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Research Article

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A Mathematical Modeling of the Dynamics of Typhoid Fever

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Abstract

A comprehensive mathematical model of typhoid fever was developed to investigate the complex transmission dynamics of the disease, shedding light on the intricate relationships between various factors influencing its spread. The model assumes a replenished population through birth and leverages existing data to validate its accuracy, ensuring a reliable representation of the disease's behavior. The primary objective of this modeling exercise is to inform and enhance strategies for preventing, controlling, and eradicating typhoid fever, ultimately leading to improved public health policy and a better quality of life. Through mathematical analysis, it was revealed that the basic reproductive number R_0 plays a crucial role in determining the global dynamics of the disease. R_0 is less than 1, the disease-free equilibrium is locally stable, indicating that the disease will eventually die out. Conversely, if R_0 exceeds 1, an endemic equilibrium exists, and the disease will persist at a stable level. A thorough sensitivity analysis of the model parameters was conducted, providing valuable insights into the impact of various factors on the spread of typhoid fever. This knowledge enables informed decision-making and effective disease management. The model was solved using the Runge-Kutta scheme of order four, with a 40-year time horizon, and implemented in MATLAB. This study showcases the potency of mathematical modeling in understanding the transmission dynamics of typhoid fever, enabling policymakers and healthcare professionals to develop evidence-based strategies for disease control and prevention. By harnessing the power of mathematical modeling, we can work towards a future where the burden of typhoid fever is significantly reduced, and public health is protected.

Keywords: Basic Reproductive Number, Sensitivity Analysis, Equilibrium Point

1. Introduction

Typhoid fever is a contagious disease that is generally caused by bacteria known as Salmonella typhi. This disease spreads through manure contamination of food or water and infects unprotected people. In this work, our focus is to numerically examine the dynamical behavior of a typhoid fever nonlinear mathematical model. Infectious diseases can be transmitted among people either directly or indirectly. These diseases take place when germs enter into the body, enhance in quantity, and then cause a reaction of the body. As of

2019, an estimated 9 million people get sick from typhoid and 11010 people die from it every year. Typhoid fever is a severe infection disease that can spread in the whole body, influencing numerous organs. If not treated on time, it can cause genuine difficulties and can be life threatening. It is caused by bacteria named Salmonella Typhi1 This bacterium is found in constipated food or water, and it causes illness when it enters the body through drinking or eating. Although, the sanitation of water coverage is improved but the spread of typhoid disease is still a noteworthy public health issue in several emergent countries [1]. The most common typhoid symptoms are migraine, stomach ache, knee pain, spine, muscular pain, lack of appetite, spew, dysentery, spots, and fever [2]. Some patients may have a rash. Some cases may lead to serious complications or even death. Typhoid fever can become more dangerous if not treated instantly. It can damage the internal flow of blood and cause infection in the tissue that lies in the stomach [3]. Typhoid fever can be diagnosed by using some simple blood or stool tests. These tests identify the existence of Salmonella typhi in blood or stool samples. Typhoid fever preventive and control strategies include antibiotic treatments, stool standard precautions, vaccination, environmental sanitation and clean water [4]. Typhoid fever maybe treated with medicines and the symptoms improve within four weeks. The symptoms may return if treatment is not completed [5]. Over 110 years ago, the first typhoid vaccine was developed. Typhoid fever vaccines are available in two forms which are oral and injectable. Among the injectable types, we have: typhoid conjugate vaccine, Tya, and Vi capsular polysaccharide vaccine. They are about 30% to 80% effective within the first 2 years of the specific vaccine in question. When a person takes on the drug resistant strain of typhoid fever and is not properly managed with effective antibiotics, then there is a high chance of it resulting in complications [6]. It is estimated that typhoid fever cases have risen from 11millionn to 21.5 million and five million cases of paratyphoid fever worldwide, with 200,000 deaths occurring each year. It is also estimated that African countries have not been left out with an increasing number of cases between 10 and 100 per 100,000 individuals, with children being the most infected due to poor hygiene and sanitation. As a result of the high rate of infection and the rising spirit of the disease strain, typhoid has become a burden that has turned into a major world health problem. However, vaccination seems to be the essential method for controlling the transmission of the disease. Typhoid fever is a global health problem. Its real impact is difficult to estimate because the clinical picture is confused with those of many other febrile infections. Additionally, the disease is underestimated because there are no bacteriology laboratories in most areas of developing countries. These factors are believed to result in many cases going undiagnosed. On the basis of the literature and the incidence of typhoid fever recorded in control groups in large vaccine field trials with good laboratory support it has been estimated that approximately 17 million cases of typhoid fever and 600 000 associated deaths occur annually. However, the estimates have been biased because study populations have usually been in areas of high incidence. Furthermore, these estimates of burden relate to the clinical syndrome of typhoid fever but not to S. typhi exposure. Since the prevalence of bacteraemia in febrile children is quite high (23%) in areas of endemicity it is suggested that exposure to the bacteria is higher than indicated by the figures that are based solely on the clinical syndrome of typhoid fever. The incidence of the disease in areas of endemicity may resemble the incidences observed in control groups in large vaccine field trials, viz. between 45 per 100 000 per year and over 1000 per 100 000 per year. Preliminary results from recent studies conducted in Bangladesh by ICDDR,B show an incidence of approximately 2000 per 100 000 per year. Typhoid fever also has a very high social and economic impact because of the hospitalization of patients with acute disease and the complications and loss of income attributable to the duration of the clinical illness. It is important to note that reports from some provinces in China and Pakistan have indicated more cases of paratyphoid fever caused by S. paratyphi A than by S. typhi. In areas of endemicity and in large outbreaks, most cases occur in persons aged between 3 and 19 years. In 1997, for example, this age range was reported during an epidemic of the disease in Tajikistan. Nevertheless, clinically apparent bacteraemic S. typhi infection in children aged under three years has been described in Bangladesh, India, Jordan, Nigeria, and elsewhere. In Indonesia there is a mean of 900 000 cases per year with over 20 000 deaths. In Indonesia, people aged 319 years accounted for 91% of cases of typhoid fever 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.

