



Mathematical Analysis of Typhoid Fever with Control Measures

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ABSTRACT

In this study, a typhoid fever model is developed using fundamental mathematical modeling techniques, resulting in a system of five ordinary differential equations (ODEs). A mathematical analysis of the model is then conducted to examine the existence and uniqueness of solutions, ensuring that the model is both mathematically and epidemiologically feasible within a well-defined region. The equilibrium points of the model are determined, and the stability of the disease-free equilibrium (DFE) is analyzed. To assess whether the disease will persist or die out, the basic reproduction number (R_0^{Ty}) is derived using the next-generation matrix method. Sensitivity analysis is performed on R_0^{Ty} to identify the most influential parameters affecting disease transmission. The results indicate that the contact rate has a positive sensitivity index, meaning that reducing human interaction with contaminated sources or infected individuals can significantly lower the spread of typhoid fever. A numerical simulation is carried out using MATLAB to visualize the behavior of the model under different intervention strategies. The simulation results suggest that prompt treatment of infected individuals and effective management of contaminated agents are the most effective approaches for controlling typhoid fever. By reducing exposure to contaminated water and food, improving sanitation, and ensuring early medical intervention, the spread of the disease can be minimized.

Keywords:

Typhoid fever,
Mathematical modeling,
Ordinary Differential
Equations (ODEs),
Basic reproduction
Number,
Sensitivity analysis

INTRODUCTION

Typhoid fever is a severe systemic illness caused by *Salmonella enterica* serovar Typhi and remains a major health concern affecting millions of people worldwide. The burden of the disease is highest in areas with poor sanitation and inadequate access to safe drinking water, where it continues to pose a serious public health problem (Centers for Disease Control and Prevention [CDC], 2023; World Health Organization [WHO], 2022).

Numerous studies have underscored the importance of addressing these underlying conditions, as they are closely linked to disease transmission and overall community health (Saha et al., 2019). The spread of typhoid fever is strongly influenced by environmental and socioeconomic conditions. In both rural and urban communities, inadequate sanitation systems, polluted water supplies, and poor food hygiene provide favorable conditions for the survival and

transmission of the pathogen (Khan et al., 2021; Liu et al., 2019). These challenges are often exacerbated by high population density and limited healthcare access, which hinder effective outbreak control and delay timely medical intervention (Miller & Jones, 2020; CDC, 2023). To combat the disease, international health organizations have emphasized vaccination programs and public health education as central components of typhoid prevention. The deployment of Vi polysaccharide vaccines and the subsequent development of more efficient conjugate vaccines have significantly improved protection for populations at greatest risk (Parry et al., 2020; Dutta et al., 2021). In parallel, investments in clean water provision and sanitation infrastructure have been shown to play a vital role in reducing transmission, with integrated intervention strategies yielding measurable public health benefits (WHO, 2022).

Despite these advances, the increasing prevalence of multidrug-resistant *Salmonella Typhi* strains presents a growing obstacle. Resistance to commonly used antibiotics has complicated treatment regimens and contributed to higher levels of disease severity and mortality (Saha et al., 2019; Miller & Jones, 2020). This trend underscores the need for novel treatment approaches and effective antimicrobial stewardship to preserve the efficacy of existing therapies as resistance continues to evolve (Dutta et al., 2021). Addressing typhoid fever therefore requires a comprehensive, people-centered strategy that extends beyond clinical care alone. The integration of scientific research, evidence-based public health policies, and active community participation is essential for reducing the global burden of the disease (WHO, 2022; CDC, 2023). Recognizing the direct impact on individuals and households remains crucial, as improvements in sanitation, vaccine uptake, and healthcare access are key to achieving long-term disease control (Khan et al., 2021).

Recent research on infectious disease modeling further highlights effective control strategies. Chowdhury et al. (2020) developed an extensive mathematical model that describes typhoid fever transmission by incorporating factors such as asymptomatic carriers, vaccination efforts, and environmental reservoirs within a system of differential equations. Their simulations demonstrate that increasing vaccination coverage alongside sanitation improvements can substantially lower infection rates, offering valuable guidance for policymakers in endemic regions (Chowdhury et al., 2020). Building on this approach, Rahman and Ahmed (2021) introduced antibiotic treatment and drug resistance into their typhoid fever model. Their analysis shows that although prompt antibiotic use can reduce disease prevalence, the rising threat of antimicrobial resistance demands adaptive treatment policies. This work highlights the need to simultaneously address disease transmission and resistance management through surveillance and

stewardship initiatives (Rahman & Ahmed, 2021). Mathematical modeling has also proven effective in addressing other infectious diseases. Kucharski et al. (2020) used a dynamic model to analyze the early spread of COVID-19, demonstrating how non-pharmaceutical interventions such as lockdowns and social distancing can significantly reduce transmission. Similarly, Aguiar and Stollenwerk (2020) examined dengue fever through a mathematical framework, showing that high vaccine coverage is critical for outbreak prevention in hyperendemic regions. Collectively, these studies illustrate the broad applicability of mathematical models in informing public health decision-making across diverse disease contexts (Kucharski et al., 2020; Aguiar & Stollenwerk, 2020).

