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RESEARCH ARTICLE

Mathematical Analysis and Control of Typhoid Fever Dynamics Using a SEITRB Fractional Model

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Abstract:
The SEITRB compartmental model was formulated to investigate the transmission dynamics and control of typhoid fever, incorporating the susceptible, exposed, infected, treatment, recovered, and environmental bacteria populations. The model was analyzed for well-posedness, establishing existence, uniqueness, positivity, and boundedness of solutions. Equilibrium states were examined under both disease-free and endemic conditions, with the basic reproduction number (R_*) derived as the threshold parameter. The analysis showed that typhoid infection dies out when $(R_* < 1)$ but persists when $(R_* > 1)$. Local and global stability analyses were established, while sensitivity analysis identified treatment rate, bacterial decay, and vaccination efficacy as the most influential parameters on (R_*). Numerical simulations, carried out using the Laplace Adomian Decomposition Method in conjunction with Caputo fractional derivatives, illustrated the impact of control measures. Findings revealed that optimal treatment effectiveness, sufficient treatment coverage, and improvements in sanitation act synergistically to minimize infection and reinfection risks. Over a multi-year horizon, these combined interventions significantly reduced disease prevalence in endemic populations. This study demonstrates that integrating mathematical analysis with practical interventions provides a robust model for understanding typhoid dynamics. By identifying the parameters most critical to disease reduction, the SEITRB model offers evidence-based guidance for health practitioners in designing localized and sustainable typhoid control strategies. Overall, the model highlights the transformative role of coordinated vaccination, treatment, and sanitation in achieving effective prevention and long-term community health improvement.

Keywords: Fractional-order model, Thypoid fever, Treatment intervention, Optimal control, Laplace adomian decomposition method (LADM)

1. Introduction

Typhoid is a highly deadly disease in remote regions of Africa, especially in regions with limited access to clean water and sanitation. Understanding the complex mechanisms that drive typhoid's transmission and control is essential for developing effective interventions. To effectively address typhoid's persistent impact, it is crucial to disentangle the relationships among these components, which may enable the design of targeted and efficient control strategies. This approach not only enhances our theoretical understanding of typhoid fever spread but also lays the foundation for improved treatment protocols and intervention schemes in [1, 2]. Decades of research have explored various facets of typhoid fever, from its pathogenesis and transmission to therapeutic approaches and community health initiatives. These studies have made substantial contributions, yielding advancements in diagnosis, treatment and prevention strategies. Drug resistance in bacteria agent (Salmonella Typhi), the causative agent of typhoid, has emerged, prompting researchers to develop new approaches for managing resistance and enhancing treatment effectiveness as discussed [3–5].

The Laplace Adomian Decomposition method in this study is applied to analyse typhoid fever dynamics, yielding insights into the impact of timely and adequate treatment, the spread of health information and the threat of reinfection as in [6]. This analysis offers practical implications for global health efforts and introduces novel approaches for minimizing typhoid's impact on affected communities. Recently, upsurge in typhoid fever cases across regions like Nigeria, Ethiopia, Ghana, Somalia, Chad, Niger and Tanzania has underscored the pressing public health hazards posed by this life-threatening disease. Typhoid fever, is an acute diarrheal illness caused by Salmonella enterica serovar Typhi, has long been a health concern in areas with limited access to clean water, sanitation and healthcare resources in [7–11]. However, recent outbreaks have been more widespread and severe, particularly impacting remote and rural populations with limited healthcare access. This resurgence places a substantial burden on public health systems, exacerbates existing vulnerabilities and highlights the urgent need for innovative, effective strategies to control and prevent the disease's spread. In these affected regions, the impact of typhoid fever has been devastating, contributing to high morbidity and mortality rates, especially among children and other vulnerable population by [12, 13]. The disease spreads rapidly through contaminated water sources, which are often the primary water supply in many communities.

The cycle of infection is further fueled by inadequate sanitation infrastructure, crowded living conditions and in some cases, the displacement of populations due to conflicts or environmental crises. As typhoid cases continue to rise, health practitioners and organizations are on high alert, working tirelessly to contain outbreaks and prevent further loss of life. Efforts to combat the spread of typhoid in these regions have included emergency responses such as establishing treatment centers, distributing oral rehydration solutions and initiating vaccination campaigns [14–16]. Additionally, health organizations are focusing on long-term solutions, such as improving water, sanitation and hygiene hand washing facilities, educating communities on prevention measures and ensuring rapid response capabilities in the event of new outbreaks [17]. Despite these efforts, the scale of recent outbreaks indicates a need for more comprehensive and coordinated interventions that address the root causes of typhoid fever transmission, including poverty, inadequate infrastructure and limited access to healthcare. This research seeks to go beyond traditional models, identifying subtle yet pivotal factors that influence cholera transmission, including the role of proper treatment and the dissemination of preventive knowledge to vulnerable populations as seen [18–20]. Also, recent progress in typhoid's research were explored as well as assessment of key developments in the field and their implications for public health. Therefore, this study examined the interaction between treatment strategies, reinfection dynamics and public health education as central factors in typhoid fever's dynamics by [21]. Estimated impact of control measures on typhoid fever spread in West Africa, the vaccination rate has been effective for 65% to 85% in reducing typhoid incidence within

high-risk populations over 3 to 5 years. For instance, in Ethiopia and Nigeria, mass vaccination campaigns have been shown to reduce local outbreaks by up to 75% when coverage exceeded 60%.

The treatment rate (Prompt antibiotics) effectiveness is 50% to 70% reduction in typhoid's mortality and disease duration e.g., in properly managed outbreaks, case fatality rates dropped from over 5% to below 1% with improved treatment access in [22–25]. Environmental sanitation (Safe water, hygiene, waste disposal) effectiveness as contributed to 40% to 60% reduction in typhoid transmission when combined with clean water supply and hand hygiene as seen in [26, 27]. However, community sanitation programs in Togo and Benin resulted in 45% fewer typhoid fever cases over one year. An in-depth analysis of these elements can provide crucial insights from previously control mechanisms of hand washing and food sanitation into how treatment adequacy, public awarenessand reinfection risk shape the trajectory of typhoid's outbreaks.

2. Materials and Methods

2.1. Preliminary Definition

Some fundamental definitions and properties in fractional calculus are given in this section.

Definition 2.1. According to [2], a real function $\varphi(t), t > 0$, is said to be in the space $C_\mu, \mu \in R$ if there exist a real number $m > \mu$ such that $\varphi(t) = t^m\varphi_1(t)$ where $\varphi_1(t) \in C(0,\infty)$ and it is said to be in the space C_μ^n, if and only if $\varphi^{(n)} \in C_\mu^n, n \in N$

Definition 2.2. The Riemann-Liouville fractional integration of order $\eta \geq 0$ of a positive real function $\varphi(t) \in C_\mu, \mu \geq -1, t > 0$ is defined as equation (2.1) as in [15]

$$I^\eta\varphi(t) = \frac{1}{\Gamma(\eta)}\int_0^t (t-x)^{\eta-1}\varphi(x)dx \text{ such that } I^0\varphi(t) = \varphi(t) \tag{2.1}$$

The following properties hold for fractional integer operator I^η for $\varphi(t) \in C_\mu, \mu \geq -1, \eta, \alpha \geq 0$ and $\beta \geq -1$:

1. $I^\eta I^\alpha\varphi(t) = I^{\eta+\alpha}\varphi(t)$,
2. $I^\eta I^\alpha\varphi(t) = I^\alpha I^\beta\varphi(t)$,
3. $I^\eta\varphi(t) = \frac{\Gamma(\beta+1)}{\Gamma(\eta+\beta+1)}t^{\eta+\beta}$

Definition 2.3. The Caputo Fractional derivatives of a positive real function $\varphi(t)$ given as $D^\eta\varphi(t)$ Is given in equation (2.2) as defined in [17]

$$D^\eta\varphi(t) = \frac{1}{\Gamma(n-\eta)}\int_0^t (t-x)^{\eta-1}\varphi^{(n)}(x)dx \text{ for } n-1 < \eta \leq n, n \in N, t > 0, \varphi \in \frac{n}{c-1} \tag{2.2}$$

The following property holds for fractional integration of the Caputo Fractional derivatives, for $n-1 < \eta \leq n, n \in N, t > 0, \varphi \in \frac{n}{c-1}$, then

$$I^\eta D^\eta\varphi(t) = \varphi(t) - \sum_{k=0}^{n-1}\varphi^{(k)}(0)\frac{t^k}{k!}$$

The theory of fractional calculus In this segment, familiarizing ourselves with the foundational principles and terminology of fractional calculus theory, essential for its utilization in analyzing the current infectious disease model. Moreover, its diverse applicability extends across various scientific disciplines.

Definition 2.4. From [16, 17] the fractional integral of Riemann–Liouville order α concerning a given function g, represented as $g : \Re^+ \to \Re$, is formally defined as

$$\Gamma g(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\phi)^{\alpha-1} g(\phi)\, d\phi$$

Definition 2.5. From [16, 17] the differential quotient of a function $p(t)$ across the time interval $[0, T]$ can be represented as the Caputo derivative, defined as

$${}_a^b D_t^\alpha = \frac{1}{\Gamma(\kappa-\alpha)} \int_0^t (t-\phi)^{\kappa-\alpha-1} p^{(\kappa)}(\phi)\, d\phi,$$

for $\kappa = [\phi] + 1$ and $[\phi]$ represents the integer part of ϕ in $[0, 1]$. km

Definition 2.6. From [16, 17] the mathematical transformation of the function differential quotient of $t^{\beta-1} E_{\alpha,\beta}(\pm\mu t^\alpha)$ through the Laplace domain can be expressed as

$$\mathcal{L}\left\{t^{\beta-1} E_{\alpha,\beta}(\pm\mu t^\alpha)\right\} = \frac{S^{\alpha-\beta}}{S^\alpha \mp \mu}.$$

The Mittag-Leffler function, characterized by its two parameters, is denoted by $E_{\alpha,\beta}(t)$, where $\alpha, \beta > 0$.

Theorem 2.1 (General Mean Value Theorem). *[20, 21]. Let $h(t) \in c[b, d]$ and ${}_d^c D_\beta^\alpha h(\beta) \in c[b, d]$ for $0 < \alpha \leq 1$. Then $h(t)$ can be written as*

$$h(t) = h(b) + \frac{1}{\Gamma(\alpha+1)} {}_b^c D_\beta^\alpha h(\beta)(t-b)^\alpha, \quad \textit{where } b \leq \beta \leq \alpha,\ \forall\, \alpha \in (b, d).$$

Also, if ${}_d^c D_\beta^\alpha h(\alpha_0) > 0$, $\alpha_0 \in (b, d)$, then $\exists$ neighborhood $h(t) < h(\alpha)$ of α_0 such that $h(t) < h(\alpha), \forall t \in N$.

