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MATHEMATICAL MODELING AND ANALYSIS OF HUMAN TO HUMAN SHIGELLOSIS TRANSMISSION WITH PUBLIC ENLIGHTENMENT

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Abstract

The shigella bacterium spreads a disease of the digestive tract known as shigellosis. The primary means of preventing shigellosis have historically been better sanitation and hygiene measures. Every year, this disease claims the lives of almost 1.1 million people, children under five years of age being the most affected. In this paper a deterministic mathematical model is proposed describing the transmission dynamics of human to human shigellosis. The model exhibits two equilibrium states, the disease-free equilibrium and the endemic equilibrium. However, the disease free equilibrium state is shown to be both locally and globally asymptotically stable under certain conditions when the control reproduction is less than unity ($\mathcal{R}_c < 1$). In contrast, endemic equilibrium is found to be globally asymptotically stable when the control reproduction number is greater than unity ($\mathcal{R}_c > 1$). The most sensitive parameters for the control of the spread of shigellosis are identified by the forward sensitivity index method (one that is very effective for the control of the disease). The contact rate β is found to be the most sensitive among all the parameters, indicating that to avoid the persistence of the disease, reducing contact between individuals should be emphasized. Finally, we obtained some numerical simulation results which that show that to eradicate shigellosis, there is need for minimizing the contact between infected individuals and susceptible ones and also minimize the number of carriers individuals that progressing to infected compartment when combined with public enlightenment and isolation of infected individuals.

Keywords: Shigellosis; public enlightenment; isolation; sensitivity analysis.

INTRODUCTION

Shigella bacteria is the cause of shigellosis, commonly referred to as bacterial dysentery, an infectious disease of the digestive tract. Shigella bacteria are responsible for most etiological causes of diarrhea, particularly bacillary dysentery. The four subgroups of the bacterial strains are S. flexneri, S. dysenteriae 1, S. sonnei, and S. boydii (Lindberg *et al.*, 1991). The majority of active cases and prevalence of shigellosis are predominantly caused by S. flexneri and S. sonnei, but the presence of S. dysenteriae 1 generally corresponds to severe dysentery outbreaks (Schroeder & Hilbi, 2008). Shigellosis is transmitted by human-to-human contact or sexual contact, contaminated food or water, and exposure to infected excrement. (CDC, 2017). In the early twentieth century, improvements in housing sanitation and hygiene significantly decreased the prevalence and incidence of dysentery and other subgroups of shigella. Reducing transmission was further aided by the development of antibiotics for the treatment of shigella bacteria (Lampel *et al.*, 2018). Shigella infections cause symptoms such as diarrhea, abdominal pain, vomiting, and fever (WHO, 2005). People with shigella infections who do not have symptoms of the disease

are called carriers. Severe cases of shigellosis can lead to reactive arthritis, sepsis, seizures, and hemolytic uremic syndrome. Symptoms usually begin one to two days after exposure and last five to seven days (WHO, 2005). Shigellosis epidemics usually occur in areas with overcrowding and poor sanitary conditions where person-to-person transmission or contamination of food or water by bacteria is common. An estimated 1.1 million deaths worldwide are attributed to it each year (Schroeder & Hilbi, 2008). Approximately 60% of deaths resulting from the disease affect children less than or equal to five years of age (Schroeder & Hilbi, 2008). In addition, it is common among travelers and men who engage in homosexual relationships (MSM) in countries with higher incomes (CDC, 2017). Shigellosis is a vaccine-preventable disease, however, despite multiple Shigella vaccines in existence, the vaccination coverage for the disease is still minimal (Levine *et al.*, 2007). Shigella species have developed an increasing resistance to the most commonly used antimicrobials such as ampicillin, cotrimoxazole, nalidixic acid and even ciprofloxacin and norfloxacin over the past few decades (Bhattacharya *et al.*, 2012). The preferred medication for multidrug-resistant Shigella infections since the late 1990s has been fluoroquinolones, such as ciprofloxacin, norfloxacin, and ofloxacin. However, certain studies have also documented the emergence of resistance to ceftriaxone in Shigella (Bhattacharya *et al.*, 2012). The treatment of shigellosis has become more difficult and has fewer therapeutic options due to the increasing level of antibiotic resistance of Shigella.

Many mathematical models have been proposed to study the dynamics of shigellosis using various control strategies among are; (Chen *et al.*, 2024) developed an age-specific susceptible-exposed-infectious / asymptomatic-recovered individuals (SEIAR) model in shigellosis. The data of the model were obtained from the Hubei province Center for Disease Control and Prevention from 2005-2017. The model divided the population into four age groups ($< 5 years$, 6-24years, 25-59 years and $> 60 years$). The model assesses the transmission of the disease in different age groups by the effective reproduction number (both for the infectivity and susceptibility). The result of the transmission of shigellosis shows that children under 5 years of age are transmitted the disease between themselves in most cases, while normal transmission occurs in the adult age (25-59) years of age. Intervention to terminate disease transmission should be taken at the age of groups. (Edward *et al.,* 2020) developed a mathematical model for shigellosis with carriers and multiple control measures were carried in the model. The effective reproduction number is computed and used to analyze the local stability analysis of the model. Global stability analysis has been proved by the comparison theorem. By lyapunov function the endemic equilibrium point is globally asymptotically stable at $R_e > 1$. The model find the parameter with higher impact in the transmission dynamics of shigellosis with both direct and indirect transmission (Direct transmission has higher infections than indirect transmission). Sensitivity analysis and numerical simulation are both performed in the model. The possibilities of eliminating the shigellosis is depend on the number of control intervention. (Bonyah*et al.*, 2018) developed a mathematical model on the effect of saturation treatment in the dynamic of spread of shigellosis. The compartmental model consist of susceptible-infected-treatment-recovered individuals (SITR). The disease free and endemic equilibriums are all exist in the model. Local and global stability are obtained from the reproduction number obtained in the model. The impact of saturation treatment function on diarrhoea spread has been demonstrated by numerical simulation results. In the complete eradication of the diarrhoea pandemic, treatment effectiveness is a major factor. (Ojaswita *et al.*, 2014) developed a continuos mathematical model for shigellosis outbreak. The model divided the population into susceptible-infected-recovered individuals (SIR). The disease-free equilibrium state and basic reproduction number were computed from the model. The result demonstrates that shigellosis will be eliminated from the community or population as long as the basic reproduction number is kept extremely low. Conversely, if the basic reproduction number is greater than one, shigellosis is going to persist in the community or population at a higher rate. By implementing several strategies, such as raising public awareness of treatment and prevention, improving workplace hygiene, and establishing improved water treatment facilities, the basic reproduction number can be kept extremely low.

Reference to the studies mentioned above, in this study we developed a mathematical modeling and analysis of human to human shigellosis transmission to assess the impact of public enlightenment. motivated by the work of (Edward *et al.,* 2020) by neglecting environmental contributions (i.e indirect transmission), incorporating only direct transmission which is human to human transmission, in addition to our work we consider aware and unaware susceptible and isolation of infected individuals which is indeed important in the dynamics of shigellosis.