2. 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_w) and individuals under hospital lock down with ongoing health monitoring (H).

The total population is given by $N(t) = S(t) + E(t) + I(t) + R(t) + C_w + H$

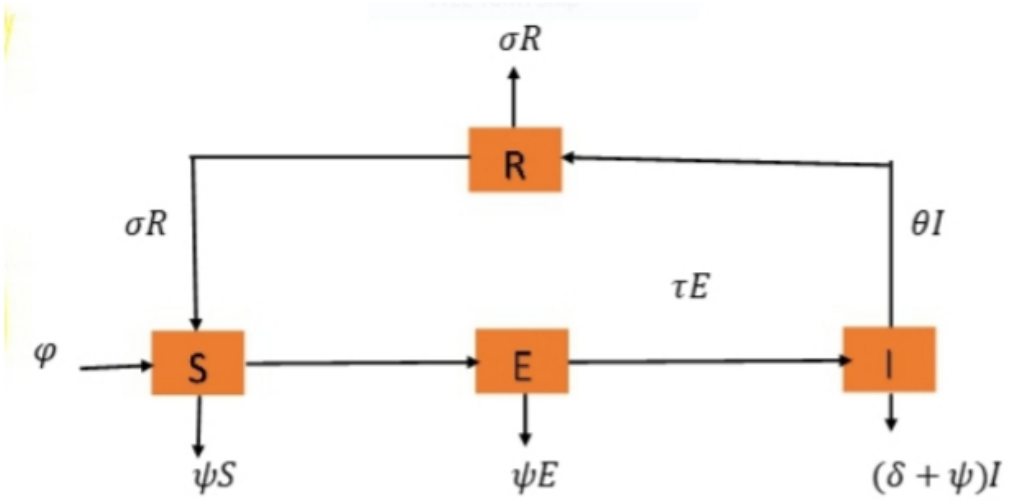

Figure 1: The model diagram for typhoid fever disease transmission (Ihsan et al. 2023)

2.1. 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.

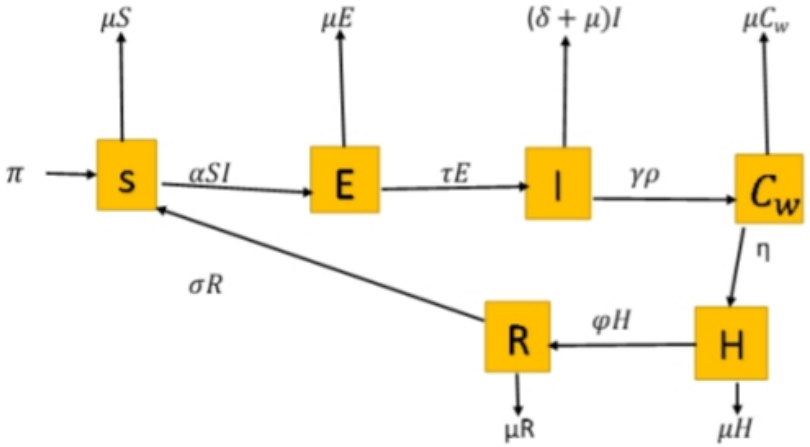

Figure 2: The detailed description of model diagram for typhoid fever transmission