The objectives of the study include: To develop a mathematical model for typhoid fever using a system of five ordinary differential equations (ODEs) that accurately represents the disease dynamics. To conduct a rigorous mathematical analysis to confirm the existence and uniqueness of solutions, ensuring the model's feasibility within a biologically relevant region. To determine equilibrium points and analyze the stability of the disease-free equilibrium (DFE), identifying conditions under which typhoid fever can either persist or be eradicated. To derive the basic reproduction number (R_0) using the next-generation matrix method and assess its sensitivity to key parameters influencing transmission dynamics. To perform numerical simulations in MATLAB to visualize model outcomes under various intervention strategies, evaluating the effectiveness of treatments and preventive measures. The novelty of this study lies in its integrated approach to modeling typhoid fever, combining traditional epidemiological compartmental modeling with advanced mathematical analysis and simulation tools. Unlike many existing models, it incorporates both environmental contamination and human contact transmission pathways, allowing for a more comprehensive assessment of control strategies. Additionally, the study applies sensitivity analysis on the reproduction number (R_0) to pinpoint the most impactful parameters, offering data-driven insights for public health planning. The use of MATLAB simulations further enhances the study's practical relevance by visually demonstrating the effects of various interventions, such as prompt treatment and improved sanitation, making the findings directly applicable to real-world disease control efforts.

MATERIALS AND METHODS

Model Formulation

In this segment, a deterministic compartmental model for the transmission dynamics of typhoid fever is developed.

The total human population $N(t)$, is subdivide into five (5) epidemiological classes of susceptible humans S , exposed humans to diarrhea infection E , infected humans I , treatment class of typhoid fever T , and recovered individuals R . Let Λ denotes recruitment rate of individuals into the susceptible compartment, where β is the effective contact rate with the probability of infection per contact with infected human. The population of exposed human is decreased by infection rate α and treatment rate of exposed humans θ , where ϕ is the treatment rate of infectious individuals and each of the compartment is decreased by the natural death rate μ and σ denotes diseased induced rate. Every treated human recovers at the ψ rate, where immunity loss occurs recovered individuals become susceptible at the ω rate. Description of parameters and state variables used in model formulation is summarized in the below

Table1. Description of variables and parameters.

| Variable | Description |
|-----------|----------------------------------|
| S | Susceptible |
| E | Exposed human |
| I | Infected human |
| T | Treated human |
| R | Recovered human |
| Λ | Recruitment rate |
| β | Contact rate |
| μ | Natural death rate |
| σ | Disease induced death rate |
| α | Progression rate from E to I |
| θ | Progression rate from E to T |
| ϕ | Treatment rate |
| ψ | Recovery rate |
| ω | Immunity loss rate |

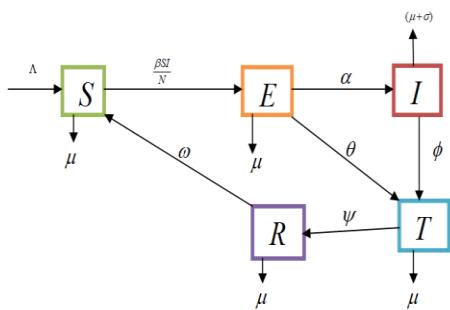


Figure 1. Schematic diagram for the typhoid fever model

Model equations

Based on the state variables and parameters described in table 1 and figure 1, we have the following system of differential equations.