2.2. Model Formulation

The epidemic model of typhoid is considered to be a control strategy for the undeviating transference of infectious disease. Due to its high accuracy and efficiency in solving nonlinear differential equations, the homotopy perturbation method is coupled with the Riemann-Liouville fractional integral operator I of order $\eta \in (0, 1)$ to obtain the approximate analytical solution of the epidemiology model presented at different level of η. A deterministic mathematical model based on the epidemiological status of population members that describes the dynamics of typhoid transmission. Susceptible population, $S(t)$ these individuals who are not infected with typhoid fever at time (t) but are at risk of infection through contact with contaminated water, food, or infected individuals. Their risk is reduced by effective water sanitation and monitoring. Exposed population, $E(t)$ have been exposed to Salmonella typhi at time (t) and are in the latent (incubation) period. They are infected but not yet infectious. Infected population, $I(t)$ these Individuals who are actively infected with typhoid fever at time (t) and can transmit the disease directly or indirectly by shedding bacteria into the environment. Treated population, $T(t)$, infected individuals who are receiving appropriate medical treatment (e.g., antibiotics) at time (t). Treatment reduces disease severity, infectiousness and bacterial shedding into the environment. Recovered population, $R(t)$, who have recovered from typhoid infection at time (t) and have acquired temporary or permanent immunity against reinfection, depending on model assumptions. Bacteria concentration in the environment, $B(t)$, the concentration of Salmonella typhi in the environment (particularly in water sources) at time (t). This concentration increases due to shedding by infected individuals and decreases through natural bacterial decay, water sanitation and environmental monitoring interventions.

The recruitment rate Λ into the susceptible population and transmission rate of typhoid β as both the transmission probability and the rate of migration/recruitment into the group that is vulnerable which are measured in terms of half-saturation constant of bacterial infection κ for typhoid fever transmission. Natural death rate μ and γ the rate at which exposed individuals become infectious is said to migrate from exposed to infected population and also τ rate of starting treatment for infected individuals when there is rampant typhoid fever disease. Recovery rate from treatment ω due to natural immunity and bacterial shedding rate by individuals under treatment ξ_2 due to medical treatment both help to increase typhoid fever resistance. Bacterial shedding rate by infected individuals ξ_1 and η natural decay rate of bacteria in the environment. Through water treatment effort, antibiotic usage control and general treatment control policy the environmental bacterial removal μ_b due to sanitation efforts on typhoid fever spread, this can easily be managed effectively in the population with time. Disease induced death rate α in infected population and ϕ additional bacteria reduction due to hygiene and food safety controls. The Figure 2.1 below depict the aforementioned model parameters and a system of differential equations in equation (2.3) denotes the model.

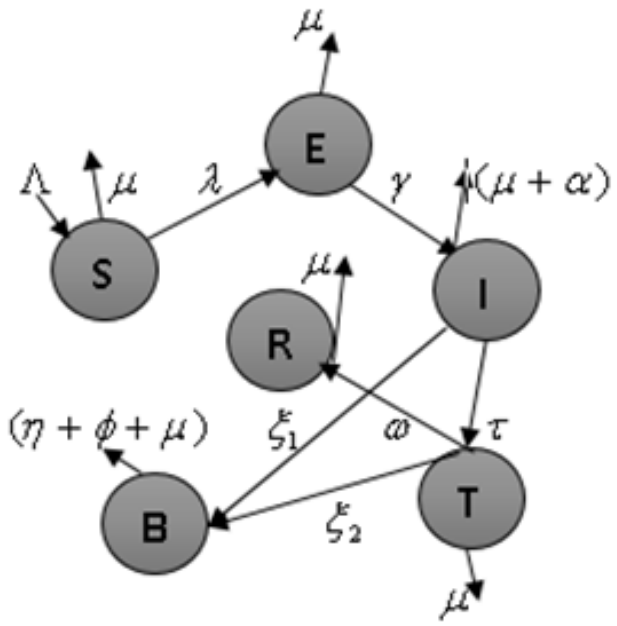

Figure 2.1: Schematic diagram of the model

$$\begin{aligned} D^{\eta_1} S(t) &= \Lambda - (\lambda + \mu) S(t) \\ D^{\eta_2} E(t) &= \lambda S(t) - (\gamma + \mu) E(t) \\ D^{\eta_3} I(t) &= \gamma E(t) - (\tau + \alpha + \xi_1 + \mu) I(t) \\ D^{\eta_4} T(t) &= \tau I(t) - (\omega + \xi_2 + \mu) T(t) \\ D^{\eta_5} R(t) &= \omega T(t) - \mu R(t) \\ D^{\eta_6} B(t) &= \xi_1 I(t) + \xi_2 T(t) - (\eta + \phi + \mu) B(t) \end{aligned} \tag{2.3}$$

Where the force of infection is defined as $\lambda = \frac{\beta B}{\kappa + B}$, subjected to the initial condition

$$S(0) = s_0 \geq 0,\ E(0) = e_0 \geq 0,\ I(0) = i_0 \geq 0,\ T(0) = t_0 \geq 0 \tag{2.4}$$

Table 2.1: Description of state variables and parameters

Variable	Description
$S(t)$	Susceptible population
$E(t)$	exposed population
$I(t)$	infected population
$T(t)$	treated population
$R(t)$	recovered population
$B(t)$	bacterial concentration
Parameters	**Description**
Λ	Recruitment rate
β	Transmission rate of typhoid environmental bacteria
α	Disease induced death
μ	Natural death
τ	Rate of starting treatment for infected individuals
ω	Recovery rate due to medical treatment
ϕ	Additional bacteria reduction due to safety hygiene
γ	Progression rate from exposed to infected population
η	Natural decay rate of bacteria in the environment
κ	Half-saturation constant of bacterial infection
ξ_1	Bacterial shedding rate by infected individuals
ξ_2	Bacterial shedding rate by individuals under treatment
μ_b	Environmental bacteria reduction due to sanitation effort

2.3. Existence and Uniqueness of Model Solution

The model in equation (2.3) which represents the spread of an epidemic disease of typhoid fever within the human population, requires that is parameters be non-negative for its existence and uniqueness of the model solution. To ensure that the system of differential equations in equation (2.3) is both mathematically valid and epidemiologically sound, it is important to establish that the model's state variables remain non-negative. Equation (2.1) is considered well-defined at the initial point if the initial conditions are non-negative. $S(0) = s_0, E(0) = e_0, I(0) = i_0, T(0) = t_0, R(0) = r_0$ and $B(0) = b_0$. In that case, the solutions of model equation (2.3) will persist in being non-negative throughtout their evolution, $t > 0$ and these positive solutions are bounded. Thus, applying the theorem below.

Theorem 2.2. *Let (x, y) be distinct points of normed linear space $(X, ||\dots||)$ over $\Re$. Then the map of $p : [0, 1] \subseteq \mathcal{R} \to (X, ||\dots||)$ such that $p(\lambda) = \lambda x + (1 - \lambda)y$ is continuous on $[0, 1]$.*

Proof. Let $\lambda_0 \in [0, 1]$ then $p(\lambda_0) = \lambda x + (1 - \lambda_0)y$ for any $\lambda_0 \in [0, 1]$.

$$\begin{aligned} ||p(\lambda) - p(\lambda_0)|| &= ||(\lambda - \lambda_0)x + (\lambda - \lambda_0)y|| \\ &\leq |\lambda - \lambda_0|(||x|| + ||y||) \end{aligned} \tag{2.5}$$

It $\varepsilon > 0$ is given, let $\delta = \frac{\varepsilon}{||x||+||y||}$. If $|\lambda - \lambda_0| < \delta$, then the $||p(\lambda) - p(\lambda_0)|| < \varepsilon$. Therefore, p is continuous at λ_0. Since λ_0 is an arbitrary point in $[0, 1]$. Then p is continuous on $[0, 1]$. Let X be a linear space over $\Re$. If (x, y) are distinct points of X, the set $\lambda x + (1 - \lambda)y$ lies in $0 \leq \lambda \leq 1$. Hence, the solutions of system of equation (2.3) are bounded if we consider the total population. The variation in the total population concerning time is given by:

$$N(t) = S(t) + E(t) + I(t) + T(t) + R(t) + B(t)$$

The variation in the total population concerning time is given by:

$$\frac{dN(t)}{dt} = \frac{d}{dt}\left(S(t) + E(t) + I(t) + T(t) + R(t) + B(t)\right) \tag{2.6}$$

Such,

$$\begin{aligned} N^*(t) &\leq \Lambda - (\lambda+\mu)S(t) + \lambda S(t) - (\gamma+\mu)E(t) + \gamma E(t) - (\tau+\alpha+\xi_1+\mu)I(t) + \tau I(t) \\ &\quad -(\omega+\xi_2+\mu)T(t) + \omega T(t) - \mu R(t) + \xi_1 I(t) + \xi_2 T(t) - (\eta+\phi+\mu)B(t) \\ N^*(t) &\leq \Lambda - \mu(S+E+I+T+R+B) \Rightarrow N^*(t) + \mu N \leq \Lambda \end{aligned} \tag{2.7}$$

$N^*(t) + \mu N \leq \Lambda$ by using integrating factor technique for the first order differential equation at time $t = 0$, $c = N(0) - \frac{\Lambda}{\mu}$ where c is the constant of integration. Hence, it is obtained that $N^*(t)$, using the integrating factor concept on the total population $N(t)$ and this leads to substituting equation (2.6) into (2.7) as time progressively increases yields:

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty} \left[\frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t}\right] = \frac{\Lambda}{\mu} \tag{2.8}$$

Then $N(0) \leq \dfrac{\Lambda}{\mu}$, then $N(t) \leq \dfrac{\Lambda}{\mu}$. This is a positive invariant set under the flow described by (2.8) so that no solution path leaves through any boundary $\Re^6_+$. Hence, it is sufficient to consider the dynamics of the model in the domain $\Re^6_+$. In this region, the model can be considered has be mathematically and epidemiologically well-posed. This shows that the total population and the subpopulation $S(t), E(t), I(t), T(t), R(t), B(t)$ of the model are bounded and is a unique solution. Hence, its applicability to studying physical systems is feasible. □

2.4. Positivity and Boundedness

This shows that the total population $N(t)$, and the subpopulation $S(t), E(t), I(t), T(t), R(t), B(t)$ of the model are bounded and is a unique solution. Hence, its applicability to study physical systems is feasible.

Theorem 2.3. *Suppose $X = x_0$ is a space of consecutive real number and which are defined as*

$$L(x,y) = \left(\sum_{i=1}^{n} |x_i|^{\Omega}\right)^{\frac{1}{\Omega}}, \qquad \Omega \geq 1.$$

X with the metric is called ξ_n^{Ω} space. If

$$\sum^{\infty} |x|^{\Omega} < \infty$$

or absolutely convergent and

$$L(x,y) = \left(\sum_{i=1}^{\infty} |x_i - y_i|^{\Omega}\right)^{\frac{1}{\Omega}},$$

then X with this metric is called an ξ^{Ω} space.

