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Effect of Environmental Precaution on the Transmission of Typhoid Fever: A Mathematical Modelling Approach

Kabir Oluwatobi Idowu[1], Latifat Morenikeji Erinle-Ibrahim[2], Joshua Oluwasegun Agbomola[3] and Sideeqoh Oluwaseun Olawale-Shosanya[4]

[1]Department of Mathematics, Purdue University, USA
[2]Department of Mathematics, Tai Solarin University of Education, Ijagun, Ogun State, Nigeria
[3]Department of Mathematics, Tulane University, New Orleans, LA, USA
[4]Department of Computer Science, Tai Solarin University of Education, Ijagun, Ogun State, Nigeria
Orcid number: [1]0000-0003-1345-4995
Orcid number: [3]0000-0002-7637-1591
Corresponding Author email: kidowu@purdue.edu

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Abstract

We proposed and analyzed a nonlinear mathematical model for typhoid fever and optimal control in a community with overpopulation. The model considered the effect of environmental precautions on the transmission of typhoid fever. We obtained the basic reproduction number denoting the epidemic indicator. We proved the local and global asymptomatic stability conditions for disease-free and endemic equilibrium. The model exhibits strategies for optimal control of typhoid fever, such as preventive strategies (environmental sanitation, proper hygiene, vaccination) and the treatment strategy. The numerical simulation of typhoid fever disease transmission and its maximum control summarized that prevention and treatment are the best methods for eradicating the disease in society. Since $R_0 = 0.1174$, which is less than one, it follows that the disease-free equilibrium is asymptomatically stable, and that the disease will always die out.

Keywords: Mathematical model; Basic reproduction number, Disease free equilibrium, hygiene, global stability

INTRODUCTION

Typhoid fever is an infectious disease caused by the bacteria *Salmonella Typhi* and *Salmonella Paratyphi* [1, 2]. The primary routes of transmission are through the consumption of food and water contaminated with the faeces and urine of infected individuals [3, 4]. Symptoms of typhoid fever include headache, stomach-ache, muscular pain, nausea, constipation, vomiting, diarrhea, loss of appetite, and fever. Enteric fever is a collective term that encompasses both typhoid and paratyphoid fever, with paratyphoid fever being clinically indistinguishable from typhoid fever. Individuals infected with *Salmonella Typhi* may suffer severe health consequences due to the infection. According to the World Health Organization (WHO) [5], typhoid fever affects between 11 and 20 million people annually, resulting in 128,000 to 161,000 deaths each year [6].

Mary Mallon, commonly known as Typhoid Mary, is the most well-known carrier of typhoid fever, being the first person in the United States identified as a carrier of the pathogen [7]. The bacillus suspected to cause typhoid fever was first described by Karl Joseph Ebert in 1880 [8]. Four years later, pathologist Georg Gaffky confirmed this link, naming the bacillus *Eberthella typhi*, now known as

Salmonella enterica [9]. The first effective vaccine for typhoid was developed by Almroth Edward Wright and introduced for military use in 1896, significantly improving the health of soldiers who were more likely to die from typhoid than in combat at that time [10].

A variety of vaccines have been recommended against typhoid infection, depending on individual age [11]. These vaccines include the injectable Typhoid Conjugate Vaccine (TCV), the injectable polysaccharide vaccine based on purified Vi antigen, and the oral live attenuated Ty21a vaccine [12]. While TCV is suitable for all ages, the other two are specifically designed for children. Additionally, TCV's enhanced immunological properties contribute to its popularity and preference among typhoid fever prevention vaccines [13]. Despite these advancements, the global burden of typhoid fever remains significant, particularly in regions with inadequate sanitation and limited access to clean water [14].

Researchers are dedicating significant efforts to developing alternative methods to prevent both the infection and transmission of typhoid fever, in addition to vaccine-based treatments [15]. This includes creating affordable preventive measures that can be widely adopted. In the field of mathematical epidemiology, numerous models have been developed to clarify the intricate dynamics of typhoid disease transmission. The aim of these models is to enhance the understanding of the disease transmission process and to identify appropriate measures that can mitigate its impact on public health [16]. By leveraging mathematical insights, the scientific community seeks to improve the methods used for preventing and controlling typhoid fever, contributing to the global effort to combat this ongoing health problem [17]. Examples of such models are reported in [18, 19, 20, 21].

Furthermore, the adoption of mathematical modeling has emerged as a crucial strategy in understanding and mitigating the spread of typhoid fever. These models assess the effectiveness of various preventative measures, such as improved sanitation and water purification, in reducing transmission [22]. By simulating the dynamics of disease spread, mathematical models provide valuable insights that aid policymakers and health professionals in devising effective control and prevention strategies [23]. This research aims to contribute to these efforts by developing a mathematical model that captures the dynamics of typhoid fever transmission, thereby offering a tool for evaluating the potential impact of various public health interventions [24]. The following authors have significantly model infectious diseases with control strategies [25-30]. Mathematical models are increasingly being utilized in various applications within the field of Mathematics [31, 32]

This study is situated within a broader context of ongoing research and innovation aimed at overcoming the multifaceted challenges posed by typhoid fever. The persistent threat of this disease, particularly in low- and middle-income countries, underscores the urgency of developing effective interventions. By leveraging the power of mathematical modeling in conjunction with cutting-edge advancements in vaccine development and comprehensive public health strategies, this research endeavors to deepen our understanding of the intricate dynamics of typhoid fever transmission. Through detailed analysis and robust simulations, we aim to uncover critical insights that can inform policy decisions and optimize resource allocation. Ultimately, this study aspires to make a significant contribution to the global effort to alleviate the burden of typhoid fever, improving health outcomes and enhancing the quality of life for affected populations around the world.

MATERIAL AND METHOD

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \varepsilon R - \beta S I_S - \mu S \\
\frac{dI_A}{dt} &= \beta S I_S - (\rho + \mu) I_A \\
\frac{dI_S}{dt} &= \rho I_A - (\gamma + \delta + \mu) I_S \\
\frac{dT}{dt} &= \gamma I_S - (\tau + \delta + \mu) T \\
\frac{dR}{dt} &= \tau T - (\varepsilon + \mu) R
\end{aligned}\right\} \quad (1)$$

Error! Reference source not found. is the formulated model for the study of dynamics of typhoid fever disease.

