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Mathematical Modeling of Typhoid Fever Transmission Dynamics: A Sensitivity Analysis and Implications for Public Health Strategies

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Abstract

A comprehensive mathematical model of typhoid fever was developed to investigate the complex transmission dynamics of the disease and clarify the relationships among factors influencing its spread. The model assumes population replenishment through births and uses existing data to validate its accuracy, thereby supporting a reliable representation of disease behavior. This study aims to inform and strengthen strategies for the prevention, control, and possible eradication of typhoid fever in order to support improved public health policy and quality of life. Mathematical analysis revealed that the basic reproductive number, R_0, plays a central role in determining the global dynamics of the disease. When R_0 is less than 1, the disease-free equilibrium is locally stable, indicating that the disease will eventually die out. Conversely, when R_0 exceeds 1, an endemic equilibrium exists, suggesting that the disease will persist at a stable level. Sensitivity analysis of the model parameters provided valuable insights into the relative influence of different factors on typhoid fever

transmission, thereby supporting informed decision-making and effective disease management. The model was solved using the fourth-order Runge–Kutta scheme over a 40-year time horizon and implemented in MATLAB. The study concludes that mathematical modeling is a powerful tool for understanding the transmission dynamics of typhoid fever and for guiding evidence-based strategies for disease control and prevention. This study contributes to infectious disease modeling by demonstrating how equilibrium analysis, reproductive number estimation, and parameter sensitivity assessment can support public health planning aimed at reducing the burden of typhoid fever.

Keywords: Typhoid Fever; Mathematical Modeling; Basic Reproductive Number; Sensitivity Analysis; Equilibrium Stability

Introduction

Typhoid fever is an infectious illness that is usually caused by bacteria referred to as Salmonella typhi. This disease is transmitted by contamination of food or water with the help of manure, and infects unprotected individuals. Here, we are interested in a numerical research of a nonlinear mathematical model of a typhoid fever. People are likely to transmit infectious diseases either directly or indirectly. These diseases occur once the germs penetrate into the body, increase in numbers, and consequently, result in a body response. As of In 2025, it is estimated that 9 million people fall ill due to typhoid and 11010 people die due to typhoid annually. Typhoid fever is a dangerous infection disease that may extend throughout the entire body, affecting a number of organs. Otherwise, it may lead to real complications and even be life threatening in case it is not treated in time. It is brought about by bacteria referred to as Salmonella Typhi1 This is a bacterium that resides in constipated food or water and causes illness when ingested in the body via drinking or eating. Though, the water coverage sanitation has been enhanced but the transmission of typhoid disease remains a significant concern in the community health of various emerging nations [1]. The symptoms of the most common type of typhoid are migraine, stomach ache, knee, spine, muscular, lack of appetite, spew, dysentery, spots, and fever [2]. Some patients may have a rash. There are those cases that can cause severe complications or even death. Failure to treat typhoid fever immediately may cause it to be more dangerous. It may cause impairment of the inner circulation of blood and tissue infection in the tissue, which is situated in the stomach [3]. It is possible to diagnose typhoid fever with the help of some simple blood or stool tests. These are tests that determine the presence of

Salmonella typhi in a blood or stool sample. Some of the preventive and control measures of typhoid fever are antibiotic therapy, stool standard precautions, vaccination, environmental sanitation and clean water [4]. Typhoid fever can be treated using medicines and the symptoms would be improved after four weeks. Failure to complete treatment can result in the recurrence of the symptoms [5]. The earliest typhoid vaccination was created more than 110 years ago. Vaccines against typhoid fever come in two forms that include oral and injectable. Some of the injectable types include: Tya, typhoid conjugate vaccine and Vi capsular polysaccharide vaccine. Their efficacy ranges between 30-80 percent in the first 2 years following administration of the particular vaccine. When an individual adopts the drug resistant strain of typhoid fever and is not well addressed using effective antibiotics, then there are high chances that it may cause complications [6]. Estimates show that cases of typhoid fever have increased between 11millionn and 21.5 million and five million cases of paratyphoid fever in the world with 200,000 death occurrences every year. It is also estimated that African states have not been left out where the number of cases in the range of between 10 and 100 per 100,000 people has been increasing with children being the most infected owing to poor hygiene and sanitation. Due to the infection rate and the increasing ethos of the disease strain, typhoid has become a bane that has become a significant issue in health in the globe. Nevertheless, vaccination appears to be the key to the process of controlling the disease spread. Typhoid fever is an international health issue. Its actual effect is not easy to determine since the clinical picture is mixed with other numerous febrile infections. As well, underestimation of the disease is due to the absence of bacteriology laboratories in most regions of the developing countries. All these are thought to cause numerous cases to be undiagnosed. Based on the literature and the incidence of typhoid fever observed in the control groups of the biggest vaccine field trials with good laboratory support it was estimated that there are about 17 million cases of typhoid fever and 600 000 deaths related to typhoid fever every year. The estimates have however been biased since study populations have most often been in high incidence areas. In addition, these burden estimates are associated with clinical syndrome of typhoid fever and not with S. typhi exposure. As the incidence of bacteraemia among febrile children is very high (23%) in endemicity areas it is implied that more exposure to the bacteria exists than the figures which are only based on the clinical manifestation of typhoid fever. The endemicity area incidence of the disease can be similar to the incidences in control groups of large vaccine field trials, viz. between 45 per 100 000 per year and over 1000 per 100 000 per year. Early findings of recent research done in Bangladesh by ICDDR,B reveals a rate of about