SHIGELLOSIS EPIDEMIC MODEL

We developed a mathematical model to study the spread of shigellosis in a human population at time $t > 0$, denoted by $N(t)$, and subdivided into seven compartments: Susceptible unaware individuals $S_u(t)$ (those who are healthy but can acquire the infection)

with the infection rate λ; susceptible aware individuals $S_a(t)$ (those who aware with shigella bacteria) but some can acquire the infection at a slower rate $\alpha\lambda$, Exposed $E(t)$ (those who are from $S_u(t)$ and $S_a(t)$ that makes effective contact with carrier and infected individuals). Carrier individuals $C(t)$ (those who are infected with no clinical symptoms and can still spread the disease). Infected individuals $I(t)$, (those who are infected with shigellosis). Isolated $H(t)$, (those who are isolated from $I(t)$). Recovered individuals $R(t)$ (those who are recovered from shigellosis). Its assume that the susceptible unaware human are recruited into the population at a constant rate π. The susceptible individuals are aware at a constant rate σ, exposed individuals may either progress to infected or carrier classes at the rate θ. A proportion ω of the exposed individuals may progress to the infected compartment, while $(1-\omega)$ to the carrier compartment. The carrier screened at rate γ_1. Infected individuals are isolated at the rate γ_2. The shigellosis-induced mortality rates assumed to be only in infected and Isolated compartments at rate δ_1 and δ_2, while the whole compartments are decreases by natural death rate μ.

The flow diagram of the model is shown in figure 1 and the variables and interpretations of parameters are summarized in table 1.

$$\begin{aligned}
\frac{dS_u}{dt} &= (1-p)\pi + \phi R - (\lambda+\sigma+\mu)S_u,\\
\frac{dS_a}{dt} &= p\pi + \sigma S_u - (\alpha\lambda+\mu)S_a,\\
\frac{dE}{dt} &= \lambda S_u + \alpha\lambda S_a - (\theta+\mu)E,\\
\frac{dC}{dt} &= (1-\omega)\theta E - (\gamma_1+\mu)C,\\
\frac{dI}{dt} &= \omega\theta E + \gamma_1 q C - (\delta_1+\gamma_2+\mu)I,\\
\frac{dH}{dt} &= \gamma_2 I - (\mu+\gamma_3+\delta_2)H,\\
\frac{dR}{dt} &= \gamma_3 H + (1-q)\gamma_1 C - (\phi+\mu)R,
\end{aligned} \tag{1}$$

Where

$$\lambda = \frac{\beta(I+\xi C)}{N}$$

THEORETICAL ANALYSIS OF THE MODEL

Boundedness and Positivity of Solution

Any of the model's parameters and state variables are non-negative for any $t \geq 0$. The theoretical framework deals with the human being population. It is now possible to demonstrate that for any $t \geq 0$, all of the state variables in the model 1 are non-negative.

Theorem 1 *The solution of the model (1) is feasible for all $t > 0$ if the solution starts and remains in the positive invariant set Ω defined by:*

$$\Omega = \left\{ (S_u(0), S_a(0), E(0), I(0), \right.$$
$$\left. C(0), H(0), R(0) \in R_+^7 : N \leq \frac{\pi}{\mu} \right\}. \tag{2}$$

Proof 1 *It is sufficient to show that the model (1) solution enters and remains in the region Ω. Let $S_u(0), S_a(0), E(0), I(0), C(0), H(0), R(0)$ are all positive. We prove by induction, suppose $S_u(0)$ and $S_a(0)$ are negatives, then there exists a time $t_1 > 0$, such that $S_u(t) > 0, S_a(t) > 0$ and $R(t) > 0$ for $t \in [0, t_1)$ and $S_u(t_1) = S_a(t_1) = R(t_1) = 0$. Now, the infection classes in (1) satisfied the following,*

$$\begin{aligned}
\frac{dE(t)}{dt} &\geq -(\theta+\mu)E(t), \quad \text{for } t \in [0,t_1),\\
\frac{dC(t)}{dt} &\geq -(\gamma_1+\mu)C(t), \quad \text{for } t \in [0,t_1),\\
\frac{dI(t)}{dt} &\geq -(\mu+\gamma_2+\delta_1)I(t) \quad \text{for } t \in [0,t_1),\\
\frac{dH(t)}{dt} &\geq -(\mu+\gamma_3+\delta_2)H(t) \quad \text{for } t \in [0,t_1).
\end{aligned} \tag{3}$$

It follows that $E(0) > 0, C(0) > 0, I(0) > 0, H(0) > 0$ and $R(0) > 0$ for $t \in [0, t_1)$. Thus, from the first equation of the system (1), we have

$$\frac{dS_u(t)}{dt} \geq -(\lambda+\sigma+\mu)S_u(t) \quad \text{for } t \in [0,t_1).$$

$$\frac{dS_a(t)}{dt} \geq -(\alpha\lambda+\mu)S_a(t) \quad \text{for } t \in [0,t_1).$$

So, we see that $S_u(0)$, $S_a(0)$ and $R(0)$ are both positive, this contradict the assumption that $S_u(t_1) = S_a(t_1) = R(t_1) = 0$. Hence $S_u(t)$, $S_a(t)$ and $R(t)$ are positive. Also, the system of equation (1) can be written as follow,

$$\frac{dX(t)}{dt} = \mathcal{N}Y(t) + \mathcal{M}(t), \tag{4}$$

with

$$\begin{aligned}
Y(t) &= \begin{pmatrix} E, & C & I, & H, & R \end{pmatrix}^T,\\
\mathcal{N} &= \begin{pmatrix} -k_3 & 0 & 0 & 0 & 0\\ (1-\omega)\,\theta & -k_4 & 0 & 0 & 0\\ \omega\theta & \gamma_1 q & -k_5 & 0 & 0\\ 0 & 0 & \gamma_2 & -k_6 & 0\\ 0 & (1-q)\gamma_1 & 0 & \gamma_3 & -k_7 \end{pmatrix},\\
\mathcal{M}(t) &= \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \end{pmatrix}^T,
\end{aligned} \tag{5}$$

where, $k_3 = (\theta+\mu)$, $k_4 = (\gamma_1+\mu)$,$k_5 = (\mu+\gamma_2)$,$k_6 = (\mu+\gamma_3)$,$k_7 = (\mu+\phi)$. Clearly $\mathcal{N}$ is a Metzler matrix