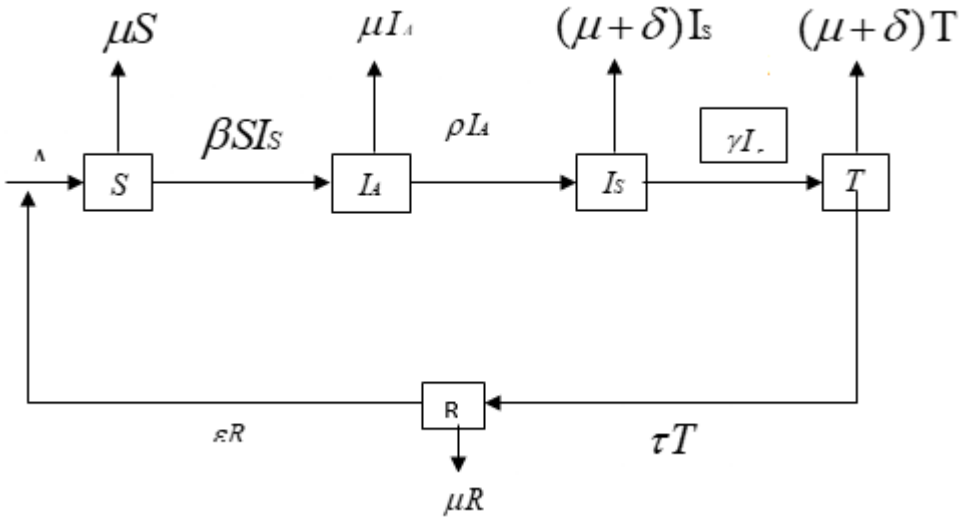

Figure 1 Schematic diagram for the transmission dynamics of Typhoid

Table 1 description of variables

Variable	Description of Variables
$S(t)$	Susceptible individuals / populations
$I_A(t)$	Asymptomatic infected individuals
$I_S(t)$	Symptomatic infected individuals
$T(t)$	Treated in factious individuals
$R(t)$	Recovered individuals

Table 2 description of parameters

Parameters	Descriptions
Λ	Recruitment rate in to the susceptible population.
ε	Rate of loss of immunity
β	Effective contact rate
μ	Natural death rate
ρ	Progression rate from $I_A(t)$ to $I_S(t)$
γ	Treatment rate for $I_A(t)$
δ	Disease-induced death
τ	Recovery rate for treated infectious individuals

Analysis of Mathematical Model

The Invariant Region

Lemma I: Let the feasible region of the model be D, Such that

$D = \left\{ \left(S, I_A, I_S, T, R\right) \in IR_+^5 : S + I_A + I_S + T + R \le \frac{\Lambda}{\mu} \right\}$ Is positively invariant and attracting.

Proof:

Consider the biologically feasible region D, Then the rate of change of the total population is Obtained by adding all the equation present in the system of (1) which gives: -

$$\frac{dN}{dt} = \Lambda + \varepsilon R - \beta S I_S - \mu S + \beta S I_S - \rho I_A - \mu I_A + \rho I_A - \gamma I_S - \delta I_S - \mu I_S + \gamma I_S - \tau T - \delta T - \mu T + \tau T - \varepsilon R - \mu R \quad (2)$$

$$\frac{dN}{dt} = \Lambda - \mu S - \mu I_A - \mu I_S - \mu T - \mu R - \delta I_S - \delta T \quad (3)$$

$$\frac{dN}{dt} = \Lambda - \mu\left(\delta + I_A + I_S + T + R\right) - \delta I_S - \delta T \quad (4)$$

Since $N = S + I_A + I_S + T + R$ and $\delta I_S = \delta T = 0$

Then, **Error! Reference source not found.** becomes: -

$$\frac{dN}{dt} = \Lambda - \mu N \quad (5)$$

Then, in the absence of mortality rate due to Typhoid, **Error! Reference source not found.** becomes:

$$\frac{dN}{dt} \le \Lambda - \mu N \quad (6)$$

Now, following the standard technique (method of integrating factor), **Error! Reference source not found.** is further Simplified as follows:

$$\frac{dN}{dt} = \Lambda - \mu N \tag{7}$$

$$N \le e^{-\mu t}\left(\frac{\Lambda e^{\mu t}}{\mu} + c\right) \tag{8}$$

$$N(t) \le N(0)ce^{-\mu t} + \frac{\Lambda}{\mu} \quad ; \quad N \le \frac{\Lambda}{\mu}$$

Hence, for all $t > 0,$ the solutions of the model with the initial conditions in the region D will remain in the region a where being epidemiologically and modeling well posed. Therefore, the biologically feasible region D is positively – invariant.

Positivity of Solution

For the typhoid model **Error! Reference source not found.** to be epidemiologically meaningful and mathematically well posed, it is worthwhile to prove that all the solutions with the non-negative for all time $t > 0$.

Theorem 1

Solution of system (1) given by the set $\{S, I_A, I_S, T, R\}$, with non-negative initial conditions $(S(0), I_A(0), I_S(0), T(0), R(0))$ remain non-negative for time $t > 0.$

Proof:-

From the first equation of the model:

$$\frac{dS}{dt} = \Lambda + \varepsilon R - \beta S I_S - \mu S$$

$$\frac{dS}{dt} + \beta S I_S + \mu S \ge 0$$

$$S(t) > S(0)\exp\left\{-\int_0^t \left[\beta I_S(\varphi)d\varphi + \mu t\right]\right\} > 0; \forall t > 0 \tag{9}$$

Using the same method for **Error! Reference source not found.**, we have

$$\left.\begin{aligned} I_A(t) &> I_A(0)\exp\left[-(\rho+\mu)t\right] > 0; \forall t > 0 \\ I_S(t) &> I_S(0)\exp\left\{(\gamma+\delta+\mu)t\right\} > 0; \forall t > 0 \\ T(t) &> T(0)\exp\left\{-(\tau+\delta+\mu)t\right\} > 0; \forall t > 0 \\ R(t) &> R(0)\exp\left\{-(\varepsilon+\mu)t\right\} > 0; \forall t > 0 \end{aligned}\right\} \tag{10}$$

Existence and Uniqueness of Solution
Statement of the theorem:

Consider the system of equations below:

$$\left.\begin{aligned} x_1^1 &= f_1(x_1, x_2, ..., x_n, t); x_1(t_o) = (x_1)_0 \\ x_2^1 &= f_2(x_1, x_2, ..., x_n, t); x_2(t_0) = (x_2)_0 \\ x_n^1 &= f_n(x_1, x_2, ...x_n, t); x_2(t_0) = (x_2)_0 \end{aligned}\right\} \quad (11)$$

The system **Error! Reference source not found.** can be written in compact form as:

$$x^1 = f(t, x); x(t_0) = x_0$$

Theorem 2: (Derrick and Grossman, 1976) Given $\frac{dx}{dt} = f(t, x); x(t_0) = x_0$