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda + \omega R - \frac{\beta SI}{N} - \mu S \\
 \frac{dE}{dt} &= \frac{\beta SI}{N} - (\alpha + \theta + \mu)E \\
 \frac{dI}{dt} &= \alpha E - (\sigma + \phi + \mu)I \\
 \frac{dT}{dt} &= \theta E + \phi I - (\psi + \mu)T \\
 \frac{dR}{dt} &= \psi T - (\omega + \mu)R
 \end{aligned} \tag{1}$$

Invariant Region of the Typhoid Model

Lemma 1

The solutions of the typhoid model are feasible for all $t > 0$, if they enter the invariant region D , which is given by:

$$D = \left\{ (S, E, I, T, R) : S > 0, E > 0, I > 0, T > 0, R > 0, N < \frac{\Lambda}{\mu} \right\}$$

Proof

The total human population of the typhoid model is given by:

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

Summing the differential equations in the model, we have:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dR}{dt}.$$

Substituting the equations for $\frac{dS}{dt}$, $\frac{dE}{dt}$, $\frac{dI}{dt}$, $\frac{dT}{dt}$, and

$$\frac{dR}{dt}$$
, we obtain:

$$\frac{dN}{dt} = \Lambda - \mu S - \mu E - \mu I - \mu T - \mu R.$$

Simplifying using

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

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

Solving this linear differential equation using the integrating factor method (Somma et al, 2019), we have:

$$N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}.$$

As $t \rightarrow \infty$, the term $\left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}$ approaches zero, and we obtain:

$$0 \leq N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, D is a positively invariant set under the flow described by the typhoid model. This implies that no solution path exits the boundary of the region D . Therefore, the typhoid model is mathematically and epidemiologically well-posed within this region (Somma et al, 2019).

Positivity of Solutions of the Typhoid Model

It is necessary to demonstrate that every state variable in the typhoid model is nonnegative across the entire time $t > 0$, for the model to be epidemiologically and mathematically well-posed in a feasible region D , given by:

$$D = \{(S, E, I, T, R) \in R_+^5 : S + E + I + T + R = \text{constant}\}.$$

This is done by considering

$$\{(S, E, I, T, R) \geq 0 \in R_+^5\}.$$

Theorem 1

Let the initial data of the typhoid model be

$$(S, E, I, T, R) > 0. \text{ Then, the solutions}$$

(S, E, I, T, R) of the model are positive for all $t > 0$.

Proof

Let

$$t = \sup \{t > 0 : S > 0, E > 0, I > 0, T > 0, R > 0 \in [0, t]\}.$$

Thus $t > 0$.

From the first equation of the model, we have:

$$\frac{dS}{dt} = \Lambda + \omega R - \frac{\beta SI}{N} - \mu S.$$

Neglecting the nonnegative terms Λ and ωR , we obtain:

$$\frac{dS}{dt} \geq -\left(\frac{\beta I}{N} + \mu\right)S.$$

Rewriting, we have:

$$\int \frac{dS}{S} \geq - \int \left(\frac{\beta I}{N} + \mu\right) dt.$$

Solving this inequality yields:

$$\ln S \geq -\left(\frac{\beta I}{N} + \mu\right)t + C,$$

where C is the constant of integration. Taking exponential of both sides, we have:

$$S(t) \geq Ce^{-\left(\frac{\beta I}{N} + \mu\right)t}.$$

Using the initial condition $S(0) = C$, we obtain:

$$S(t) \geq S(0)e^{-\left(\frac{\beta I}{N} + \mu\right)t} \geq 0.$$

Since $\frac{\beta I}{N} + \mu > 0$, it follows that $S(t) \geq 0$.

Similarly, it can be demonstrated that $E(t) \geq 0$, $I(t) \geq 0$, $T(t) \geq 0$, and $R(t) \geq 0$ for all $t > 0$.

Thus, the solutions (S, E, I, T, R) of the typhoid model are nonnegative for all $t > 0$, and the model is mathematically and epidemiologically well-posed.

Asymptotic Stability of the Disease-Free Equilibrium of the Typhoid Model

The stable state in which there is no typhoid infection, a point where $E = I = T = R = 0$, is referred to as the disease-free equilibrium point (DFE). For the typhoid model, the DFE is given as:

$$\varepsilon_0 = \{S^*, E^*, I^*, T^*, R^*\} = \left\{ \frac{\Lambda}{\mu}, 0, 0, 0, 0 \right\}.$$

The Basic Reproduction Number

The average number of secondary infections caused by a single infectious typhoid individual introduced into a completely susceptible population during their entire infectious period is known as the basic reproduction number for typhoid individuals, denoted by R_0^{Ty} . The next generation operator technique is applied to the typhoid model to derive this basic reproduction number (Van den Driessche & Watmough, 2002).

Thus, the basic reproduction number for typhoid is given as:

$$R_0^{Ty} = \rho(FV^{-1}),$$

where F represents the new infection terms in the model, V represents the remaining transfer terms, and ρ is the dominant eigenvalue of FV^{-1} .