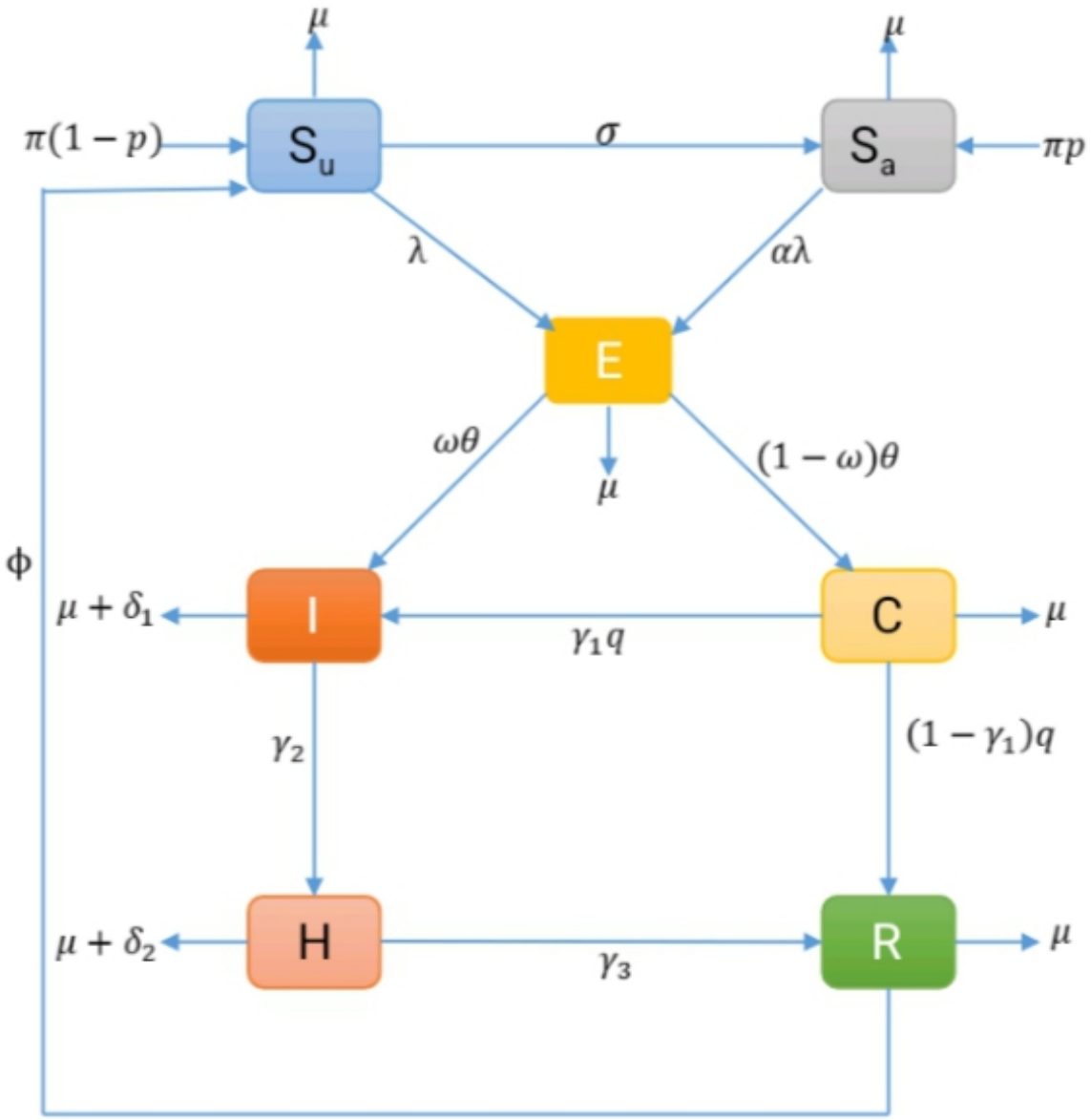

Figure 1: The flow diagram describing the model (1).

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for the fact that $S_u(t)$ and $S_a(t)$ are positive. This suggests that 4, the subsystem, is a monotone system. Therefore, under the flow of subsystem (4), R_+^7 is invariant.

Shigellosis-free Equilibrium

A state of equilibrium known as "shigellosis-free equilibrium" takes place when there is no infection in the society. By setting the equations for the right-hand side of the system 1 to zero, the shigellosis-free equilibrium can be obtained as follows:

$$\epsilon^0 = (S_u^0, S_a^0, E^0, C^0, I^0, H^0, R^0) = \left(\frac{(1-p)\,\pi}{\mu+\sigma}, \frac{\pi\,(\mu\,p+\sigma)}{\mu\,(\mu+\sigma)}, 0, 0, 0, 0, 0\right). \tag{6}$$

Basic Reproduction Number

When an infected person interacts with a fully susceptible population in the absence of vaccination and awareness, the number of new infections they cause is known as the basic reproduction number (represented by $R_0 = \rho(FV^{-1})$ in the model 1. Here, ρ represents the spectral radius of the next generation

Table 1: Interpretation of the state variables and parameters used in the model (1).

Variable	Description
N	Total population
S_u	Susceptible unaware individuals
S_a	Susceptible aware individuals
E	Exposed individuals
C	Carrier individuals
I	Infected individuals
H	Isolated individuals
R	Recovered individuals
Parameter	
π	Recruitment rate
μ	Natural mortality rate
σ	Awareness rate
θ	Progression rate of E to I or C
ω	Proportion of E that are carrier C
q, γ_3	Recovery rate of C and H
γ_2	Isolation rate
δ_1	Disease induced death rates of I
δ_2	Disease induced death rates of H
γ_1	Progression rate of C to I or R
ϕ	Immunity wining rate

matrix, FV^{-1}). The stability of the equilibrium is established using the next-generation matrix technique (Abubakar *et al.*,2025, Andrawus *et al.,* 2025 and Ibrahim *et al.,* 2025). The new infection terms are represented by the matrix F, while the existing transition terms are represented by the matrix V.

$$F = \begin{bmatrix} 0 & \frac{\beta\,\xi\,(S_a\,\alpha+S_u)}{N} & \frac{\beta\,(S_a\,\alpha+S_u)}{N} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{7}$$

and

$$V = \begin{bmatrix} k_3 & 0 & 0 & 0 \\ (\omega-1)\,\theta & k_4 & 0 & 0 \\ -\omega\,\theta & -\gamma_1\,q & k_5 & 0 \\ 0 & 0 & -\gamma_2 & k_6 \end{bmatrix} \tag{8}$$

$$V^{-1} = \begin{bmatrix} {k_3}^{-1} & 0 & 0 & 0 \\ -\frac{(\omega-1)\theta}{k_3\,k_4} & {k_4}^{-1} & 0 & 0 \\ -\frac{\theta\,(q(\omega-1)\gamma_1-k_4\,\omega)}{k_3\,k_4\,k_5} & \frac{\gamma_1\,q}{k_4\,k_5} & {k_5}^{-1} & 0 \\ -\frac{(q(\omega-1)\gamma_1-k_4\,\omega)\gamma_2\,\theta}{k_3\,k_4\,k_5\,k_6} & \frac{\gamma_2\,\gamma_1\,q}{k_4\,k_5\,k_6} & \frac{\gamma_2}{k_5\,k_6} & {k_6}^{-1} \end{bmatrix} \tag{9}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta\,(S_a\,\alpha+S_u)\theta\,(-\xi\,k_5\,\omega-\omega\,q\gamma_1+k_4\,\omega+\xi\,k_5+\gamma_1\,q)}{Nk_3\,k_4\,k_5} & \frac{\beta\,(S_a\,\alpha+S_u)(\xi\,k_5+\gamma_1\,q)}{Nk_4\,k_5} & \frac{\beta\,(S_a\,\alpha+S_u)}{Nk_5} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{10}$$

Equation (11) below gives the eigenvalues of the matrix in (10)

$$\begin{bmatrix} 0 \\ 0 \\ 0 \\ \frac{\beta\,(S_a\,\alpha+S_u)\theta\,(-\xi\,k_5\,\omega-\omega\,q\gamma_1+k_4\,\omega+\xi\,k_5+\gamma_1\,q)}{Nk_3\,k_4\,k_5} \end{bmatrix} \tag{11}$$

Where;

$$S_u = \frac{\pi\,(1-p)}{\mu+\sigma}, \qquad S_a = \frac{\pi(\mu p+\sigma)}{\mu(\mu+\sigma)} \tag{12}$$

The dominant eigenvalue is

$$\mathcal{R}_c = \frac{\beta\theta((1-p)\mu + \alpha(\mu p+\sigma))[(1-\omega)\xi k_5 + (1-\omega)\gamma_1 q + \omega k_4]}{k_3 k_4 k_5 \mu(\mu+\sigma)} \tag{13}$$

Epidemiological Interpretation of control reproduction number ($\mathcal{R}_c$) : Is the number of secondary cases produced by shigellosis infected individuals during the entire infection period in a population with presence of enlightenment and isolation.