Let D denote the region, such that: $D = \{(x, t) : |t - t_0| \le a, \|x - x_0\| \le b\}$ where $x = (x_1, x_2, ..., x_n)_0; x_0[(x_1)_0, (x_2)_0, ..., (x_n)_0]$ and suppose that $f(t, x)$ satisfies Lipschitz condition

$$\|f(t, x_1) - f(t, x_2)\| \le \|x_1 - x_2\| \quad (12)$$

Whenever the pairs (t, x_1) and (t, x_2) belongs to D, where K is positive constant. Then, there exists a constant $S > 0$ such that, there exist is a unique continuous vector solution $\underline{X}(t)$ of the solution of the equation (12) in the interval $|t - t_0| \le S$
It is of great importance to note that the condition in equation (3.28) is satisfied by the
Requirement that

$$\frac{\partial f_i}{\partial x_j}, i, j = 1,2,...n \text{ be continuous and bounded in } D$$

Theorem 3:
Let:

$$f_i = f_i(S, I_A, I_s, T, R), \text{where } i = 1,2,..., n, \text{such that}$$

$$f_1 = \frac{dS}{dt} = \Lambda + \varepsilon R - \beta S I_S - \mu S, S(t_0) = S_0$$
$$f_2 = \frac{dI_A}{dt} = \beta S I_S - (\rho + \mu) I_A, I_A(t_0) = I_{A0}$$
$$f_3 = \frac{dI_S}{dt} = \rho I_A - (\gamma + \delta + \mu) I_S, I_S(t_0) = I_{S0} \quad (13)$$
$$f_4 = \frac{dT}{dt} = \gamma I_S - (\tau + \delta + \mu) T, T(t_0) = T_0$$
$$f_5 = \frac{dR}{dt} = \tau T - (\varepsilon + \mu) R, R(t_0) = R_0$$

Now Let

$$D = \{(S, I_A, I_S, T, R); |s - s_0| \le a, |I_A - I_{A0}| \le b, |I_S - I_{S0}| \le c, |T - T_0| \le d, |R - R_0| \le e\}$$

Then, the system **Error! Reference source not found.** has a unique solution in D. Thus, we have the following proofs. Following Derrick and Grossman (1976), There exists a unique solution if the partial derivatives:

$$\frac{\partial f_i}{\partial x_j}|(x_1, x_2, ..., x_n) = 0| \text{ and } \frac{\partial f_i}{\partial x_j}|(x_1, x_2, ..., x_n)| < \infty, i, j = 1,2,..., n \in D$$

Now, for the first compartment of the model, when $f_1 = \frac{ds}{dt}$

Find the partial derivative of f_1 with respect t_0.

$f_i = f_1(S, I_A, I_S, T, R)$ respectively

Then the following results are obtained:

$$f_i = \frac{dS}{dt} = \Lambda + \varepsilon R - \beta S I_S - \mu S$$
$$f_1 = \frac{dS}{dt} = \Lambda + \varepsilon R - (\beta I_S + \mu) S$$
$$\frac{\partial f_1}{\partial S}|(0,0,0,0,0)| = -(\beta I_S + \mu) \Rightarrow |-(\beta I_S + \mu)| < \infty$$
$$\frac{\partial f_1}{\partial I_A}|(0,0,0,0,0)| = 0 \Rightarrow |0| < \infty \quad (14)$$
$$\frac{\partial f_1}{\partial I_S}|(0,0,0,0,0)| = -\beta S \Rightarrow |-\beta S| < \infty$$
$$\frac{\partial f_1}{\partial T}|(0,0,0,0,0)| = 0 \Rightarrow |0| < \infty$$
$$\frac{\partial f_1}{\partial T}|(0,0,0,0,0)| = \varepsilon \Rightarrow |\varepsilon| < \infty$$

Therefore $\left|\frac{\partial f_1}{\partial xj}\right|$, where $I, j = 1,2,...,5$ and $x = S, I_A, I_S, T, R,$ are continuous and bounded. Hence, the system of equation (3.29) has a unique solution and so, the model is mathematically and epidemiologically well posed.

Endemic Equilibrium Point (EEP)

Let the endemic equilibrium point of typhoid present equilibrium of the model **1** denoted by $\varepsilon_0{}^*$ when $\varepsilon_0{}^* = \left(S^*, I_A{}^*, I_S{}^*, T^*, R^*\right)$ are the EEP.

Then the model **1** becomes

$$\left.\begin{aligned}
\frac{dS^*}{dt} &= \Lambda + \varepsilon R^* - \beta S^* I_S^* - \mu S^* \\
\frac{dI_A^*}{dt} &= \beta S^* I_S^* - (\rho + \mu) I_A^* \\
\frac{dI_S^*}{dt} &= \rho I_A^* - (\gamma + \delta + \mu) I_S^* \\
\frac{dT^*}{dt} &= \gamma I_A^* - (\tau + \delta + \mu) T^* \\
\frac{dR^*}{dt} &= \tau T^* - (\varepsilon + \mu) R^*
\end{aligned}\right\}$$

$(\rho + \mu) = K_1, (\gamma + \delta + \mu) = K_2, (\tau + \delta + \mu) = K_3$ and $(\varepsilon + \mu) = K_4$ then, **Error! Reference source not found.** becomes:

$$\Lambda + \varepsilon R^* - \beta S^* I_S^* - \mu S^* = 0$$

$$\beta S^* I_S^* - K_1 I_A^* = 0$$

$$\rho I_A^* - K_2 I_S^* = 0$$

$$\gamma I_S^* - K_3 T^* = 0$$

$$\tau T^* - K_4 R^* = 0$$

Where, $\frac{dS^*}{dt} = \frac{dI_A^*}{dt} = \frac{dI_S^*}{dt} = \frac{dT^*}{dt} = \frac{dR^*}{dt} = 0$

Then the endemic equilibrium denoted by $\varepsilon_0^* = \left(S^*, I_A^*, I_S^*, T^*, R^*\right)$ is obtained as;

$$\begin{aligned}
S^* &= \frac{\Lambda K_1 K_2 K_3 K_4}{K_1 K_2 K_3 K_4 (\lambda^* + \mu) - \varepsilon\tau\gamma\rho\lambda^*} \\
I_S^* &= \frac{\Lambda\rho K_3 K_4 \lambda^*}{K_1 K_2 K_3 K_4 (\lambda^* + \mu) - \varepsilon\tau\gamma\rho\lambda^*} \\
I_A^* &= \frac{\Lambda K_2 K_3 K_4 \lambda^*}{K_1 K_2 K_3 K_4 (\lambda^* + \mu) - \varepsilon\tau\gamma\rho\lambda^*} \\
T^* &= \frac{\Lambda\gamma\rho K_4 \lambda^*}{K_1 K_2 K_3 K_4 (\lambda^* + \mu) - \varepsilon\tau\gamma\rho\lambda^*} \\
R^* &= \frac{\Lambda\tau\gamma\rho\lambda^*}{K_1 K_2 K_3 K_4 (\lambda^* + \mu) - \varepsilon\tau\gamma\rho\lambda^*}
\end{aligned} \tag{15}$$