Proof. It can be checked that for each n:

$$0 \leq x_1^2 + x_2^2 + x_3^2 + \cdots + x_n^2 \leq (|x_1| + |x_2| + |x_3| + \cdots + |x_n|)^2 .$$

This will result to; $x_1^2 + x_2^2 \leq (|x_1| + |x_2|)^2$. Therefore,

$$0 \leq \left(x_1^2 + x_2^2 + x_3^2 + \cdots + x_n^2\right)^{\frac{1}{2}} \leq |x_1| + |x_2| + |x_3| + \cdots + |x_n|$$

If $\sum_{n=1}^{\infty}|x_n|$ converges, that $\sum_{n=1}^{\infty}|x_n|$ is absolutely convergent. Then,

$$0 \leq \left(x_1^2+x_2^2+x_3^2+\cdots+x_n^2\right)^{\frac{1}{2}} \leq |x_1|+|x_2|+|x_3|+\cdots+|x_n| = \sum_{n=1}^{\infty}|x_n| < \infty \tag{2.9}$$

Therefore, $0 \leq x_1^2+x_2^2+x_3^2+\cdots+x_n^2 \leq [\sum_{n=1}^{\infty}|x_n|] < \infty$. These sequences x_n is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty}x_n$ converges absolutely, if $x_n \in \xi^1$, then $x_n \in \xi^2$ where $\xi^1 \leq \xi^2$. In case of ξ^1 denote the set of all sequences of x_n real numbers such that $\sum_{n=1}^{\infty}x_n$ is convergent absolutely. I.e. $\sum_{n=1}^{\infty}|x_n| < \infty$ whereas ξ^2 denote the set of all sequence x_n of real numbers such that $\sum_{n=1}^{\infty}x_n^2 < \infty$ converges. From the proceeding $x_n \in \xi^1 \Leftrightarrow x_n \in \xi^2$ i.e. $\xi^1 \subseteq \xi^2$.

Further, if $x_n = \frac{1}{n^{\frac{4}{3}}}$, then $\sum_{n=1}^{\infty}|x_n|$ diverges and thus $x_n \notin \xi^1$. But $\sum_{n=1}^{\infty}x_n^2 = \sum_{n=1}^{\infty}\frac{1}{n^{\frac{8}{3}}}$ converges, implying that $x_n \in \xi^2$. We conclude that $\xi^1 \subseteq \xi^2$ and thus $\xi^1 \neq \xi^2$. If (x_n, y_n) are sequences of real numbers, then;

$$\sum_{n=1}^{\infty}(x_i-y_i)^2 \quad \leq \quad \sum_{n=1}^{\infty}x_i^2+\sum_{n=1}^{\infty}y_i^2+2\left[\sum_{n=1}^{\infty}x_i^2\right]^{\frac{1}{2}}\left[\sum_{n=1}^{\infty}y_i^2\right]^{\frac{1}{2}} \tag{2.10}$$

Therefore if $\sum_{n=1}^{\infty}x_i^2 < \infty$ and $\sum_{n=1}^{\infty}y_i^2 < \infty$ then $\sum_{n=1}^{\infty}(x_i-y_i)^2 < \infty$ for all n. The monotone increasing sequence $\left[\sum_{n=1}^{\infty}(x_i-y_i)^2\right]$ is then bounded above and hence converges, i.e. $\sum_{n=1}^{\infty}(x_i-y_i)^2 < \infty$. Thus $(x_i-y_i)^2 \in \xi^2$ if $(x_n, y_n) \in \xi^2$. Consequently, considering the compartmental disposition

$$\Psi = \left(S(t), E(t), I(t), T(t), R(t), B(t) : N(t)\frac{\Lambda}{\mu} \in \Re_+^6\right)$$

it is obtained as;

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda-(\lambda+\mu)S(t) \Rightarrow \frac{dS}{dt} \geq -S(t)(\lambda+\mu), \qquad \frac{dS}{S(t)} \geq -(\lambda+\mu)\,dt,\\
\int\frac{dS}{S(t)} &\geq -\int(\lambda+\mu)\,dt \Rightarrow S(t) \geq S_0\ell^{-(\lambda+\mu)t} > 0. \quad \text{At } t>0,\ S(t)>0
\end{aligned} \tag{2.11}$$

In the second compartment as deduced from the disease free equilibrium, it is obtained for $E(t)$, $I(t)$ and $R(t)$.

$$\begin{aligned}
\frac{dE}{dt} &= \lambda S(t)-(\gamma+\mu)E(t),\\
\frac{dS}{dt} &\geq -(\gamma+\mu)E(t)\\
\frac{dE}{E(t)} &\geq -(\gamma+\mu)\,dt \Rightarrow \int\frac{dE}{E(t)} \geq -\int(\gamma+\mu)dt\\
E(t) &\geq E_0e^{-(\gamma+\mu)t} > 0\ t>0\ E(t)>0
\end{aligned}$$

Subsequently this obtained for other compartments of the model equation (2.3) that;

$$\begin{aligned}
I(t) &\geq I_0e^{-(\tau+\alpha+\xi_1+\mu)t} > 0\\
T(t) &\geq I_0e^{-(\omega+\xi_2+\mu)t} > 0\\
R(t) &\geq R_0e^{-(\mu)t} > 0,\\
B(t) &\geq R_0e^{-(\eta+\phi+\mu)t} > 0
\end{aligned} \tag{2.12}$$

Equation (2.11) and (2.12) above shows that equation (2.4) is in the positive quadrant of $\Re_+^5$, persisting in the attracting subset Ψ, which is compact, positively invariant, and influential, with a well-posed, epidemiologically and mathematically represented solution. □

2.5. Disease Free Equilibrium

The equilibrium state of non-infected individuals with typhoid fever signifies a system devoid of Salmonella Typhi, encompassing individuals categorized as infected (I), exposed (E).

$$\begin{aligned}
\Lambda - (\lambda + \mu)S(t) &= 0 \\
\lambda S(t) - (\gamma + \mu)E(t) &= 0 \\
\gamma E(t) - (\tau + \alpha + \xi_1 + \mu)I(t) &= 0 \\
\tau I(t) - (\omega + \xi_2 + \mu)T(t) &= 0 \\
\omega T(t) - \mu R(t) &= 0 \\
\xi_1 I(t) + \xi_2 T(t) - (\eta + \phi + \mu)B(t) &= 0
\end{aligned}$$

When no spread of typhoid fever is observed, the disease classes are at $t = 0$, $\lambda S(t) - (\gamma + \mu)E(t), R(0)$, where, $S_0 = \frac{\Lambda}{(\lambda+\mu)}$. Thus, the disease-free equilibrium when state variables of $E = I = 0$ yields:

$$(S, E, I, T, R, B) = \left(S_0 = \frac{\Lambda}{\lambda + \mu}, E_0 = 0, I_0 = 0, T = 0, R_0 = 0, B = 0\right) \tag{2.13}$$

2.6. Endemic Equilibrium Point

Crucially the dynamic nature of typhoid prevalence, especially its central role in sustaining outbreaks within a population. To analyse the system at equilibrium, consider the set of equation (2.3), where the equilibrium points represent the endemic states of cholera prevalence. Let $\Phi = (S^*, E^*, I^*, T^*, R^*, B^*)$ and $t > 0$, disease endemicity where $I \neq 0$,

$$\begin{aligned}
\Lambda - (\lambda + \mu)S^*(t) &= 0 \\
\lambda S^*(t) - (\gamma + \mu)E^*(t) &= 0 \\
\gamma E^*(t) - (\tau + \alpha + \mu)I^*(t) &= 0 \\
\tau I^*(t) - (\omega + \xi_2 + \mu)T^*(t) &= 0 \\
\omega T^*(t) - \mu R^*(t) &= 0 \\
\xi_1 I^*(t) + \xi_2 T^*(t) - (\eta + \phi + \mu)B^*(t) &= 0
\end{aligned}$$

Each state variables are resolved for in respect of others where perseverance of typhoid fever spread is observed. It is obtained that $\Phi = (S^*, E^*, I^*, T^*, R^*, B^*) \neq 0$. Hence, equation (2.14) below depicts as follows;

$$\begin{aligned}
S^* &= \frac{\lambda(1+\alpha)^2\big(\tau E + \omega R^*\big)(\phi + \gamma + \mu)}{\Lambda - \mu(1+\alpha)(\xi_1 + \gamma + \mu) + (\mu + \tau + \xi_2)}, \\
E^* &= \frac{\sqrt{(\mu + \phi)(1 + \xi_1) + (1 + \xi_2)(1 + \alpha)}}{\eta - \mu(1+\alpha) + (1 - \xi_2)(\xi_1 + \gamma + \mu) - (\tau + \mu)}, \\
I^* &= \frac{(\mu + \omega + \xi_1) + \alpha(1 - \gamma)}{(\mu + \phi + \eta)\sqrt{(1+\alpha) + (1 + \xi_1)}}, \\
R^* &= \frac{(1 + \xi_1)(\gamma + \eta)I^*}{(\mu + \omega)(1 + \xi_2)(\mu + \eta + \tau)(\mu + \gamma + \xi_2)} \\
B^* &= \frac{(1 + \xi_2)(\gamma + \omega)I^*}{(\mu + \omega)\eta(1 + \xi_1)(\mu + \eta + \delta)(\mu + \gamma + \xi_1)} \\
T^* &= \frac{\Lambda\beta\rho^2(\mu + \gamma + \delta)\rho^2}{\mu^2(\delta + \mu)} - \frac{\sqrt{(\sigma + \mu + \delta)(\delta + \mu)}}{\rho(\gamma + \mu + \delta)\rho}
\end{aligned} \tag{2.14}$$

2.7. Basic Reproduction Number

The basic reproduction number, denoted R_*, measures the potential for new typhoid fever patient observed from an infected individual in a population with no prior infections. To determine the system of equations (2.3), the next generation matrix method, focusing on the infectious classes of individuals is resolved for the basic reproduction number. This involves calculating the F and V matrices representing the rates of new infections and transitions in and out of the infected compartment respectively. From the model equation (2.3), deriving these matrices as $R_* = \rho|G - \lambda I|$ where $G = FV^{-1}$ and ρ is the spectral radius of the matrix $|G - \lambda I|$. It is obtained from matrix F and V as;

$$F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j}\right), \qquad V_i = \left(\frac{\partial v_i(x_i)}{\partial x_j}\right)$$

at disease free equilibrium state,

$$\left(S = \frac{\Lambda}{(\lambda+\mu)}, 0, 0, 0, 0, 0\right), \qquad f = \begin{pmatrix} \lambda S_0 \\ 0 \end{pmatrix}, \qquad v = \begin{pmatrix} (\gamma+\mu)E(t) \\ -\gamma E(t) + (\tau+\alpha+\xi_1+\mu)I(t) \end{pmatrix}$$

Then,

$$F = \begin{pmatrix} \frac{\lambda\Lambda}{(\lambda+\mu)} & 0 \\ 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} (\gamma+\mu) & 0 \\ -\gamma & (\tau+\alpha+\xi_1+\mu) \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{\tau+\alpha+\xi_1+\mu}{\gamma+\mu} & 0 \\ \frac{\gamma}{(\gamma+\mu)(\tau+\alpha+\xi_1+\mu)} & \frac{1}{\tau+\alpha+\xi_1+\mu} \end{pmatrix}$$

Thus, the R_* is obtained as

$$R_* = \frac{\Lambda\,\phi\lambda}{(\xi_1+\tau+\alpha)(\mu+\eta+\gamma)} \tag{2.15}$$

It results that the basic reproductive ratio determines the number of infected individual migrating to the sub-population of exposed and infected, as this affect the level of recovery form the spread of typhoid fever. The leading eigenvalue of the non-invariant is the basic reproduction number of the disease model.