2.2. Model Equation

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

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 1: Variable Description

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

Table 2: Parameter Description

3. Model Analysis

3.1. Possitivity and Boundness

Considering the system equation,

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

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

$$IF = e^{\int(\mu+\alpha I)dt}$$

On multiplying $e^{\int(\mu+\alpha I)dt}$ we get

$$\frac{d}{dt}[S(t)e^{\int(\mu+\alpha I)t}] = (\lambda + \sigma R)e^{\int(\mu+\alpha I)t}$$

$$\int_{t_1} e^{(\mu+\alpha I)t_1} - S_{t_0} = \int_{t_0}^{T_1}(\lambda + \sigma R)e^{\int(\mu+\alpha I)t}dt$$

Therefore,

$$\int_{t_1} = S_{t_0}e^{-(\mu+\sigma I)t_1} + e^{-(\mu+\sigma I)t_1}\int_{t_0}^{t_1}(\lambda + \sigma R)e^{\int(\mu+\alpha I)t}dt \geq 0$$

The same can be done for other state variables for any given time. Hence, the system is well posed.

3.2. Disease-Free Equilibrium Point

For the disease free equilibrium of the system, we set each of the equation to zero(0) and noting that, in the absence of the disease,

$E = I = C_w = H = 0$

Thus,

$\pi + \sigma R - (\mu + \alpha I)S = 0$ (3.1)

$\omega H - (\sigma + \mu)$ (3.2)

Since I = H = 0 then (3.1) and (3.2) becomes,

$\pi + \sigma R - \mu = 0$ (3.3)

$-(\sigma + \mu)R = 0$ (3.4)

From (3.3) and (3.4),

$$R^0 = 0, S^0 = \frac{\pi}{\mu}$$

∴ The disease free equilibrium is;

$D_0 = \{S^0,0,0,0,0,0\}$
$= \{\frac{\pi}{\mu},0,0,0,0,0\}$

3.3. The Basic Reproductive Number R_0

This is an important thresshold needed to analysis a dynamical system.From the system we will consider the new infection F and the incidence terms V respectively.

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

$$V = \begin{bmatrix} \alpha SI - (\tau + \mu)E \\ \tau E - (\delta + \mu + \gamma\rho)I \\ \gamma\rho I - (\eta + \mu)C_w \end{bmatrix} V^* = \begin{bmatrix} -(\tau + \mu) & 0 & 0 \\ \tau & -(\delta + \mu + \gamma\rho) & 0 \\ 0 & \gamma\rho & -(\eta + \mu)C_w \end{bmatrix}$$

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

$$F^*V^{-1} = \begin{bmatrix} \frac{-\alpha\tau S^0}{(\mu+\eta)(\delta+\mu+\gamma\rho)} & \frac{-\alpha S^0}{\delta+\mu+\gamma\rho} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (3.7)$$

The largest spectra of the matrix above is,

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

The equilibrium point (D_0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$
Proof: The Jacobian matrix at DFE associated with the system is,

$$J_{D_0} = \begin{bmatrix} -\mu & 0 & \frac{-\alpha\tau}{\mu} & 0 & 0 & 0 \\ 0 & -(\tau+\mu) & 0 & 0 & 0 & 0 \\ 0 & \tau & -(\delta+\mu+\gamma\rho) & 0 & 0 & 0 \\ 0 & 0 & \gamma\rho & -(\eta+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\sigma+\mu) & \varphi \\ 0 & 0 & 0 & \eta & 0 & -(\varphi+\mu) \end{bmatrix} \quad (3.9)$$

On expanding the matrix $[JD_0 - \lambda I]$ we have;
$\lambda_1 = -\mu, \lambda_2 = -\mu - \eta, \lambda_3 = -\mu - \varphi$, $\lambda_4 = -\mu - \sigma, \lambda_5 = -\mu - \eta, \lambda_6 = -\delta - \mu - \gamma\rho$ All the eigen values are negative. therefore, the disease equilibrium point D_0 is locally asymptotically stable.