2000 per 100 000 per year. Typhoid fever is also very high in social and economic effects due to hospitalization of patients with acute illness and complications and loss of income that can be attributed to the period of the clinical illness. It should be mentioned that in some provinces in China and Pakistan, reports have mentioned higher cases of paratyphoid fever S. paratyphi A than S. typhi. Most cases are found in persons aged 3-19 years in the endemic areas as well as in massive outbreaks. The example is in 1997, when an epidemic of the disease in Tajikistan was reported in this age range. However, bacteraemic S. typhi infection clinically apparent amongst children younger than three years has been reported in Bangladesh, India, Jordan, Nigeria and other countries. There is a mean of 900 000 cases annually and more than 20 000 deaths in Indonesia. The attack rate of typhoid fever by people of 319 years old in Indonesia was 91% and the attack rate of blood-culture-positive typhoid fever was 1026 per 100 000 per year. A similar situation was reported from Papua New Guinea. When typhoid fever was highly endemic in certain countries in South America the incidence of clinical typhoid fever in children aged under 3 years was low. In Chile, however, single blood cultures for all children aged under 24 months who presented at health centres with fever, regardless of other clinical symptoms, showed that 3.5% had unrecognized bacteraemic infections caused by S. typhi or S. paratyphi [7] . Enteric fever had not been suspected on clinical grounds in any of the children. In South America the peak incidence occurred in school students aged 519 years and in adults aged over 35 years. This kind of study has not been conducted in other areas of endemicity.

Material and Methods

The model is a modification of the model proposed by Ihsan et al. (2023). They presumed that the whole population N(t) is separated into four sections. Susceptible (S), Exposed E, infected (I) and recovered (R). The work i.e N(t) = S(t) + E(t) + I(t) + R(t). This work was extended by adding two (2) compartments namely individuals afflicted with complications but without underlying chronic disease (C_{η}) and individuals under hospital lock down with ongoing health monitoring (H).

The total population is given by N(t) = S(t) + E(t) + I(t) + R(t) + C_{η} + H

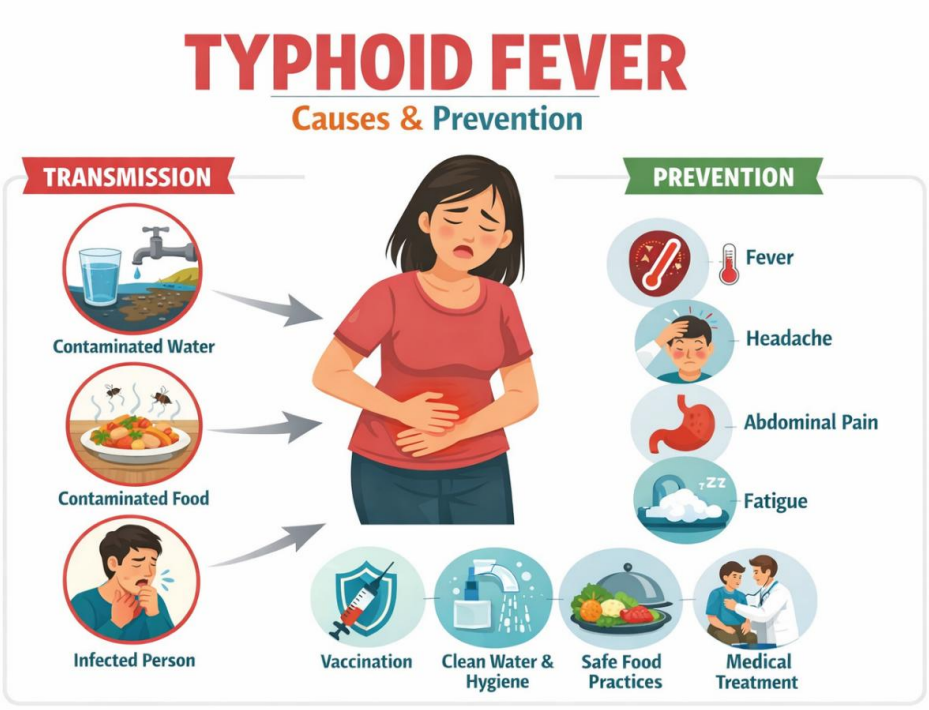

Figure 1: The model diagram for typhoid fever disease transmission (Ihsan et al. 2023)

Basic Assumptions for Model

• Those that are hospitalized after treatment recovers but does not recover completely.

• Individuals are all recruited in the susceptible compartment.

• Infectious disease can be transmitted among people directly or indirectly.

• Individual symptoms may return if treatment is not completed.

$$\begin{aligned}
\frac{dS}{dt} &= \pi + \sigma R - (\mu + \alpha I)S, \\
\frac{dE}{dt} &= \alpha SI - (\tau + \mu)E, \\
\frac{dI}{dt} &= \tau E - (\delta + \mu + \gamma\rho)I, \\
\frac{dC_w}{dt} &= \gamma\rho I - (\eta + \mu)C_w, \\
\frac{dR}{dt} &= \phi H - (\sigma + \mu)R, \\
\frac{dH}{dt} &= \eta C_w - (\phi + \mu)H.
\end{aligned} \qquad (2.1)$$

Table 1: Variable Description

Variable	Description
S	Susceptible Human
E	Exposed Human
I	Infected Human
R	Recovered Human
C_w	Individuals affected with Complications
R	Population of recovered individuals
H	Hospitalized Human

Table 2: Parameter Description

Parameter	Description	Value	References
π	Recruitment rate of human	200	Assumed
γ	Rate of individuals with severe complications	0.08	Hicham et al. 2023
η	Rate of transmission of humans from susceptible to exposed	0.05	Hicham et al. 2023
δ	Rate at which people become infected without chronic disease	0.02	Assumed
ρ	The number of people who die as a result of the disease or illness	0.625	Ihsan et al. 2023
σ	Rate of patients admitted in the hospital recovers	0.75	Ihsan et al. 2023
ϕ	Rate at which recovered humans loses temporary immunity	0.125	Ihsan et al. 2023
μ	Natural death rate	0.02	Ihsan et al. 2023

Model Analysis

1. Positivity and Boundedness of Solutions

To establish that the model is epidemiologically meaningful, it is necessary to show that all state variables remain non-negative for all $t \geq 0$, provided the initial conditions are non-negative. Also, we show that the total population remains bounded. Consider equation (2.1)

with initial conditions

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, C_w(0) \geq 0, R(0) \geq 0, H(0) \geq 0.$$

Positivity of Solutions

We show that each state variable remains non-negative for all time.

