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Mathematical Model for the Dynamics of *Paragonimiasis* Disease in Human, Snail, and Crustacean Populations

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ABSTRACT

This paper proposes a mathematical model for the dynamics of *Paragonimiasis* transmission in human, snail, and crustacean populations. We proved that the solutions to the differential equations are both positive and bounded. In addition, we calculated the equilibrium states of *Paragonimiasis* in both its free and present forms. We computed the basic reproduction number using the next-generation matrix. Furthermore, we used the Routh-Hurwitz criteria to determine local stability, which revealed that the system is locally asymptotically stable (LAS). Similarly, we assessed the global stability of the disease-free equilibrium and found that the system is globally asymptotically stable (GAS). To better understand the dynamics of *Paragonimiasis* transmission, we carried out sensitivity index analyses and computational simulations. Our data show varied indices of rise and decline as parameters are changed.

1. Introduction

Paragonimiasis, commonly known as lung fluke sickness, is a tropical neglected disease that occurs in tropical, subtropical, and temperate regions across the world (WHO, 2023). *Paragonimiasis* is a life-threatening condition that causes morbidity and has the potential to hurt one's socioeconomic status. The respiratory disease is caused by the trematode Paragonimus, which grows in the lungs of people or animals that eat raw or undercooked freshwater crustaceans, usually brachyuran crabs, infected with the fluke's larval stages. Bakuza (2020), Banzai *et al.* (2021) and Rabone *et al.* (2021) have identified various *Paragonimus* species, with *P. westermani*, *P. africanus*, *P. heterotremus*, *P. kellicotti*, *P. mexicanus*, *P. siamensis*, *P. skrjabini*, *P. skrjabini miyazakii*, and *P. uterobilateralis* causing human infections. The prevalence of human *Paragonimiasis* cases is notable in Asia, Africa, and North and South America (Narain, Agatsuma and Blair, 2010; Chai, 2013). The disease's impact is evident in different regions, including Southeast Nigeria, where the Nigerian Civil War (1967-1970) triggered an epidemic due to food shortages and altered eating habits, particularly the consumption of raw crabs. Noteworthy spikes in prevalence have been reported, emphasizing the disease's persistence and potential for long-term infections (Eke *et al*., 2013).

Recent researches on *Paragonimiasis* and the *Paragonimus* genus investigate various aspects of its biology, evolution, and medical significance. Several studies by Adams (2018), Doanh *et al.* (2020), Chai and Jung (2019), Zhou *et al.* (2021), Rabone *et al.* (2021), and Tenorio and Molina (2021) provide insights from Asia, China, Africa, the Philippines, North America, and Ecuador. Yuan *et al.* (2018) investigated the transmission dynamics of *Paragonimus* in Foshan, China, using a deterministic model to grasp its spread across human snail-fish populations. However, underscoring the need for a broader approach, this paper examines Yuan *et al.* (2018) weaknesses. In contrast to their failure to include the whole range of symptoms associated with food-borne trematodiasis (*Paragonimus* /*Paragonimiasis*) as described by WHO (2023) the current study provides an improved model. This model includes asymptomatic, symptomatic, and chronic infectious phases for humans, as well as an exposed compartment for intermediate hosts (snails and crayfish), to improve the existing framework.

2. Formulation of *Paragonimiasis* Model

This paper studied the dynamics of *Paragonimiasis* disease. Five (5) populations of interest were considered i.e all the intermediate and definite hosts for the transmission dynamics of *Paragonimiasis* which includes humans, eggs, snails, cercariae, and crayfish. The human population is subdivided into five (5) compartments namely susceptible human $S_h(t)$, exposed human $E_h(t)$, acutely infected human $I_A(t)$, chronically infected human, and recovered human $R_h(t)$. The human population has the (SEI_AI_CR) type of model hence, the total number of human

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populations is given by $N_h(t) = S_h + E_h + I_A + I_C + R_h$. The population of susceptible individuals increases by recruitment into the class at a constant rate Λ_h and also from inflow of individuals who have received treatment and have lost immunity to the disease at the rates ϕ. Susceptible human population reduced due to infection acquired via ingestion of contaminated and not properly cooked crayfish at $\frac{\beta_h S_h I_c}{N_h}$ force of infection rate, where β_h is the transmission rate from crayfish to human. The population of susceptible individuals further reduced by natural death at a rate μ_h. The exposed individuals become asymptomatically infectious to the disease due to progression of the disease at $\frac{\beta_h S_h I_c}{N_h}$ progression rate and reduced by r progression rate from exposed to acutely infectious human or natural death at a rate μ_h. The population of acutely infected individuals increases by r progression rate from exposed to acute infected and reduces due to disease induced death at a rate d_1 or ψr_1, where ψr_1 is the proportion of acute that received treatment and recovered from the disease and by natural death at a rate μ_h. The chronically infected individuals population increases by $(1-\psi)r_1$, this shows proportion of acute infected individuals who become carriers and move to chronic infected class. The population is reduced by disease induced death at a rate d_2, natural death at a rate μ_h or chronically infected individuals that received treatment and recovered from the disease at a rate r_2. The recovered individuals population increases at a rate r_2 and ψr_1. This denotes fraction of those that received treatment from chronic infection and acute infection individuals. The recovered population reduced by ϕ and natural death at a rate μ_h.

The snail's population is subdivided into three (3) compartments namely susceptible snails $S_s(t)$, exposed snails $E_s(t)$ and infected snails $I_s(t)$ respectively. The snails population has (SEI) type of model hence, the total number of snails population is given by $N_s(t) = S_s(t) + I_s(t)$. The population of susceptible class increases by recruitment into the compartments at a rate Λ_s. Susceptible snails population reduced by $\frac{\beta_s S_s G}{N_s}$ force of infection rate, where β_s is the transmission rate from eggs to snails. The population of susceptible snails further reduced by natural death at a rate μ_s. The exposed snails class increases due to progression at $\frac{\beta_s S_s G}{N_s}$ progression rate and reduced by natural death at a rate μ_s. The population of infected snails increases at a rate ε_1 and reduced by natural death at a rate μ_s. Similarly, $N_c(t) = S_c(t) + E_c(t) + I_c(t)$ denote total number of crayfish population. Where $S_s(t)$ susceptible crayfish, $E_s(t)$ exposed crayfish and $I_s(t)$ infectious crayfish. The population of susceptible class increases by recruitment into the compartments at a recruitment rate Λ_c. Susceptible crayfish population reduced by $\frac{\beta_c S_c C}{N_c}$ force of infection rate, where β_c is the transmission rate from cercariae to crayfish. The population of susceptible snails further reduced by predation rate p_c and natural death at a rate μ_c. The exposed snails class increases due to progression at $\frac{\beta_c S_c C}{N_c}$ progression rate and reduced by predation rate p_c and natural death rate. The population of infected crayfish increases at a rate ε_2 and reduced by predation rate p_c and natural death at a rate μ_c .The eggs population is denoted by $G(t)$. Recruitment into these compartments is that, a portion P_g of eggs leaves the infectious body via sputum, faeces or urine at a rate θ_g finds their way into fresh water. A susceptible aquatic snail if come in contact with the release eggs becomes exposed to producing cercariae. After the incubation period from first contact matured, the exposed snails become infected. The infected snails will then release a second form of free-swimming larva called cercariae, a portion P_c at a rate θ_c. The number of eggs consumed by snails compared to the number of eggs in the environment cannot give rise to a susceptible snail. Thus, this $\frac{\delta_g \beta_s S_s(t) G(t)}{N_s(t)}$ proportion of larva eggs (*miracidium*) is consumed by susceptible snails' population. Similarly, the number of cercariae consumed by crayfish can't give rise to a susceptible

crayfish. This $\frac{\delta_c \beta_c S_c(t) C(t)}{N_c(t)}$ proportion of cercariae is being consumed by susceptible crayfish population.

The schematic diagram and model equations for the transmission dynamics of *Paragonimiasis* are presented in figure 1 and system of equation 1 respectively.