Determination of Basic Reproduction Number (R_0)

Consider the infection related compartment in the typhoid model. Using the next generation matrix method (NGM),

where:

ρ = Spectral radius

F = New infection terms A

V = Transmission of infection

Then, $R_0 = \rho FV^{-1}$

$\therefore R_0 = Max\lambda_i \; of \; FV^{-1}$

$$J = \begin{pmatrix} \beta S I_S \\ 0 \end{pmatrix}, V = \begin{pmatrix} -K_1 I_A \\ \rho I_A - K_2 I_S \end{pmatrix}$$

$$F = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix} \Rightarrow F\big|_{\varepsilon 0} = \begin{pmatrix} 0 & \frac{\beta\Lambda}{\mu} \\ 0 & 0 \end{pmatrix} \quad V = \begin{pmatrix} K_1 & 0 \\ \rho & K_2 \end{pmatrix} \quad ;$$

$$V^{-1} = \begin{pmatrix} \frac{1}{K_1} & 0 \\ \frac{\rho}{K_1 K_2} & \frac{1}{K_2} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{\rho\beta\Lambda}{\mu K_1 K_2} & \frac{\beta\Lambda}{\mu K_2} \\ 0 & 0 \end{pmatrix}$$

And

$$|FV^{-1} - \lambda I| = \left| \begin{pmatrix} \frac{\rho\beta\Lambda}{\mu K_1 K_2} - \lambda & \frac{\beta\Lambda}{\mu K_2} \\ 0 & -\lambda \end{pmatrix} \right|$$

$$\lambda = 0 \text{ and } \lambda = \frac{\rho\beta\Lambda}{\mu K_1 K_2}$$

Since $R_0 = Max\lambda_i \; of \; FV^{-1}$, it follows that

$$R_0 = \frac{\rho\beta\Lambda}{\mu(\rho+\mu)(\gamma+\delta+\mu)} \tag{16}$$

Local Stability of Disease-Free Equilibrium

Theorem 4:- The disease free equilibrium of the system **Error! Reference source not found.** is locally asymptotically stable (LAS) if the threshold quantity $R_0 < 1$ and unstable if otherwise $(R_0 > 1)$.

Then, the Jacobian matrix defined for the system is as follows: -

$$J=\begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I_A} & \frac{\partial f_1}{\partial I_S} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I_A} & \frac{\partial f_2}{\partial I_S} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I_A} & \frac{\partial f_3}{\partial I_S} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial I_A} & \frac{\partial f_4}{\partial I_S} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial I_A} & \frac{\partial f_5}{\partial I_S} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} \end{pmatrix}$$

following Descartes Rule of signs, the Polynomial of order five Obtained in **Error! Reference source not found.** has no positive roots of

(i) $\mu+K_1+K_2+K_3+K_4>0$

(ii) $\mu(K_1+K_2+K_3+K_4)+K_1(K_2+K_3+K_4)+K_2(K_3+K_4)+K_3K_4>0$

(iii) $\mu K_1(K_2+K_3+K_4)+\mu K_2(K_3+K_4)+\mu K_3K_4+K_1K_2(K_3+K_4)+K_3K_4(K_1+K_2)>\frac{\rho\beta\Lambda}{\mu}$

(iv) $\mu K_1(K_2K_3+K_2K_4+K_3K_4)+K_2K_3K_4(\mu+K_1)>\frac{\rho\beta\Lambda}{\mu}(K_3+K_4)$

(v) $\mu K_1K_2K_3K_4>\frac{\rho\beta\Lambda K_3K_4}{\mu}$

Hence, the disease-free equilibrium is locally asymptotically stable, if the associated basic reproduction Number (R_0) is less then unity $(R_0<1)$ and unstable if otherwise when $(R_0>1)$

Sensitivity Analysis of the Model

To see the effects or behavior of each parameter present in the basic reproduction number, it is worthwhile to investigate how sensitive the parameters are in the basic reproduction number,

Following the normalized forward sensitivity index, the sensitivity analysis of the model is carried out as follows.

$$\gamma_{\rho}^{R_0}=\frac{\partial R_0}{\partial\rho}\times\frac{\rho}{R_0}$$

Then, the sensitivity indices of R_0 to each of the parameters are calculated or determined.

Table 3 list of parameters used for the numerical analysis.

PARAMETERS	VALUES
Λ	0.75
ε	0.0925
ρ	0.01503
μ	0.015
τ	0.35
γ	0.0625
δ	0.125
β	0.00095

$$R_0 = \frac{\rho\beta\Lambda}{\mu(\rho+\mu)(\gamma+\delta+\mu)}$$

$$R_0 = \frac{0.01503\times 0.00095\times 0.75}{0.015[(0.01503+0.015)\times(0.0626+0.125+0.015)]}$$

$$R_0 = \frac{0.0000107089}{0.015[(0.03003)\times(0.2025)]}$$

$$R_0 = \frac{0.0000107089}{0.015[0.006081075]}$$

$$R_0 = \frac{0.0000107089}{0.0000912161}$$

$$R_0 = 0.1174014237$$

Table 4 Sensitivity indices of the models parameters with R_0

Parameter	Sensitivity Index
Λ	1.0
ρ	0.997
μ	-1.0
τ	-0.061
γ	-0.021
δ	-0.043
β	1.0