For the susceptible class,

$$\frac{dS}{dt} = \pi + \sigma R - (\mu + \alpha I)S,$$

which can be written as

$$\frac{dS}{dt} + (\mu + \alpha I)S = \pi + \sigma R.$$

Using the integrating factor

$$\text{IF} = e^{\int_0^t (\mu + \alpha I(u))\, du},$$

we obtain

$$\frac{d}{dt}\left[S(t)e^{\int_0^t (\mu + \alpha I(u))\, du}\right] = (\pi + \sigma R)e^{\int_0^t (\mu + \alpha I(u))\, du}.$$

Integrating from 0 to t gives

$$S(t)e^{\int_0^t (\mu + \alpha I(u))\, du} = S(0) + \int_0^t (\pi + \sigma R(s))\, e^{\int_0^s (\mu + \alpha I(u))\, du}\, ds.$$

Hence,

$$S(t) = S(0)e^{-\int_0^t (\mu + \alpha I(u))\, du} + e^{-\int_0^t (\mu + \alpha I(u))\, du} \int_0^t (\pi + \sigma R(s))\, e^{\int_0^s (\mu + \alpha I(u))\, du}\, ds.$$

Since $\pi \geq 0$, $\sigma \geq 0$, $R(t) \geq 0$, and $S(0) \geq 0$, it follows that

$$S(t) \geq 0 \text{for all } t \geq 0.$$

Similarly, for the exposed class,

$$\frac{dE}{dt} + (\tau + \mu)E = \alpha SI.$$

Applying the integrating factor $e^{(\tau+\mu)t}$, we obtain

$$E(t) = E(0)e^{-(\tau+\mu)t} + e^{-(\tau+\mu)t} \int_0^t \alpha S(s)I(s)\, e^{(\tau+\mu)s}\, ds \geq 0.$$

For the infected class,

$$\frac{dI}{dt} + (\delta + \mu + \gamma\rho)I = \tau E,$$

so that

$$I(t) = I(0)e^{-(\delta+\mu+\gamma\rho)t} + e^{-(\delta+\mu+\gamma\rho)t}\int_0^t \tau E(s)\, e^{(\delta+\mu+\gamma\rho)s}\, ds \geq 0.$$

For the complication class,

$$\frac{dC_w}{dt} + (\eta + \mu)C_w = \gamma\rho I,$$

hence

$$C_w(t) = C_w(0)e^{-(\eta+\mu)t} + e^{-(\eta+\mu)t}\int_0^t \gamma\rho I(s)\, e^{(\eta+\mu)s}\, ds \geq 0.$$

For the recovered class,

$$\frac{dR}{dt} + (\sigma + \mu)R = \phi H,$$

thus

$$R(t) = R(0)e^{-(\sigma+\mu)t} + e^{-(\sigma+\mu)t}\int_0^t \phi H(s)\, e^{(\sigma+\mu)s}\, ds \geq 0.$$

For the hospitalized class,

$$\frac{dH}{dt} + (\phi + \mu)H = \eta C_w,$$

which gives

$$H(t) = H(0)e^{-(\phi+\mu)t} + e^{-(\phi+\mu)t}\int_0^t \eta\, C_w(s)e^{(\phi+\mu)s}\, ds \geq 0.$$

Therefore, all state variables remain non-negative for all $t \geq 0$whenever the initial data are non-negative. Hence, the feasible region of the model is positively invariant. The model is therefore epidemiologically well posed.

Boundedness of Solutions

Let the total population be

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

Differentiating with respect to time gives

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

Substituting from the model equations, we obtain

$$\begin{aligned}\frac{dN}{dt} &= [\pi + \sigma R - (\mu + \alpha I)S] + [\alpha SI - (\tau + \mu)E] \\ &+[\tau E - (\delta + \mu + \gamma\rho)I] + [\gamma\rho I - (\eta + \mu)C_w] \\ &+[\phi H - (\sigma + \mu)R] + [\eta C_w - (\phi + \mu)H].\end{aligned}$$

On simplification, all transition terms cancel, yielding

$$\frac{dN}{dt} = \pi - \mu(S + E + I + C_w + R + H) - \delta I.$$

That is,

$$\frac{dN}{dt} = \pi - \mu N - \delta I.$$

Since $\delta I \geq 0$, it follows that

$$\frac{dN}{dt} \leq \pi - \mu N.$$

Consider the comparison equation

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

Its solution is

$$N(t) = N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}).$$

Hence,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}).$$

Therefore, as $t \to \infty$,

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

Thus, all solutions of the model remain in the region

$$\Omega = \left\{(S, E, I, C_w, R, H) \in \mathbb{R}_+^6 : N(t) \leq \frac{\pi}{\mu}\right\}.$$

Hence, the biologically feasible region

$$\Omega = \left\{(S, E, I, C_w, R, H) \in \mathbb{R}_+^6 : 0 \leq N(t) \leq \frac{\pi}{\mu}\right\}$$

is positively invariant and attracting. Therefore, the model is both mathematically and epidemiologically meaningful.