The basic reproduction number can be obtained if the control parameters are zero $\sigma = \gamma_2 = 0$.

$$\mathcal{R}_0 = \frac{\beta\theta((1-p)+\alpha p)[(1-\omega)\xi(\mu+\delta_1) + (1-\omega)\gamma_1 q + \omega(\gamma_1+\mu)]}{\mu(\theta+\mu)(\gamma_1+\mu)(\mu+\delta_1)} \tag{14}$$

Epidemiological Interpretation of basic reproduction number ($\mathcal{R}_0$) : Is the number of secondary cases produced by shigellosis infected individuals during the entire period of infection in a population with absence of enlightenment and isolation.

Local Stability of The DFE

When a minor perturbation does not affect the equilibrium state of a system, then we say the system is locally asymptotically stable. Therefore, a locally asymptotically stable shegallosis-free equilibrium refers to the state by which a small number of infections will not lead to a larger outbreak. Mathematically, this condition is met if the real parts of all the linearized system's eigenvalues are negative negative. This applies to Theorem (2) below.

Theorem 2 *The shigellosis free equilibrium (SFE) ϵ^0, of the model (1), is locally-asymptotically stable (LAS) in Ω if $\mathcal{R}_e < 1$, and unstable if $\mathcal{R}_e > 1$.*

Proof 2 *The system (1) is linearized by computing the Jacobian matrix at the shigellosis-free equilibrium, as follows,*

$$J(\epsilon^0) = \begin{bmatrix} -k_1 & 0 & 0 & -\beta\,\xi\,L_1 & -\beta\,L_1 & 0 & 0 \\ \sigma & -\mu & 0 & -\beta\,\alpha\,\xi\,L_2 & -\beta\,\alpha\,L_2 & 0 & 0 \\ 0 & 0 & -k_3 & \beta\,\xi\,(\alpha\,L_2 + L_1) & \beta\,(\alpha\,L_2 + L_1) & 0 & 0 \\ 0 & 0 & (1-\omega)\,\theta & -k_4 & 0 & 0 & 0 \\ 0 & 0 & \theta\,\omega & \gamma_1 q & -k_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & -k_6 & 0 \\ 0 & 0 & 0 & (1-q)\,\gamma_1 & 0 & \gamma_3 & -k_7 \end{bmatrix} \tag{15}$$

where

$$L_1 = \frac{(1-p)\mu}{\mu+\sigma}, L_2 = \frac{(\mu p+\sigma)}{(\mu+\sigma)}$$

The matrix in equation (15) is reduced by row-echelon form as,

$$J(\epsilon^0) = \begin{bmatrix} d_{11} & 0 & 0 & d_{14} & d_{15} & 0 & 0 \\ 0 & d_{22} & 0 & \frac{d_{11}\,d_{24}-d_{14}\,d_{21}}{d_{11}} & \frac{d_{11}\,d_{25}-d_{15}\,d_{21}}{d_{11}} & 0 & 0 \\ 0 & 0 & d_{33} & d_{34} & d_{35} & 0 & 0 \\ 0 & 0 & 0 & \frac{d_{33}\,d_{44}-d_{34}\,d_{43}}{d_{33}} & -\frac{d_{43}\,d_{35}}{d_{33}} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{d_{33}\,d_{44}\,d_{55}-d_{34}\,d_{43}\,d_{55}+d_{35}\,d_{43}\,d_{54}-d_{35}\,d_{44}\,d_{53}}{d_{33}\,d_{44}-d_{34}\,d_{43}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & d_{66} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & d_{77} \end{bmatrix} \tag{16}$$

Where,

$d_{11} = -k_1,\ d_{13} = -\beta\xi L_1,\ d_{14} = -\beta L_2,\ d_{21} = \sigma,$

$d_{22} = -\mu,\ d_{24} = -\beta\alpha\xi L_2,\ d_{33} = -k_3,$
$d_{34} = \beta\xi(L_1+\alpha L_2),\ d_{35} = \beta(L_1+\alpha L_2),\ d_{43} = (1-\omega)\theta,$
$d_{44} = -k_4,\ d_{53} = \theta\omega,\ d_{54} = \gamma_1 q,$

$d_{55} = -k_5$, $d_{65} = \gamma_2$, $d_{66} = -k_6$, $d_{74} = (1-q)\gamma_1$, $d_{76}\gamma_3$, $d_{77} = -k_7$.

From (16) we obtained the eigenvalues as

$$\begin{bmatrix} d_{77} \\ d_{66} \\ d_{33} \\ \frac{d_{33}\,d_{44} - d_{34}\,d_{43}}{d_{33}} \\ \frac{d_{33}\,d_{44}\,d_{55} - d_{34}\,d_{43}\,d_{55} + d_{35}\,d_{43}\,d_{54} - d_{35}\,d_{44}\,d_{53}}{d_{33}\,d_{44} - d_{34}\,d_{43}} \\ d_{22} \\ d_{11} \end{bmatrix} \tag{17}$$

Obviously, $\lambda_1, \lambda_2, \lambda_3, \lambda_6$ and λ_7 are all negatives from (17). Now for the remaining eigenvalues after simplifications, we have

$$\iff \frac{-k_3k_4k_5 + \beta\theta\xi(L_1 + \alpha L_2)[(1-\omega)k_5 + (1-\omega)\gamma_1 q + \omega k_4]}{k_3k_4 - \beta\theta\xi(L_1 + \alpha L_2)} < 0, \tag{22}$$

$$\iff -k_3k_4k_5 + \beta\theta\xi(L_1 + \alpha L_2)[(1-\omega)k_5 + (1-\omega)\gamma_1 q + \omega k_4] < 0, \tag{23}$$

$$\iff \beta\theta\xi(L_1 + \alpha L_2)[(1-\omega)k_5 + (1-\omega)\gamma_1 q + \omega k_4] < k_3k_4k_5, \tag{24}$$

$$\iff \frac{\beta\theta\xi(L_1 + \alpha L_2)[(1-\omega)k_5 + (1-\omega)\gamma_1 q + \omega k_4]}{k_3k_4k_5} < 1, \tag{25}$$

after substituting L_1 and L_2 in (25) we have

$$\frac{\beta\theta((1-p)\mu + \alpha(\mu p + \sigma))[(1-\omega)\xi k_5 + (1-\omega)\gamma_1 q + \omega k_4]}{\mu k_3k_4k_5(\mu + \sigma)} = \mathcal{R}_c < 1. \tag{26}$$

This completes the proof if $\mathcal{R}_c < 1$, which indicates that all the eigenvalues are negative; if $\mathcal{R}_c > 1$, it indicates instability.

Global Stability of Disease-Free Equilibrium

In order to determine whether the interventions included in the model can effectively control shigellosis infection in a large number of infected individuals, it is essential to consider the global stability of a dynamical system, which is defined as its stability even under large perturbations. Consequently, we propose the following theorem to guarantee the stability of the system ((1)):

λ_4 is also negative.

$$\iff \frac{-k_3k_4 + \beta\theta\xi(1-\omega)(L_1 + \alpha L_2)}{k_3} < 0, \tag{18}$$

$$\iff -k_3k_4 + \beta\theta\xi(1-\omega)(L_1 + \alpha L_2) < 0, \tag{19}$$

$$\iff \beta\theta\xi(1-\omega)(L_1 + \alpha L_2) < k_3k_4, \tag{20}$$

$$\iff \frac{\beta\theta\xi(1-\omega)(L_1 + \alpha L_2)}{k_3k_4} < 1. \tag{21}$$

Also λ_5 is negative

Theorem 3 *The disease-free equilibrium ϵ^0 of the model (1) is globally-asymptotically stable in Ω if $\mathcal{R}_c < 1$ and unstable if $\mathcal{R}_c > 1$.*

Proof 3 *To prove the global asymptotic stability of the disease-free equilibrium, the two axioms $[N_1]$ and $[N_2]$ for $\mathcal{R}_c < 1$ must be satisfied (Castillo-Charez and Son, 2004). System (1) is separated into two subsystems, $X_1 = (S_u^0, S_a^0, R^0)$ and $X_2 = (E^0, C^0, I^0, H^0)$, to make analysis easier. $X_2 \in \mathcal{R}_+^4$ represents the infected classes, whereas $X_1 \in R_+^3$ represents the uninfected subpopulation.*

This partition allows the system (1) to be rewritten as two connected differential equations, one governing X_1 dynamics and the other governing X_2 dynamics.

$$\begin{aligned} \frac{dX_1}{dt} &= F(X_1, X_2), \\ \frac{dX_2}{dt} &= G(X_1, X_2) : N(X_1, 0) = 0. \end{aligned} \tag{27}$$

N_1***: global stability of*** X_1.