When the disease is present in the population we have an endemic state (E_0) where the diseas settles into a stable state when perturbed. The endemic point (E_0) for this state are ;

3.4. Endemic Equilibrium Point

$$S = \frac{(\delta\mu+\delta\tau+\mu\tau\mu^2+\mu\rho\gamma+\rho\gamma\tau)}{\alpha\tau} \quad (3.10)$$

$$\text{Let } E = \frac{a}{b} \quad (3.11)$$

$a = -(\mu+\eta)(\mu+\phi)(\delta+\mu+\rho\gamma)(\delta\mu^3 + mu^3\sigma + \mu^3\tau + \mu^4 + \delta\mu^2\sigma + \delta\mu^2\tau + \mu^3\rho\gamma + \mu^2\sigma\tau - \alpha\mu\eta^1\tau - \alpha\eta^1\sigma\tau + \delta\mu\sigma\tau + \mu^2\rho\gamma\sigma + \mu^2\rho\gamma\tau + \mu\rho\gamma\sigma\tau)$ and
$b = (\tau(\alpha\mu^5 + \alpha\mu^4\sigma + \alpha\mu^4\tau + \alpha\delta\mu^4 + \alpha\mu^4\eta + \alpha\mu^4\phi + \alpha\delta\mu^3\eta + \alpha\delta\mu^3\phi + \alpha\delta\mu^3\sigma + \alpha\delta\mu^3\tau + \alpha\mu^3\eta\phi + \alpha\mu^3\eta\sigma + \alpha\mu^3\eta\tau + \alpha\mu^4\rho\gamma + \alpha\mu^3\phi\sigma + \alpha\mu^3\phi\tau + \alpha\mu^3\sigma\tau + \alpha\delta\mu^2\eta\phi + \alpha\delta\mu^2\eta\sigma + \alpha\delta\mu^2\eta\tau + \alpha\delta\mu^2\phi\sigma + \alpha\delta\mu^2\phi\tau + \alpha\delta\mu^2\sigma\tau + \alpha\mu^3\eta\rho\gamma + \alpha\mu^2\eta\phi\sigma + \alpha\mu^2\eta\phi\tau + \alpha\mu^3\rho\phi\gamma + \alpha\mu^2\eta\sigma\tau + \alpha\mu^3\rho\gamma\sigma + \alpha\mu^3\rho\gamma\tau + \alpha\mu^2\phi\sigma\tau + \alpha\mu^2\eta\rho\phi\gamma + \alpha\mu^2\eta\rho\gamma\sigma + \alpha\mu^2\eta\rho\gamma\tau + \alpha\mu^2\rho\phi\gamma\sigma + \alpha\mu^2\rho\phi\gamma\tau + \alpha\mu^2\rho\gamma\sigma\tau + \alpha\delta\mu\eta\phi\sigma + \alpha\delta\mu\eta\phi\tau + \alpha\delta\mu\eta\sigma\tau + \alpha\delta\mu\phi\sigma\tau + \alpha\delta\eta\phi\sigma\tau + \alpha\mu\eta\phi\sigma\tau + \alpha\mu\eta\rho\phi\gamma\sigma + \alpha\mu\eta\rho\phi\gamma\tau + \alpha\mu\eta\rho\gamma\sigma\tau + \alpha\mu\rho\phi\gamma\sigma\tau)$

$$I = \frac{c}{d} \quad (3.12)$$

where
$c = -(\mu+\eta)(\mu+\phi)(\delta\mu^3 + \mu^3\sigma + \mu^3\tau + \mu^4 + \delta\mu^2\sigma + \delta\mu^2\tau + \mu^3\rho\gamma + \mu^2\sigma\tau - \alpha\mu\eta 1\tau - \alpha\eta^1\sigma\tau + \delta\mu\sigma\tau + \mu^2\rho\gamma\sigma + \mu^2\rho\gamma\tau + \mu\rho\gamma\sigma\tau)$ and $d = (\alpha\mu^5 + \alpha\mu^4\sigma + \alpha\mu^4\tau + \alpha\delta\mu^4 + \alpha\mu^4\eta + \alpha\mu^4\phi + \alpha\delta\mu^3\eta + \alpha\delta\mu^3\phi + \alpha\delta\mu^3\sigma + \alpha\delta\mu^3\tau + \alpha\mu^3\eta\phi + \alpha\mu^3\eta\sigma + \alpha\mu^3\eta\tau + \alpha\mu^4\rho\gamma + \alpha\mu^3\phi\sigma + \alpha\mu^3\phi\tau + \alpha\mu^3\sigma\tau + \alpha\delta\mu^2\eta\phi + \alpha\delta\mu^2\eta\sigma + \alpha\delta\mu^2\eta\tau + \alpha\delta\mu^2\phi\sigma + \alpha\delta\mu^2\phi\tau + \alpha\delta\mu^2\sigma\tau + \alpha\mu^3\eta\rho\gamma + \alpha\mu^2\eta\phi\sigma + \alpha\mu^2\eta\phi\tau + \alpha\mu^3\rho\phi\gamma + \alpha\mu^2\eta\sigma\tau + \alpha\mu^3\rho\gamma\sigma + \alpha\mu^3\rho\gamma\tau + \alpha\mu^2\phi\sigma\tau + \alpha\mu^2\eta\rho\phi\gamma + \alpha\mu^2\eta\rho\gamma\sigma + \alpha\mu^2\eta\rho\gamma\tau + \alpha\mu^2\rho\phi\gamma\sigma + \alpha\mu^2\rho\phi\gamma\tau + \alpha\mu^2\rho\gamma\sigma\tau + \alpha\delta\mu\eta\phi\sigma + \alpha\delta\mu\eta\phi\tau + \alpha\delta\mu\eta\sigma\tau + \alpha\delta\mu\phi\sigma\tau + \alpha\delta\eta\phi\sigma\tau + \alpha\mu\eta\phi\sigma\tau + \alpha\mu\eta\rho\phi\gamma\sigma + \alpha\mu\eta\rho\phi\gamma\tau + \alpha\mu\eta\rho\gamma\sigma\tau + \alpha\mu\rho\phi\gamma\sigma\tau)$