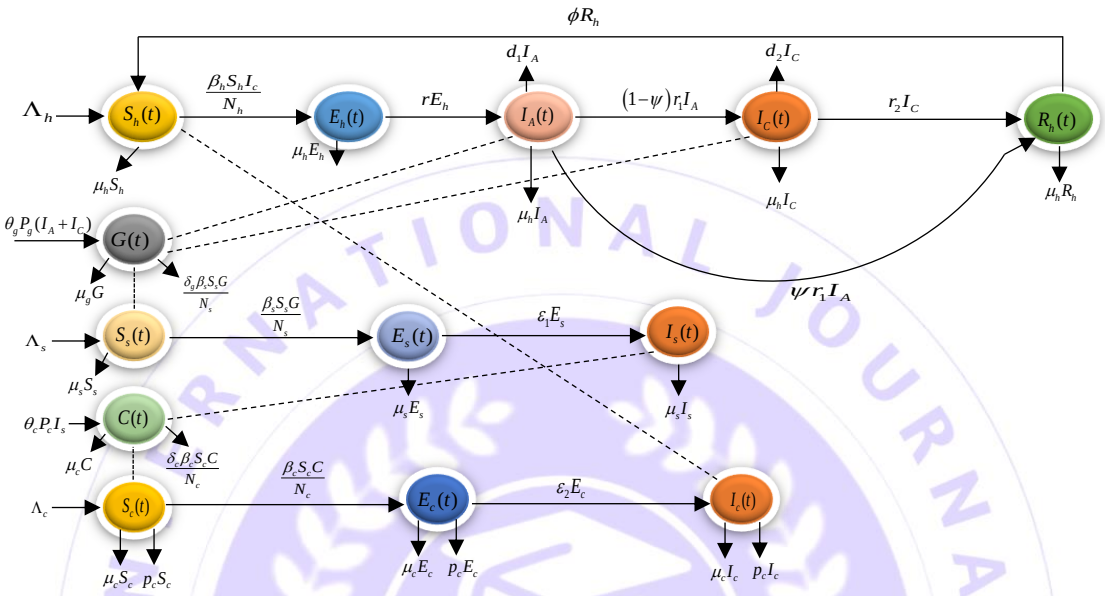


FIGURE 1: Schematic diagram of *Paragonimiasis*

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \phi R_h - \left(\frac{\beta_h I_c}{N_h} + \mu_h\right) S_h \\
\frac{dE_h}{dt} &= \frac{\beta_h S_h I_c}{N_h} - (r + \mu_h) E_h \\
\frac{dI_A}{dt} &= rE_h - \left[(1-\psi) r_1 + d_1 + \psi r_1 + \mu_h\right] I_A \\
\frac{dI_C}{dt} &= (1-\psi) r_1 I_A - (r_2 + d_2 + \mu_h) I_C \\
\frac{dR_h}{dt} &= \psi r_1 I_A + r_2 I_c - (\phi + \mu_h) R_h \\
\frac{dG}{dt} &= \theta_g P_g (I_A + I_C) - \left(\frac{\delta_g \beta_s S_s}{N_s} + \mu_g\right) G \\
\frac{dS_s}{dt} &= \Lambda_s - \left(\frac{\beta_s G}{N_s} + \mu_s\right) S_s \\
\frac{dE_s}{dt} &= \frac{\beta_s S_s G}{N_s} - (\varepsilon_1 + \mu_s) E_s \\
\frac{dI_s}{dt} &= \varepsilon_1 E_s - \mu_s I_s \\
\frac{dC}{dt} &= \theta_c P_c I_s - \left(\frac{\delta_c \beta_c S_c}{N_c} + \mu_c\right) C \\
\frac{dS_c}{dt} &= \Lambda_c - \left(\frac{\beta_c C}{N_c} + p_c + \mu_c\right) S_c \\
\frac{dE_c}{dt} &= \frac{\beta_c S_c C}{N_c} - (\varepsilon_2 + p_c + \mu_c) E_c \\
\frac{dI_c}{dt} &= \varepsilon_2 E_c - (p_c + \mu_c) I_c
\end{aligned}\right\} \quad (1)$$

with initial condition:

$$\begin{cases}
S_h(0) \geq 0, E_h(0) \geq 0, I_A(0) \geq 0, I_C(0) \geq 0, R_h(0) \geq 0, \\
G(0) \geq 0, \\
S_s(0) \geq 0, E_s(0) \geq 0, I_s(0) \geq 0, \\
C(0) \geq 0, \\
S_c(0) \geq 0, E_c(0) \geq 0, I_c(0) \geq 0.
\end{cases} \quad (2)$$

3. Analysis

In this section, the researchers established the positivity and boundedness of solutions to the Paragonimiasis model. In addition, they also determined the equilibrium states of the Paragonimiasis disease and examined the fundamental reproduction number and stability analysis of the disease.

3.1 Positivity of the solutions of *Paragonimiasis* model

Theorem 1: The solution set $\{S_h, E_h, I_A, I_c, R_h, G, S_s, E_s, I_s, C, S_c, E_c, I_c\}$ of the model equations (1) with non-negative initial conditions remain non-negative at $t > 0.$ Hence, the initial set will be:

$$\Omega = \left\{\left(S_h(t), E_h(t), I_A(t), I_c(t), R_h(t), G(t), S_s(t), E_s(t), I_s(t), C(t), S_c(t), E_c(t), I_c(t)\right) \in \mathbb{R}_+^{13}\right\}$$

which is positive for all time $t.$

Proof:

Given the initial conditions of the model equations (1) as $S_h(0)$, $E_h(0)$, $I_A(0)$, $I_c(0)$, $R_h(0)$, $G(0)$, $S_s(0)$, $E_s(0)$, $I_s(0)$, $C(0)$, $S_c(0)$, $E_c(0)$, $I_c(0)$ are non-negative. Then, the equations of the system with only term related to the state variables are considered. Thus, yielded

$E_h(t) \geq E_h(0)e^{-(r+\mu_h)t}$, $I_A(t) \geq I_A(0)e^{-(d_1+r_1+\mu_h)t}$, $I_C(t) \geq I_C(0)e^{-(r_2+d_2+\mu_h)t}$, $R_h(t) \geq R_h(t)e^{-(\phi+\mu_h)t}$,

$G(t) \geq G(0)e^{-(\lambda_2+\mu_g)t}$, $S_s(t) \geq S_s(0)e^{-(\lambda_3+\mu_s)t}$, $E_s(t) \geq E_s(0)e^{-(\varepsilon_1+\mu_s)t}$, $I_s(t) \geq I_s(0)e^{-\mu_s t}$,

$C(t) \geq C(0)e^{-(\lambda_4+\mu_c)t}$, $S_c(t) \geq S_c(t)e^{-(\lambda_5+p_c+\mu_c)t}$, $E_c(t) \geq E_c(0)e^{-(\varepsilon_2+P_c+\mu_c)t}$ and $I_c(t) \geq I_c(t)e^{-(P_c+\mu_c)t}$

$\forall t > 0$

3.2 Boundedness of the solution of *Paragonimiasis* model

The model equation (1) is analyzed in a biologically feasible region.