2. Disease-Free Equilibrium Point (Modified Model)

The disease-free equilibrium (DFE) corresponds to the state where no infection persists in the population. Thus, all disease-related compartments are set to zero. That is,

$$E = I = C_w = H = 0.$$

At equilibrium, all time derivatives are set to zero. Hence, from the model equations, we obtain the steady-state system.

From the susceptible equation,

$$\frac{dS}{dt} = \pi + \phi R - (\mu + \alpha I)S = 0. \qquad (3.1)$$

From the recovered equation,

$$\frac{dR}{dt} = \sigma H - (\phi + \mu)R = 0. \qquad (3.2)$$

Substituting the disease-free conditions $I = 0$and $H = 0$into equations (3.1) and (3.2), we obtain

$$\pi + \phi R - \mu S = 0, \qquad (3.3)$$

$$-(\phi + \mu)R = 0. \qquad (3.4)$$

From equation (3.4), it follows that

$$R_0 = 0$$

Substituting $R_0 = 0$into equation (3.3), we obtain

$$\pi - \mu S = 0,$$

which gives

$$S_0 = \frac{\pi}{\mu}.$$

Since all infected and disease-related compartments vanish at the disease-free state, we also have

$$E_0 = I_0 = C_{w0} = H_0 = 0.$$

Therefore, the disease-free equilibrium of the modified system is given by

$$D_0 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right).$$

3. The Basic Reproductive Number R_0(Modified)

New infection matrix:

$$F = \begin{bmatrix} (\mu+\alpha I) \\ 0 \\ 0 \end{bmatrix}, F^* = S^* \begin{bmatrix} 0 & \alpha & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \qquad (3.5)$$

Transition matrix:

$$V = \begin{bmatrix} \alpha SI - (\tau+\mu)E \\ \tau E - (\rho+\mu+\eta\delta)I \\ \eta\delta I - (\gamma+\mu)C_w \end{bmatrix}$$

$$V^* = \begin{bmatrix} -(\tau+\mu) & 0 & 0 \\ \tau & -(\rho+\mu+\eta\delta) & 0 \\ 0 & \eta\delta & -(\gamma+\mu) \end{bmatrix} \qquad (3.6)$$

Inverse:

$$V^{-1} = \begin{bmatrix} -\frac{1}{\tau+\mu} & 0 & 0 \\ -\frac{\tau}{(\mu+\gamma)(\rho+\mu+\eta\delta)} & -\frac{1}{\rho+\mu+\eta\delta} & -\frac{1}{\mu+\gamma} \\ -\frac{\delta\eta\gamma}{(\mu+\gamma)(\mu+\tau)(\rho+\mu+\eta\delta)} & -\frac{\eta\delta}{(\mu+\gamma)(\rho+\mu+\eta\delta)} & -\frac{1}{\mu+\gamma} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} -\frac{\alpha\tau S_0}{(\mu+\gamma)(\rho+\mu+\eta\delta)} & -\frac{\alpha S_0}{\rho+\mu+\eta\delta} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \qquad (3.7)$$

Thus, the spectral radius:

$$R_0 = \frac{\alpha\tau\pi}{\mu(\rho\mu + \rho\tau + \mu^2 + \mu\eta\delta + \eta\delta\tau)} \qquad (3.8)$$

The disease-free equilibrium point

$$D_0 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right)$$

of the modified system is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof

To examine the local stability of the disease-free equilibrium point D_0, we linearize the system around D_0. The Jacobian matrix of the system evaluated at the disease-free equilibrium is given by

$$J(D_0) = \begin{pmatrix} -\mu & 0 & -\frac{\alpha\pi}{\mu} & 0 & \phi & 0 \\ 0 & -(\tau+\mu) & \frac{\alpha\pi}{\mu} & 0 & 0 & 0 \\ 0 & \tau & -(\rho+\mu+\eta\delta) & 0 & 0 & 0 \\ 0 & 0 & \eta\delta & -(\gamma+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\phi+\mu) & \sigma \\ 0 & 0 & 0 & \gamma & 0 & -(\sigma+\mu) \end{pmatrix}. \quad (3.9)$$

To determine the eigenvalues, we form the characteristic equation

$$\det(J(D_0) - \lambda I) = 0.$$

Thus,

$$J(D_0) - \lambda I$$

$$= \begin{pmatrix} -\mu-\lambda & 0 & -\frac{\alpha\pi}{\mu} & 0 & \phi & 0 \\ 0 & -(\tau+\mu)-\lambda & \frac{\alpha\pi}{\mu} & 0 & 0 & 0 \\ 0 & \tau & -(\rho+\mu+\eta\delta)-\lambda & 0 & 0 & 0 \\ 0 & 0 & \eta\delta & -(\gamma+\mu)-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\phi+\mu)-\lambda & \sigma \\ 0 & 0 & 0 & \gamma & 0 & -(\sigma+\mu)-\lambda \end{pmatrix}.$$

On expanding the matrix $[J(D_0) - \lambda I]$, we obtain the characteristic polynomial in the form

$$(-\mu-\lambda)\,[-(\phi+\mu)-\lambda]\;|\,A\,| = 0,$$

where

$$A = \begin{pmatrix} -(\tau+\mu)-\lambda & \frac{\alpha\pi}{\mu} & 0 & 0 \\ \tau & -(\rho+\mu+\eta\delta)-\lambda & 0 & 0 \\ 0 & \eta\delta & -(\gamma+\mu)-\lambda & 0 \\ 0 & 0 & \gamma & -(\sigma+\mu)-\lambda \end{pmatrix}.$$

Since $A$is block lower triangular, its determinant is the product of the determinants of its diagonal blocks. Hence,

$$\det(A) = \left[(\lambda+\tau+\mu)(\lambda+\rho+\mu+\eta\delta) - \frac{\alpha\pi\tau}{\mu}\right](\lambda+\gamma+\mu)(\lambda+\sigma+\mu).$$

Therefore, the characteristic equation becomes

$$(\lambda+\mu)(\lambda+\phi+\mu)(\lambda+\gamma+\mu)(\lambda+\sigma+\mu)\left[(\lambda+\tau+\mu)(\lambda+\rho+\mu+\eta\delta) - \frac{\alpha\pi\tau}{\mu}\right] = 0. \quad (3.10)$$

Thus, four eigenvalues are immediately obtained as

$$\lambda_1 = -\mu, \lambda_2 = -(\phi+\mu), \lambda_3 = -(\gamma+\mu), \lambda_4 = -(\sigma+\mu).$$

These are clearly negative.