$$C = \frac{e}{f} \quad (3.13)$$

where
$e = -(\mu+\phi)(\mu^4\rho\gamma + \mu^3\rho^2\gamma^2 + \mu^2\rho^2\gamma + \mu^2\rho^2\gamma^2\tau + \delta\mu^3\rho\gamma + \mu^3\rho\gamma\sigma + \mu^3\rho\gamma\tau + \mu\rho\gamma^2\sigma\tau + \delta\mu^2\rho\gamma\sigma + \delta\mu^2\rho\gamma\tau + \mu^2\rho\gamma\sigma\tau - \alpha\mu\eta^1\rho\gamma\tau - \alpha\eta^1\rho\gamma\sigma\tau + \delta\mu\rho\gamma\sigma\tau)$
and $f = (\alpha\mu^5 + \alpha\mu^4\sigma + \alpha\mu^4\tau + \alpha\delta\mu^4 + \alpha\mu^4\eta + \alpha\mu^4\phi + \alpha\delta\mu^3\eta + \alpha\delta\mu^3\phi + \alpha\delta\mu^3\sigma + \alpha\delta\mu^3\tau + \alpha\mu^3\eta\phi + \alpha\mu^3\eta\sigma + \alpha\mu^3\eta\tau + \alpha\mu^4\rho\gamma + \alpha\mu^3\phi\sigma + \alpha\mu^3\phi\tau + \alpha\mu^3\sigma\tau + \alpha\delta\mu^2\eta\phi + \alpha\delta\mu^2\eta\sigma + \alpha\delta\mu^2\eta\tau + \alpha\delta\mu^2\phi\sigma + \alpha\delta\mu^2\phi\tau + \alpha\delta\mu^2\sigma\tau + \alpha\mu^3\eta\rho\gamma + \alpha\mu^2\eta\phi\sigma + \alpha\mu^2\eta\phi\tau + \alpha\mu^3\rho\phi\gamma + \alpha\mu^2\eta\sigma\tau + \alpha\mu^3\rho\gamma\sigma + \alpha\mu^3\rho\gamma\tau + \alpha\mu^2\phi\sigma\tau + \alpha\mu^2\eta\rho\phi\gamma + \alpha\mu^2\eta\rho\gamma\sigma + \alpha\mu^2\eta\rho\gamma\tau + \alpha\mu^2\rho\phi\gamma\sigma + \alpha\mu^2\rho\phi\gamma\tau + \alpha\mu^2\rho\gamma\sigma\tau + \alpha\delta\mu\eta\phi\sigma + \alpha\delta\mu\eta\phi\tau + \alpha\delta\mu\eta\sigma\tau + \alpha\delta\mu\phi\sigma\tau + \alpha\delta\eta\phi\sigma\tau + \alpha\mu\eta\phi\sigma\tau + \alpha\mu\eta\rho\phi\gamma\sigma + \alpha\mu\eta\rho\phi\gamma\tau + \alpha\mu\eta\rho\gamma\sigma\tau + \alpha\mu\rho\phi\gamma\sigma\tau)$