Theorem 2: The solution set $\{S_h, E_h, I_A, I_c, R_h, G, S_s, E_s, I_s, C, S_c, E_c, I_c\}$ of the model equations (1) is assumed to be non-negative at $t > 0.$ using standard analysis, the solutions to the system of equations (1) are non-negative in the region:

$$\Omega = \left\{\left(S_h(t), E_h(t), I_A(t), I_c(t), R_h(t), G(t), S_s(t), E_s(t), I_s(t), C(t), S_c(t), E_c(t), I_c(t)\right) \in R_+^{13}:\right.$$

$$0 \leq S_h(t) + E_h(t) + I_A(t) + I_c(t) + R_h(t) \leq \frac{\Lambda_h}{\mu_h}, 0 \leq G(t) \leq \frac{2\theta_g p_g \Lambda_h}{(\delta_g \beta_s + \mu_g)\mu_h},$$

$$\left. 0 \leq S_s(t) + E_s(t) + I_s(t) \leq \frac{\Lambda_s}{\mu_s}, 0 \leq C(t) \leq \frac{\theta_c p_c \Lambda_s}{(\delta_c \beta_c + \mu_c)\mu_s}, 0 \leq S_c(t) + E_c(t) + I_c(t) \leq \frac{\Lambda_c}{P_c + \mu_c}\right\}$$

which is positively invariant and attracting for the system (1) for all time? $t.$

Proof:

The total summation of the human population yields `

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_A}{dt} + \frac{dI_c}{dt} + \frac{dR_h}{dt}$$

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h \leq \Lambda_h - \mu_h N_h$$

On solving the above inequality, we obtained

$$\lim_{x\to\infty} SupN_h \leq \frac{\Lambda_h}{\mu_h}$$

Also, following the same procedure for the egg, snail, cercariae and crayfish population, we obtained,

$$\lim_{x\to\infty} SupN_G \leq \frac{2\theta_g P_g \Lambda_h}{\left(\delta_g \beta_s + \mu_g\right)\mu_h}, \lim_{x\to\infty} SupN_s \leq \frac{\Lambda_s}{\mu_s}, \lim_{x\to\infty} SupN_C \leq \frac{\theta_c p_c \Lambda_s}{\left(\delta_c \beta_c + \mu_c\right)\mu_s} \text{ and}$$

$$\lim_{x\to\infty} SupN_c \leq \frac{\Lambda_c}{\left(p_c + \mu_c\right)}$$

Therefore, the feasible solution set of *Paragonimiasis* model (1) is positively invariant in the region Ω. Thus, the *Paragonimiasis* model is epidemiologically and mathematically well posed and is sufficient to study the dynamic of the model in the region Ω.

Table 1: Variables and parameter description of *Paragonimiasis* model

Variables	Description
$S_h(t)$	Susceptible Human at time t.
$E_h(t)$	Exposed Human at time t.
$I_A(t)$	Acute Infected Human at time t.
$I_C(t)$	Chronic Infected Human at time t.
$R_h(t)$	Recovered Human at time t.
$G(t)$	Population of eggs at time t.
$S_s(t)$	Population of susceptible Snail at time t.
$E_s(t)$	Population of exposed Snail at time t.
$I_s(t)$	Population of Infected Snail at time t.
$C(t)$	Population of Cercariae at time t.
$S_c(t)$	Population of susceptible crayfish at time t.
$E_c(t)$	Population of exposed crayfish at time t.
$I_c(t)$	Population of Infected crayfish at time t.
Λ_h	Recruitment rate into susceptible human compartment
Λ_s	Recruitment rate into susceptible snails compartment
Λ_c	Recruitment rate into susceptible crayfish compartment
β_h	Transmission rate from infected crayfish to humans
β_s	Transmission rate from eggs to snails
β_c	Transmission rate from cercariae to crayfish
μ_h	Death rate of human host
μ_s	Death rate of snails
$\mu_c + p$	Death rate and predation rate of crayfish
μ_g	Death rate of meta cercariae eggs
μ_c	Death rate of cercariae
P_c	Number of cercariae in every infected snail
P_g	Number of embryonated eggs passed by each infected human
θ_g	Rate of eggs into fresh water (snail)
θ_c	Rate of cercariae released from infected snails
r	Progression rate from exposed to infectious human
d_1	Death rate from acute infection
d_2	Death rate from chronic infection
r_1	Rate of progression to chronic from acute infected individual
δ_c	Per consumption rate of the eggs by crayfish

δ_g	Per consumption rate of the eggs by snail
r_2	Rate of progression from chronic to recovered.
ε_1	Proportion of snails in latent that become infectious
ε_2	Proportion of crayfish in latent that become infectious
ϕ	Rate of human recovery due to permanent immunity.
ψ	Rate of acute infection human that recovered after receiving treatment

3.3 *Paragonimiasis* equilibrium states

The *Paragonimiasis* has two equilibrium states namely *Paragonimiasis* free equilibrium state and *Paragonimiasis* present equilibrium state obtained by setting the model differential equations to zero:

$$\frac{dS_h}{dt}=\frac{dE_h}{dt}=\frac{dI_A}{dt}=\frac{dI_C}{dt}=\frac{dR}{dt}=\frac{dG}{dt}=\frac{dS_s}{dt}=\frac{dE_s}{dt}=\frac{dI_s}{dt}=\frac{dC}{dt}=\frac{dS_c}{dt}=\frac{dE_c}{dt}=\frac{dI_c}{dt}=0 \tag{3}$$

3.3.1 *Paragonimiasis* free equilibrium state

The *Paragonimiasis* free equilibrium state $(E_q^{\,0})$ for the model equations (1) is given by

$$E_q^{\,0}=\left(\frac{\Lambda_h}{\mu_h},0,0,0,0,0,\frac{\Lambda_s}{\mu_s},0,0,0,\frac{\Lambda_c}{p_c+\mu_c},0,0,0\right) \tag{4}$$

3.3.2 *Paragonimiasis* present equilibrium state

We obtained *Paragonimiasis* present equilibrium state $(E_q^{\,*})$ of the model equations (1) by

$$
\begin{aligned}
S_h^* &= \frac{\Lambda_h\left(\left(\phi+\mu_h\right)\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)\left(r_2+d_2+\mu_h\right)\right)+\phi\left(rr_1\lambda_1\psi\left(r_2+d_2+\mu_h\right)+rr_1r_2\lambda_1\left(1-\psi\right)\right)}{\left(\lambda_1+\mu_h\right)\left(\phi+\mu_h\right)\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)\left(r_2+d_2+\mu_h\right)} \\
E_h^* &= \frac{\lambda_1}{\left(r+\mu_h\right)} \\
I_A^* &= \frac{r\lambda_1}{\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)} \\
I_C^* &= \frac{rr_1\lambda_1\left(1-\psi\right)}{\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)\left(r_2+d_2+\mu_h\right)} \\
R_h^* &= \frac{rr_1\lambda_1\psi\left(r_2+d_2+\mu_h\right)+rr_1r_2\lambda_1\left(1-\psi\right)}{\left(\phi+\mu_h\right)\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)\left(r_2+d_2+\mu_h\right)} \\
G^* &= \frac{\theta_g P_g r\lambda_1\left[\left(r_2+d_2+\mu_h\right)+r_1\left(1-\psi\right)\right]}{\left(\lambda_2+\mu_g\right)\left(r+\mu_h\right)\left(r_1+d_1+\mu_h\right)\left(r_2+d_2+\mu_h\right)} \\
S_s^* &= \frac{\Lambda_s}{\left(\lambda_3+\mu_s\right)} \\
E_s^* &= \frac{\lambda_3}{\left(\varepsilon_1+\mu_s\right)} \\
I_s^* &= \frac{\varepsilon_1\lambda_3}{\mu_s\left(\varepsilon_1+\mu_s\right)} \\
C^* &= \frac{\theta_c P_c\varepsilon_1\lambda_3\left(\lambda_4+p_c+\mu_c\right)}{\mu_s\left(\varepsilon_1+\mu_s\right)\left[\delta_c\beta_c\mu_c+\mu_c\left(\lambda_4+p_c+\mu_c\right)\right]} \\
S_c^* &= \frac{\Lambda_c}{\left(\lambda_4+p_c+\mu_c\right)} \\
E_c^* &= \frac{\lambda_4\Lambda_c}{\left(\lambda_4+p_c+\mu_c\right)\left(\varepsilon_2+p_c+\mu_c\right)} \\
I_c^* &= \frac{\varepsilon_2\Lambda_c\lambda_4}{\left(p_c+\mu_c\right)\left(\lambda_4+p_c+\mu_c\right)\left(\varepsilon_2+p_c+\mu_c\right)}
\end{aligned} \tag{5}
$$

where:

$$
\lambda_1=\frac{\beta_h S^*{}_h I^*{}_c}{N_h^*},\ \lambda_2=\frac{\delta_g\beta_s S^*{}_s}{N_s^*},\ \lambda_3=\frac{\beta_s S^*{}_s G^*}{N^*{}_s},\ \lambda_4=\frac{\beta_c C^*}{N_c^*},\ N_h^*=S_h^*+E_h^*+I_A^*+I_C^*+R_h^*\quad,
$$

$$
N_s^*=S_s^*+E_s^*+I_s^* \text{ and } N_c^*=S_c^*+E_c^*+I_c^*
$$

3.4 Reproduction number

The number of secondary infection cases brought on by the introduction of a single infectious agent into a population that is entirely susceptible is the basic reproduction number. The researchers opt for a generalized approach propounded by Diekmann, Heesterbeek, and Roberts, 2010. The threshold R_0 is represented by $R_0=\rho(FV^{-1})$

where ρ is the spectral radius.

