of a variable, u, that depends differentiably on index of a parameter, z, is defined as $K_z^v = \partial v/\partial z \times z/v$. As we have an implicit formula for $\mathscr{R}_{MS}$ we derive analytically for the sensitivity of $\mathscr{R}_{MS}$ as $K_z^{\mathscr{R}_{MS}} = \partial\mathscr{R}_{MS}/\partial z \times z/\mathscr{R}_{MS}$ to each parameter involved in $\mathscr{R}_{MS}$.

Important indices are given thus:

$$K_{\beta_1}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\beta_1} \times \frac{\beta_1}{\mathscr{R}_{MS}} = +1.0$$

$$K_{\beta_2}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\beta_2} \times \frac{\beta_2}{\mathscr{R}_{MS}} = +1.0$$

$$K_{\beta_3}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\beta_3} \times \frac{\beta_3}{\mathscr{R}_{MS}} = +1.0$$

$$K_{\beta_4}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\beta_4} \times \frac{\beta_4}{\mathscr{R}_{MS}} = +0.99$$

$$K_{b_v}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial b_v} \times \frac{b_v}{\mathscr{R}_{MS}} = +0.74$$

$$K_{d_H}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial d_H} \times \frac{d_H}{\mathscr{R}_{MS}} = -2.0$$

$$K_{\epsilon_v}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\epsilon_v} \times \frac{\epsilon_v}{\mathscr{R}_{MS}} = +1.0$$

$$K_{\epsilon_h}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\epsilon_h} \times \frac{\epsilon_h}{\mathscr{R}_{MS}} = +1.0$$

$$K_{\omega}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\omega} \times \frac{\omega}{\mathscr{R}_{MS}} = -0.91$$

$$K_{\theta}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\theta} \times \frac{\theta}{\mathscr{R}_{MS}} = -0.00000000000186$$

$$K_{\sigma}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\gamma} \times \frac{\sigma}{\mathscr{R}_{MS}} = +2.0$$

$$K_{d_v}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial d_v} \times \frac{d_v}{\mathscr{R}_{MS}} = -0.23$$

$$K_{b_H}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial b_H} \times \frac{b_H}{\mathscr{R}_{MS}} = +2.0$$

$$K_{b_s}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial b_s} \times \frac{b_s}{\mathscr{R}_{MS}} = +1.0$$

$$K_{d_s}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial d_s} \times \frac{d_s}{\mathscr{R}_{MS}} = -0.11$$

$$K_{\rho_s}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\rho_s} \times \frac{\rho_s}{\mathscr{R}_{MS}} = -0.876$$

$$K_{\rho_H}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial\rho_H} \times \frac{\rho_H}{\mathscr{R}_{MS}} = -0.0069$$

$$K_{q_H}^{\mathscr{R}_{MS}} = \frac{\partial\mathscr{R}_{MS}}{\partial q_H} \times \frac{q_H}{\mathscr{R}_{MS}} = -0.99. \tag{91}$$

5.1. Interpretation of Sensitivity Indices. It is observed that the positive signs of sensitivity indices of the basic reproduction number to the model parameters reveal that an increase or reduction in the value of each of the parameter in this case will point to an increase or decrease in the basic reproduction number of the disease [22]. The case of the $K_{\epsilon_v}^{\mathscr{R}_{MS}} = 1.0$ shows that increasing or reducing the mosquito biting rate by 10 percent increases or reduces the basic reproduction number, $\mathscr{R}_{MS}$, by 10 percent. However, the negative signs of the sensitivity indices of the basic reproduction number to the model parameters show that an increase or reduction in the value of each of the parameter in this class indicates a corresponding reduction or increase in the basic reproduction number of the disease [22]. Hence, the sensitivity analysis of the malaria-schistosomiasis coinfection model (12) provides an effective means of knowing how the dynamics of the coinfection model respond to changes in parameters of the model [22]. More importantly, it helps as a guide to policy makers and public health authorities in focusing on an appropriate intervention strategy for preventing and controlling the spread of the disease. The sensitivity analysis is applied to study the relative importance of model parameters to malaria-schistosomiasis coinfection. The sensitivity analysis reveals how each parameters influence the malaria-schistosomiasis coinfection output. However, the analysis will be carried out with response to the most crucial and sensitive parameters to the malaria-schistosomiasis coinfection model.