$$R = \frac{g}{h} \quad (3.14)$$

where
$g = -(\phi(\mu^2\eta\rho^2\gamma^2 + \mu^3\eta\rho\gamma + \mu\eta\rho^2\gamma^2\tau + \delta\mu^2\eta\rho\gamma + \mu^2\eta\rho\gamma\tau - \alpha\eta\eta 1\rho\gamma\tau + \delta\mu\eta\rho\gamma\tau)$ and $h = (\alpha\mu^5 + \alpha\mu^4\sigma + \alpha\mu^4\tau + \alpha\delta\mu^4 + \alpha\mu^4\eta + \alpha\mu^4\phi + \alpha\delta\mu^3\eta + \alpha\delta\mu^3\phi + \alpha\delta\mu^3\sigma + \alpha\delta\mu^3\tau + \alpha\mu^3\eta\phi + \alpha\mu^3\eta\sigma + \alpha\mu^3\eta\tau + \alpha\mu^4\rho\gamma + \alpha\mu^3\phi\sigma + \alpha\mu^3\phi\tau + \alpha\mu^3\sigma\tau + \alpha\delta\mu^2\eta\phi + \alpha\delta\mu^2\eta\sigma + \alpha\delta\mu^2\eta\tau + \alpha\delta\mu^2\phi\sigma + \alpha\delta\mu^2\phi\tau + \alpha\delta\mu^2\sigma\tau + \alpha\mu^3\eta\rho\gamma + \alpha\mu^2\eta\phi\sigma + \alpha\mu^2\eta\phi\tau + \alpha\mu^3\rho\phi\gamma + \alpha\mu^2\eta\sigma\tau + \alpha\mu^3\rho\gamma\sigma + \alpha\mu^3\rho\gamma\tau + \alpha\mu^2\phi\sigma\tau + \alpha\mu^2\eta\rho\phi\gamma + \alpha\mu^2\eta\rho\gamma\sigma + \alpha\mu^2\eta\rho\gamma\tau + \alpha\mu^2\rho\phi\gamma\sigma + \alpha\mu^2\rho\phi\gamma\tau + \alpha\mu^2\rho\gamma\sigma\tau + \alpha\delta\mu\eta\phi\sigma + \alpha\delta\mu\eta\phi\tau + \alpha\delta\mu\eta\sigma\tau + \alpha\delta\mu\phi\sigma\tau + \alpha\delta\eta\phi\sigma\tau + \alpha\mu\eta\phi\sigma\tau + \alpha\mu\eta\rho\phi\gamma\sigma + \alpha\mu\eta\rho\phi\gamma\tau + \alpha\mu\eta\rho\gamma\sigma\tau + \alpha\mu\rho\phi\gamma\sigma\tau)$

$$H = \frac{\iota}{j} \tag{3.15}$$

where
$\iota = -(\mu^3\eta\rho^2\gamma^2 + \mu^4\eta\rho\gamma + \mu^2\eta\rho^2\gamma^2\sigma + \mu^2\eta\rho^2\gamma^2\tau + \delta\mu^3\eta\rho\gamma + \mu^3\eta\rho\gamma\sigma + \mu^3\eta\rho\gamma\tau + \delta\mu^2\eta\rho\gamma\sigma + \delta\mu^2\eta\rho\gamma\tau + \mu^2\eta\rho\gamma\sigma\tau + \mu\eta\rho^2\gamma^2\sigma\tau - \alpha\mu\eta\eta^1\rho\gamma\tau - \alpha\eta\eta^1\rho\gamma\sigma\tau + \delta\mu\eta\rho\gamma\sigma\tau)$ and $j = (\alpha\mu^5 + \alpha\mu^4\sigma + \alpha\mu^4\tau + \alpha\delta\mu^4 + \alpha\mu^4\eta + \alpha\mu^4\phi + \alpha\delta\mu^3\tau + \alpha\delta\mu^3\phi + \alpha\delta\mu^3\sigma + \alpha\delta\mu^3\tau + \alpha\mu^3\eta\phi + \alpha\mu^3\eta\sigma + \alpha\mu^3\eta\tau + \alpha\mu^4\rho\gamma + \alpha\mu^3\phi\sigma + \alpha\mu^3\phi\tau + \alpha\mu^3\sigma\tau + \alpha\delta\mu^2\eta\phi + \alpha\delta\mu^2\eta\sigma + \alpha\delta\mu^2\eta\tau + \alpha\delta\mu^2\phi\sigma + \alpha\delta\mu^2\phi\tau + \alpha\delta\mu^2\sigma\tau + \alpha\mu^3\eta\rho\gamma + \alpha\mu^2\eta\phi\sigma + \alpha\mu^2\eta\phi\tau + \alpha\mu^3\rho\phi\gamma + \alpha\mu^2\eta\sigma\tau + \alpha\mu^3\rho\gamma\sigma + \alpha\mu^3\rho\gamma\tau + \alpha\mu^2\phi\sigma\tau + \alpha\mu^2\eta\rho\phi\gamma + \alpha\mu^2\eta\rho\gamma\sigma + \alpha\mu^2\eta\rho\gamma\tau + \alpha\mu^2\rho\phi\gamma\sigma + \alpha\mu^2\rho\phi\gamma\tau + \alpha\mu^2\rho\gamma\sigma\tau + \alpha\delta\mu\eta\phi\sigma + \alpha\delta\mu\eta\phi\tau + \alpha\delta\mu\eta\sigma\tau + \alpha\delta\mu\phi\sigma\tau + \alpha\delta\eta\phi\sigma\tau + \alpha\mu\eta\phi\sigma\tau + \alpha\mu\eta\rho\phi\gamma\sigma + \alpha\mu\eta\rho\phi\gamma\tau + \alpha\mu\eta\rho\gamma\sigma\tau + \alpha\mu\rho\phi\gamma\sigma\tau)$