Given the infectious stages as $E_h, I_A, I_C, G, E_s, I_s, C, E_c, I_c$ in (1) we can create a vector $\mathcal{F}$ that represents the new infections. The components of the vector $\mathcal{F}$ are obtained by considering the terms denoting new infections from the susceptible entering the exposed compartment:

$$F = \frac{\partial F_i}{\partial x_j} = \; F = \begin{bmatrix} 0 & 0 & \beta_h & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\delta_g \beta_s & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_s & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\delta_c \beta_c & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_c & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{6}$$

Along with $\mathcal{F}$, the researchers also consider V vectors which denote the outflow from the infectious compartments in (1) given by:

$$V = \frac{\partial V_i}{\partial x_j} = V = \begin{bmatrix} -(r+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ r & -(r_1+d_1+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\psi)r & -(r_2+d_2+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \theta_g P_g & \theta_g P_g & -\mu_g & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\varepsilon_1+\mu_s) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon_1 & -\mu_s & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta_c P_c & -\mu_c & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(\varepsilon_2+p_c+\mu_c) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_2 & -(p_c+\mu_c) \end{bmatrix} \tag{7}$$

Therefore, the basic reproduction number for *Paragonimiasis* model (1) is given by:

$$R_0 = \rho(FV^{-1}) = \frac{\beta_h r r_1 (1-\psi)}{(r+\mu_h)(r_1+d_1+\mu_h)(r_2+d_2+\mu_h)} \tag{8}$$

3.5 Local stability of *Paragonimiasis* free equilibrium state

Theorem 3: The *Paragonimiasis* free equilibrium state of the model equation (1) is Locally and Asymptotically Stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

Substituting the system of equations (1) and evaluating the result at DFE we obtained:

$$\left|J_{E_0}\right| = \begin{vmatrix} -\mu_h & 0 & 0 & -\frac{\beta_h S_h}{N_h} & \phi & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(r+\mu_h) & 0 & \frac{\beta_h S_h}{N_h} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & r & -(r_1+d_1+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\psi)r_1 & -(r_2+d_2+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \psi r_1 & r_2 & -(\phi+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_g P_g & \theta_g P_g & 0 & -\left(\frac{\delta_g \beta_s S_s}{N_s}+\mu_s\right) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\frac{\beta_s S_s}{N_s} & -\mu_s & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_s S_s}{N_s} & 0 & -(\varepsilon_1+\mu_s) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_1 & -\mu_s & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_c P_c & -\left(\frac{\delta_c \beta_c S_c}{N_c}+\mu_c\right) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_c S_c}{N_c} & -(p_c+\mu_c) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_c S_c}{N_c} & 0 & -(\varepsilon_2+p_c+\mu_c) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_2 & -(p_c+\mu_c) \end{vmatrix} \tag{9}$$

From (9) we have $\lambda_1 = -\mu_h$, $\lambda_2 = -(r+\mu_h)$, $\lambda_3 = -(r_1+d_1+\mu_h)$, $\lambda_4 = -(r_2+d_2+\mu_h)$, $\lambda_5 = -(\phi+\mu_h)$, $\lambda_6 = -\left(\frac{\delta_g \beta_s S_s}{N_s}+\mu_g\right)$, $\lambda_7 = -\mu_s$, $\lambda_8 = -(\varepsilon_1+\mu_s)$, $\lambda_9 = -\mu_s$, $\lambda_{10} = -\left(\frac{\delta_c \beta_c S_c}{N_c}+\mu_c\right)$, $\lambda_{11} = -(p_c+\mu_c)$, $\lambda_{12} = -(\varepsilon_2+p_c+\mu_c)$ and $\lambda_{13} = -(p_c+\mu_c) < 0$ only if $R_0 < 0$, thus, (9) is then reduces to a 3×3 matrix given by:

$$\begin{pmatrix} -(r+\mu_h) & 0 & \beta_h \\ r & -(r_1+d_1+\mu_h) & 0 \\ 0 & (1-\psi)r_1 & -(r_2+d_2+\mu_h) \end{pmatrix} \tag{10}$$

Similarly, equation (10) can be simplified as:

$$\begin{pmatrix} -K_1 & 0 & K_5 \\ r & -K_2 & 0 \\ 0 & K_4 & -K_3 \end{pmatrix} \tag{11}$$

where

$K_1 = -(r+\mu_h)$, $K_2 = -(r_1+d_1+\mu_h)$, $K_3 = -(r_2+d_2+\mu_h)$, $K_4 = -(1-\psi)r_1$ and $K_5 = \beta_h$

The characteristic polynomial of (11) is given by:

$$A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 \tag{12}$$

where the coefficients of are given by:

$A_1 = 1, A_2 = (K_1+K_2+K_3)$, $A_3 = \left[K_3(K_1+K_2) + \left((r+\mu_h)(r_1+d_1+\mu_h)(r_2+d_2+\mu_h)(1-R_0)\right)K_1K_2\right]$ and

$$A_4 = (r+\mu_h)(r_1+d_1+\mu_h)(r_2+d_2+\mu_h)(1-R_0)$$

By setting up Routh-Hurwitz array matrix we obtained:

$$\Rightarrow \begin{vmatrix} A_1 & A_3 & 0 & 0 \\ 1 & A_2 & A_4 & 0 \\ 0 & A_1 & A_3 & 0 \\ 0 & 1 & A_2 & A_4 \end{vmatrix} \tag{13}$$

$$\therefore H_1 > 0,\ |H_2| = A_1A_2 > A_3, |H_3| = A_1A_2A_3 > A_4A_1^2 + A_3^2, |H_4| = A_1A_2A_3A_4 > A_1^2A_4^2 + A_3^2A_4$$

Since $H_1, H_2, H_3, H_4 > 0$ as observed, by applying Routh-Hurwitz criteria which asserts that all roots of the polynomial have a negative real component if and only if the coefficient are positive and the determinant of the matrix $H_i > 0$ for $i = 1,...,3.$ It is clear that $A_i > 0$ for $i = 1,...,3$ are positive and A_3, A_4 will be positive when $R_0 < 0.$ Since the necessary condition for Routh-Hurwitz criteria for the third-order characteristics polynomial is satisfied, we conclude that, the *Paragonimiasis* free equilibrium is locally asymptotically stable (LAS) when $R_0 < 1.$

3.6 Global stability of *Paragonimiasis* free equilibrium state

The method of Castillo-Chavez, Feng and Huang (2002) was used to investigate the Global Asymptotic Stability (GAS) of the *Paragonimiasis* free equilibrium state for the system (1).