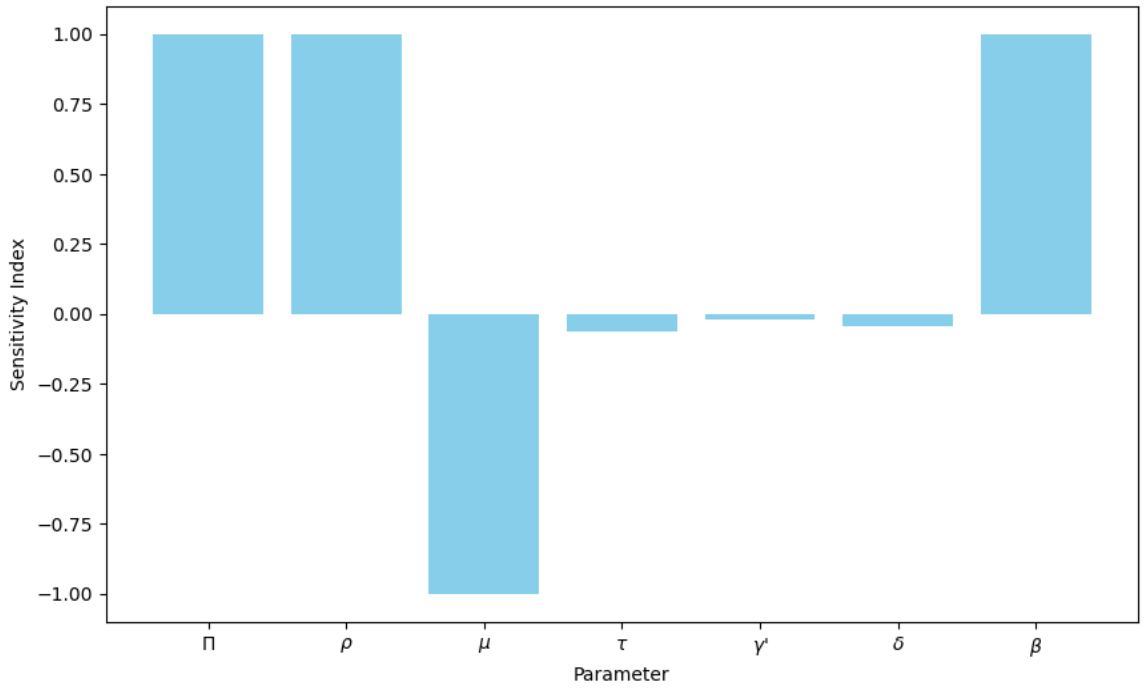

Figure 2a: Sensitivity analysis of the model system

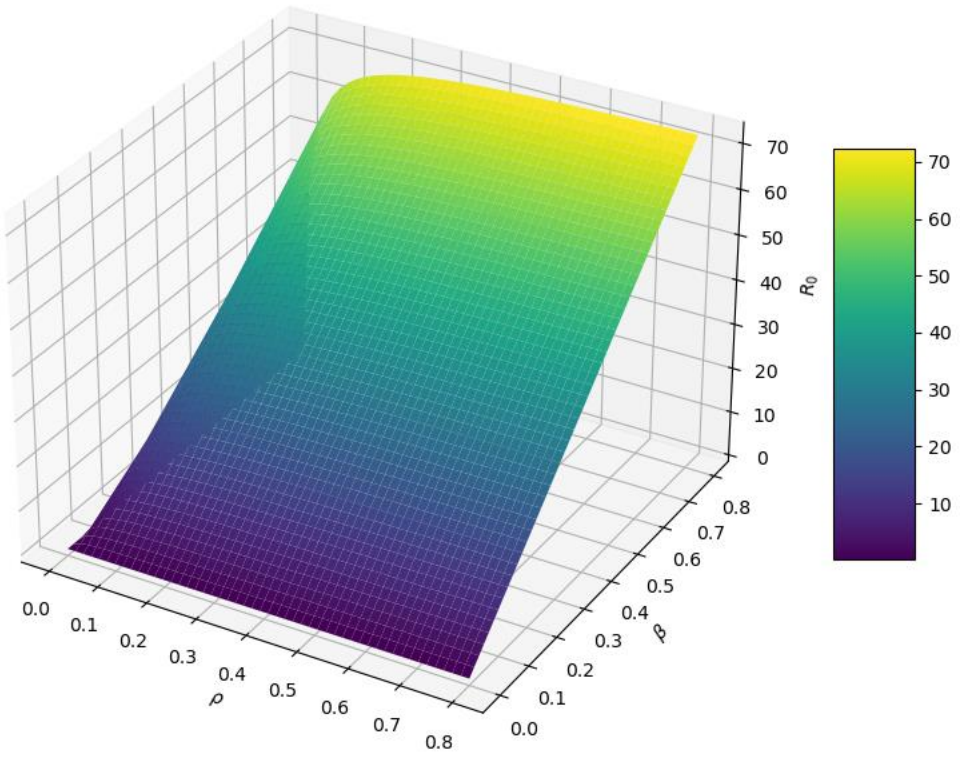

Figure 2b: 3-dimensional plot of the basic reproduction number versus the most sensitive parameters ρ and β

Numerical Simulation

Since, the **Error! Reference source not found.** cannot solve analytically because of its linearity, therefore, the solution was obtained by using a numerical method [25,26]. The initial conditions used for the simulations are as follows; $S(0)=3000$, $I_A(0)=2100$, $I_S(0)=2000$, $T(0)=1000$ and $R(0)=500$ at $t=0$.