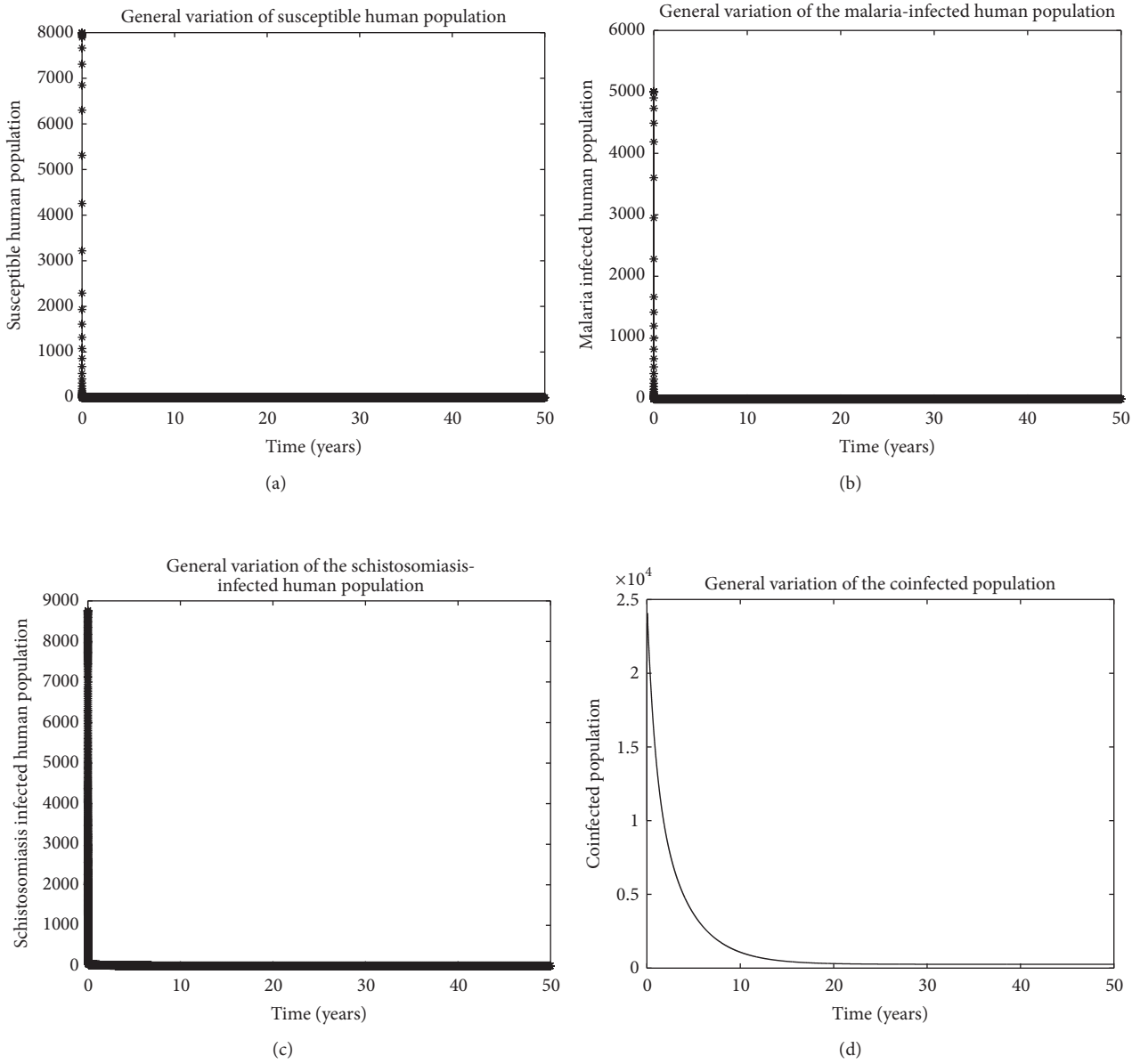

Figure 1: Simulation showing the variation of susceptible human (S_h) in (a), infected human with malaria only (I_m) in (b), infected human with schistosomiasis only (I_{hs}) in (c), and humans infected with both malaria and schistosomiasis (V_{ms}) in (d).