Theorem 4: The *Paragonimiasis* free equilibrium state of the model equation (1) is globally and asymptotically stable (GAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: We begin by establishing that the conditions H_1 and H_2 hold when $R_0 < 1.$ Consider:

$$K(X,0) = \begin{pmatrix} \Lambda_h - \mu_h S_h & 0 \\ \Lambda_s - \mu_s S_s & 0 \\ \Lambda_w - A_{is} S_w & 0 \\ \Lambda_c - (p_c + \mu_c) S_c & 0 \end{pmatrix} \text{ and}$$

$X \in x^4$ denotes the *Paragonimiasis* compartments

in the system (1) , we have: $B(X,Z) = AZ - B(X,Z)$

where

$$A = \begin{bmatrix} -(r+\mu_h) & 0 & \frac{\beta_h S^0{}_h}{N^0{}_h} & 0 & 0 & 0 & 0 & 0 & 0 \\ r & -(r_1+d_1+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\psi)r_1 & -(r_2+d_2+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \theta_g P_g & \theta_g P_g & -\left(\frac{\delta_g \beta_s S^0{}_s}{N^0_s} + \mu_g\right) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_s S^0{}_s}{N^0_s} & -(\varepsilon_1+\mu_s) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon_1 & -\mu_s & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta_c P_c & -\left(\frac{\delta_c \beta_c S^0_c}{N^0_c} + \mu_c\right) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_c S^0{}_c}{N^0_c} & -(\varepsilon_2+p_c+\mu_c) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_2 & -(p_c+\mu_c) \end{bmatrix}$$

(14)

$$\begin{pmatrix} B_1(X,Z) \\ B_2(X,Z) \\ B_3(X,Z) \\ B_4(X,Z) \\ B_5(X,Z) \\ B_6(X,Z) \\ B_7(X,Z) \\ B_8(X,Z) \\ B_9(X,Z) \end{pmatrix} = \begin{pmatrix} \beta_h I_C\left(1-\frac{S_h}{N_h}\right) \\ 0 \\ 0 \\ \delta_g \beta_s G\left(\frac{S_s}{N_s}-1\right) \\ \beta_h G\left(1-\frac{S_s}{N_s}\right) \\ 0 \\ \delta_c \beta_c C\left(\frac{S_C}{N_C}-1\right) \\ \beta_c C\left(1-\frac{S_c}{N_c}\right) \\ 0 \end{pmatrix}$$

Thus, (15)

Then $B_1(X,Z), B_2(X,Z), B_3(X,Z), B_5(X,Z), B_8(X,Z), B_9(X,Z) \geq 0$ if $S_h \leq N_h$, $S_s \leq N_s$, $S_c \leq N_c$ and $B_4(X,Z), B_7(X,Z) = 0,$ if $S_s = N_s$, and $S_C = N_C$ thus, the global stability of DFE of the system $\frac{dX}{dt} = K(X,0)$ is easy to verify. Therefore, (E^0) is globally asymptotically stable (GAS).

3.7 Sensitivity Analysis of *Paragonimiasis* Model

In this section, we investigate the relevance of each transmission parameter of the *Paragonimiasis* model (1) on the dynamics of the subpopulation of humans infected by the virus. To answer this key question, we compare the impact of those parameters on the outcome of the infected. The threshold R_0 parameter plays a determinant role in the different subpopulations of the model. This was used to check for and identify parameters that may influence basic reproduction R_0 and, in turn, the transmission of *Paragonimiasis*. The sensitivity index was computed using the formula:

$$\alpha_{\chi}^{R_0} = \frac{\partial R_0}{\partial \chi} \times \frac{\chi}{R_0} \tag{16}$$

where:

R_0 Is the basic reproduction number

χ Is the parameter of interest

Solving for the parameters of interest, we obtained the results as presented in table 2.

Table 2: Parameter values for R_0 used in the model and their sensitivity index

Parameter	Solutions	Index	Baseline Value	Sensitivity Index	Sources for Baseline Values
β_h	1	+	9.69×10^{-4}	1	Yuan *et al.* (2018)
r	$-\frac{\mu_h}{r+\mu_h}$	-	0.2405	-5.82×10^{-4}	Yuan *et al.* (2018)

r_1	$\frac{d_1+\mu_h}{r_1+d_1+\mu_h}$	+	0.125	0.0750	Assumed
r_2	$-\frac{r_2}{r_2+d_2+\mu_h}$	-	0.73	−0.9836	Assumed
d_1	$-\frac{d_1}{r_1+d_1+\mu_h}$	-	$0.1 year^{-1}$	-7.4×10^{-2}	Assumed
d_2	$-\frac{d_2}{r_2+d_2+\mu_h}$	-	$0.12 year^{-1}$	-1.62×10^{-2}	Assumed
ψ	$\frac{\psi}{\psi-1}$	+	0.3	0.4286	Assumed
μ_h		-	1.4×10^{-4}	-3.6×10^{-3}	Gao, Liu, Luo, and Xie, (2011)

The sensitivity indices results presented in table 2 show that, those parameters with positive indices increase the R_0 , that means; the parameters have great impact of expanding the disease in the population if their values are increased. Similarly, those parameters with negative sensitivity indices decreases the R_0 , which also implies that, the parameters has the ability of minimizing the disease in the population if their values are increased while others are kept constant.

3.8 Numerical Simulation

In this section, we performed numerical simulation on the system of equations (1) using the values in Table 3. The results are presented in Figure 2 to Figure 7.

Table 3: Initial and parameter values used for numerical simulation

Variables	Initial values	Reference
$S_h(0)$	1.9×10^5	Yuan *et al.* (2018)
$E_h(0)$	2882	Yuan *et al.* (2018)
$I_A(0)$	360	Assumed
$I_C(0)$	430	Assumed
$R_h(0)$	568	Yuan *et al.* (2018)
$G(0)$	25	Yuan *et al.* (2018)
$S_s(0)$	1000	Yuan *et al.* (2018)
$E_s(0)$	20	Assumed
$I_s(0)$	12	Yuan *et al.* (2018)
$C(0)$	14	Yuan *et al.* (2018)
$S_c(0)$	9.99×10^6	Yuan *et al.* (2018)
$E_c(0)$	2.13×10^5	Assumed
$I_c(0)$	1.9×10^5	Yuan *et al.* (2018)
Λ_h	5×10^6	Yuan *et al.* (2018)
Λ_s	3.12×10^6	Yuan *et al.* (2018)
Λ_c	1×10^3	Yuan *et al.* (2018)
β_s	5.54×10^{-4}	Yuan *et al.* (2018)
β_c	3.59×10^{-3}	Yuan *et al.* (2018)
μ_s	1	Chen *et al.* (2011)
μ_c	0.2030	Qian, *et al.* (2016)
μ_g	3.85×10^{-2}	Qian, *et al.* (2016)
μ_c	2.614	Qian, *et al.* (2016)
P_c	452	Yuan *et al.* (2018)
P_g	1.46×10^6	Yuan *et al.* (2018)
θ_c	1×10^{-2}	Yuan *et al.* (2018)
θ_g	0.1564	Yuan *et al.* (2018)
δ_c	0.2156	Assumed
δ_g	0.1714	Assumed
ε_1	0.234	Assumed
ε_2	0.605	Assumed
ϕ	0.67	Assumed
p_c	0.1001	Assumed

3.8.1 Dynamics of sub-populations plotted against time.

The dynamics of sub-population plotted against time is presented in Figure 2-7. All the graphs were generated using values in Table 3.