4. Endemic Equilibrium Point

The endemic equilibrium point of the system is obtained by setting the right-hand side of the system (2.1) to zero and solving simultaneously when the disease persists in the population, that is,

$$E^* \neq 0, I^* \neq 0, C_w^* \neq 0, H^* \neq 0$$

Thus, the steady-state system becomes:

$$\pi + \phi R^* - (\mu + \alpha I^*)S^* = 0 \quad (3.10)$$

$$\alpha S^* I^* - (\tau+\mu)E^* = 0 \quad (3.11)$$

$$\tau E^* - (\rho+\mu+\eta\delta)I^* = 0 \quad (3.12)$$

$$\eta\delta I^* - (\gamma+\mu)C_w^* = 0 \quad (3.13)$$

$$\sigma H^* - (\phi+\mu)R^* = 0 \quad (3.14)$$

$$\gamma C_w^* - (\sigma+\mu)H^* = 0 \quad (3.15)$$

From equation (3.11):

$$E^* = \frac{\alpha S^* I^*}{\tau + \mu}$$

Substituting into equation (3.12):

$$I^* = \frac{\tau E^*}{\rho + \mu + \eta\delta} = \frac{\alpha\tau S^* I^*}{(\tau + \mu)(\rho + \mu + \eta\delta)}$$

This yields the equilibrium condition:

$$S^* = \frac{(\tau + \mu)(\rho + \mu + \eta\delta)}{\alpha\tau}$$

From equation (3.13):

$$C_w^* = \frac{\eta\delta I^*}{\gamma + \mu}$$

From equation (3.15):

$$H^* = \frac{\gamma C_w^*}{\sigma + \mu}$$

Substituting C_w^*:

$$H^* = \frac{\gamma\eta\delta I^*}{(\gamma + \mu)(\sigma + \mu)}$$

From equation (3.14):

$$R^* = \frac{\sigma H^*}{\phi + \mu}$$

Substituting H^*:

$$R^* = \frac{\sigma\gamma\eta\delta I^*}{(\phi + \mu)(\gamma + \mu)(\sigma + \mu)}$$

Finally, substituting into equation (3.10):

$$S^* = \frac{\pi + \phi R^*}{\mu + \alpha I^*}$$

Thus, the endemic equilibrium of the system is given by:

$$E^* = \frac{\alpha S^* I^*}{\tau + \mu}, I^* = I^*, C_w^* = \frac{\eta\delta I^*}{\gamma + \mu}$$

$$H^* = \frac{\gamma\eta\delta I^*}{(\gamma+\mu)(\sigma+\mu)}, R^* = \frac{\sigma\gamma\eta\delta I^*}{(\phi+\mu)(\gamma+\mu)(\sigma+\mu)}$$
$$S^* = \frac{(\tau+\mu)(\rho+\mu+\eta\delta)}{\alpha\tau}$$

5. Sensitivity Analysis

Sensitivity analysis is carried out to determine the impact of the model parameters on the basic reproductive number R_0. The normalized forward sensitivity index of a variable R_0, with respect to a parameter p, is defined as:

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

Using the expression for the basic reproductive number obtained in equation (3.8):

$$R_0 = \frac{\alpha\tau\pi}{\mu(\rho\mu + \rho\tau + \mu^2 + \mu\eta\delta + \eta\delta\tau)}$$

we compute the sensitivity indices of the model parameters as follows.

6. Sensitivity Indices Computation

To understand how different parameters affects the burden of the disease: we present the sensitivity each parameter that appeared in the R_0 as follows:

The sensitivity index of $R_0$with respect to the transmission rate α is:

$$\Upsilon_\alpha^{R_0} = \frac{\partial R_0}{\partial \alpha} \cdot \frac{\alpha}{R_0} = 1 \qquad (3.16)$$

Similarly, the sensitivity index with respect to the progression rate $\tau$is:

$$\Upsilon_\tau^{R_0} = 1 \qquad (3.17)$$

The sensitivity index with respect to the recruitment rate $\pi$is:

$$\Upsilon_\pi^{R_0} = 1 \qquad (3.18)$$

The sensitivity index with respect to the natural death rate $\mu$is obtained as:

$$\Upsilon_\mu^{R_0} = -\frac{\mu(\rho+\tau+\mu+\eta\delta)}{\rho\mu + \rho\tau + \mu^2 + \mu\eta\delta + \eta\delta\tau} \qquad (3.19)$$

The sensitivity index with respect to $\rho$is:

$$\Upsilon_{\rho}^{R_0} = -\frac{\rho(\mu+\tau)}{\rho\mu+\rho\tau+\mu^2+\mu\eta\delta+\eta\delta\tau} \quad (3.20)$$

The sensitivity index with respect to $\eta$is:

$$\Upsilon_{\eta}^{R_0} = -\frac{\eta\delta(\mu+\tau)}{\rho\mu+\rho\tau+\mu^2+\mu\eta\delta+\eta\delta\tau} \quad (3.21)$$

The sensitivity index with respect to $\delta$is:

$$\Upsilon_{\delta}^{R_0} = -\frac{\eta\delta(\mu+\tau)}{\rho\mu+\rho\tau+\mu^2+\mu\eta\delta+\eta\delta\tau} \quad (3.22)$$