6. Numerical Simulation and Graphical Illustration of the Malaria-Schistosomiasis Coinfection Model

Numerical simulation and graphical illustrations are carried out in order to verify some of the analytical results. Different initial starts have been used from Table 2 to perform the computer simulations for different cases and displayed graphically in Figures 1–4. Figure 1 shows the variation of susceptible human (S_h), infected human with malaria only (I_m), infected human with schistosomiasis only (I_{hs}), and humans infected with both malaria and schistosomiasis (V_{ms}). It is observed that, in Figure 1(a), the susceptible human population decreases with time due to the malaria-schistosomiasis coinfections and in Figure 1(b), the population of malaria-infected human decreases with time due to treatment of the infected population. In Figure 1(c), the population of the schistosomiasis-infected human population also decreases due to the treatment while the population of the coinfected decreases to a particular level and then reaches its equilibrium point due to treatments. Figure 2 shows the variation of humans who recovered from malaria only (R_m), humans who recovered from schistosomiasis only (R_{hs}), and humans who recovered from both malaria and schistosomiasis (R_{ms}). It

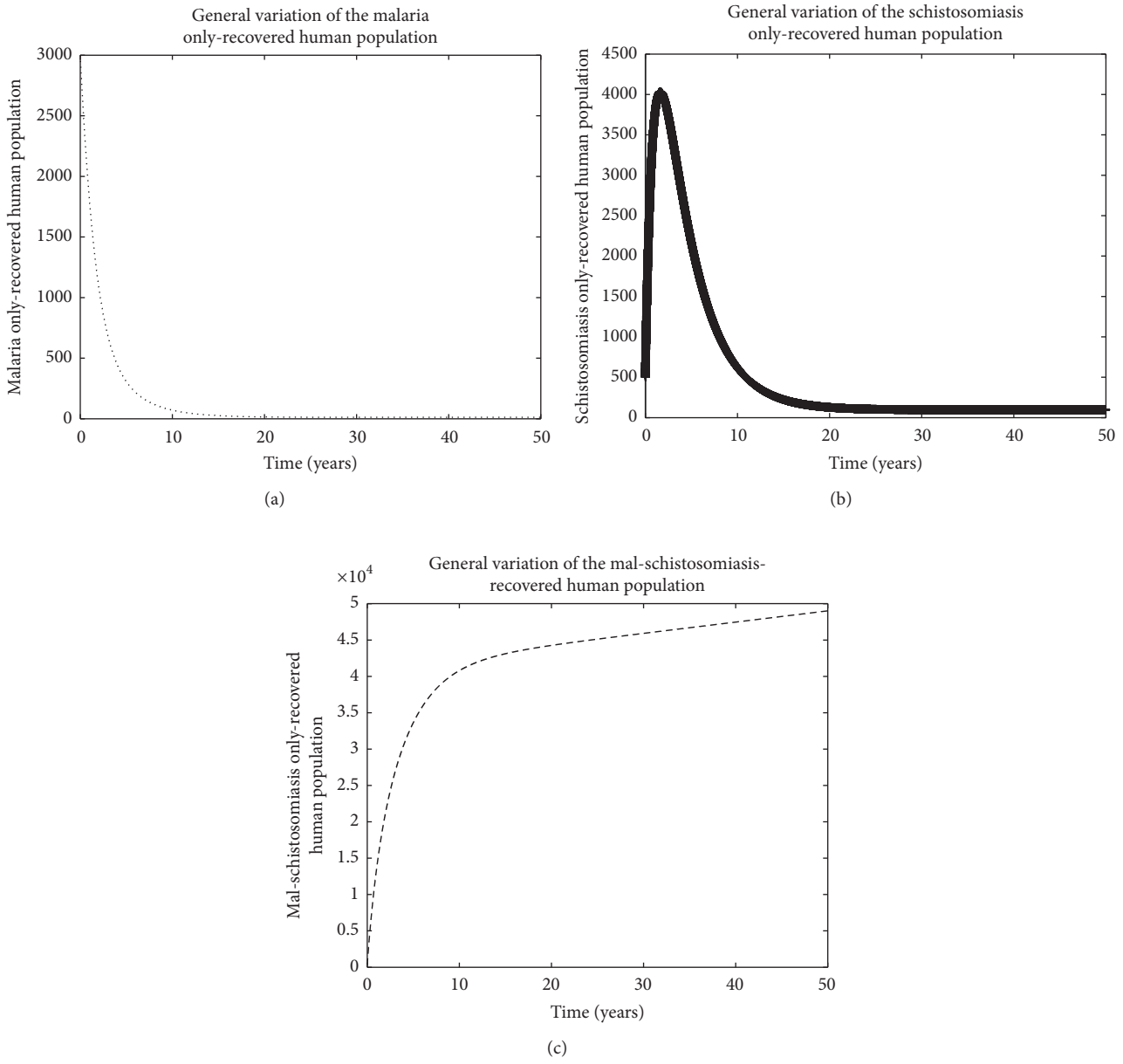

Figure 2: Simulation showing the variation of humans who recovered from malaria only (R_m) in (a), humans who recovered from schistosomiasis only (R_{hs}) in (b), and humans who recovered from both malaria and schistosomiasis (R_{ms}) in (c).