2.8. Quantitative Analysis of R_ε

Here, the quantitative analysis of R_* was conducted to assess its metric progression concerning each interventio method. Excluding the values of intervention parameters, assessing equation (2.15) using the stallion baseline values for its quantitative analysis provided in Table 2.1, yielding equation (2.15), subsequently the results obtained in the outcomes of these quantitative analysis is presented in Figure 2.2.

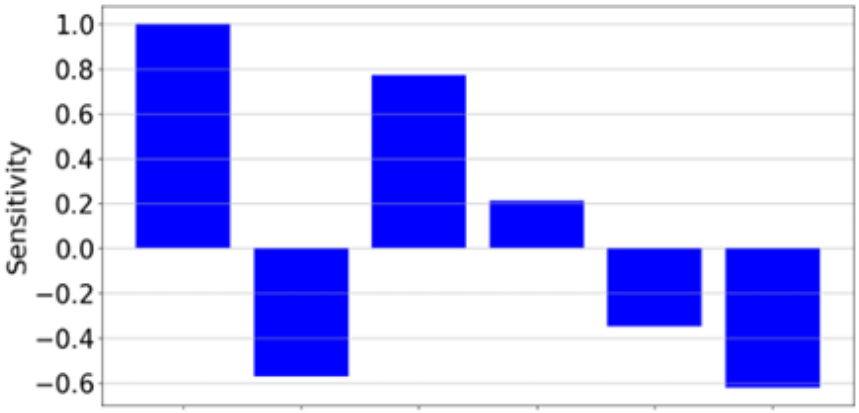

Figure 2.2: Sensitivity index of relative parameter of R_*

2.9. Local Stability of Disease Free State

Local stability of the disease free equilibrium state for typhoid fever was conducted by analyzing the minimal recurrence rate impact. When the recurrence rate is less than unity it is asymptotically stable and if more than unity is unstable and the disease prevails. To deduce when disease declines and to determine the stability using the Jacobian matrix, the characteristic equation. The disease-free state of the model is locally asymptotically stable $R_* < 1$, otherwise $R_* > 1$ if and only if the disease state prevails. The disease-free equilibrium obtained as the Jacobian matrix of the system of equation (2.3) is evaluated at the disease free state using the linearization thus;

$$J_{\varepsilon_0} = \begin{pmatrix} -(\lambda+\mu) & 0 & 0 & 0 & 0 & 0 \\ \lambda & -(\gamma+\mu) & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\tau+\alpha+\xi_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\omega+\xi_2+\mu) & 0 & 0 \\ 0 & 0 & 0 & \omega & -\mu & 0 \\ 0 & 0 & \xi_1 & \xi_2 & 0 & -(\eta+\phi+\mu) \end{pmatrix}$$

Respectively at disease free equilibrium of the model solution: computing for the eigenvalues, $|J_{E_1} - \lambda_i I| = 0$, from the Jacobian matrix the respective eigen values of the matrix can be obtained as;

$$\begin{vmatrix} -(\lambda+\mu) & 0 & 0 & 0 & 0 & 0 \\ \lambda & -(\gamma+\mu) & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\tau+\alpha+\xi_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\omega+\xi_2+\mu) & 0 & 0 \\ 0 & 0 & 0 & \omega & -\mu & 0 \\ 0 & 0 & \xi_1 & \xi_2 & 0 & -(\eta+\phi+\mu) \end{vmatrix} = 0$$

The negativity of the invariants region with respective eigen values obtained for the model equation is said to define that of asymptotical stability. The respective eigen values obtained are all negatively invariant in $\Re^6_+$

$$\begin{aligned} \lambda_1 &= -(\lambda+\mu), \quad \lambda_2 = -(\gamma+\mu), \quad \lambda_3 = -(\mu+\alpha+\xi_1+\tau), \\ \lambda_4 &= -(\mu+\xi_2+\omega), \quad \lambda_5 = -\mu, \quad \lambda_6 = -(\eta+\phi+\mu) \end{aligned} \tag{2.16}$$

2.9.1. Local Stability of Endemic Equilibrium

The regional resilience of the persistent equilibrium point of the model equation (2.3) is locally asymptotically stable if $\Re_* < 1$ and unstable otherwise if $\Re_* > 1$. Suppose,

$$S = x + S^*, \qquad E = y + E^*, \qquad I = z + I^*, \qquad T = a + T^*, \qquad R = b + R^*, \qquad B = c + B^*.$$

Linearizing equation (2.3), is then obtained as

$$\begin{aligned}
S^*(t) &= \Lambda - (\lambda + \mu)S(t) + \text{higher order} + \text{nonlinear terms}\ldots \\
E^*(t) &= \lambda S(t) - (\gamma + \mu)E(t) + \text{higher order} + \text{nonlinear terms}\ldots \\
I^*(t) &= \gamma E(t) - (\tau + \alpha + \xi_1 + \mu)I(t) + \text{higher order} + \text{nonlinear terms}\ldots \\
T^*(t) &= \tau I(t) - (\omega + \xi_2 + \mu)T(t) + \text{higher order} + \text{nonlinear terms}\ldots \\
R^*(t) &= \omega T(t) - \mu R(t) + \text{higher order} + \text{nonlinear terms}\ldots \\
B^*(t) &= \xi_1 I(t) + \xi_2 T(t) - (\eta + \phi + \mu)B(t) + \text{higher order} + \text{nonlinear terms}\ldots
\end{aligned}$$

Jacobian matrix of the system,

$$\begin{vmatrix}
-(\lambda+\mu) & 0 & 0 & 0 & 0 & 0 \\
\lambda & -(\gamma+\mu) & 0 & 0 & 0 & 0 \\
0 & \gamma & -(\tau+\alpha+\xi_1+\mu) & 0 & 0 & 0 \\
0 & 0 & \tau & -(\omega+\xi_2+\mu) & 0 & 0 \\
0 & 0 & 0 & \omega & -\mu & 0 \\
0 & 0 & \xi_1 & \xi_2 & 0 & -(\eta+\phi+\mu)
\end{vmatrix} = 0 \quad (2.17)$$

The resulting eigenvalue of the above matrix is obtained as;

$$\begin{aligned}
a &= -(\lambda+\mu), \qquad b = -(\gamma+\mu), \qquad c = -(\mu+\alpha+\xi_1+\tau), \\
d &= -(\mu+\xi_2+\omega), \qquad e = -\mu, \qquad f = -(\eta+\phi+\mu).
\end{aligned}$$

Therefore, the persistent resilience of the respective eigen values obtained for the model whose trace result from the Jacobian matrix representation of individual characteristic values are in the model invariance region of $\Re_6^+$ and is asymptotically stable for all $\lambda_i < 0$.

2.9.2. Global Stability of Disease Free Equilibrium

In this section, we employ the concept of Lyapunov function to establish the global asymptotic stability for the disease dynamics at equilibrium point. However, if $\Re_* < 1$ and unstable otherwise if $\Re_* > 1$. Therefore,

$$\Psi(t, S, E, I, T, R, B) = C_1 I_1 + C_2 I_2 \quad (2.18)$$

$$\begin{aligned}
\frac{d\Psi}{dt} &= C_1\dot{I}_1 + C_2\dot{I}_2 = C_1\big(\lambda S_0(t) - (\gamma+\mu)I_1\big) + C_2\big(\gamma I_1(t) - (\tau+\alpha+\xi_1+\mu)I_2\big) \\
\frac{d\Psi}{dt} &\leq \left(C_2\frac{\lambda\Lambda}{(\mu+\lambda)(\alpha+\xi_1+\xi_2)} - (\gamma+\mu+\phi)\right)I_1 - C_2\big((\tau+\mu)+(\mu+\gamma+\eta)\big)I_2 \\
\frac{d\Psi}{dt} &\leq \left(C_2\frac{\lambda\Lambda}{(\mu+\lambda)(\alpha+\xi_1+\xi_2)} - C_1(\gamma+\mu+\phi)\right)I_1 - \big(C_1(\tau+\mu)+(\mu+\gamma+\eta)\big)I_2
\end{aligned}$$

$$C_1 = \frac{1}{(\gamma+\mu+\phi)}, \qquad C_2 = \frac{(\gamma+\eta+\mu)}{\mu(\mu+\xi_1)(\mu+\gamma+\alpha)}, \qquad B \leq 0$$

$$\begin{aligned}\frac{d\Psi}{dt} &\leq \left(\frac{\Lambda\phi\lambda}{(\xi_1+\tau+\alpha)(\mu+\eta+\gamma)} - \frac{(\gamma+\mu+\phi)}{(\gamma+\mu+\phi)}\right) I_1 \\ &\quad + \left(\frac{(\gamma+\eta+\mu)}{\mu(\mu+\xi_1)(\mu+\gamma+\alpha)} - \frac{(\gamma+\eta+\mu)}{\mu(\mu+\xi_1)(\mu+\gamma+\alpha)}\right) I_2 \\ \frac{d\Psi}{dt} &\leq (R_0-1) \end{aligned} \tag{2.19}$$

It is pertinent that when $t \to \infty$, $\Psi^* \leq 0$. Substituting into the model equation (2.4) reveals that based on LaSalle's principle $\Psi^* = 0$. Hence, it is globally asymptotically stable whenever $R_* > 1$.

2.9.3. Global Stability of Endemic Equilibrium

Theorem 2.4. *The Dulac criterion is a method used in dynamical systems to determine the absence of periodic orbits in a given region of the phase plane, which can be extended to analyze the global stability of an equilibrium point.*

Proof. For a dynamical system described by the differential equations:

$$\frac{dx}{dt} = f(x,y) \;\Leftrightarrow\; \frac{dy}{dt} = g(x,y) \tag{2.20}$$

The Dulac criterion states that if there exists a continuously differentiable function $B(x,y)$ (called the Dulac function) such that the expression:

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) \tag{2.21}$$

is either strictly positive or strictly negative throughout a simply connected region D of the phase plane, then there are no closed trajectories (periodic orbits) contained entirely within D.