Sensitivity Index with Respect to Parameters

Sensitivity index with respect to γat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= -2037887360000000$

Sensitivity index with respect to ρat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= -5094718400000000$

Sensitivity index with respect to δat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= -101894368000000000$

Sensitivity index with respect to μat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= -83232712204800000000$

Sensitivity index with respect to τat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= 32906640000$

Sensitivity index with respect to αat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= 1600000000$

Sensitivity index with respect to ρat

$\gamma = 0.05, \rho = 0.02, \delta = 0.625, \mu = 0.02, \tau = 1.99, \alpha = 0.0125, \pi = 0.02$:

Sensitivity Index $= 509440000$

Table 3: Adopted Data, Reference, and Sensitivity

PARAMETER	VALUE	REFERENCE	SENSITIVITY INDEX
γ	0.05	Hicham et al. 2023	-1.92
μ	0.02	Ihsan et al. 2023	-7796.83
π	200	Assumed	+155.92
δ	0.625	Ihsan et al. 2023	-95.44
τ	1.99	Ihsan et al. 2023	+0.000031
ρ	Assumed	-4.8	
α	0.0125	Ihsan et al. 2023	+49.97

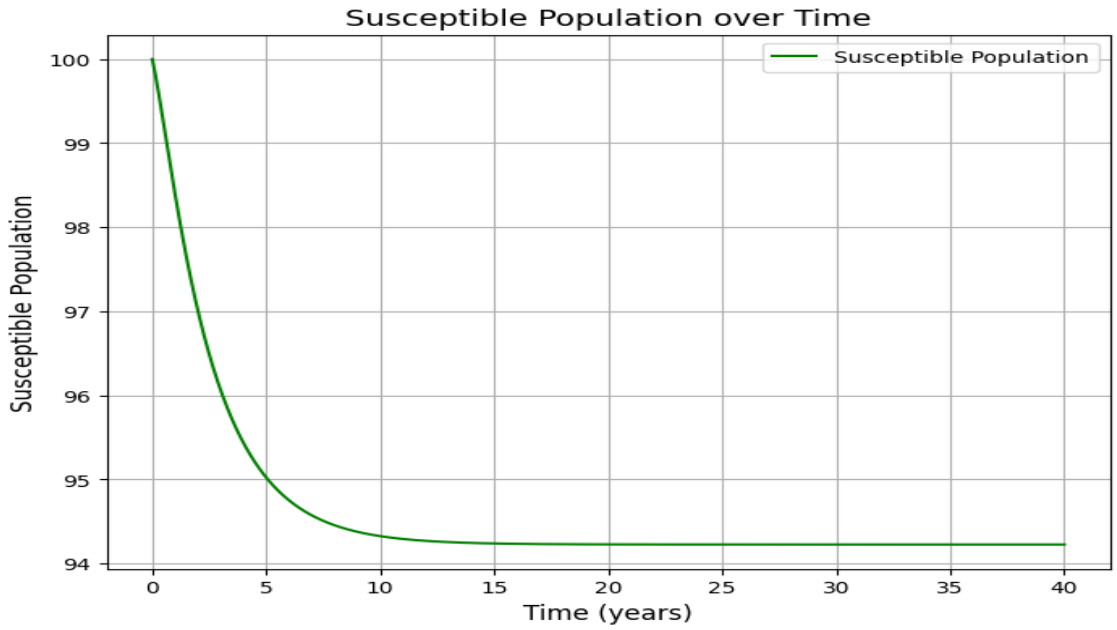

Figure 3: Graph of Susceptible Population

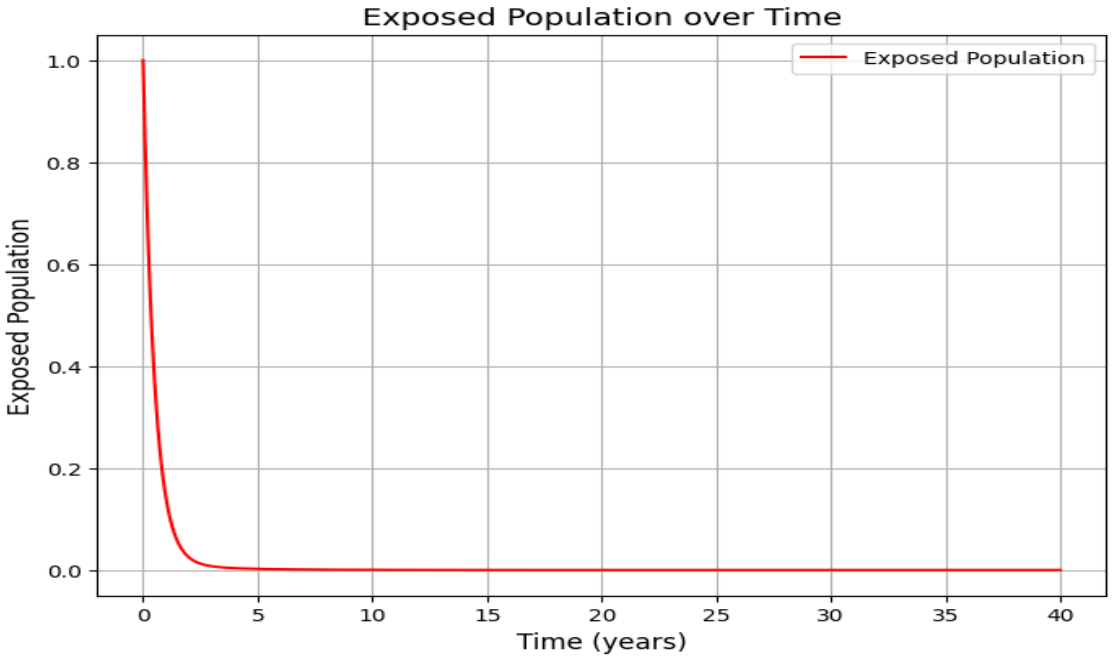

Figure 4: Graph of Exposed Population

Figure 5: Graph of Infectious Population

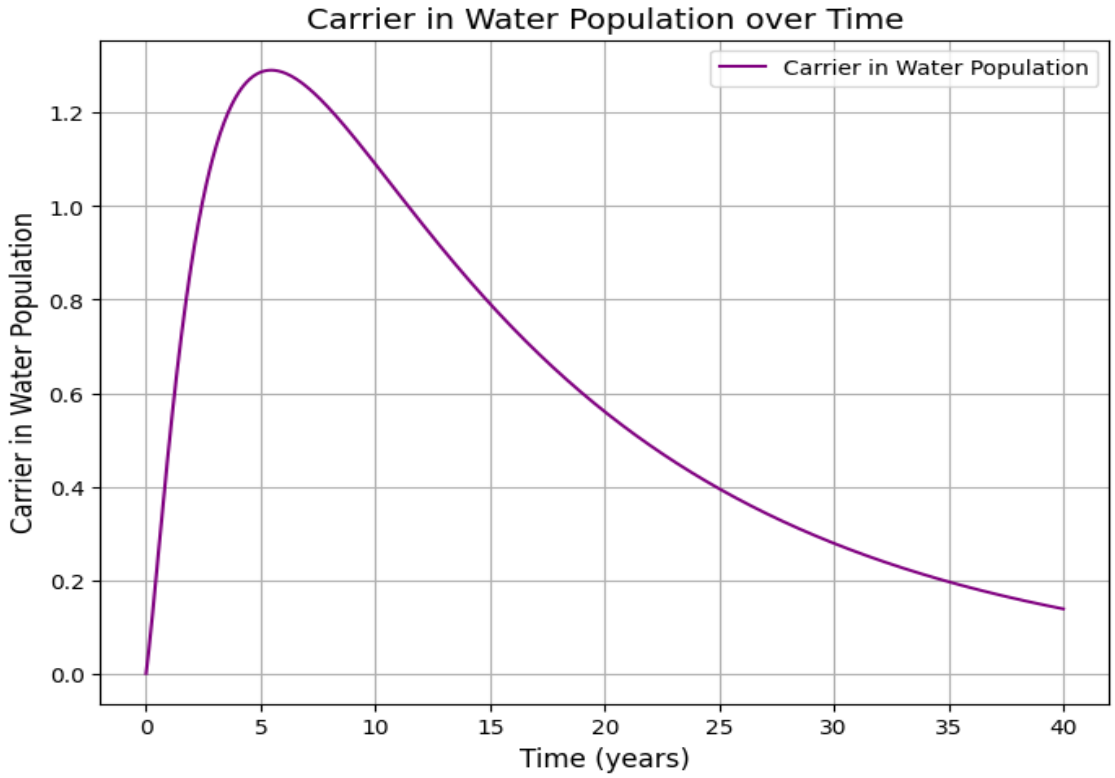

Figure 6: Graph of Carrier in water Population

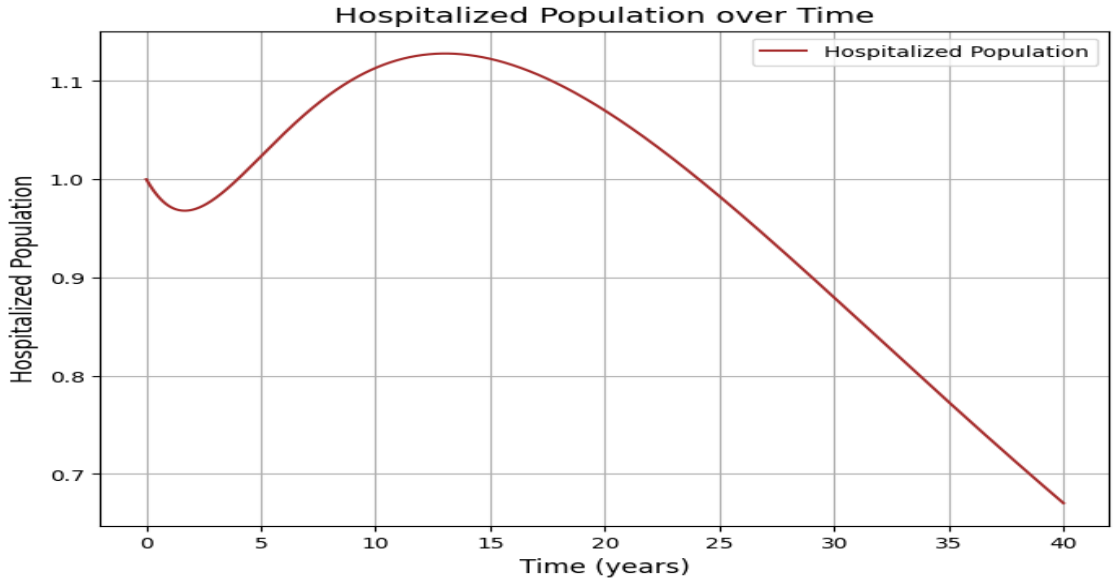

Figure 7: Graph of Hospitalized Population

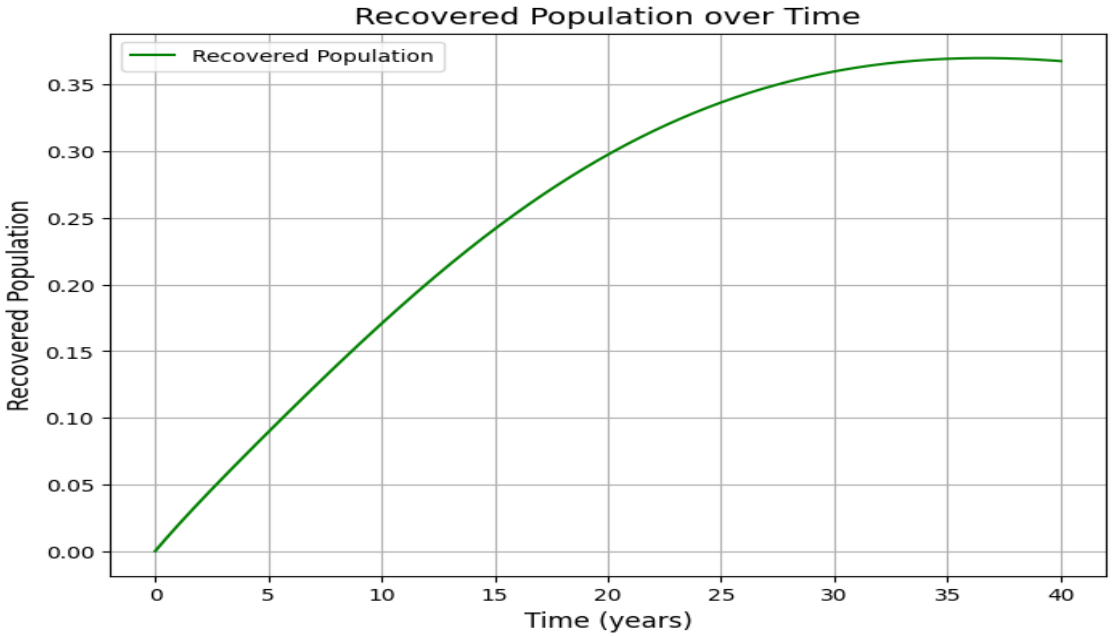

Figure 8: Graph of Recovered Population

Discussion

We have made significant progress by expanding a mathematical model for typhoid fever, based on the existing model, by introducing two new compartments: the chronic and hospital lockdown compartments. As shown in Figure 4, we observed that the number of individuals in the susceptible population decreases over time as some individuals contract typhoid fever and move into the exposed and infectious classes. A more detailed analysis of the exposed (Figure 4) and infectious (Figure 5) compartments reveals an initial rise followed by a drop as individuals are admitted to the hospital and