is observed that, in Figure 2(a), the population of malaria-recovered human population decreases with time to a level and in Figure 2(b), the population of the schistosomiasis-only-recovered human population increases with time and then decreases with time until it reaches its equilibrium point while, in Figure 2(c), the population of malaria-schistosomiasis recovered human population increases with time and then reaches its equilibrium point. Figure 3 shows the variation of susceptible mosquito (S_v) and infected mosquito with malaria (I_v). It is observed that, in Figure 3(a), the susceptible snail population decreases with time due to infection with schistosomiasis while, in Figure 3(b), the population of the infected snail maintains a constant equilibrium and then increases with time. Figure 4 shows the variation of susceptible mosquito (S_v) and infected mosquito with malaria (I_v). It is observed that, in Figure 4(a), the population of susceptible mosquito decreases and then increases to maintain a steady state while the infected mosquito population increases with time because of the presence of the infection.

7. Concluding Remarks

In this paper, we developed and analysed a deterministic model for the transmission of malaria-schistosomiasis coinfection without intervention strategies. The model was rigorously analysed to gain insights into its qualitative

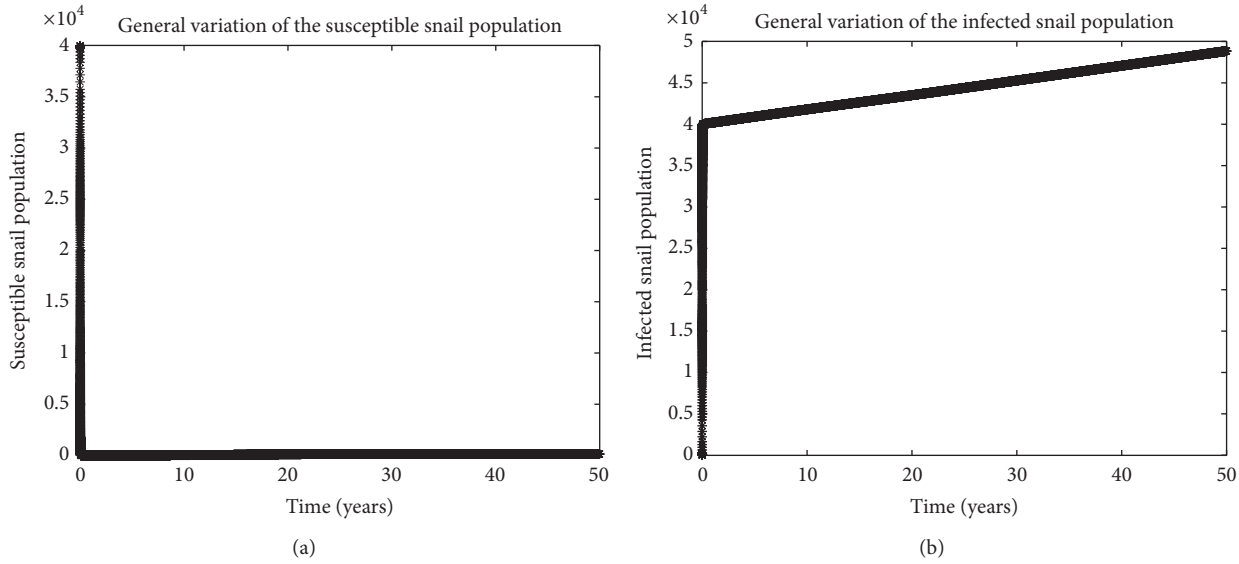

Figure 3: Simulation showing the variation of Susceptible Snail (S_s) in (a), and Infected Snail with schistosomiasis (I_s) in (b).