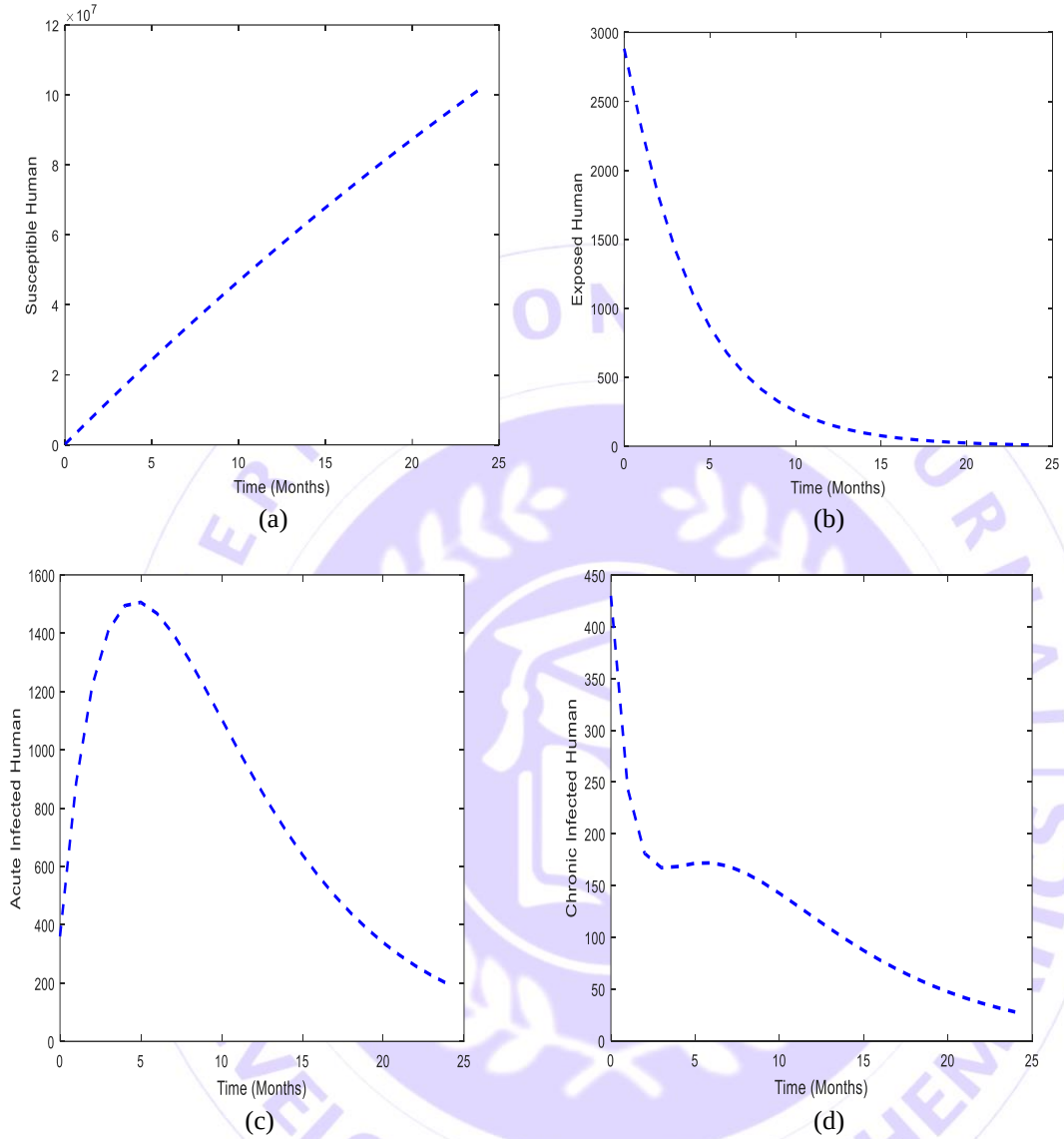
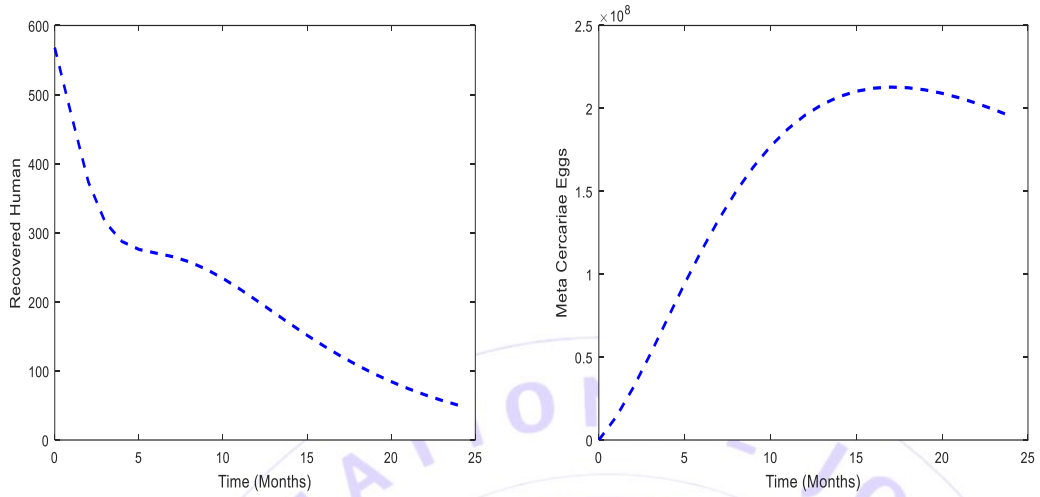
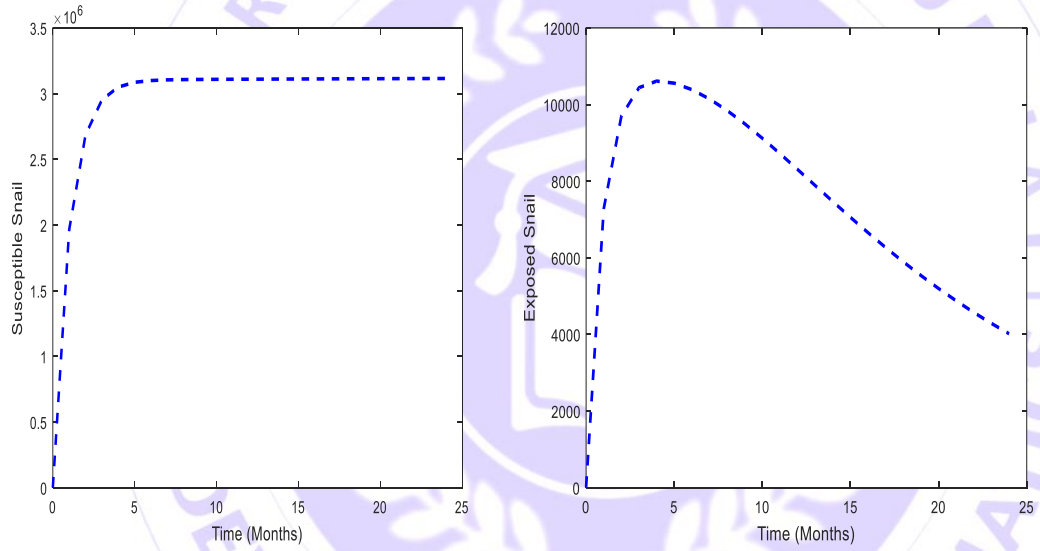


FIGURE 2: Dynamics of susceptible humans, exposed humans, acute infected humans and Chronic infected humans plotted against time.

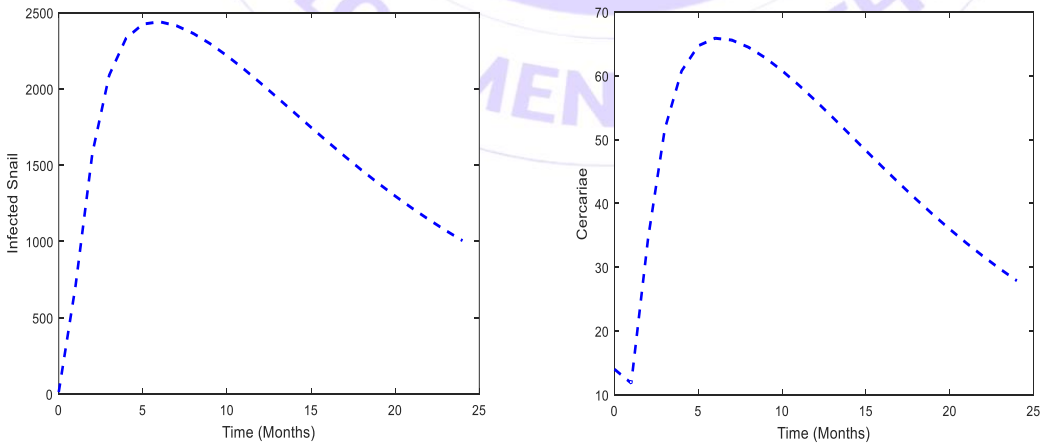


(e) (f)

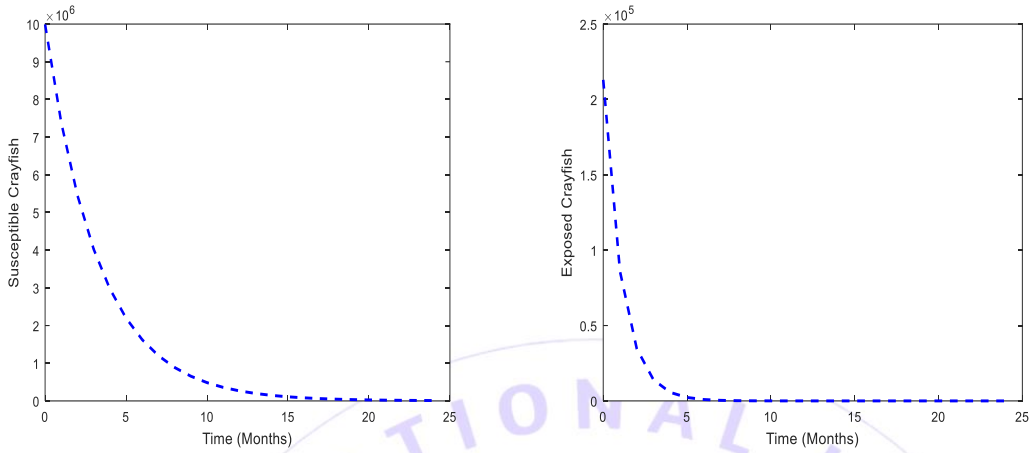


(g) (h)