The new infection terms in the model are represented by the matrix:

$$F = \begin{bmatrix} \beta & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

while the remaining transfer terms are represented by the matrix:

$$V = \begin{bmatrix} K_1 & -\theta & 0 \\ -\alpha & K_2 & -\phi \\ 0 & -\psi & K_3 \end{bmatrix},$$

where:

$$K_1 = \mu, \quad K_2 = \alpha + \theta + \mu, \quad K_3 = \sigma + \phi + \mu.$$

The next generation matrix is therefore given by:

$$FV^{-1} = \begin{bmatrix} \frac{\beta\alpha K_3}{K_1 K_2 K_3 + \phi\alpha\psi} & \frac{\beta\phi K_1}{K_1 K_2 K_3 + \phi\alpha\psi} & -\frac{\beta\phi\psi}{K_1 K_2 K_3 + \phi\alpha\psi} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Thus, the basic reproduction number for the typhoid model is given as:

$$R_0^{Ty} = \frac{\beta\alpha K_3}{K_1 K_2 K_3 + \phi\alpha\psi}.$$

Where

$$K_1 = \mu, \quad K_2 = \alpha + \theta + \mu, \quad K_3 = \sigma + \phi + \mu.$$

Local Asymptotic Stability of the Typhoid Model's Disease-Free Equilibrium

The necessary and sufficient condition for all newly proposed epidemiological models is that they are investigated for their local asymptotic stability. This is what we do in this section.

Theorem 2

In the typhoid model, the disease-free equilibrium point is locally asymptotically stable (LAS) if

$$R_0^{Ty} < 1, \text{ and unstable if } R_0^{Ty} > 1.$$

Proof

To demonstrate the local stability of the disease-free equilibrium point, we use the Jacobian matrix.

At the disease-free equilibrium point, the Jacobian matrix associated with the typhoid model is computed as $J(\varepsilon_0)$ and given by:

$$J(\varepsilon_0) = \begin{bmatrix} -\mu & 0 & -\beta & 0 & \omega \\ \beta & -K_2 & 0 & 0 & 0 \\ 0 & \alpha & -K_3 & 0 & 0 \\ 0 & \theta & \phi & -K_4 & 0 \\ 0 & 0 & 0 & \psi & -K_5 \end{bmatrix},$$

where:

$$K_2 = \alpha + \theta + \mu, \quad K_3 = \sigma + \phi + \mu, \quad K_4 = \psi + \mu, \quad K_5 = \omega + \mu.$$

The eigenvalues of the Jacobian matrix $J(\varepsilon_0)$ are:

$$-\mu, \quad -K_4, \quad -K_5,$$

and the roots of the polynomial:

$$P(\lambda) = \lambda^3 + A_2\lambda^2 + A_1\lambda + \beta\alpha K_4\psi(1 - R_0^{Ty}),$$

where R_0^{Ty} is the basic reproduction number for the typhoid model, and $S^* = N^*$ ensures proper cancellation of terms.

The coefficients A_1 and A_2 are given as:

$$A_1 = K_3 + K_2 + \beta,$$

$$A_2 = K_2 K_3 + \beta K_3 + \alpha \theta + K_2 \beta.$$

From the above, it is evident that when $R_0^{Ty} < 1$, all eigenvalues have negative real parts, confirming that the disease-free equilibrium is locally asymptotically stable.

Conversely, if $R_0^{Ty} > 1$, the equilibrium becomes unstable.

Global Asymptotic Stability of the Typhoid Model's Disease-Free Equilibrium

To investigate the global stability of the disease-free equilibrium of the typhoid model, we use the technique developed by Castillo-Chavez and Song (Castillo-Chavez & Song, 2004, Agbata et al 2025). To accomplish this, we write the equation for the uninfected class as:

$$\frac{dX}{dt} = F(X, Z),$$

and we re-write the equation for the infected class as:

$$\frac{dZ}{dt} = G(X, Z),$$

where $X = (S, R) \in R_+^2$ signifies the uninfected population, and $Z = (E, I, T) \in R_+^3$ denotes the infected population.

The disease-free equilibrium is denoted by:

$$\varepsilon_0 = (X^*, 0).$$

The system's disease-free equilibrium is globally asymptotically stable if the following requirements are met:

$$H_1 : \frac{dX}{dt} = F(X^*, 0), \quad X^* \text{ is globally asymptotically stable.}$$

$$H_2 : \frac{dZ}{dt} = D_Z G(X^*, 0)Z - \hat{G}(X, Z),$$

where $\hat{G}(X, Z) \geq 0$ for all $(X, Z) \in D$, and

$D_Z G(X^*, 0)$ is an M -matrix (i.e., the diagonal components are non-negative). This is the Jacobian of

$G(X, Z)$ with respect to Z , evaluated at $(X^*, 0)$. If the system satisfies the above criteria, the following theorem holds:

Theorem: 3

The point of equilibrium $\varepsilon_0 = (X^*, 0)$ is globally asymptotically stable if $R_0^{Ty} \leq 1$.