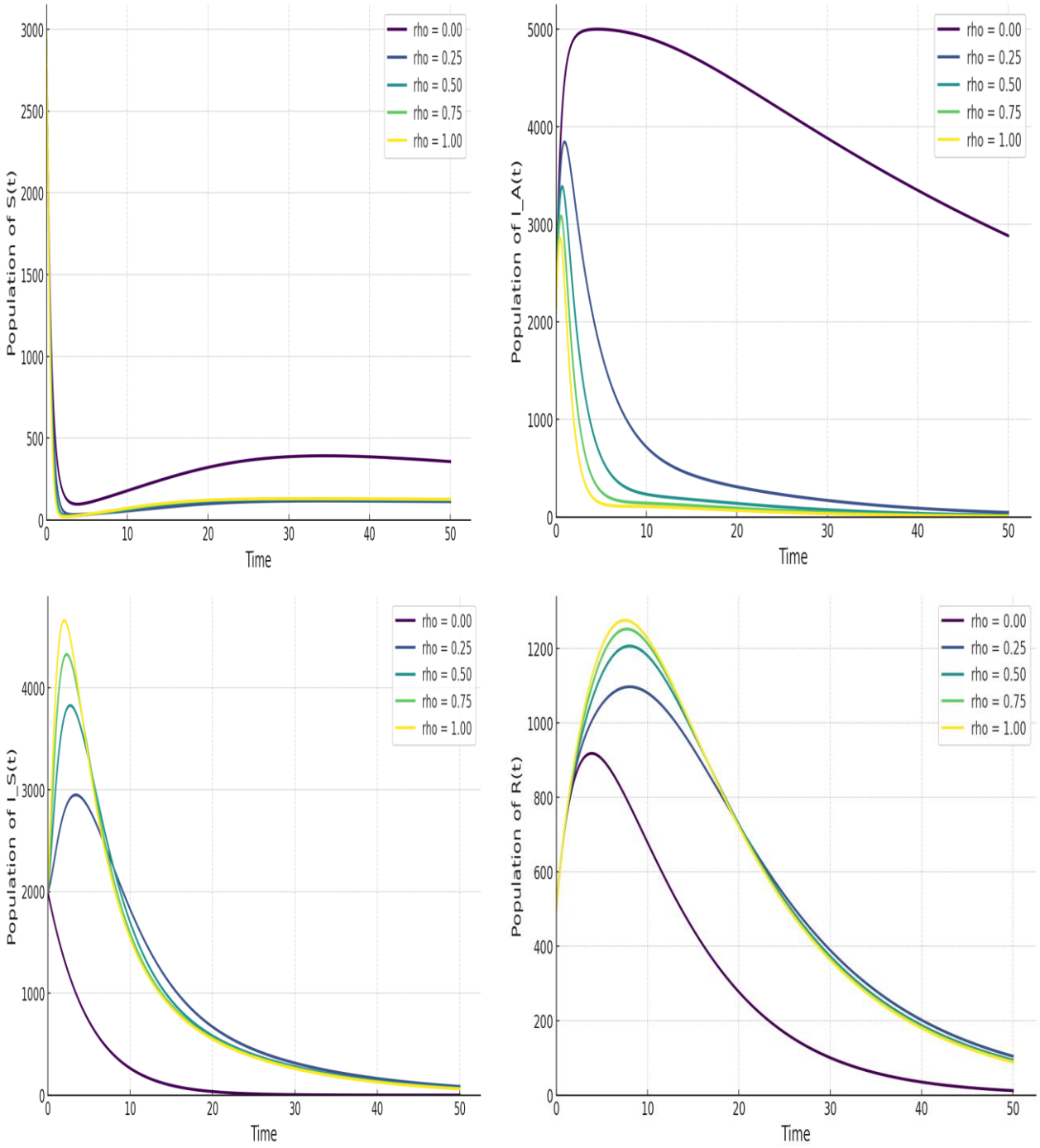

Figure 3: Varying parameter ρ in different states of the model

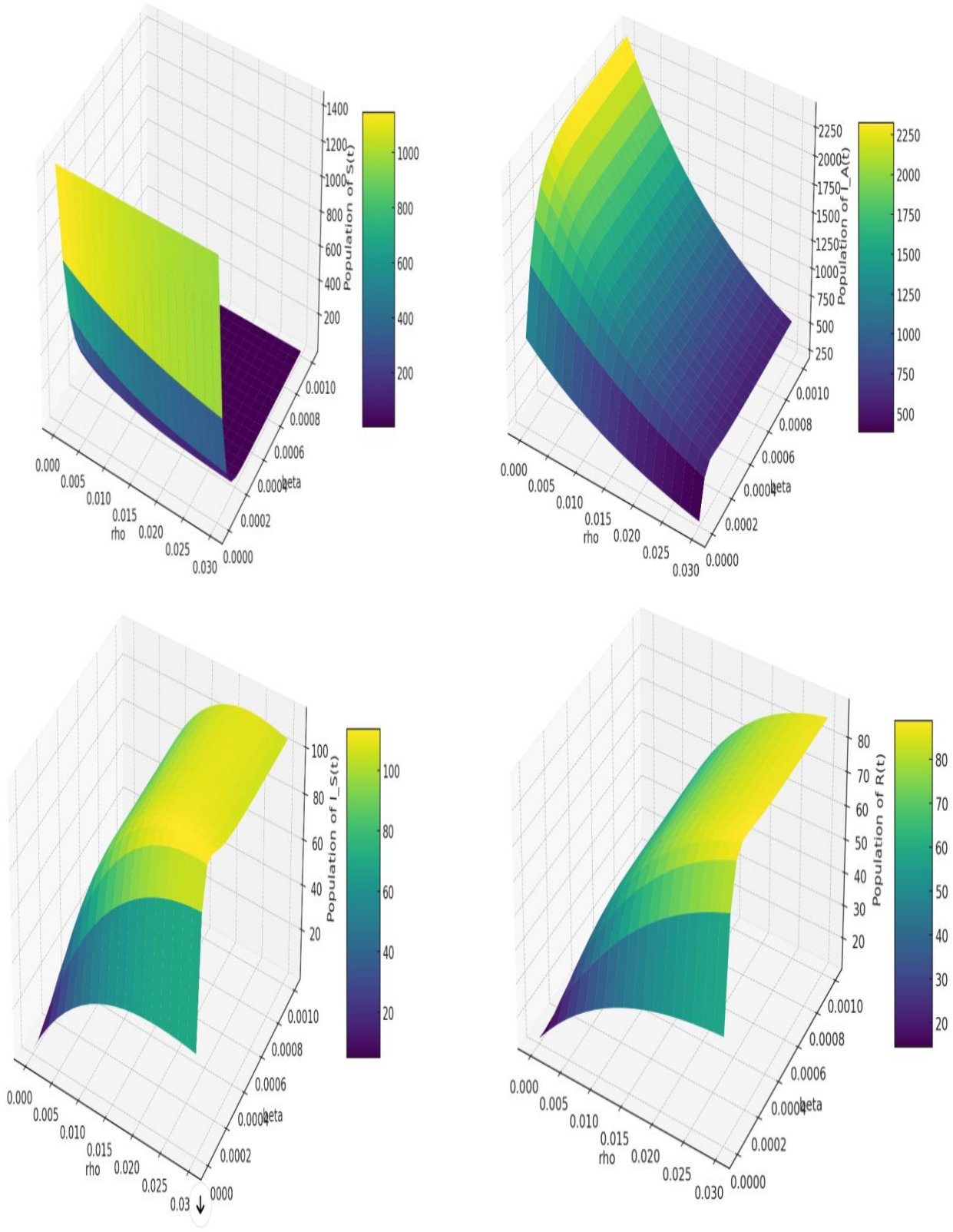

Figure 4 3D plot of parameters ρ and β in different states of the model

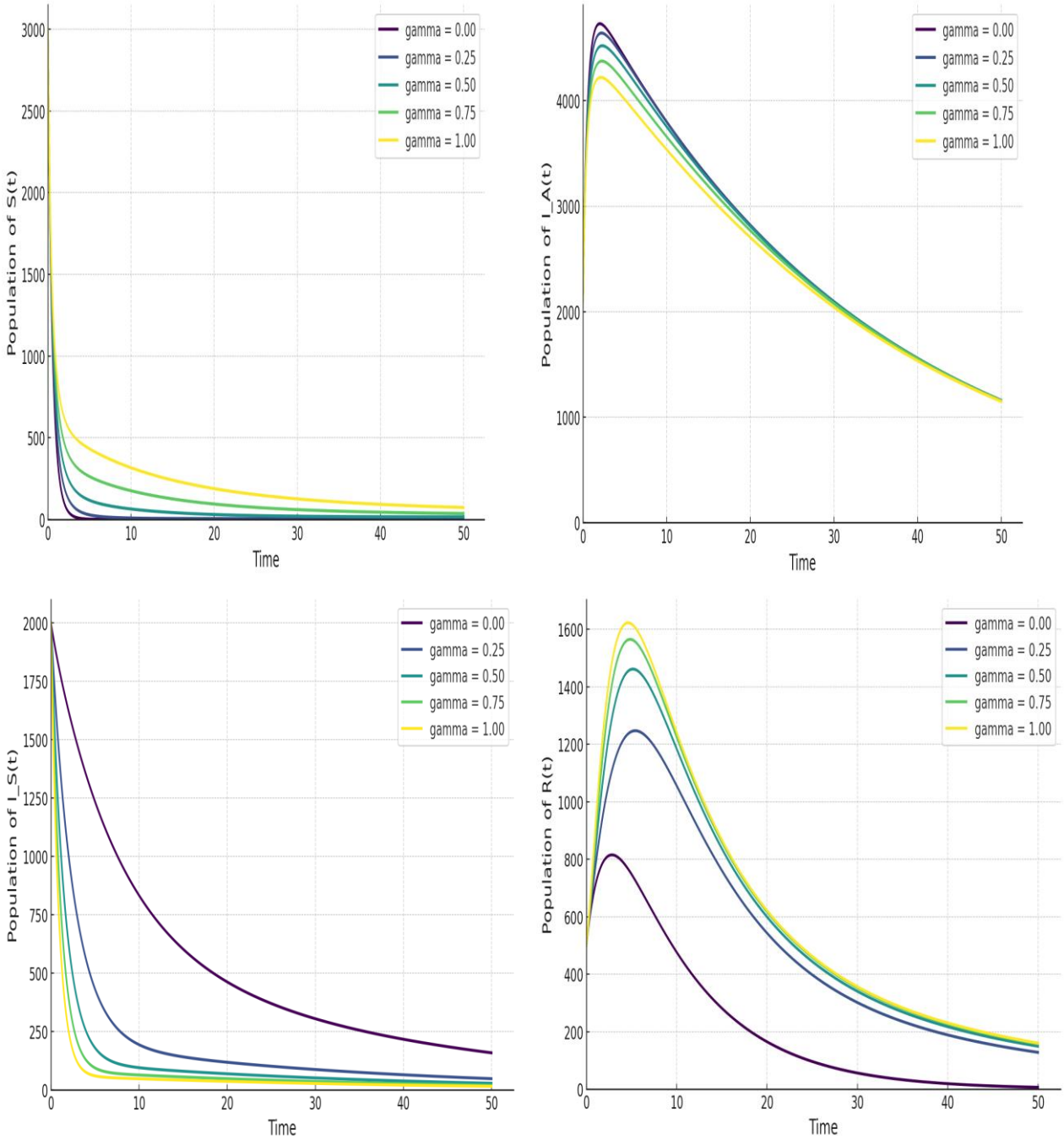

Figure 5: Varying parameter γ in different states of the model

DISCUSSION

Figure 2a & 2b show the sensitivity analysis of the model system. Succinctly, parameter β & ρ contribute more to the Basic reproductive number R_0. So, to curb the transmission dynamic of typhoid fever in the population, it is therefore encouraged to minimize the contact rate and the progression from $I_A(t)$ to $I_S(t)$.

Figure 3 demonstrates that an augmentation in the rate of progression among individuals afflicted with typhoid fever exerts a pronounced influence on the transmission dynamics of the ailment within the population. In other words, when ρ is zero, the curve in the susceptible and asymptomatic classes increase rapidly. So, the higher the ρ the more increase its curve and vice versa. It is observed that the

fever will go into extinction in about 50 days if proper class is identified for each patient and if proper treatment is given to infected patient. This impact is discernible through the concurrent augmentation in the populace comprising individuals in the clinical stage of the infection. Evidently, the affirmative influence of the progression rate on this class is poised to amplify the exponential surge of typhoid fever within the population, unless decisive measures are promptly instituted to mitigate its transmission dynamics.

Figure 4 illustrates the outcomes of a sensitivity analysis, elucidating the paramount parameters contributing to the Basic Reproduction Number (R_0) in the compartmentalized model system. The four graphs delineate the pronounced influence of the two most pivotal parameters on the transmission dynamics, as their values are systematically incremented. It is obvious to see that the more increased the values of these two parameters, the higher its curves as shown on the susceptible, asymptomatic, symptomatic, recovered compartments. The effect was shown by consecutively increasing the values of ρ & β. It is strongly recommended that individuals afflicted with typhoid fever exercise caution near non-infected (susceptible) individuals. Moreover, the prompt identification of symptomatic cases should be facilitated through enhanced mechanisms. This approach aims to significantly curtail the progression rate from the asymptomatic to the symptomatic compartment $I_A(t)$ to $I_S(t)$, thereby potentially achieving a disease-free equilibrium.