eventually transition to the recovered compartment (R). This transition is reflected in the substantial increase in the hospital lockdown (H) and recovered (R) populations as shown in Figure 6 and Figure 7, respectively. These findings highlight the changing dynamics among the compartments and demonstrate the role of hospitalization and recovery in limiting the spread of typhoid fever.

Our model provides a deeper insight into the course of the disease and emphasizes the importance of combining measures to address infectious diseases. The model analysis reveals key observations about the local stability of both the disease-free equilibrium and the endemic equilibrium, indicating that when the basic reproduction number, R_0, is less than 1, the disease-free equilibrium is stable. However, when R_0exceeds 1, the equilibrium becomes unstable, as shown through the Jacobian matrix method. This underscores the critical role of R_0in the

disease's transmission.

Sensitivity analysis further highlights the importance of specific parameters, showing both positive and negative sensitivity signs. A positive sensitivity index indicates a direct proportional relationship, while a negative index implies an inverse relationship between key parameters and R_0. These findings emphasize the need for coordinated efforts to combat typhoid fever. Governments should prioritize awareness campaigns and ensure that adequate medical facilities are available to address the growing challenges of the epidemic and prevent further spread of typhoid fever.

Conclusion

Our extensive investigation has made it clear that different parameter values have a strong influence on the population distribution of typhoid fever. In particular, our results suggest that raising parameters with a positive sensitivity index will significantly increase the burden of typhoid fever, whereas raising parameters with negative sensitivity index will significantly decrease it. The reproductive number R_O is a key determinant in typhoid fever dynamics, and a positive relationship exists between changes.in R_O and the disease's burden. It is worth noting, our model has shown that combined interventions, such as vaccination, hospitalization and quarantine, can effectively control