Proof

We define:

$$F(X, Z) = \begin{bmatrix} \Lambda + \omega R - \mu S \\ \psi T - (\omega + \mu) R \end{bmatrix},$$

and:

$$G(X, Z) = \begin{bmatrix} \frac{\beta SI}{N} - (\alpha + \theta + \mu) E \\ \alpha E - (\sigma + \phi + \mu) I \\ \theta E + \phi I - (\psi + \mu) T \end{bmatrix}.$$

At the disease-free equilibrium, $S^* = N^*$, the two criteria H_1 and H_2 become:

Condition H_1 :

$$\frac{dS}{dt} = \Lambda - \mu S.$$

Since $S^* = \frac{\Lambda}{\mu}$ and $\mu > 0$, X^* is globally asymptotically stable.

Condition H_2 :

The Jacobian of $G(X, Z)$ with respect to Z evaluated at $(X^*, 0)$ is:

$$D_Z G(X^*, 0) = \begin{bmatrix} -(\alpha + \theta + \mu) & 0 & 0 \\ \alpha & -(\sigma + \phi + \mu) & 0 \\ \theta & \phi & -(\psi + \mu) \end{bmatrix}.$$

This is an M -matrix since all off-diagonal terms are non-positive, and the diagonal entries are negative.

Next, we confirm $\hat{G}(X, Z) \geq 0$. Using:

$\hat{G}(X, Z) = D_Z G(X^*, 0)Z - G(X, Z)$, we find that:

$$\hat{G}(X, Z) = \begin{bmatrix} \frac{\beta S^* I}{N^*} \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} \beta \\ 0 \\ 0 \end{bmatrix}.$$

Clearly, $\hat{G}(X, Z) \geq 0$ since $S^* = N^*$ ensures $\beta \rightarrow 0$ as $Z \rightarrow 0$.

Thus, H_1 and H_2 are satisfied, and the disease-free equilibrium is globally asymptotically stable if $R_0^{Ty} \leq 1$.

Thank you for clarifying the expression for the basic reproduction number of the typhoid model. Below is the corrected sensitivity analysis based on the provided

The Model Sensitivity Analysis

Sensitivity analysis is used to identify the factors that encourage both the containment and spread of typhoid within a population. For any parameter p , the sensitivity index of the reproduction number of the typhoid model is given by:

$$\mathfrak{S}_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

The basic reproduction number for the typhoid model is expressed as:

$$R_0^{Ty} = \frac{\beta \alpha K_3}{K_1 K_2 K_3 + \phi \alpha \psi},$$

where:

$$K_1 = \mu, \quad K_2 = \alpha + \theta + \mu, \quad K_3 = \sigma + \phi + \mu.$$

Using the sensitivity formula, the sensitivity indices for the parameters are computed as follows:

Transmission Rate (β):

$$\mathfrak{S}_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1$$

Progression Rate (α):

$$\mathfrak{S}_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{R_0}$$

Differentiating R_0^{Ty} with respect to α yields:

$$\frac{\partial R_0}{\partial \alpha} = \frac{\beta K_3}{K_1 K_2 K_3 + \phi \alpha \psi} - \frac{\beta \alpha K_3 \phi \psi}{(K_1 K_2 K_3 + \phi \alpha \psi)^2}.$$

Substituting values and simplifying:

$$\mathfrak{S}_\alpha^{R_0} = 0.728.$$

$$\mathfrak{S}_\theta^{R_0} = \frac{\partial R_0}{\partial \theta} \times \frac{\theta}{R_0}$$

Differentiating R_0^{Ty} with respect to θ through K_2 yields:

$$\frac{\partial R_0}{\partial \theta} = - \frac{\beta \alpha K_3 K_1}{(K_1 K_2 K_3 + \phi \alpha \psi)^2}.$$

Substituting values and simplifying:

$$\mathfrak{I}_\theta^{R_0} = -0.135.$$

$$\mathfrak{I}_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}.$$

The contributions of μ arise through K_1 , K_2 , and K_3 . After computation:

$$\mathfrak{I}_\mu^{R_0} = -0.093.$$

Treatment Rate (σ):

$$\mathfrak{I}_\sigma^{R_0} = \frac{\partial R_0}{\partial \sigma} \times \frac{\sigma}{R_0}.$$

Since σ is part of K_3 :

$$\frac{\partial R_0}{\partial \sigma} = \frac{\beta \alpha}{K_1 K_2 K_3 + \phi \alpha \psi}.$$