The disease-free equilibrium can now be represented in terms of the partitioned system as:

$\epsilon^0 = (X_1, \mathbf{0})$,

where the infected compartments are all set to zero.

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} (1-p)\pi + \phi R^0 - (\sigma + \mu)S_u^0 \\ p\pi + \sigma S_u^0 - \mu S_a^0 \\ -(\phi + \mu)R^0 \end{bmatrix}. \tag{28}$$

The solution of a linear ODE in (28) yields,

$$\begin{aligned}\frac{(1-p)\pi+\phi R^0}{(\sigma+\mu)}-\frac{(1-p)\pi+\phi R^0}{(\sigma+\mu)}e^{-(\sigma+\mu)t}+S_u^0(0)e^{-(\sigma+\mu)t}&=S_u^0(t),\\ \frac{p\pi+\sigma S_u^0}{\mu}-\frac{p\pi+\sigma S_u^0}{\mu}e^{-\mu t}+S_a^0(0)e^{-\mu t}&=S_a^0(t),\\ R^0(0)e^{-(\phi+\mu)t}&=R^0(t).\end{aligned} \tag{29}$$

It is evident from system (1) that as $t\to\infty$, the sum $S_u^0(t)+S_a^0(t)+R^0(t)$ approaches the total population $N^0(t)$, regardless of the individual values of $S_u^0(t)$, $S_a^0(t)$, and $R^0(t)$. Consequently, the uninfected subsystem converges to $X_1^*=(N^0,0)$, which is globally asymptotically stable.

N_2**:**$\tilde{N}(X_1,X_2)=AX_2-N(X_1,X_2)\geq 0$

$$A=\begin{pmatrix}-(\theta+\mu) & \frac{\beta S_u}{N}+\frac{\alpha\beta S_a}{N} & \frac{\beta\xi S_u}{N}+\frac{\alpha\beta\xi S_a}{N} & 0\\ (1-\omega\theta) & -(\gamma_1+\mu) & 0 & 0\\ \omega\theta & \gamma_1 q & -(\delta_1+\gamma_2+\mu) & 0\\ 0 & 0 & \gamma_2 & -(\delta_2+\gamma_3+\mu)\end{pmatrix}. \tag{30}$$

The matrix A qualifies as a Metzler matrix due to the non-negativity of its off-diagonal elements.

$$N(X_1,X_2)=\begin{pmatrix}\frac{\beta(C^0+\xi I^0)}{N^0}S_u^0+\frac{\beta\alpha(C^0+\xi I^0)}{N^0}S_a^0-(\theta+\mu)E^0\\ (1-\omega\theta)E^0-(\gamma_1+\mu)C^0\\ \omega\theta E^0+\gamma_1 qC^0-(\mu+\gamma_2+\delta_1)I^0\\ \gamma_2 I^0-(\mu+\gamma_3+\delta_2)H^0\end{pmatrix} \tag{31}$$

Then,

$$\tilde{N}(X_1,X_2)=AX_2-G(X_1,X_2)=\begin{bmatrix}0\\0\\0\\0\end{bmatrix}.$$

therefore $\tilde{N}(X_1,X_2)=0$.

Theorem (3) has important epidemiological implications: it indicates that maintaining the control reproduction number below unity ($\mathcal{R}_c<1$) is sufficient to eliminate shigellosis, regardless of the initial number of infected individuals.

Shigellosis Endemic Equilibrium Point

When $C\neq 0$ and $I\neq 0$ this implies that the shigellosis invades the population at . As such, setting the vector field of (1) to zero, we obtain the equilibrium point at endemic state, as:

$$\epsilon^*=(S_u^{**},S_a^{**}E^{**},C^{**},I^{**},H^{**},H^{**})$$

$$\begin{aligned}S_u^{**}&=-\frac{(p-1)\,\pi}{\lambda+\mu+\sigma}\\ S_a^{**}&=\frac{\pi\,((\mu+\lambda)\,p+\sigma)}{(\lambda+\mu+\sigma)\,(\alpha\,\lambda+\mu)}\\ E^{**}&=\frac{\pi\,\lambda\,((\alpha\,p-p+1)\,\mu+\alpha\,(\sigma+\lambda))}{k_3\,(\lambda+\mu+\sigma)\,(\alpha\,\lambda+\mu)}\\ C^{**}&=-\frac{\theta\,\pi\,\lambda\,(\omega-1)\,((\alpha\,p-p+1)\,\mu+\alpha\,(\sigma+\lambda))}{k_4\,k_3\,(\lambda+\mu+\sigma)\,(\alpha\,\lambda+\mu)}\\ I^{**}&=-\frac{\pi\,\theta\,(q\,(\omega-1)\,\gamma_1-k_4\,\omega)\,\lambda\,((\alpha\,p-p+1)\,\mu+\alpha\,(\sigma+\lambda))}{k_3\,k_4\,k_5\,(\alpha\,\lambda+\mu)\,(\lambda+\mu+\sigma)}\\ H^{**}&=-\frac{\pi\,\gamma_2\theta\,\lambda\,(q\,(\omega-1)\,\gamma_1-\omega\,k_4)\,((\alpha\,p-p+1)\,\mu+\alpha\,(\sigma+\lambda))}{k_3\,k_4\,k_5\,k_6\,(\alpha\,\lambda+\mu)\,(\lambda+\mu+\sigma)}\end{aligned} \tag{32}$$

The model does not include the R term, which refers to recovered individuals. This exclusion is reasonable because once individuals recover, they are no longer susceptible and hence do not contribute to the endemic dynamics. However, waning immunity could cause some recovered individuals to become susceptible again, returning to the susceptible population. While waning immunity is not directly incorporated into the

model, it is an important consideration when analyzing the disease dynamics. If immunity wanes, recovered individuals may become susceptible, potentially altering the dynamics of the disease.