To apply this to determine the global stability of an endemic equilibrium (x^*, y^*) of a mathematical model, the endemic equilibrium point (x^*, y^*). Also define the Dulac function $B(x,y)$ and the expression

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) = B(x,y)g(x,y) \tag{2.22}$$

This shows that this expression is of one sign (either strictly positive or strictly negative) in the region of interest. If such a Dulac function $B(x,y)$ can be found, the system has no periodic orbits in that region, suggesting the global stability of the endemic equilibrium if no other attractors exist. Hence, if $\exists B(x,y) \in C^1$ such that

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) \neq 0 \quad \text{in } D.$$

Then there are no closed trajectories in D. This criterion is useful in proving the global stability of the endemic equilibrium when combined with other stability analysis techniques.

The concept of Dulac's criterion was employed. Let $\varpi = (S, E, I, T, R, B)$ define the Dulac's function $G = \frac{1}{SI}$. The following system of equation are obtained;

$$\begin{aligned}
G\frac{dS}{dt} &= \frac{1}{SI}\left\{\Lambda - (\lambda+\mu)S(t)\right\},\\
G\frac{dE}{dt} &= \frac{1}{SI}\left\{\lambda S(t) - (\gamma+\mu)E(t)\right\},\\
G\frac{dI}{dt} &= \frac{1}{SI}\left\{\gamma E(t) - (\tau+\alpha+\xi_1+\mu)I(t)\right\},\\
G\frac{dT}{dt} &= \frac{1}{SI}\left\{\pi I(t) - (\omega+\xi_2+\mu)T(t)\right\},\\
G\frac{dR}{dt} &= \frac{1}{SI}\left\{\omega T(t) - \mu R(t)\right\},\\
G\frac{dB}{dt} &= \frac{1}{SI}\left\{\xi_1 I(t) + \xi_2 T(t) - (\eta+\phi+\mu)B(t)\right\}
\end{aligned}$$

The above system of equations results when resolved by Dulac derivatives yield to;

$$\begin{aligned}
G\frac{dS}{dt} &= \left\{\frac{\Lambda}{SI} - \frac{(\mu+\alpha)}{1+\alpha} + \frac{\xi_1}{SI} - \frac{\mu}{I}\right\},\\
G\frac{dE}{dt} &= \left\{\frac{(\eta+\phi+\xi_2)}{(\mu+\alpha)} - \frac{(\xi_2+\mu)(\mu+\eta+\phi)E}{SI}\right\},\\
G\frac{dI}{dt} &= \left\{\frac{(1-\xi_2)E}{SI} - \frac{(1+\xi_1)(\mu+\gamma+\tau)}{S} - \frac{\delta R}{SI}\right\},\\
G\frac{dR}{dt} &= \left\{\frac{(1-u_3)(\gamma+T)}{S} + \frac{(\mu+\delta+\eta)R}{SI}\right\},\\
G\frac{dB}{dt} &= \left\{\frac{\eta R}{SI} - \frac{(\mu+\gamma+\tau)}{SI}\right\}
\end{aligned}$$

At $t > 0$ orbital resolution of the system of equations is given by $\frac{d(G\varpi)}{dt}$ as obtained below.

$$\begin{aligned}
\frac{d(G\varpi)}{dt} &= \frac{\partial}{\partial S}\left\{G\frac{dS}{dt}\right\} + \frac{\partial}{\partial E}\left\{G\frac{dE}{dt}\right\} + \frac{\partial}{\partial I}\left\{G\frac{dI}{dt}\right\} + \frac{\partial}{\partial R}\left\{G\frac{dR}{dt}\right\} + \frac{\partial}{\partial M}\left\{G\frac{dB}{dt}\right\}\\
&= \frac{\partial}{\partial S}\left\{\frac{\lambda}{SI} - \frac{\lambda-(1-\eta)\phi}{1+\alpha} + \frac{\pi B}{SI} - \frac{\mu}{I}\right\} + \frac{\partial}{\partial E}\left\{\frac{(1-\xi_1)\phi}{1+\alpha} - \frac{(1-\xi_2)(\mu+\alpha)(\mu+\tau)E}{SI}\right\}\\
&\quad + \frac{\partial}{\partial I}\left\{\frac{(1-\xi_1)E}{SI} - \frac{(1-\xi_2)(\mu+\gamma+\alpha)}{S} - \frac{\delta R}{SI}\right\} + \frac{\partial}{\partial R}\left\{\frac{(1-\xi_1)(\gamma+\eta)}{S} + \frac{(\mu+\alpha+\eta)R}{SI}\right\}\\
&\quad + \frac{\partial}{\partial R}\left\{\frac{\eta R}{SI} - \frac{(1-\xi_1)B}{SI}\right\}\\
\frac{d(G\varpi)}{dt} &= \frac{\partial}{\partial S}\left\{G\frac{dS}{dt}\right\} + \frac{\partial}{\partial E}\left\{G\frac{dE}{dt}\right\} + \frac{\partial}{\partial I}\left\{G\frac{dI}{dt}\right\} + \frac{\partial}{\partial R}\left\{G\frac{dR}{dt}\right\} + \frac{\partial}{\partial R}\left\{G\frac{dB}{dt}\right\}\\
\frac{d(GX)}{dt} &= \frac{\partial}{\partial S}\left\{\frac{\lambda}{SI} - \frac{\lambda-(1-\xi_1)\beta}{1+\alpha} + \frac{\eta B}{SI} - \frac{\mu}{I}\right\} + \frac{\partial}{\partial E}\left\{\frac{(1-\xi_2)}{1+\alpha} - \frac{(\mu+\alpha)(\mu+\phi)E}{SI}\right\}\\
&\quad + \frac{\partial}{\partial I}\left\{\frac{(1-\xi_2)\varepsilon E}{SI} - \frac{(1-\xi_1)(\mu+\gamma+\phi)}{S} - \frac{\delta R}{SI}\right\} + \frac{\partial}{\partial R}\left\{\frac{(\gamma+\eta)}{S} + \frac{(\mu+\xi_2+\eta)R}{SI}\right\}\\
&\quad + \frac{\partial}{\partial R}\left\{\frac{\eta R}{SI} - \frac{\alpha B}{SI}\right\}
\end{aligned}$$

$$\begin{aligned}
\frac{d(G\varpi)}{dt} &= \frac{\partial}{\partial S}\left\{\frac{\lambda-(1-\xi_1)\gamma+\kappa M_t-\mu}{SI(1+\alpha)}\right\}+\frac{\partial}{\partial E}\left\{\frac{(1-\xi_2)\gamma+(1-\xi_1)\gamma(\mu+\gamma)E}{SI(1+\alpha)}\right\} \\
&+\frac{\partial}{\partial I}\left\{\frac{-(1-u_2)\varepsilon+(1-u_3)(\mu+\gamma+T)-\delta}{SI}\right\}+\frac{\partial}{\partial R}\left\{\frac{(1-u_3)(\gamma+T)+(\mu+\delta+\eta)}{S}\right\} \\
&+\frac{\partial}{\partial R}\left\{\frac{\phi R-\gamma B}{SI}\right\} \\
\frac{d(GX)}{dt} &= -\left\{\frac{\lambda-(1-\xi_1)(1-\xi_2)(\mu+\gamma)+\alpha B-\lambda}{SI}-\sqrt{(1-\xi_2)(\mu+\gamma+\bar{\phi})(1-\xi_1)}\right\}<0 \qquad (2.23)
\end{aligned}$$

This result indicates that the system lacks closed orbits, meaning there are no periodic fluctuations in the number of infected individuals. Epidemiologically, this suggests that sustained oscillations in typhoid fever cases do not occur, underscoring the importance of treatment as a primary control strategy. By focusing on typhoid fever treatment, resource allocation can be optimized to effectively reduce and eventually halt the rapid spread of the disease with time. □

2.9.4. Sensitivity Analysis of R_*

The primary aim is to assess the sensitivity of the basic reproduction number, by computing its derivative concerning all relevant parameters. This analysis will result in the determination of the normalized forward sensitivity index, denoted as in equation (2.13) and depicted in Table 2.2 below.

$$\begin{aligned}
\frac{\partial R_*}{\partial \tau} &= \frac{\partial R_*}{\partial \tau}\times\frac{\tau}{R_*}=0.2873, \quad \frac{\partial R_0}{\partial \alpha}=\frac{\partial R_0}{\partial \alpha}\times\frac{\alpha}{R_0}=1.9343, \quad \frac{\partial R_*}{\partial \lambda}=\frac{\partial R_*}{\partial \lambda}\times\frac{\lambda}{R_*}=1.0938 \\
\frac{\partial R_*}{\partial \gamma} &= \frac{\partial R_*}{\partial \gamma}\times\frac{\gamma}{R_*}=0.2013, \quad \frac{\partial R_*}{\partial \xi_2}=\frac{\partial R_*}{\partial \xi_2}\times\frac{\xi_2}{R_*}=0.1874, \\
\frac{\partial R_*}{\partial \mu} &= \frac{\partial R_*}{\partial \mu}\times\frac{\mu}{R_*}=-0.1535, \quad \frac{\partial R_*}{\partial \omega}=\frac{\partial R_*}{\partial \omega}\times\frac{\omega}{R_*}=1.7321, \\
\frac{\partial R_*}{\partial \xi_1} &= \frac{\partial R_*}{\partial \xi_1}\times\frac{\xi_1}{R_*}=0.4210, \quad \frac{\partial R_*}{\partial \phi}=\frac{\partial R_*}{\partial \phi}\times\frac{\phi}{R_*}=0.0006 \qquad (2.24)
\end{aligned}$$

Table 2.2: Sensitivity analysis and parameter indices

Parameters	Sensitivity indices
τ	0.2873 per week
α	1.9343 per week
μ	-0.1535 per week
λ	1.0938 per week
ξ_1	0.4210 per week
γ	0.2013 per week
ξ_2	0.1874 per week
ϕ	0.0006 per week
ω	1.7321 per week

As obtained from Table 2.2 above, it shows that the sensitivity indices are positively invariant in $\Re^5_+$. The sensitivity indices depend on the values of each parameter of R_*, and this brings about changes in the values that will affect the behaviour of the threshold on the spread or vanity of typhoid disease. Based on the table, we can conclude that parameters ω and α are the most sensitive to the basic reproduction number of the typhoid model. Particularly, increasing the value of ξ_2 will result in a 96.96% increase in R_*, while increasing the value of ξ_1 will lead to a 91.52% decrease in R_*.