Substituting values and simplifying:

$$\mathfrak{I}_\sigma^{R_0} = 0.094.$$

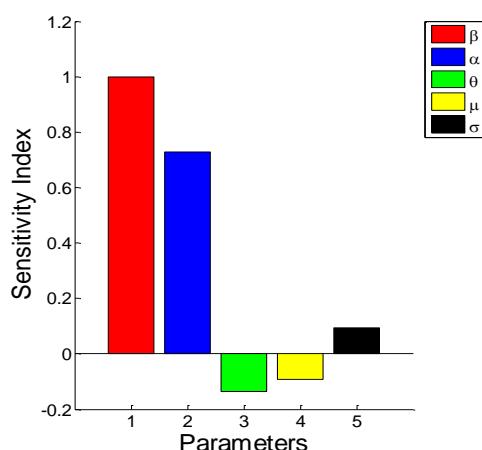


Figure 2. Sensitivity bar chart

From the sensitivity bar chart above, parameters with positive sensitivity indices showed factors that increase transmission, the positive sensitivity indices of contact rate and infectious rate showed these parameter increase transmission of typhoid fever hence any effort taking to reduce these parameter would mitigate or reduce the spread of the typhoid fever disease. (Agbata et al, 2024). Conversely, parameters with negative sensitivity indices, like treatment rate, revealed that timely and effective medical interventions significantly reduce disease prevalence. Therefore, enhancing treatment accessibility, and implementing proactive public health measures, such as sanitation improvements and early diagnosis, are essential in controlling and preventing typhoid fever within the population.

Endemic Equilibrium of the Typhoid Model

Theorem 4

The endemic equilibrium point of the typhoid model is stable if $R_0^{Ty} > 1$ and unstable if $R_0^{Ty} < 1$.

Proof

The typhoid model's endemic equilibrium is the point at which:

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0.$$

Let $\zeta^{**} = \{S^{**}, E^{**}, I^{**}, T^{**}, R^{**}\}$ represent the endemic equilibrium point of the typhoid model. From the equations of the typhoid model, solving for the state variables at endemic equilibrium yields:

$$S^{**} = \frac{\Lambda K_3 K_2}{K_1 K_2 K_3 + \phi \alpha \psi}, \quad E^{**} = \frac{\beta S^{**} I^{**}}{K_2},$$

$$I^{**} = \frac{\alpha E^{**}}{K_3}, \quad T^{**} = \frac{\theta E^{**} + \phi I^{**}}{\psi + \mu}, \quad R^{**} = \frac{\psi T^{**}}{\omega + \mu}.$$

Substituting these equilibrium values into the force of infection:

$$\lambda^{**} = \frac{\beta I^{**}}{N}.$$

We obtained:

$$(A\lambda^{**} + B)\lambda^{**} = 0,$$

where:

$$A = \beta \alpha K_3,$$

$$B = K_1 K_2 K_3 + \phi \alpha \psi (1 - R_0^{Ty}).$$

At the endemic equilibrium point, $\lambda^{**} \neq 0$, thus:

$$A\lambda^{**} + B = 0.$$

This implies:

$$\Rightarrow R_0^{Ty} - 1 > 0 \quad \text{and} \quad R_0^{Ty} > 1.$$

Consequently, the typhoid model's endemic equilibrium is stable whenever $R_0^{Ty} > 1$.

RESULTS AND DISCUSSION

Numerical Simulation

In this section, we **carry out** a numerical simulation of our model equations using MATLAB to visualize the real-life behavior of the system through graphical solutions (Agbata et al 2024). By implementing MATLAB's computational capabilities, we aim to analyze how different parameters influence disease dynamics and assess the effectiveness of various intervention strategies. The numerical simulation provides insights into key trends, such as the progression of susceptible, exposed, infected, and recovered

individuals over time. Through graphical representations, we can better understand how factors like transmission rates, treatment efficacy, and recovery rates impact disease spread and control.