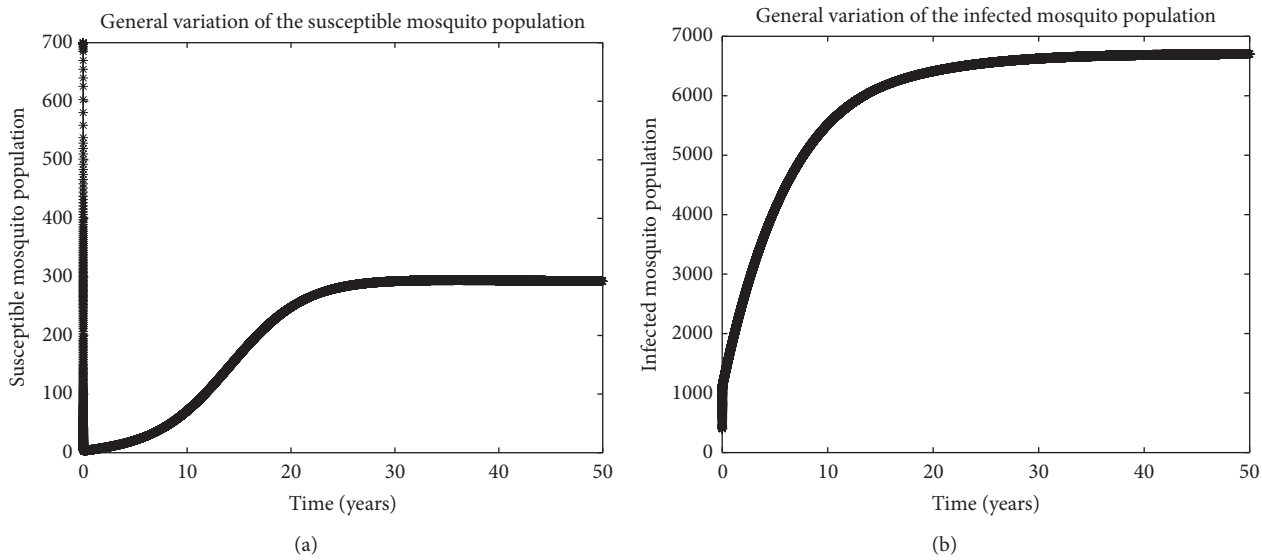

Figure 4: Simulation showing the variation of susceptible mosquito (S_v) in (a) and infected mosquito with malaria (I_v) in (b).

dynamics. We obtained the following results: (i) the malaria-schistosomiasis coinfection model has a locally stable disease-free equilibrium whenever the associated reproduction number is less than unity. (ii) We observed from our analysis of the effect of schistosomiasis on malaria that schistosomiasis infection may be associated with an increased risk of malaria. (iii) We show that the interaction between schistosomiasis and malaria may reduce the effectiveness of malaria treatment for controlling malaria transmission. Sensitivity analysis of the reproduction number to the model parameter was performed to investigate the parameters that possess greater influence on the model. Sensitivity analysis also showed that mosquito biting rate ϵ_v and rate of acquiring schistosomiasis through contacts with infected snails β_3 among other parameters contribute most significantly to the transmission and spread of the malaria disease. Hence the intervention strategy that inhibits the human-mosquito contact and human-snail contact should be encouraged in order to achieve a malaria-schistosomiasis-free population.

Table 2: Table showing numerical values of parameters used in the model.

Parameter	Symbol	Value	Source
Disease induced death rate due to malaria only	θ	0.05	[23]
Probability of human getting infected with malaria	β_1	0.8333	[24, 25]
Rate of acquiring schistosomiasis through contact with infected snails	β_2	0.406	[26]
Rate of acquiring schistosomiasis through contacts with infected humans	β_3	0.0004	[26]
Probability of mosquito getting infected by an infectious human	β_4	0.09	[24, 25]
Malaria schistosomiasis immunity waning rate	φ	0.0005	[24]
Schistosomiasis induced death rate	r	0.02	[assumed]
Malaria disease induced death rate	m	0.06	[assumed]
Coinfected population who recovered from malaria only	ϕ	0.12	[assumed]
Disease induced death rate of human due to schistosomiasis only	ρ_H	0.0039	[27]
Disease induced death rate of snails due to schistosomiasis only	ρ_s	0.0004012	[28]
Human spontaneous recovery	ω	0.005	[13]
Rate of loss of immunity to the schistosomiasis disease only	k	0.7	[assumed]
Rate of loss of immunity to malaria and schistosomiasis disease	φ	0.0005	[24, 25]
Human spontaneous recovery from schistosomiasis only	q_H	0.56	[assumed]
Recovery rate of coinfected individual	α	0.7	[assumed]
Modification parameter	η_1	1.3	[assumed]
Modification parameter	η_2	1.4	[assumed]
Per capita birth rate of mosquitoes	b_v	1000	[24, 29]
Per capita birth rate of snails	b_s	200	[8]
Natural death rate of humans	d_H	0.00004	[10]
Per capita biting rate of mosquitoes	ϵ_v	0.2	[30–32]
Contact rate of vector per human per unit time	σ	0.502	[33]
Per capita biting rate of humans	ϵ_h	0.2	[30, 34]
Natural death rate of mosquitoes	d_v	0.1429	[24]
Natural death rate of snails	d_s	0.0000569	[26]
Rate of loss of immunity to the malaria disease only	γ	0.7902	[24, 29, 35]
Per capita birth rate of humans	b_h	100	[36]

Competing Interests

The authors declare that they have no competing interests.

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