3. Numerical Simulation

Consider the differential equation in the general form

$$Ly(x) + Ry(x) + Ny(x) = f(x) \tag{3.25}$$

where L is the linear operator of the highest-order derivative which is assumed to be invertible easily, R is also a linear operator of order less than L, and $Ny(x)$ indicates the non-linear term and f is the source term. Thus applying the inverse operator L^{-1} to the above equation to obtain

$$y(x) = g_0 + L^{-1}\big(f(x) - Ry(x) - Ny(x)\big) \tag{3.26}$$

where g_0 is the solution of the homogeneous equation, $Ly(x) = 0$ The constants of integration involved in the solution of homogeneous are to be determined by the initial conditions, according to the problem, whether it is an initial value problem or boundary value problem. According to Laplace Adomian Decomposition Method (LADM), the solution of the unknown function $y(x)$ can be expressed by an infinite series of the form

$$y(x) = \sum_{n=0}^{\infty} y_n x \tag{3.27}$$

and the non-linear term can be decomposed by the infinite series of the form

$$Ny(x) = \sum_{n=0}^{\infty} A_n,$$

and $A_n x$ are called Adomian polynomials, which can be determined by the algorithm. Substituting into the above equation to obtain;

$$\sum_{n=0}^{\infty} y_n x = g_0 + L^{-1}\left[f(x) - R\sum_{n=0}^{\infty} y_n x - \sum_{n=0}^{\infty} A_n\right] \tag{3.28}$$

where the components $(y_0, y_1, y_2, y_3, y_4 \dots y_n)$, are determined by the recursive relation $y_0 = g_0$, $y_{k+1} = L^{-1}(Ry_k) - L^{-1}(A_k),\ k \geq 0$. Given the model solution via the application of Laplace Adomian Method. Conducting a numerical simulation on the typhoid disease model, the following iterative scheme of Laplace Adomian decomposition method for the model equation is considered. The Laplace Adomian decomposition method was employed to computationally analyse the epidemic model. Maple software facilitated the generation of iteration formulas for each compartment. These formulas were then iteratively solved, enabling the numerical evaluation of the model's dynamics and providing insights into the epidemic's behaviour and progression. Taking the Laplace transform of both sides of the above equation.

$$\begin{aligned}
L\left[\frac{dS}{dt}\right] &= L[\Lambda] - L[(\lambda+\mu)S(t)] \\
L\left[\frac{dE}{dt}\right] &= L[\lambda S(t)] - L[(\gamma+\mu)E(t)] \\
L\left[\frac{dI}{dt}\right] &= L[\gamma E(t)] - L[(\tau+\alpha+\xi_1+\mu)I(t)] \\
L\left[\frac{dT}{dt}\right] &= L[\pi I(t)] - L[(\omega+\xi_2+\mu)T(t)] \\
L\left[\frac{dR}{dt}\right] &= L[\omega T(t)] - L[\mu R(t)] \\
L\left[\frac{dB}{dt}\right] &= L[\xi_1 I(t) + \xi_2 T(t)] - L[(\eta+\phi+\mu)B(t)]
\end{aligned} \tag{3.29}$$

Substituting from equation (2.2) into (3.29) to yield

$$\begin{aligned} mL[S(t)] &= S(0) + L[\Lambda] - L[(\lambda+\mu)S(t)] \\ mL[E(t)] &= E(0) + L[\lambda S(t)] - L[(\gamma+\mu)E(t)] \\ mL[I(t)] &= I(0) + L[\gamma E(t)] - L[(\tau+\alpha+\xi_1+\mu)I(t)] \\ mL[T(t)] &= T(0) + L[\pi I(t)] - L[(\omega+\xi_2+\mu)T(t)] \\ mL[R(t)] &= R(0) + L[\omega T(t)] - L[\mu R(t)] \\ mL[B(t)] &= B(0) + L[\xi_1 I(t) + \xi_2 T(t)] - L[(\eta+\phi+\mu)B(t)] \end{aligned} \tag{3.30}$$

where $S(0) = s_0,\ E(0) = e_0,\ T(0) = t_0,\ I(0) = i_0,\ R(0) = r_0,\ B(0) = b_0.$

$$\begin{aligned} L[S(t)] &= \frac{s_0}{m} + \frac{\Lambda}{m^2} + L\big[(\lambda+\mu)S(t)\big] \\ L[E(t)] &= \frac{e_0}{m} + \frac{1}{m}L\big[\lambda S(t)\big] - L\big[(\gamma+\mu)E(t)\big] \\ L[I(t)] &= \frac{i_0}{m} + \frac{1}{m}L\big[\gamma E(t)\big] - L\big[(\tau+\alpha+\xi_1+\mu)I(t)\big] \\ L[T(t)] &= \frac{t_0}{m} + \frac{1}{m}L\big[\pi I(t)\big] - L\big[(\omega+\xi_2+\mu)T(t)\big] \\ L[R(t)] &= \frac{r_0}{m} + \frac{1}{m}L\big[\omega T(t)\big] - L\big[\mu R(t)\big] \\ L[B(t)] &= \frac{b_0}{m} + \frac{1}{m}L\big[\xi_1 I(t) + \xi_2 T(t)\big] - L\big[(\eta+\phi+\mu)B(t)\big] \end{aligned}$$

Letting the non-linear terms in the above iteration and substitutes by taking the inverse Laplace transform of both sides,

$$\begin{aligned} S(t) &= s_0 + \Pi t + L^{-1}\left(\frac{1}{m}[\beta_2 V] - L[\beta_1 S + \beta S I_1 + \alpha S I_2 + (\tau+\omega)S + \mu S]\right) \\ V(t) &= v_0 + L^{-1}\left(\frac{1}{m}L[\beta_1 S] - L[(\beta_2+\mu)V]\right) \\ E_1(t) &= e_0 + L^{-1}\left(\frac{1}{m} + L[\beta S I_1] - L[(c+\eta+\mu)E_1]\right) \\ E_2(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L[\alpha S I_2] - L[(\sigma+\mu)E_2]\right) \\ I_1(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L[\tau S + (c+\eta)E_1] - L[(\varepsilon+\delta_1+r+\mu)I_1]\right) \\ I_2(t) &= r_0 + L^{-1}\left(\frac{1}{m} + L[\sigma E_2] - L[(\delta_2\rho+\mu)I_2]\right) \\ R(t) &= r_0 + L^{-1}\left(\frac{1}{m}L[\alpha S + (r+\varepsilon)I_1 + \rho I_2] - L[\mu R]\right) \end{aligned} \tag{3.31}$$

Subsequently, iteration result obtained from the above equation of systems is deduced as;

$$\begin{aligned} \sum_{k=0}^{\infty} S_n(t) &= s_0 + \Lambda t + L^{-1}\left(\frac{1}{m}\left[\beta_2 \sum_{k=0}^{\infty} E_n\right] - L\left[\beta_1 \sum_{k=0}^{\infty} S_n + \phi \sum_{k=0}^{\infty} S I_n + \alpha \sum_{k=0}^{\infty} S I_n \right.\right. \\ &\quad \left.\left. + \big[(\tau+\omega+\mu)\big] \sum_{k=0}^{\infty} S_n\right]\right) \\ \sum_{k=0}^{\infty} E_n(t) &= e_0 + L^{-1}\left(\frac{1}{m}L\left[\xi_1 \sum_{k=0}^{\infty} S_n\right] - L\left[(\xi_2+\mu)\sum_{k=0}^{\infty} E_n\right]\right) \end{aligned}$$

$$\begin{aligned}
\sum_{k=0}^{\infty} I(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L\left[\gamma\sum_{k=0}^{\infty} SI_n\right] - L\left[(\alpha+\eta+\mu)\sum_{k=0}^{\infty} I\right]\right)\\
\sum_{k=0}^{\infty} T(t) &= t_0 + L^{-1}\left(\frac{1}{m} + L\left[\alpha\sum_{k=0}^{\infty} SI_2\right] - L\left[(\eta+\mu)\sum_{k=0}^{\infty} T\right]\right)\\
\sum_{k=0}^{\infty} R(t) &= r_0 + L^{-1}\left(\frac{1}{m} + L\left[\tau\sum_{k=0}^{\infty} S_n + (\omega+\eta)\sum_{k=0}^{\infty} E_1\right] - L\left[(\phi+\xi_1+\xi_2+\mu)\sum_{k=0}^{\infty} R\right]\right)\\
\sum_{k=0}^{\infty} B(t) &= b_0 + L^{-1}\left(\frac{1}{m}L\left[\omega\sum_{k=0}^{\infty} S + (\alpha+\mu)\sum_{k=0}^{\infty} I_1 + \lambda\sum_{k=0}^{\infty} I\right] - L\left[\mu\sum_{k=0}^{\infty} B\right]\right) \qquad (3.32)
\end{aligned}$$

The initial approximations of each class are given by;

$$S_0(t) = s_0,\ E_0(t) = e_0,\ I_0(t) = i_0,\ T(t) = t_0,\ R_0(t) = r_0,\ B_0(t) = b_0$$

Now, comparing the coefficients at $n = 1, 2, 3$. Using the recurrence relations obtained from the iterations. Compartmentally it is obtained that

$$\begin{aligned}
D^{\eta_1}S(t) &= \Lambda - \frac{1}{2}\xi_1 s_0 i_0 - \frac{2}{3}\xi_2 s_1 e_1 - \alpha s_1 s_2 - \frac{3}{5}(\tau+\omega)s_0 + \xi_1 v_0 - \mu s_0\\
D^{\eta_1}E(t) &= \xi_1 s_0 - \frac{1}{3}(\xi_2+\mu)e_0\\
D^{\eta_1}I(t) &= \frac{1}{2}\phi s_0 i_0 - \frac{2}{3}(c+\eta+\mu)e_0 I\\
D^{\eta_1}T(t) &= \alpha s_0 i_0 + \tau t_0 e_0 - (\gamma+\mu)t_0\\
D^{\eta_1}R^*(t) &= \phi e_0 s_0 - \frac{1}{5}(\phi+\omega+\mu)r_0\\
D^{\eta_1}B(t) &= -\frac{1}{2}\tau s_0 r_0 + (\gamma+\eta+\mu)e_1 i_1 - (\xi_1+\eta+\mu)i_0 b_1
\end{aligned}$$