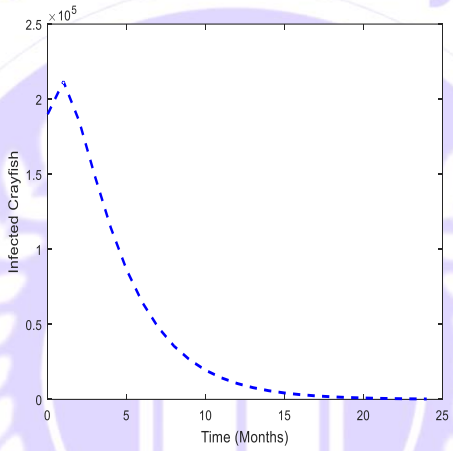
FIGURE 3: Dynamics of recovered humans, meta cercariae eggs, susceptible snail and exposed snail plotted against time.



(i) (j)



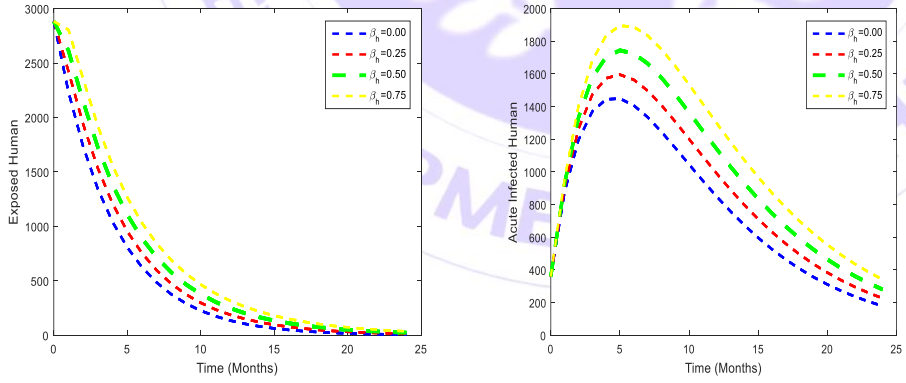
(k) (l)



(m)

FIGURE 4: Dynamics of infected snail, meta cercariae eggs, susceptible crayfish exposed crayfish and infected crayfish plotted against time.

3.8.2 Dynamics of sub-populations plotted against time with varied β_h



(a) (b)

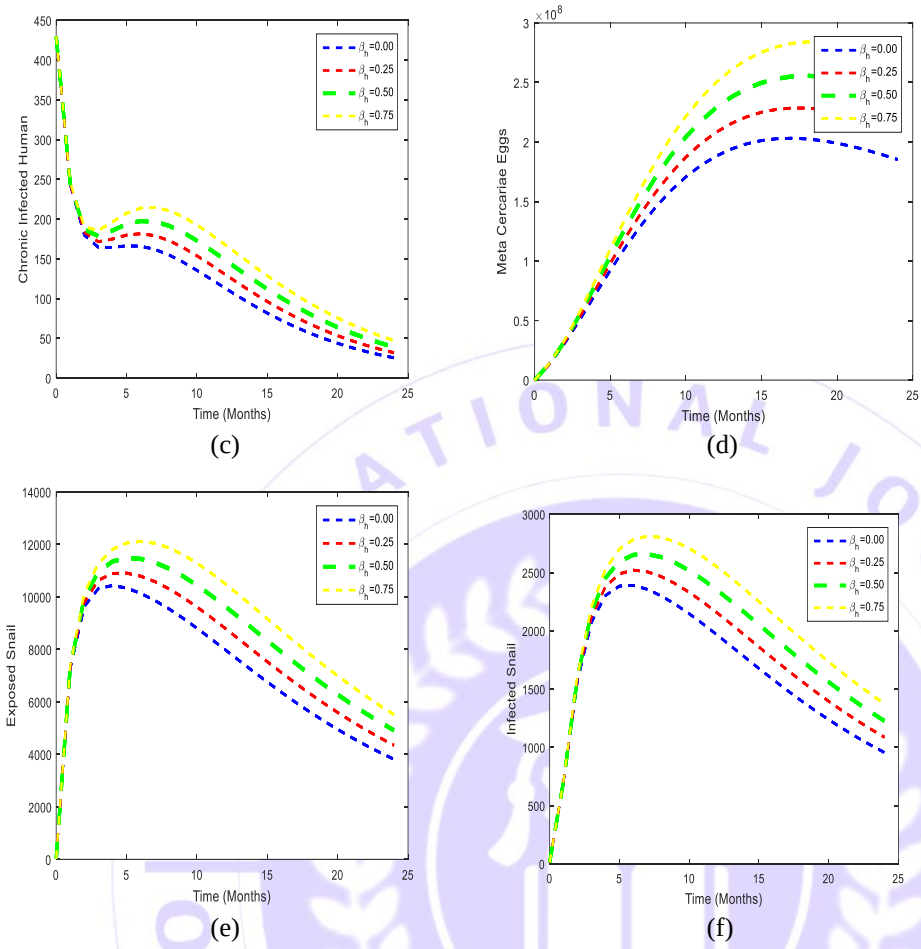
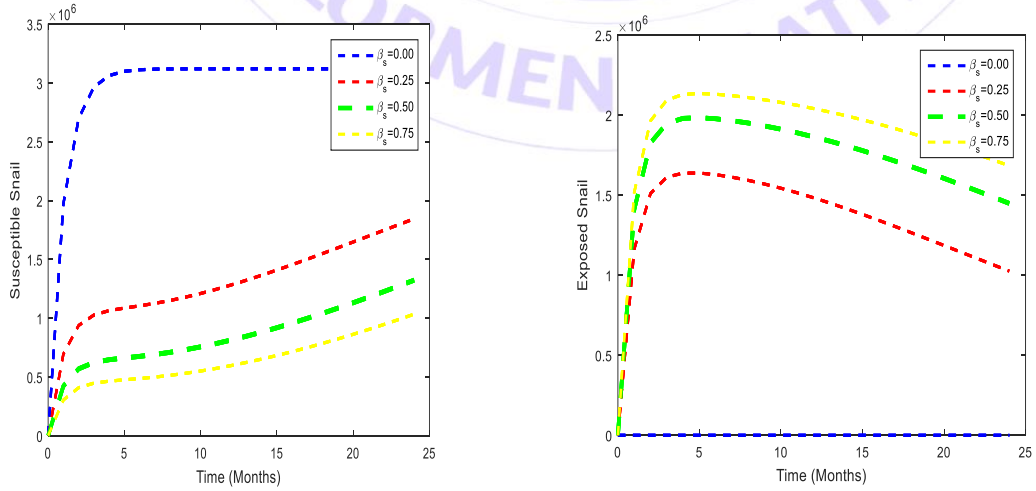


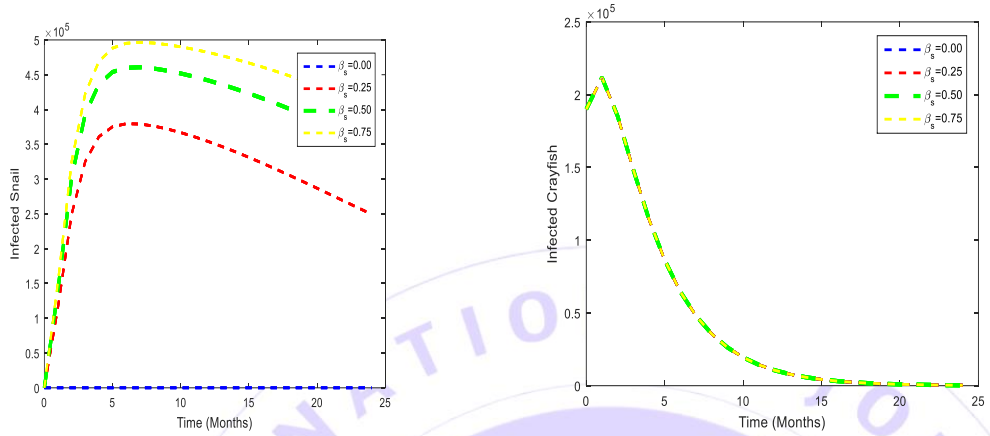
FIGURE 5: Dynamics of exposed human, acute infected human, chronic infect human, meta cercariae eggs, exposed snail and infected snail with varied β_h plotted against time.