Figure 5 elucidates the pivotal role played by the treatment rate of infectious individuals in the extirpation of typhoid fever from the population. The empirical findings indicate a positive correlation between the augmentation of the treatment rate and the concurrent increase in the cohort of treated in factious individuals, juxtaposed with a concomitant decrease in the population of symptomatically, asymptomatically infected individuals. Also, the recovered compartment increases drastically as infected individuals get treated. These observations underscore the imperative for medical health practitioners, governmental bodies, and non-governmental agencies to ensure the accessibility of efficacious medications or pharmaceutical interventions for treating typhoid fever. Such measures are essential for fortifying the effective control mechanisms governing the transmission dynamics of this fever within the population.

CONCLUSION

This study successfully developed and analysed a nonlinear mathematical model to understand and control the transmission of typhoid fever in overpopulated communities. By incorporating environmental precautions and determining the basic reproduction number R_0, we established the local and global asymptotic stability conditions for both disease-free and endemic equilibria. Our findings emphasize the importance of minimizing the contact rate and the progression from asymptomatic to symptomatic states to effectively control the spread of typhoid fever. Sensitivity analysis revealed that parameters β and ρ significantly impact R_0, indicating that targeted efforts to reduce these parameters are crucial.

Furthermore, our model highlights the critical role of treatment rates in reducing the population of symptomatically infected individuals. There is a positive correlation between increased treatment rates and the decline of symptomatic cases, underscoring the need for accessible and effective medical interventions. This research provides valuable insights into the transmission dynamics of typhoid fever and the effectiveness of various control strategies. By integrating mathematical modelling with advancements in vaccine development and public health measures, this study contributes to the global effort to mitigate the burden of typhoid fever. Future research should focus on refining these models and exploring new strategies to enhance control and prevention efforts.

References

[1] J.W. Karunditu, G. Kimathi, S.Osman. (2019). Mathematical modeling of typhoid fever disease incorporating unprotected humans in the spread dynamics. *Journal of Advance in Mathematics and Computer science*, 32(3): 1- 11.