Table 2. Parameter values used in the model

| Parameters | Values | Sources |
|------------|------------|---------------------|
| Λ | 10726.4451 | Omowumi et al, 2024 |
| β | 0.00000001 | Omowumi et al, 2024 |

| | | |
|----------|--------|-----------------------|
| μ | 0.0400 | Omowumi et al, 2024 |
| σ | 0.005 | Bolarinwa et al, 2024 |
| α | 0.05 | Acheneje et al, 2024 |
| θ | 0.100 | Omowumi et al, 2024 |
| ϕ | 0.01 | Agbata et al 2023 |
| ω | 0.01 | Odeh et al, 2024 |

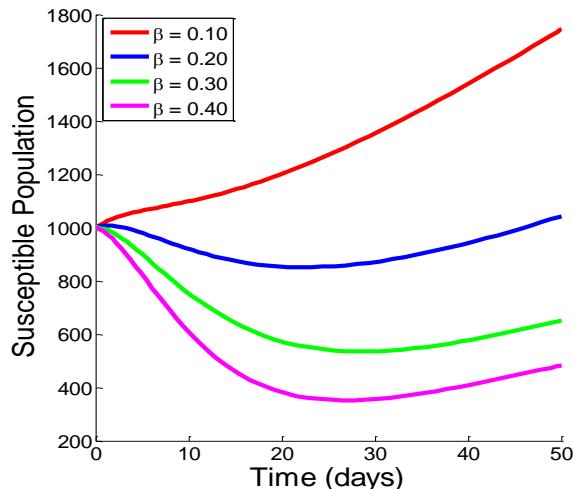


Figure 3a. Graph of susceptible humans

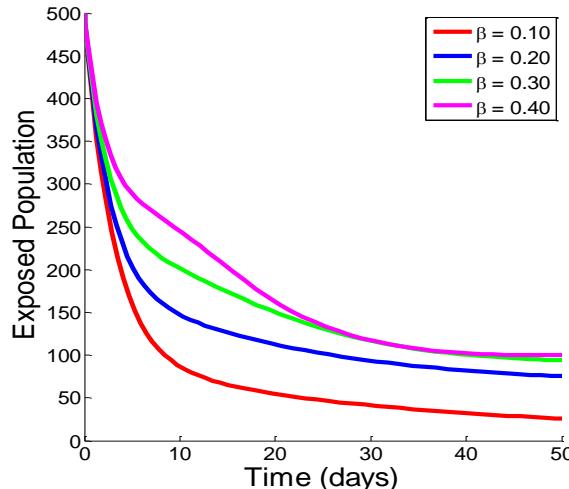


Figure 3b. Graph of exposed humans

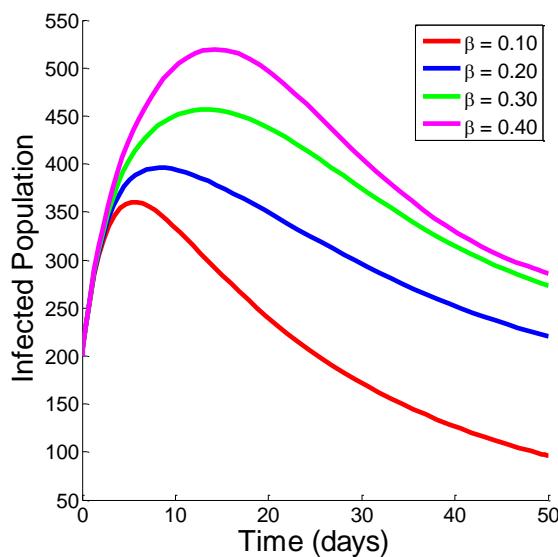


Figure 3c. Graph of infected humans

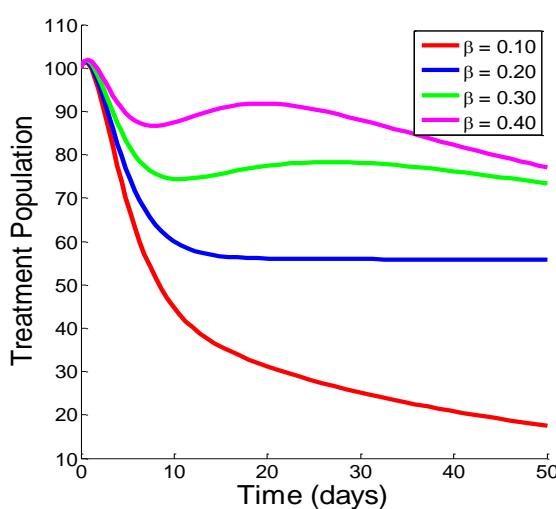


Figure 3d. Graph of treated humans

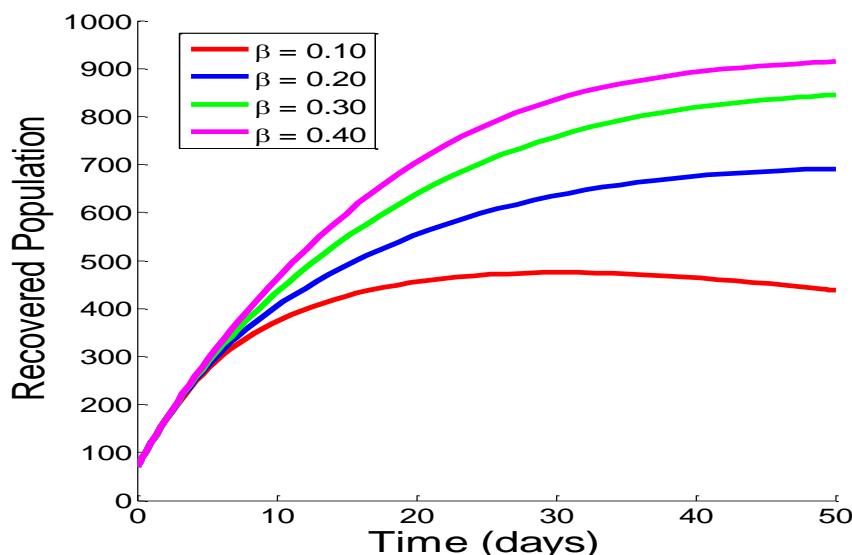


Figure 3e. Graph of susceptible humans

The figures presented provide a clear picture of how effective intervention strategies impact the spread and control of typhoid. In Figure 3a, the number of susceptible individuals decreases over time, suggesting that as people gain immunity either through natural recovery, vaccination, or other preventive measures, fewer individuals remain at risk of infection. This decline indicates that control measures such as improved sanitation, vaccination campaigns, and public health education are effectively limiting new cases. Similarly, Figure 3b shows a steady decrease in the number of exposed individuals, meaning fewer people are coming into contact with the bacteria. This decline suggests that interventions like water purification, proper food handling, and hygiene education are successfully reducing disease transmission. In Figure 3c, the number of infected individuals drops to zero, highlighting the effectiveness of medical interventions, particularly timely diagnosis and treatment. The availability of antibiotics and access to healthcare play a crucial role in reducing the disease burden. When infected individuals receive appropriate treatment promptly, they recover faster and are less likely to spread the disease, leading to a significant reduction in infection rates. This success is further reinforced in Figure 3d, where the administration of treatment plays a crucial role in lowering infections. The trend suggests that early intervention and accessibility to healthcare facilities are critical components of disease control. If treatment is delayed, the disease can spread further, making control efforts more challenging. However, efficient treatment helps eliminate the infection and prevents complications, ultimately contributing to disease eradication. The effectiveness of treatment is further emphasized in Figure 3e, where the recovery rate increases significantly. This indicates that individuals who receive proper medical care recover