3.8.3 Dynamics of sub-populations plotted against time with varied β_s

The dynamics of sub-population plotted against time with varied β_s is presented in figure 6. All the graphs were generated using values in table 3.



(a) (b)

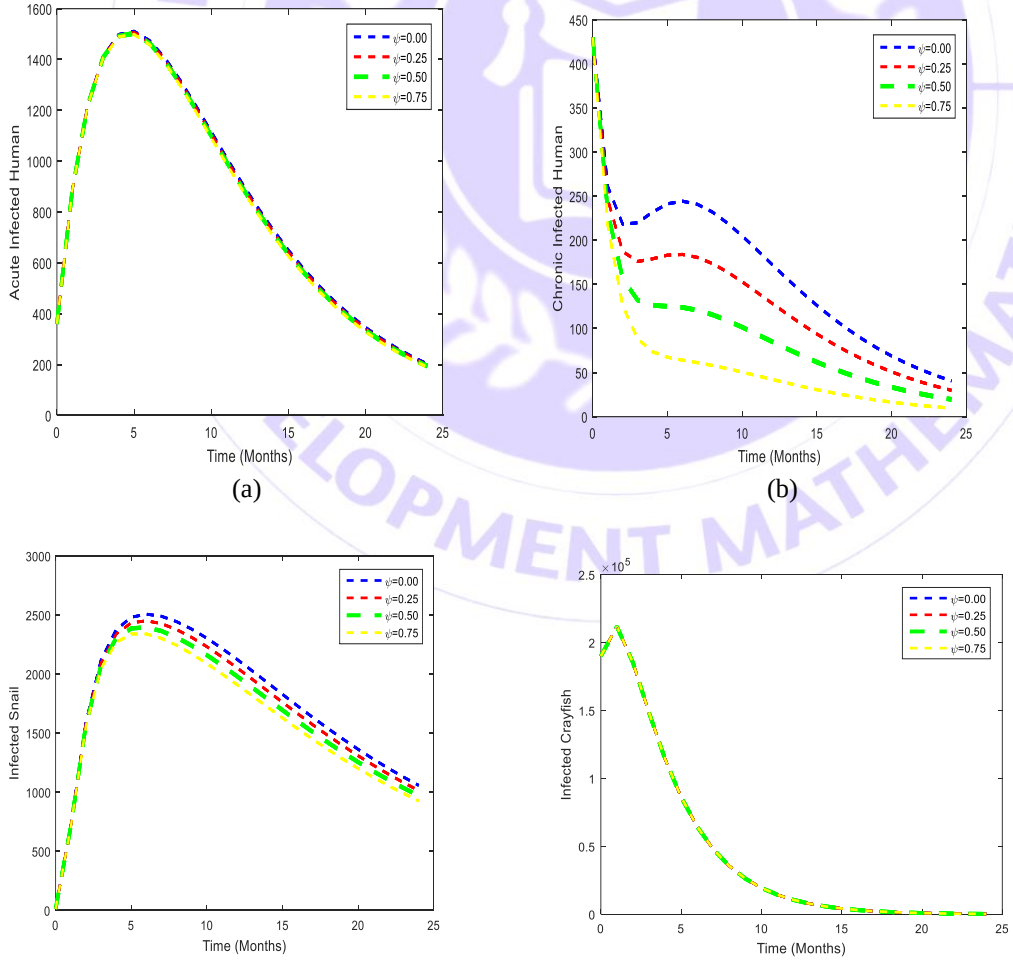


(c) (d)

FIGURE 6: Dynamics of susceptible snail, exposed snail, infected snail and infected crayfish with varied β_s plotted against time.

3.8.4 Dynamics of sub-populations plotted against time with varied ψ

The dynamics of sub-population plotted against time with varied ψ is presented in figure 7. All the graphs were generated using values in table 1 and table 3 respectively.



(a) (b)

(c) (d)

FIGURE 7: Dynamics of acute infected human, chronic infected human, infected snail and Infected crayfish with varied Ψ plotted against time.

4. Results Discussion

In this section, we discussed analytical and numerical results. The *Paragonimiasis* model is made up of thirteen (13) systems of differential equations. The positivity of the solutions to the system of differential equations must be non-negative; hence we showed that the solution of the model is positively invariant and attracting for the system (1) for all time *t.* The *Paragonimiasis* free and *Paragonimiasis* present equilibrium states were obtained. The basic reproduction number was also obtained using the next generation matrix. We applied the method of Routh Hurwitz criterion and Castillo-Chavez, Feng and Huang (2002) to investigate the Local and Global Asymptotic Stability of the *Paragonimiasis* free equilibrium state for the system. The Routh-Hurwitz criteria for Local stability result shows that it is Locally Asymptotically Stable (LAS). Similarly, the global stability of DFE was also computed, it shows that the system is Globally Asymptotically Stable. The sensitivity index was carried out and the result shows that, those parameters with positive indices increase the reproduction number while those parameters with negative sensitivity indices decreases it, which implies that, the parameters has the ability of increasing or decreasing the disease in the population.

This section further explored mathematical models that explain a comprehensive simulation of the dynamics of several subpopulations involved in the spread of *Paragonimiasis*. The simulation plots the changes over time for susceptible individual, exposed humans, acutely infected humans, chronically infected humans, recovered humans, Meta cercariae eggs, susceptible snail, exposed snail, infected snail, susceptible crayfish, exposed crayfish, and infected crayfish. The first portion (Section 3.8.1) focuses on the dynamics of susceptible individuals, exposed individuals, acutely infected humans, and chronically infected humans. The simulation, run using MATLAB R2015a, depicts the interaction of different human groups, with a substantial increase in the number of vulnerable individuals over time. The decrease in infectious people is due to interactions between diseased humans, snails, crayfish, and vulnerable humans. The next sections (Section 3.8.2 to Section 3.8.4) show simulations of various sub-populations involving recovered people, meta cercariae eggs, susceptible snail, exposed snail, infected snail, susceptible crayfish, exposed crayfish, and infected crayfish. The findings emphasize variations in behavior over time, including observations such as a decline in recovered persons after 5 months, consistent increases in meta cercariae and snail populations, and the effect of varied transmission rates on disease propagation. The sentence underlines the need of knowing these processes in order to better understand how the disease spreads and changes over time. It explores how altering transmission rates, progression rates, and other environmental variables affect the equilibrium points and overall prevalence of *Paragonimiasis* in distinct subpopulations.

5. Conclusion

Conclusively, this paper gives a thorough picture of *Paragonimiasis* transmission patterns, building on past studies and providing new insights. By exploring the positivity and boundedness of solutions, finding equilibrium states, and computing the fundamental reproduction number, the study provides a solid foundation for understanding disease propagation. Furthermore, stability assessments, both locally and globally, demonstrate the persistence of the Paragonimiasis-free equilibrium state under a variety of situations. Sensitivity analysis reveals crucial characteristics that influence *Paragonimiasis* patterns, guiding prospective therapeutic methods. The numerical models provide a useful tool for measuring and predicting the impact of *Paragonimiasis* on human populations, therefore assisting in the development of appropriate public health policies. Thus, this study provides useful knowledge to the field of infectious disease epidemiology, helping efforts to reduce the impact of *Paragonimiasis* on afflicted populations.

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