Further iterations are done to obtain successive iterative terms at $n = 2$

$$\begin{aligned}
S_2(t) &= \left(\frac{1}{2}\alpha^2 i^2 s_0 + \frac{1}{2}\alpha i s_0 + \frac{1}{2}\alpha i s_0\mu_0 + \frac{1}{2}\alpha i s_0\rho_0 - \frac{1}{2}\alpha i s_0 e_0 + \frac{1}{2}\alpha i s_0\beta_1 - \frac{1}{2}\alpha i s_0\xi_2\right.\\
&\qquad \left. + \frac{1}{2}\mu^2 s_0 + \xi_2\mu s_0 + \xi_2\mu v_0 + \frac{1}{2}\xi_2 s_0 + \frac{1}{2}\xi_2 s_0 - \frac{1}{2}\xi_2 v_0 - \frac{1}{2}\xi_2 v_0\right)t^2\\
E_2(t) &= \left(-\frac{1}{2}\alpha i s_0\xi_2 + \frac{1}{2}\mu^2 v_0 - \xi_2\mu s_0 + \xi_1\mu s_0 - \frac{1}{2}\xi_1 s_0 + \frac{1}{2}\xi_2 s_0 - \frac{1}{2}\gamma v_0 + \frac{1}{2}\phi^2 v_0\right)t^2\\
&\quad + \left(-\frac{1}{6}\alpha i_0\pi\xi_2 - \frac{1}{3}\xi_2\mu\eta - \frac{1}{6}\xi_1^2\omega + \frac{1}{6}\omega\eta\right)t^3\\
I(t) &= \left(-\frac{1}{6}\alpha^2 i^2\omega - \frac{1}{3}\alpha i_0\omega - \frac{2}{3}\alpha i_0\mu - \frac{1}{3}\alpha i_0\omega + \frac{1}{3}\alpha e_0\pi\omega_1 - \frac{1}{6}\mu^2\pi - \frac{1}{6}\alpha i_0\omega\gamma_1\right)t^3\\
&\quad + \left(-\frac{1}{2}\alpha^2 i^2 s_0 - \frac{1}{2}\sigma i s_0 - \frac{2}{3}\alpha i s_0\mu_0 - \frac{1}{2}\alpha i s_0\omega_0 + \frac{1}{2}\alpha i s_0 b_0 - \mu^2 i e_0\omega + \frac{1}{2}\alpha i e_0\omega^2\right)t^2
\end{aligned}$$

$$
\begin{aligned}
T(t) &= \left(-\frac{1}{6}\alpha^2 i^2\omega - \frac{1}{3}\alpha i_0\gamma - \frac{2}{3}\alpha i_0\pi\mu - \frac{1}{3}\alpha i_0\omega + \frac{1}{3}\alpha e_0\eta_1 - \frac{1}{6}\mu^2\pi - \frac{1}{6}\alpha i_0\omega_1\right)t^3 \\
&\quad + \left(-\frac{1}{2}\alpha^2 i^2 s_0 - \frac{1}{2}\sigma i s_0 - \frac{2}{3}\alpha i s_0\mu_0 - \frac{1}{2}\alpha i s_0 + \frac{1}{2}\alpha i s_0 v_0 - \mu^2 i e_0\beta_1 + \frac{1}{2}\alpha i e_0\alpha^2\right)t^2 \\
R(t) &= -\frac{1}{6}\alpha^2 i^2 + \left(\frac{1}{2}\alpha i s_0 + \frac{1}{2}\gamma^2 i_0 + \xi\mu i_0 - \frac{1}{2}\xi\sigma i e_0 + \frac{1}{2}\mu^2 i_0 - \mu\omega i_0\right. \\
&\quad \left. -\mu\sigma i_0 + \frac{1}{2}\omega^2 i_0 - \frac{1}{2}\phi\sigma i e_0 - \frac{1}{2}\xi^2 e_0\right)t^2
\end{aligned} \tag{3.33}
$$

and so on. This can be further till desired number of iterations are obtained. Thus, the obtained raw solution to each model compartment is obtained as;

$$
\begin{aligned}
S(t) &= \sum_{k=0}^{3} s_k(t), \qquad E(t) = \sum_{k=0}^{3} e_k(t), \qquad I(t) = \sum_{k=0}^{3} i_k(t), \\
T(t) &= \sum_{k=0}^{3} t_k(t), \qquad R(t) = \sum_{k=0}^{3} r_k(t), \qquad B(t) = \sum_{k=0}^{3} b_k(t)
\end{aligned} \tag{3.34}
$$

Evaluating these series results using the corresponding variables and parameter values for the iterative terms of (3.34), the following equation is obtained.

$$
\begin{aligned}
S(t) &= 0.004671030532t^5 + 2.913084517t^4 + 97.15316451t^3 - 89.2522072t^2 + 38.399597t + 23.09, \\
E(t) &= -3.781930532t^5 + 2.9130844517t^4 + 97.15341651t^3 + 89.25220726t^2 - 38.399597t + 1369, \\
I(t) &= 3.78193052t^5 - 3.2742949239t^4 - 97.15341651t^3 - 89.25220726t^2 - 38.399597t + 163.93, \\
T(t) &= -3.924584378t^5 + 3.931713891t^4 + 72.783672672t^3 + 88.257658358t^2 + 40.538721t + 27.15, \\
R(t) &= 0.004671030532t^5 + 3.052597940t^4 + 107.1176562t^3 - 91.6099077t^2 + 38.399597t + 883.2, \\
B(t) &= -3.7813532t^5 - 3.482003841t^4 + 138.4166085t^3 + 98.68300935t^2 - 38.399597t + 209.4.
\end{aligned}
$$

4. Optimal Control Analysis

This mathematical model of typhoid fever disease control mechanism was modified by incorporating four control interventions. This will help us to identify the best intervention strategies that needs to eradicate the disease within the community. The control intervention are defined as follows; ξ_1 typhoid prevention efforts such as, proper use of water purifying agents to protect susceptible individuals from contracting the disease, ξ_2 typhoid fever prevention efforts by consuming well cooked and hygienic food substance. With the above assumptions, to study the optimal levels of the controls, the control set U is Lebesgue measurable and it is define as:

$$U : \{(\xi_1(t), \xi_2(t)) : 0 \leq \xi_1 < 1, 0 \leq \xi_1 < 1, 0 \leq u_3 \leq t \leq 0\}$$

The aim is to obtain a control U and state variables $(seitrb)$ and I_V that minimize the cost of control and the objective functional J given by:

$$J(\xi_1, \xi_2) = \int_{n=1}^{t} \left(H_S I_S + H_E E_E + H_I I_I + H_T R_T + H_B B_B + \frac{b_1}{2}\xi_1^2 + \frac{b_2}{2}\xi_2^2\right) dt \tag{4.35}$$

where H_1, H_2 and b_i are positive. The expression $\frac{1}{2}b_i u_i^2$, such that $i = 1, 2 \ldots 5$, represents cost which are associated with the controls u_i. The quadratic form is chosen because it assumes that costs are non-linear in nature. The aim is to minimize the number of infectious and costs of control. Thus, intention is to obtain an optimal controls (ξ_1^*, ξ_2^*) in which:

$$J(\xi_1^*, \xi_2^*) = \min\left\{J\left[\left(\frac{(\xi_1, \xi_2)}{u_i}\right) \in U\right]\right\}, \tag{4.36}$$

where $U = (\xi_1, \xi_2) \in \xi_i$ is measurable with $0 \leq \xi_i < 1$, for $0 \leq t \leq t_f$.

The Hamilton and Optimality System of Typhoid Disease

Consider using the principle of Pontryagin's Maximum Principle, obtaining a Hamiltonian (H) defined as:

$$\begin{aligned} H_i &= H_S I_S + H_E E_E + H_I I_I + H_T R_T + H_B B_B + \frac{b_1}{2}\xi_1^2 + \frac{b_2}{2}\xi_2^2 + \lambda_1\frac{dS}{dt} + \lambda_2\frac{dE}{dt} \\ &\quad +\lambda_3\frac{dI}{dt} + \lambda_4\frac{dT}{dt} + \lambda_5\frac{dR}{dt} + \lambda_6\frac{dB}{dt} \end{aligned}$$

to determine the adjoint variable by applying Pontryagin's Maximum Principle for existence of the optimal control pairs.

Theorem 4.1. *Given an optimal control (ξ_1^*, ξ_2^*) and the solutions $(s^*, e^*, i^*, t^*, r^*, b^*)$ of the corresponding state system of the formulated model that minimizes $J(\xi_1, \xi_2)$ over U. Then, there exist adjoint variables $(\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6)$ such that;*

$$\begin{aligned} \frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial S} - H_1 + \lambda_1\big(\Lambda - (\lambda + \mu)S(t)\big), \\ \frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial E} - H_2 + \lambda_2\big(\lambda S(t) - (\gamma + \mu)E(t)\big), \\ \frac{d\lambda_3}{dt} &= -\frac{\partial H}{\partial I} - H_2 + \lambda_4\big(\gamma E(t) - (\tau + \alpha + \xi_1 + \mu)I(t)\big), \\ \frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial R} - H_3 - \lambda_2\big(\pi I(t) - (\omega + \xi_2 + \mu)T(t)\big) \\ \frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial R} - -H_3 - \lambda_2\big(\omega T(t) - \mu R(t)\big) \\ \frac{d\lambda_6}{dt} &= -\frac{\partial H}{\partial B} - H_3 - \lambda_2\big(\xi_1 I(t) + \xi_2 T(t) - (\eta + \phi + \mu)B(t)\big) \end{aligned} \tag{4.37}$$

with transversally conditions, $\lambda_i(t_i) = 0,\ i = 1, 2, 3 \ldots$ and the characterized control set of (ξ_1^*, ξ_2^*) is;

$$\begin{aligned} u_1^* &= \max\left\{0, \min\left[1, \left(\frac{(\lambda_3(1 + \xi_1) + \lambda_2\xi_1 - \lambda_1)(s + e + i + t + r + b)}{a_1}\right)\right]\right\}, \\ u_2^* &= \max\left\{0, \min\left[1, \left(\frac{(\lambda_1(1 + \xi_2) + \lambda_2\xi_2 - \lambda_1)\eta\phi I}{a_2}\right)\right]\right\}, \\ u_3^* &= \max\left\{0, \min\left[1, \left(\frac{(\lambda_2(1 + \xi_1) + \lambda_3\xi_2 - \lambda_1)\eta(\phi + \mu)}{a_3}\right)\right]\right\} \end{aligned} \tag{4.38}$$

Proof. The adjoint equation and transversality conditions are standard results from Pontryagin's maximum principle. Differentiate the Hamiltonian with respect to each states variable as follow: And also for characterization of the optimal control taking the following partial differential equation:

$$\frac{\partial H}{\partial u_i} = 0,\ u_i = u_i^*,\ \text{where } i = 1,2,3.\ \text{At } i = 1,\ \frac{\partial H}{\partial u_1} = 0,\ \text{when } u_1 = u_1^*$$

$$\begin{aligned}
u_1^* &= \frac{\big(\lambda_3(1+\xi_1) + \lambda_2\xi_1 - \lambda_1\big)(s+e+i+t+r+b)}{a_1},\ \text{at } i = 1,\ \frac{\partial H}{\partial u_2} = 0,\ \text{when } u_1 = u_1^* \\
u_2^* &= \frac{\big(\lambda_1(1+\xi_2) + \lambda_2\xi_2 - \lambda_1\big)\eta\phi I}{a_2},\ \text{also at } i = 2,\ \frac{\partial H}{\partial u_2} = 0,\ \text{when } u_2 = u_2^* \\
u_3^* &= \frac{\big(\lambda_2(1+\xi_1) + \lambda_3\xi_2 - \lambda_1\big)\eta(\phi+\mu)}{a_3},\ \text{at } i = 3,\ \frac{\partial H}{\partial u_3} = 0,\ \text{when } u_3 = u_3^*
\end{aligned} \tag{4.39}$$

Since $0 < u_i^* \leq 1$ the compact form to obtain the equation is depicted as; □

The optimality system for the (SEITRB) model addressing typhoid spread combines the state equations (describing the disease dynamics) and the adjoint equations (capturing sensitivity information), along with the characterized control set and specified initial and boundary conditions. This model enables precise identification and implementation of optimal control strategies such as treatment or sanitation to minimize the disease's impact. By integrating mathematical rigor with practical interventions, the model provides a robust tool for designing effective public health policies to mitigate typhoid transmission and improve community health outcomes.