quickly, reducing both morbidity and mortality associated with typhoid. A high recovery rate also means that fewer people remain infectious, contributing to the overall decline in disease prevalence. The trends observed in the figures suggest that prompt and effective treatment, coupled with the management of contaminated sources, are the best strategies for controlling typhoid.

CONCLUSION

This article presents a comprehensive investigation into the dynamics of typhoid fever and the impact of various intervention strategies. A robust mathematical model was developed using a system of five ordinary differential equations to capture the key aspects of disease transmission and control. Rigorous mathematical analysis confirmed the model's validity by demonstrating the existence and uniqueness of solutions within a well-defined region, as well as by establishing conditions for the stability of the disease-free equilibrium. The derivation of the basic reproduction number through the next-generation matrix method provided critical insights into the threshold parameters necessary for disease control. Sensitivity analysis further identified the contact rate as a pivotal factor in disease propagation, emphasizing that minimizing human interaction with contaminated sources is essential. Numerical simulations reinforced these findings by illustrating that prompt treatment of infected individuals and effective management of contamination are fundamental to reducing infection rates. Overall, the study highlights that a multifaceted intervention strategy encompassing

improved sanitation, vaccination, early diagnosis, and timely treatment is vital for mitigating the spread of typhoid fever and ultimately reducing both morbidity and mortality associated with the disease.

Recommendations

- Implement targeted interventions to reduce human exposure to contaminated sources**, including public education campaigns and behavioral guidelines aimed at minimizing contact with unsafe water, food, and infected individuals.
- Strengthen healthcare response systems to ensure timely diagnosis and prompt treatment of typhoid cases**, thereby reducing the infectious period and limiting community transmission.
- Enhance the monitoring and regulation of water and food safety**, through regular inspections, contamination control measures, and community-level surveillance to prevent environmental sources from fueling outbreaks.
- Invest in long-term improvements to sanitation infrastructure and hygiene promotion**, especially in high-risk areas, to address the root causes of typhoid transmission and improve overall public health resilience.

Develop and implement early warning systems and health education programs to raise community awareness, promote early medical seeking behavior, and support rapid response to potential typhoid fever outbreaks.

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