3.5. Sensitivity Analysis

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:

sensitivity with respect to γ : $\frac{-(\alpha\rho\tau(\mu\rho+\rho\tau))}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\mu\rho\gamma+\rho\tau\gamma)^2)}$

sensitivity with respect to ρ : $\frac{-(\alpha\pi\tau(\gamma\mu+\gamma\tau))}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\mu\rho\gamma+\rho\tau\gamma)^2)}$

sensitivity with respect to δ : $\frac{-(\alpha\pi\tau(\mu+\tau))}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\mu\rho\gamma+\rho\tau\gamma)^2)}$

sensitivity with respect to μ : $\frac{-(\alpha\pi\tau)}{(\mu^2(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\gamma\mu\rho+\gamma\rho\tau))-(\alpha\pi\tau(\delta+2\mu+\gamma\rho+1))(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\mu\rho\gamma+\rho\tau\gamma)^2)}$

sensitivity with respect to τ : $\frac{(\alpha\pi)/(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\gamma\mu\rho+\gamma\rho\tau))-(\alpha\rho\tau(\delta+\gamma\rho+1))}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\mu\rho\gamma+\rho\tau\gamma)^2)}$

sensitivity with respect to α : $\frac{(\pi\tau)}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\gamma\mu\rho+\gamma\rho\tau))}$

sensitivity with respect to π : $\frac{(\alpha\tau)}{(\mu(\mu+\tau+\delta\mu+\delta\tau+\mu^2+\gamma\mu\rho+\gamma\rho\tau))}$

sensitivity index with respect to γ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{-2037887360000000}{1067594654320489}$

sensitivity index with respect to ρ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{-5094718400000000}{1067594654320489}$

sensitivity index with respect to δ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{-101894368000000000}{1067594654320489}$

sensitivity index with respect to μ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{-83232712204800000000}{1067594654320489}$

sensitivity index with respect to τ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{32906640000}{1067594654320489}$

sensitivity index with respect to α at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$ $\frac{1600000000}{32674067}$

sensitivity index with respect to ρ at $\gamma = 0.05$ $\rho = 0.02$ $\delta = 0.625$ $\mu = 0.02$ $\tau = 1.99$ $\alpha = 0.0125$ and $\pi = 0.02$: $\frac{5094400000}{32674067}$