Existence of Endemic Equilibrium

The existence of an endemic equilibrium was established using Descartes' Rule of Signs, which states that the number of real roots of a polynomial is equal to the number of sign changes in the polynomial. Additional details can be found in Andrawus et al. (2024) and Ibrahim et al. (2025). In the endemic state, the force of infection is given by:

$$\lambda^* = \frac{\beta(I^* + \xi C^*)}{N^*}, \tag{33}$$

where,

$$N^* = S_u^* + S_a^* + E^* + C^* + I^* + H^*. \tag{34}$$

Substituting (32) in equation (33), we have the following quadratic equation,

$$M_1 {\lambda^*}^2 + M_2 \lambda^* + M_3 = 0 \tag{35}$$

where,

$$\begin{aligned} M_1 &= \alpha\left(k_6\, k_4\, k_5 + \theta\ (1-\omega)\, k_6\, k_5 + \theta\left(q\,(1-\omega)\,\gamma_1 + k_4\,\omega\right) k_6 + \gamma_2\theta\left(q\,(1-\omega)\,\gamma_1 + k_4\,\omega\right)\right), \\ M_2 &= k_6\, k_4\, k_5 + \theta\ (\omega-1)\, k_6\, k_5 - \theta\left(q\,(\omega-1)\,\gamma_1 - k_4\,\omega\right) k_6 - \gamma_2\theta\left(q\,(\omega-1)\,\gamma_1 - k_4\,\omega\right) \\ &\quad (\alpha\sigma + (\alpha\, p - p + 1)\,\mu + (1-p)\, k_6\, k_5\, k_4\, k_3), \\ M_3 &= \mu k_3 k_4 k_5[1 - \mathcal{R}_c]. \end{aligned} \tag{36}$$

It is evident that $M_1 > 0$ since all the parameters are nonnegative and $0 < \omega < 1$. Hence, depending on the sign of M_2 and M_3 we claim the following theorem.

Theorem 4 *The endemic equilibrium point of the model (1) has a positive equilibrium whenever $\mathcal{R}_c > 1$.*
i. If $M_2 > 0$ and $M_3 > 0 \Longleftrightarrow R_0 \leq 1$. The quadratic equation (35) has no positive real root, implies the model has no positive equilibrium.
ii. If $M_2 > 0$ and $M_3 < 0 \Longleftrightarrow R_0 > 1$. The quadratic equation (35) has one positive real root, implies the model has unique positive equilibrium.
iii. If $M_2 < 0$ and $M < 0 \Longleftrightarrow R_0 > 1$. The quadratic equation (35) has one positive real root, implies the model has unique positive equilibrium.
iv If $M_2 < 0$ and $M_3 > 0 \Longleftrightarrow R_0 < 1$. The quadratic equation (35) has either two, one or no positive real root depend on $M_2^2 - 4M_1M_3$, implies the model has either two positive equilibria, unique positive equilibrium or no positive equilibrium.

For reference, the following theorem was developed using items (ii) and (iii) of theorem (4).

Theorem 5 *The system (1) has a unique positive endemic equilibrium if $\mathcal{R}_c > 1$.*

Global Stability of EE Point

Theorem 6 *If $\mathcal{R}_c > 1$, the endemic equilibrium ϵ^*, is globally asymptotically stable.*

Proof 4 *We construct a Lyapunov function*

$$\begin{aligned} F &= \left(S_u - S_u^{**} - S_u^{**} ln \frac{S_u}{S_u}^{**}\right) + \left(S_a - S_a^{**} - S_a^{**} ln \frac{S_a}{S_a}^{**}\right) + \left(E - E^{**} - E^{**} ln \frac{E}{E}^{**}\right) \\ &+ \frac{(\theta+\mu)}{\theta}\left(C - C^{**} - C^{**} ln \frac{C}{C}^{**}\right) + \frac{(\theta+\mu)(q+\mu)}{\theta q}\left(I - I^{**} - I^{**} ln \frac{I}{I}^{**}\right) \\ &+ \frac{(\theta+\mu)(q+\mu)(\gamma_2+\mu)}{\theta\gamma_2 q}\left(H - H^{**} - H^{**} ln \frac{H}{H}^{**}\right). \end{aligned} \tag{37}$$

When (37) is differentiated in relation to time, we obtain

$$\begin{aligned} \dot{F} &= \left(1 - \frac{S_u^{**}}{S_u}\right)\dot{S}_u + \left(1 - \frac{S_a^{**}}{S_a}\right)\dot{S}_a + \left(1 - \frac{E^{**}}{E}\right)\dot{E} + \frac{(\theta+\mu)}{\theta}\left(1 - \frac{C^{**}}{C}\right)\dot{C} \\ &\frac{(\theta+\mu)(q+\mu)}{\theta q}\left(1 - \frac{I^{**}}{I}\right)\dot{I} + \frac{(\theta+\mu)(q+\mu)(\gamma_2+\mu)}{\theta\gamma_2 q}\left(1 - \frac{H^{**}}{H}\right)\dot{H} \end{aligned} \tag{38}$$

with

$$N = \frac{\pi}{\mu} \tag{39}$$

As we alter the infection's force, we have

$$\bar{\lambda} = \bar{\beta}(I + \xi C) \tag{40}$$

where

$$\bar{\beta} = \beta\frac{\pi}{\mu} \tag{41}$$

When (1) is substituted with (38), we obtain

$$\begin{aligned}\dot{F} &= \left(1-\frac{S_u^{**}}{S_u}\right)((1-p)\pi+\phi R-(\lambda+\sigma+\mu)S_u)+\left(1-\frac{S_a^{**}}{S_a}\right)(p\pi+\sigma S_u-(\alpha\lambda+\mu)S_a)\\ &+\left(1-\frac{E^{**}}{E}\right)(\lambda S_u+\alpha\lambda S_a-(\theta+\mu)E)+\frac{(\theta+\mu)}{\theta}\left(1-\frac{C^{**}}{C}\right)((1-\omega)\theta E-(\gamma_1+\mu)C)\\ &+\frac{(\theta+\mu)(q+\mu)}{\theta q}\left(1-\frac{I^{**}}{I}\right)(\omega\theta E+\gamma_1 qC-(\delta_1+\gamma_2+\mu)I)\\ &+\frac{(\theta+\mu)(q+\mu)(\gamma_2+\mu)}{\theta\gamma_2 q}\left(1-\frac{H^{**}}{H}\right)(\gamma_2 I-(\mu+\gamma_3+\delta_2)H)\end{aligned} \tag{42}$$

With relationships

$$\begin{aligned}(1-p)\pi &= (\lambda^{**}+\mu)S_u^{**},\\ p\pi &= (\alpha\lambda^{**}+\mu)S_a^{**},\\ \lambda S_u^{**}+\alpha\lambda^{**}S_a^{**} &= (\theta+\mu)E^{**},\\ \theta E^{**} &= (\gamma_1+\mu)C^{**},\\ \gamma_1 C^{**} &= (\gamma_2+\mu)I^{**},\\ \gamma_2 I^{**} &= (\mu+\gamma_3)H^{**}.\end{aligned} \tag{43}$$

We may simplify by changing the relations in (43) to (42).

$$\begin{aligned}\dot{F} &\le \mu S_u^{**}\left(2-\frac{S_u}{S_u^{**}}-\frac{S_u^{**}}{S_u}\right)+\mu S_a^{**}\left(2-\frac{S_a}{S_a^{**}}-\frac{S_a^{**}}{S_a}\right)\\ &+\lambda S_u^{**}\left(6-\frac{S_u^{**}}{S_u}-\frac{S_u E^{**}}{S_u^{**}E}-\frac{EC^{**}}{E^{**}C}-\frac{CI^{**}}{C^{**}I}-\frac{IH^{**}}{I^{**}H}-\frac{H}{H^{**}}\right)\\ &+\alpha\lambda S_a^{**}\left(6-\frac{S_a^{**}}{S_a}-\frac{S_a E^{**}}{S_a^{**}E}-\frac{EC^{**}}{E^{**}C}-\frac{CI^{**}}{C^{**}I}-\frac{IH^{**}}{I^{**}H}-\frac{H}{H^{**}}\right)\end{aligned} \tag{44}$$