the transmission of typhoid fever. The number of people who are recovered increases with time as more people are vaccinated and treatment is administered, showing that these strategies are effective in reducing the effects of the disease. We can strive to mitigate the challenges of typhoid fever and protect the health of the population by explaining the effect of these parameters and working towards strategies that help to decrease the burden of the illness. Our results highlight the significance of a complex approach, including medical mitigation and public health strategies, to successfully curb the transmission of typhoid fever and safeguard a population at risk.

The coupled model with its sensitivity analysis provides an in-depth insight into the multifaceted nature of typhoid fever and is an invaluable tool in the fight against this condition. In order to efficiently counteract the transmission of typhoid fever, one must make awareness and education his top priority, and make sure that the population is properly educated about the dangers and impacts of this illness. Such empowerment through knowledge will result in a culture of early intervention and prevention.

In addition there should be maximum vaccination, which should start at birth to offer early protection against typhoid fever. To reduce the risk of contact and decrease the chance of transmission, vaccination must be accompanied by social distancing. It is important to note that in case of an infected or failed vaccination, hospitalization or quarantine as soon as possible is necessary to avoid the further spread and provide adequate treatment. A combination of these measures will play a great role in the control of the spread of typhoid fever, enhance the standard of living of people, and finally, eliminate this disease among people. With the recognition of the value of a multi-faceted approach, we can strive to achieve a future in which the risk of typhoid fever is greatly reduced, and the health of the people is ensured. With this holistic approach, we would develop a society in which people are encouraged to manage their own health, and where the incidence of typhoid fever is greatly reduced [8-23].

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