4.1. Results

The interpretation of numerical simulation results as conducted through iterative steps using Laplace Adomian Decomposition Method is depicted pictorially below in Figure 4.3 to Figure 4.7.

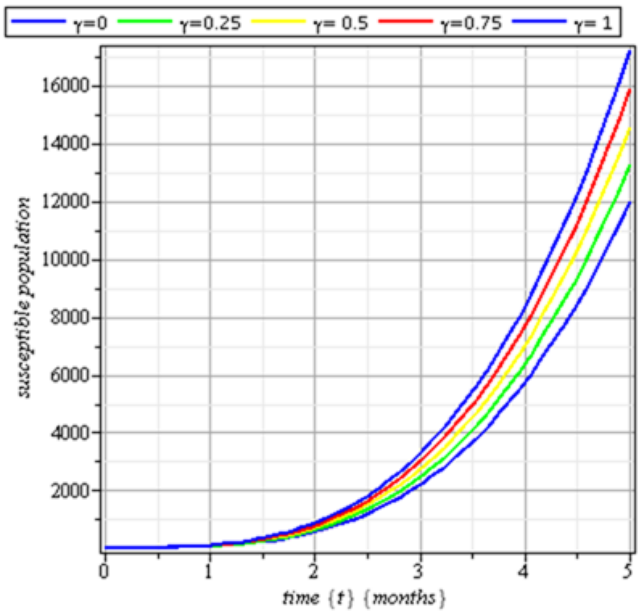

Figure 4.3: Higher vaccination rates reduce re-infected individuals, showing an inverse relationship. This indicates vaccination lowers susceptibility and prevents secondary infection, with stronger herd immunity limiting pathogen spread in the population

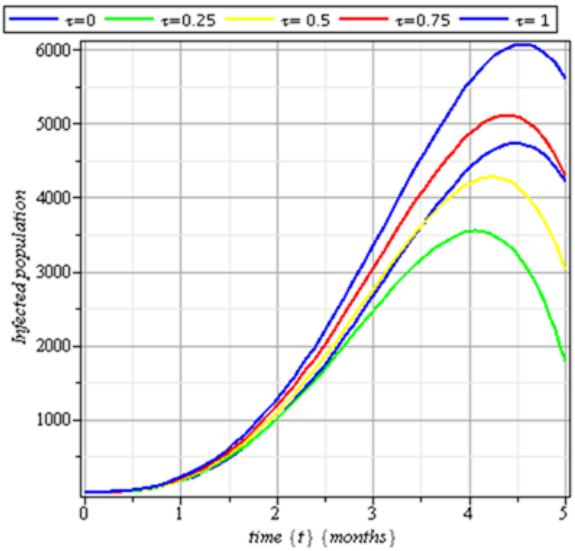

Figure 4.4: Treatment increases recovered individuals, accelerating recovery and reducing infectious duration. This shows timely medical care improves outcomes and highlights the importance of effective healthcare access and intervention

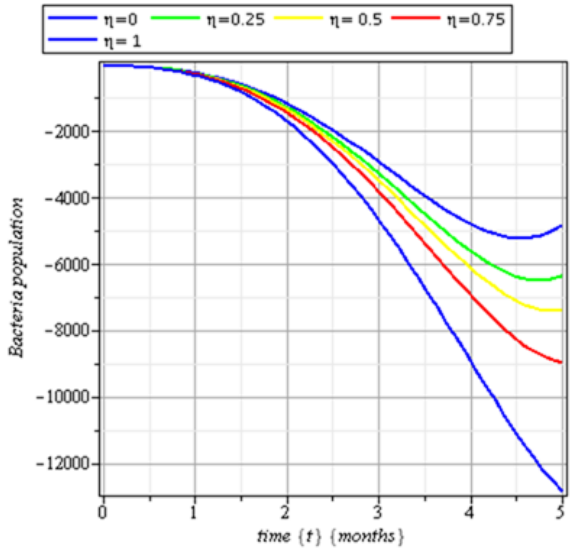

Figure 4.5: Treatment reduces exposed individuals as intensity increases, indicating that early intervention limits progression to active infection. It also reflects reduced transmission pressure, thereby decreasing the number of individuals moving through disease stages in the population

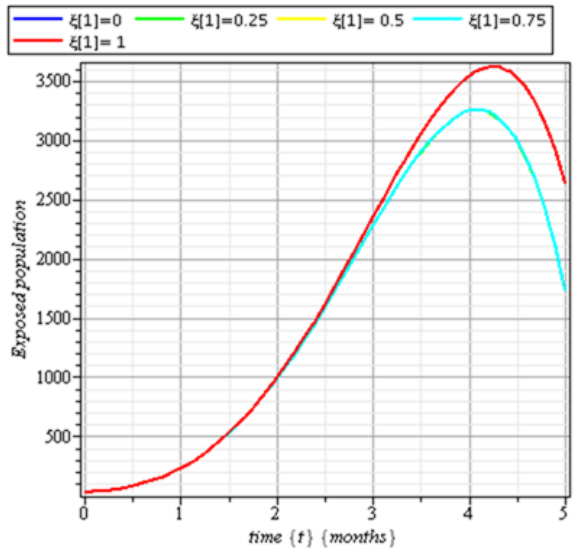

Figure 4.6: Treatment effectiveness increases under optimal control and stabilizes at a high level over time. This shows sustained intervention improves efficiency, optimizes resource allocation, and leads to a steady quasi-equilibrium where treatment impact becomes consistent and sustained

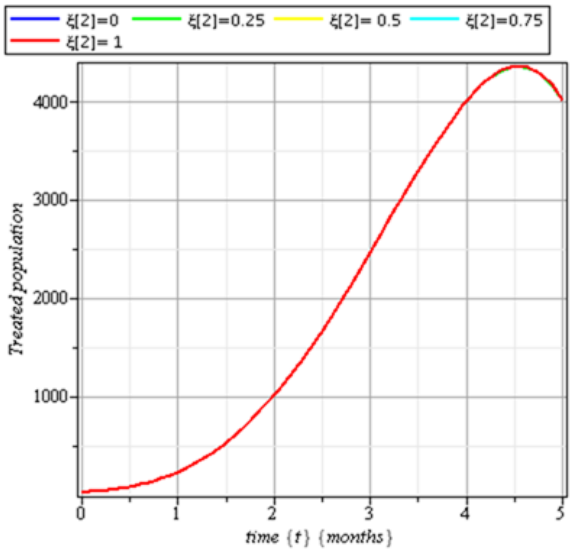

Figure 4.7: Treatment and sanitation jointly reduce infected individuals, showing a strong synergistic effect. Improved hygiene and clean water lower transmission, while treatment shortens infection duration, together greatly suppressing disease prevalence

5. Discussion of Results

The graphical results presented in Figure 4.3 to Figure 4.7 illustrate the dynamical impact of different control strategies vaccination, treatment and sanitation on the transmission and progression of typhoid fever within the selected population. Overall, the simulations demonstrate that the incorporation of optimal control measures significantly improves disease outcomes by reducing infection prevalence and enhancing recovery dynamics. Figure 4.3 shows a clear inverse relationship between vaccination rate and the number of re-infected individuals in the population. As the vaccination coverage increases, the population of re-infected individuals declines steadily over time. This indicates that vaccination not only reduces initial susceptibility but also plays a crucial role in preventing relapse or secondary infection. The observed decline suggests that herd immunity effects become more pronounced at higher vaccination rates, thereby limiting pathogen circulation within the population. In Figure 4.4, the effect of treatment on the recovered class is prominently positive. The graph demonstrates that an increase in treatment rate leads to a higher number of recovered individuals over time. This implies that effective medical intervention accelerates recovery and reduces the duration of infectiousness. The upward trend in the recovered population highlights the importance of timely diagnosis and adequate access to healthcare facilities in improving recovery outcomes.

Figure 4.5 presents the impact of treatment on the exposed (latent) class of the population. The results indicate a gradual decline in the number of exposed individuals as treatment intensity increases. This suggests that early treatment intervention may reduce progression from the exposed stage to active infection. It may also reflect the combined effect of treatment and reduced transmission pressure, thereby limiting the pool of individuals progressing through the disease stages. Figure 4.6 depicts the effectiveness of treatment under an optimal control framework. The curve shows an increasing trend in treatment effectiveness over the intervention period, eventually stabilizing at a higher level. This behavior suggests that sustained application of optimal control strategies enhances treatment efficiency and ensures better allocation of health resources. The stabilization phase indicates that the system reaches a quasi-equilibrium where treatment impact becomes consistent and sustained. Figure 4.7 demonstrates a strong synergistic effect between treatment and sanitation strategies on the infected population. As both intervention rates increase, there is a marked reduction in the number of infected individuals. The steep decline suggests that sanitation practices such as improved hygiene, clean water supply, and environmental cleanliness significantly reduce transmission, while treatment reduces infectious duration. Together, these interventions amplify each other's effectiveness, leading to a substantial suppression of disease prevalence.

6. Conclusion

This study successfully employed the Laplace Adomian decomposition method to derive numerical solutions for assessing the impact of high treatment and sanitation on typhoid fever, using an optimal control strategy based on the (susceptible, exposed, infected, treated, recovered and bacteria) mathematical model. The approach demonstrated strong effectiveness, significantly reducing the basic reproduction number below unity a crucial threshold for disease eradication. Through numerical simulations results, the influence of varying sanitation and treatment rates on typhoid transmission within the population was examined. Detailed behavioural analysis of the sub-population responses over time, supported by graphical illustrations, revealed key epidemiological patterns and biological insights. Importantly, the findings underscore the crucial role of combining awareness efforts with improved environmental sanitation in curbing the spread of typhoid disease.

These measures serve as essential components in developing sustainable strategies for disease containment and eventual eradication. Furthermore, the implementation of awareness campaigns and health education programs remains vital. Behavioural interventions, when integrated into public health policy can substantially enhance the long-term effectiveness of biomedical control strate-

gies. This underscores the need for health practitioners in endemic regions of West Africa to adopt a multifaceted approach that includes both medical and behavioural components to combat typhoid effectively.

Data Availability Statment

Data sets generating during this research are available from the corresponding author upon request.

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