Since the arithmetic mean is greater than the geometric mean we have:

$$\begin{aligned}&\left(2-\frac{S_u}{S_u^{**}}-\frac{S_u^{**}}{S_u}\right)\le 0,\left(2-\frac{S_a}{S_a^{**}}-\frac{S_a^{**}}{S_a}\right)\le 0,\\ &\left(6-\frac{S_u^{**}}{S_u}-\frac{S_u E^{**}}{S_u^{**}E}-\frac{EC^{**}}{E^{**}C}-\frac{CI^{**}}{C^{**}I}-\frac{IH^{**}}{I^{**}H}-\frac{H}{H^{**}}\right)\le 0,\\ &\left(6-\frac{S_a^{**}}{S_a}-\frac{S_a E^{**}}{S_a^{**}E}-\frac{EC^{**}}{E^{**}C}-\frac{CI^{**}}{C^{**}I}-\frac{IH^{**}}{I^{**}H}-\frac{H}{H^{**}}\right)\le 0.\end{aligned} \tag{45}$$

Thus, we have that $F^{'} \le 0$ for $\mathcal{R}_c > 1$ since the relevant variables in the equations for $S_u^{**}(t), S_a^{**}(t), E^{**}(t), C^{**}(t), I^{**}(t), H^{**}(t), R^{**}(t)$ are at endemic steady state it follows that these can be substituted into the equations for $S_u(t), S_a(t), E(t), C(t), I(t), H(t)$ and $R(t)$.Therefore, the result follows by applying Lasalle invariance principle (Laselle, 1976). Hence the endemic equilibrium (EE) ϵ^* of the model (1) is globally asymptotically stable (GAS).

Sensitivity analysis

Sensitivity analysis is employed to identify the most influential parameters that affect the reproduction number's value. Through this analysis, we gain a better understanding of the factors that impact the dynamics of disease transmission. Each parameter is classified based on its sign, with the most sensitive parameters being identified as those with negative signs, which contribute to a decrease in the reproduction number, and those with positive signs, which increase the reproduction number. The $\mathcal{R}_c$ normalized local sensitivity index with regard to the parameters is provided by,

$$\eta_{\Omega}^{\mathcal{R}_c} = \frac{\Omega}{\mathcal{R}_c}\times\frac{\partial\mathcal{R}_c}{\partial\Omega}, \tag{46}$$

Table 2: Forward Normalized Sensitivity Indices

Parameter	Elasticity Indices	Values of the Elasticity index
θ	$\eta_{\theta}^{\mathcal{R}_c}$	0.5305
ϕ	$\eta_{\phi}^{\mathcal{R}_c}$	0.2653
μ	$\eta_{\mu}^{\mathcal{R}_c}$	-0.4256
σ	$\eta_{\sigma}^{\mathcal{R}_c}$	0.3112
β	$\eta_{\beta}^{R_c}$	9.9893
ω	$\eta_{\omega}^{\mathcal{R}_c}$	0.3876
q	$\eta_{q}^{\mathcal{R}_c}$	-0.3912
γ_1	$\eta_{\gamma_1}^{\mathcal{R}_c}$	-0.5192
γ_2	$\eta_{\gamma}^{\mathcal{R}_c}$	-0.7406
γ_3	$\eta_{\gamma}^{R_c}$	-0.2318
α	$\eta_{\alpha}^{\mathcal{R}_c}$	0.2218
p	$\eta_{p}^{\mathcal{R}_c}$	0.6231
δ_1	$\eta_{\delta_1}^{\mathcal{R}_c}$	-0.3421
δ_2	$\eta_{\delta_2}^{\mathcal{R}_c}$	-0.3421

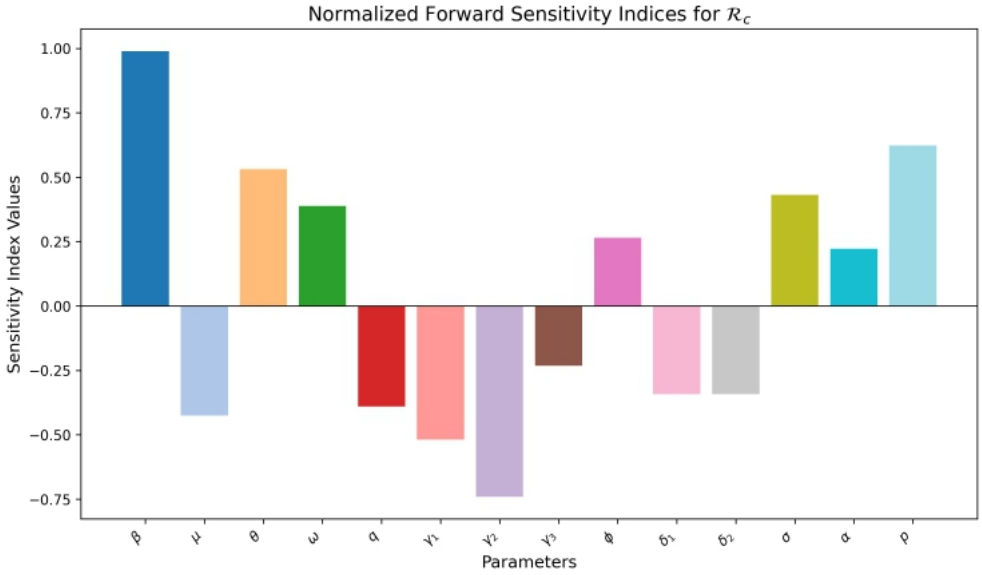

Figure 2: Bar chart graph showing PRCC of model (1) parameters.

NUMERICAL SIMULATIONS

Numerical simulations of model (1) offer a valuable tool for understanding the dynamics of shigellosis by visualizing the effects of various factors on disease transmission and control. Parameters used in the model are either derived from existing literature or assumed. Two control parameters, each varying from 0 to 1, provide the ability to observe the impact of different parameter values on the spread and control of the disease in a population. The simulations offer a dynamic representation of the complex interactions between factors influencing the disease dynamics, aiding in the development of effective control strategies.

DISCUSSION

Table ?? parameters and state variables were used to simulate the model's transmission dynamics of shigellosis. The way the state variables behave and the flow from one compartment to another are both investigated. Figure 3 described the behavior of unaware and aware susceptible with different levels of awareness as the rate increases the number of unaware drastically decreases, and as the rate increases the number of aware increases significantly. Figure 4 described the behavior of infected and Isolated individuals with different values of γ_2 as the rate increases the number of infected drastically decreases, and as the rate increases the number of isolated increases significantly. Figure 5 described the behavior of carrier and infected individuals respectively with different values of contact rate β. The graph raises

Table 3: Ranges and baseline values of parameters of model (1).