[2] WHO (2007) Background paper on Vaccination against typhoid fever using new generation Vaccines.

Presented at the SAGE meeting 11.

[3] WHO (2021) Immunization Vaccines and biological. Available at https://www.int/teams/immuniztion - vaccines-and - biological /diseases/ typhoid.

[4] J.K. Nihiiri, G.O. Lawi, C.O. Akinyi, D.O. Oganga, W.C. Muriuki, M.J. Musyka, P.O. Otieno, L. Koech.(2016). Mathematical modeling of typhoid fever disease incorporating protection against infection. *Br. J. Math. Comput.Sci*. 14(1); 1- 10.

[5] J.M. Mutua, F.B. wang, N.K. Vaidya. (2015). Modeling Malaria and typhoid fever Co- infection dynamics. *Math .Biosci*. 264: 128- 144.

[6] O.O. Temitope, M.O. Oluwatayo, N.O. Segun, E.F. Grace, O.O. Joshua, R.A. Opeyemi, Y.H. Omobolaji, O.O. Adenike . (2020). On the effect of vaccination, Screening and treatment in controlling typhoid fever spread dynamics deterministic and stochastic application. *Math.Stat*. 8(6): 621- 630.

[7] M.N. Chamuchi, J.K. Sigee, J.A. Okello, J.M. Okwoyo. (2014) SII_CR model and simulation of the effects of carriers on the transmission dynamics of typhoid fever in KISII town Kenya. 2014: 2(3).

[8] O.J. Peter, M.O. Ibrahim, O.B Akinduko, M.Rabiu. (2017). Mathematics model for the control of typhoid fever. *ISOR Journal of* M*athematics* 13(14)VerII: 60- 66.

[9] O.D Makinde and K.O. Okosun . (2011). Impact of chemotherapy on optimal control of malaria disease with infected immigrants . *Biosystems*, 104(1): 32-41.

[10] K.O Okosun and O.D. Makinde (2012). On a drug-resistant malaria model with susceptible individuals without access to basic amenities. *Journal of Biological Physics*, 38(3): 507-530.

[11] K.O Okosun and O.D Makinde (2013). Optimal control analysis of malaria in the presence of non-linear incidence rate. Applied and Computational Mathematics. *An International Journal*, 12(1): 20- 32.

[12] K.O. Okasun and O.D. Makinde. (2014). Optimal control analysis of hepatitis C virus with acute and chronic stages in the presence of treatment and infected immigrants. *International Journal of Biomathematics*, 7(2), Article ID 1450019, 23pp.

[13] K.O. Okasun and O.D. Makinde. (2014). A Co- infection model of Malaria and Cholera diseases with optimal control. *Mathematical Biosciences*, 258: 19- 32.

[14] World Health Organization (2000), Typhoid fever fact sheet ,http://www.who.int/mediacenter/factsheet/.

[15] World Health Organization (2003), The Diagnosis, Treatment and prevention of Typhoid fever.

[16] A. Alhassan, A.A. Momoh, S.A. Abdullahi and A. Audu. (2021). Mathematical Model for the Transmission Dynamics of Typhoid Fever Infection with Treatment. *International Journal of Science for Global Sustainability*, 7(2): 37- 49

[17] Halson O. Nyaberi and Jane S. Musaili. (2021). Mathematical modeling of the impact of treatment on the dynamics of typhoid. 29(15): 1- 11.

[18] E. Stephen and N. Nyerere. (2016). Modeling typhoid fever with education, vaccination and applied Mathematics. *Am.J. Trop. Med.Hyg*.2: 156- 164.

[19] S. Muhayabasa, C.P. Bhunu, and N.A. Mhlanga. (2014). Modeling the Transmission Dynamics of Typhoid in Malaria Endemic Settings. *An International Journal of Applications and Applied Mathematics* (AMM), 9(1): 121-140.

[20] H. Abboubakar and R. Racke. (2019). Mathematical Modeling and Optimal Control of Typhoid Fever. *Downloaded from Konstanzer Online- Publication- System* (KOPS); http://nbn-resolving.de/ 8(21): 352-367.

[21] G.T. Tilahum, O.D. Makinde. (2017). Modeling and Optimal Control of Typhoid Fever Disease with Cost – Effective Straggles. *Computational and Mathematical Methods in Medicine* volume 2017: 1- 16.

[22] O.J. Peter, O.A. Afolabi, F.A. Oguntolu, C.Y. Ishola and A.A Victor. (2018). Solution of a Deterministic Mathematical Model of Typhoid Fever by Variational Iteration Method. *Science World Journal* 13(2): 64-68.

[23] Oluwatobi, I. K., & Chris, L. A. (2023). Qualitative Analysis of the Transmission Dynamics and Optimal Control of Covid-19.

[24] I.K. Oluwatobi, L.M. Erinle-Ibrahim (2021), Mathematical modelling of pneumonia dynamics of children under the age of five.

[25] M. Erinle-Ibrahim Latifat, I.K. Oluwatobi, I. Sulola Abigail (2021), Mathematical modelling of the transmission dynamics of malaria infection with optimal control, Kathmandu Univ. J. Sci. Eng. Technol. 15.

[26] A.C. Loyinmi, K.O. Idowu (2023), Semi-Analytic Approach to Solving Rosenau-Hyman and Korteweg-De Vries Equations Using Integral Transform, Tanzania J. Sci. 49: 26–40.

[27] K.O. Idowu, T.G. Akinwande, I. Fayemi, U.M. Adam, A.C. Loyinmi (2023), Laplace Homotopy Perturbation Method (LHPM) for Solving Systems of N-Dimensional Non-Linear Partial Differential Equation, https://doi.org/https://doi.org/10.55810/2313-0083.1031.

[28] E.-I.L. Morenikeji, A.O. Babajide, I.K. Oluwatobi (2021), Application of Homotopy Perturbation Method to the Mathematical Modelling of Temperature Rise during Microwave Hyperthermia, FUDMA J. Sci.

5:273–282.

[29] Agbomola, J. O., & Loyinmi, A. C. (2022). Modelling the impact of some control strategies on the transmission dynamics of Ebola virus in human-bat population: An optimal control analysis. *Heliyon*, *8*(12).

[30] Erinle-Ibrahim, L. M., Adebimpe, O., Lawal, W. O., & Agbomola, J. O. (2022). A mathematical model and sensitivity analysis of Lassa fever with relapse and reinfection rate. *Tanzania Journal of Science*, *48*(2), 414-426.

[31] Agbomola, J., & Loyinmi, A. (2022). A Mathematical Model For The Dynamical Behavior Of Ebola Virus Transmission In Human-Bat Population: Implication Of Immediate Discharge Of Recovered Individuals.