Parameter	Ranges (Baseline)	Unit	Reference
π	462	per years	
θ	0.35		Chen *et al.*, (2014)
μ	0.0012	per years	(Edward *et al.*, 2020)
ω	0.9	per years	Chen *et al.*, (2014)
q	0.0286	per years	(Edward *et al.*, 2020)
γ_1	0.56	per years	(Edward *et al.*, 2020)
γ_2	0.0011	per years	Chen *et al.*, (2014)
γ_3	0.021283	per years	Estimated
ϕ	0.000684	per years	(Edward *et al.*, 2020)
δ_1	0.000055	per years	(Edward *et al.*, 2020)
δ_2	0.059351	per years	Estimated
σ	0.6356	per years	Estimated
p	0.67	per years	(Edward *et al.*, 2020)
α	0.445	per years	(Edward *et al.*, 2020)

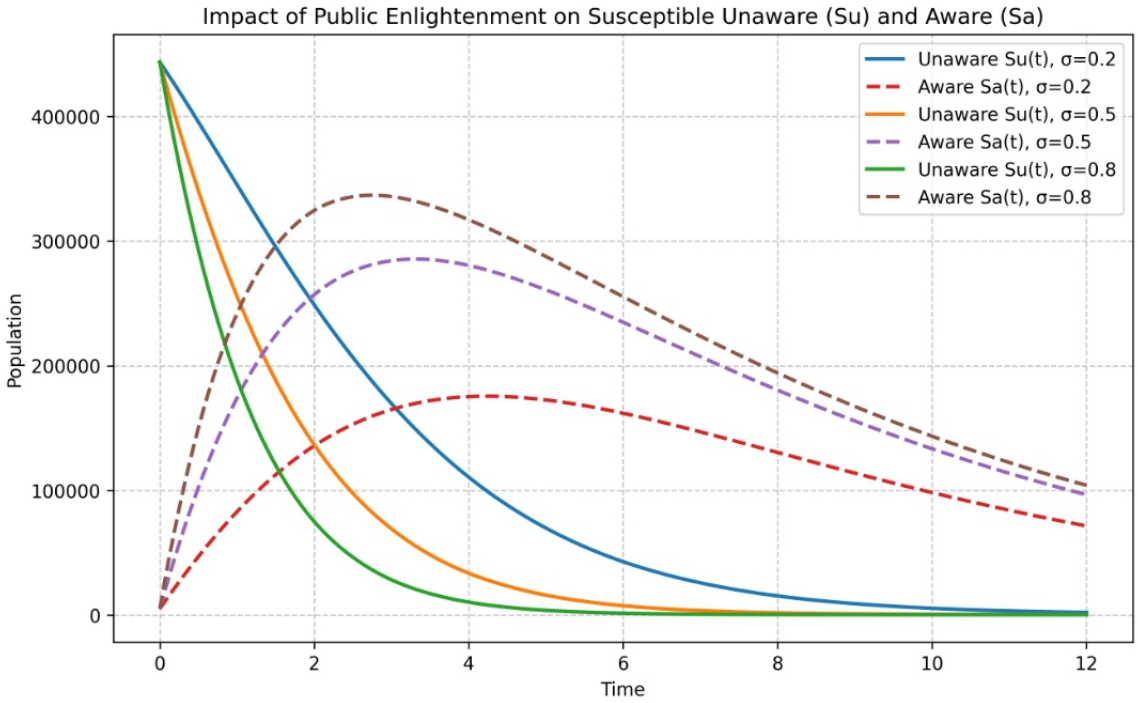

Figure 3: Pattern of susceptible aware and unaware individuals with different values of σ.

when the contact rate is greater. The more the contact rate, the more the number of infected individuals

CONCLUSION

In this paper we developed a deterministic mathematical model describing the transmission dynamics of human to human shigellosis. The model exhibit two equilibrium state, the disease free equilibrium and the endemic equilibrium. However, the disease free equilibrium state is shown to be both locally and globally asymptotically stable under certain conditions when the basic reproduction is less than unity ($\mathcal{R}_c < 1$). The endemic equilibrium on the contrary is found to be globally asymptotically stable when the basic reproduction number is greater than unity ($\mathcal{R}_c < 1$). The most sensitive parameters for the control of the spread of shigellosis are identified by forward sensitivity index method as shown in figure 2, and found contact rate β to be the most sensitive parameter. Further more, the numerical simulations carried out in figure 3 show the impact of public enlightenment. Similarly, figure 4 show the impact of isolation and figure 5 show how contact rate increasing the carrier and infected compartments respectively. Finally the result shows that to eradicate Shigellosis, there is need for minimizing the contact between the infected individuals and susceptible ones and also minimize the number of carrier individuals progressing

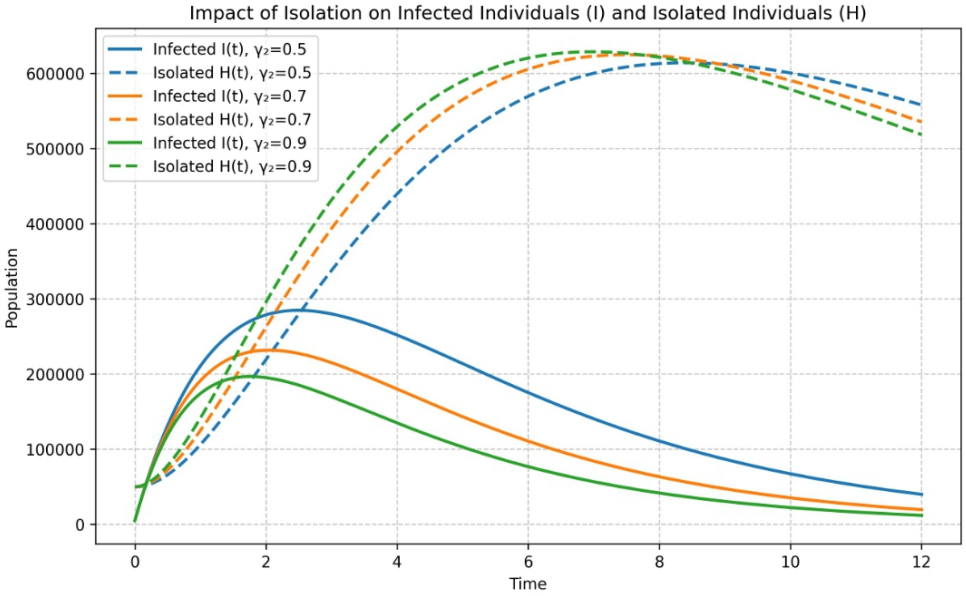

Figure 4: Pattern of infected and isolated individuals with different values of γ_2

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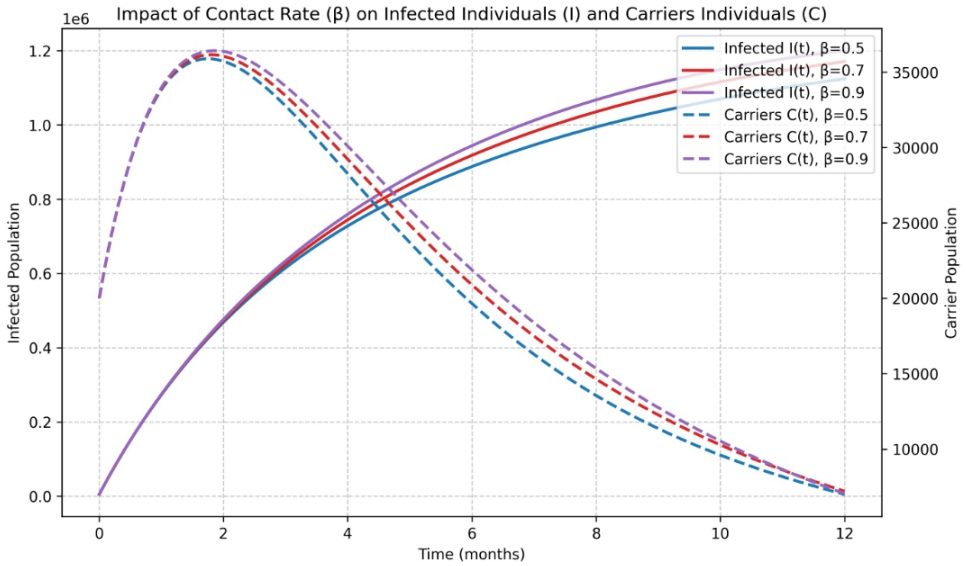

Figure 5: Pattern of carrier and infected individuals with different values of β

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into infected compartment when combined with public enlightenment and isolation of infected individuals.

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