PARAMETERS	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

Table 3: Adopted data and Reference

4. Results

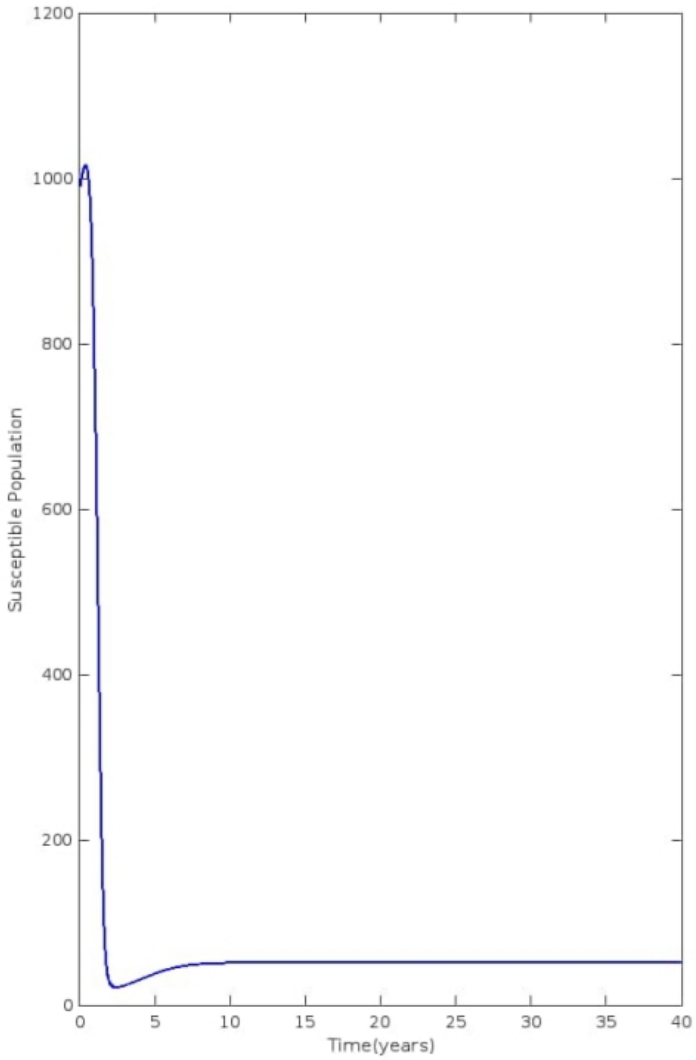

Figure 3: Graph of Susceptible Population

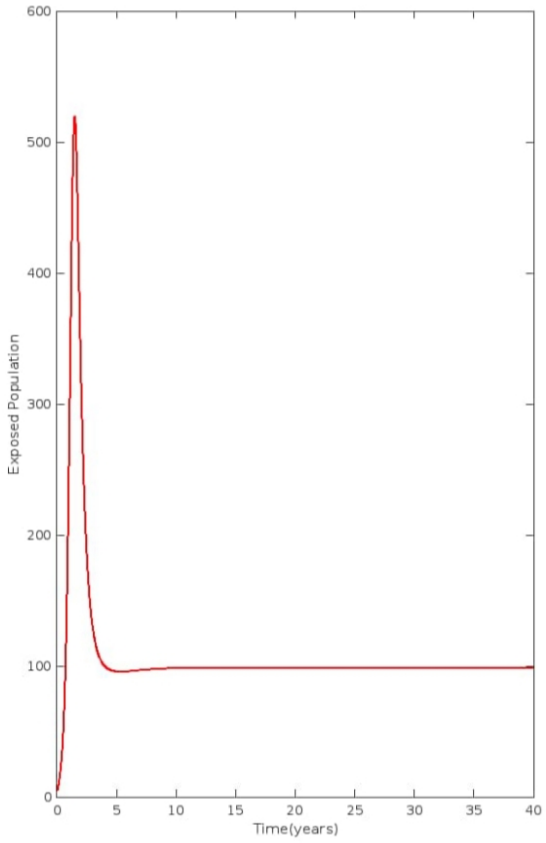

Figure 4: Graph of Exposed Population

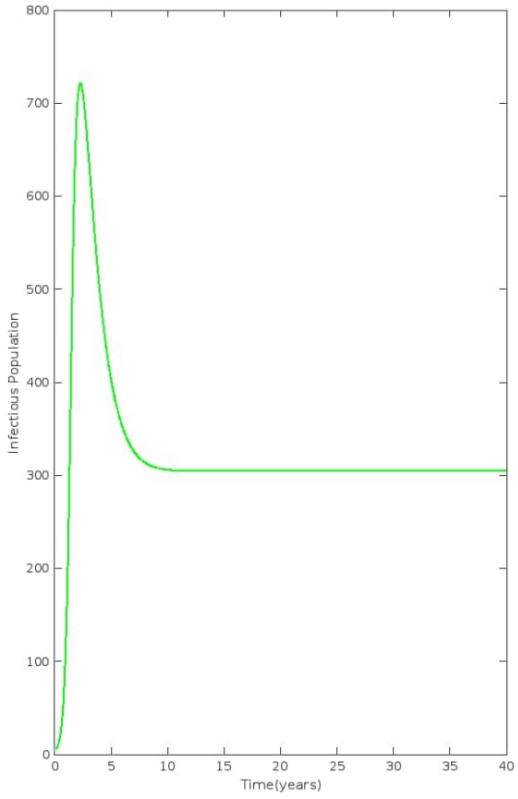

Figure 5: Graph of Infectious Population

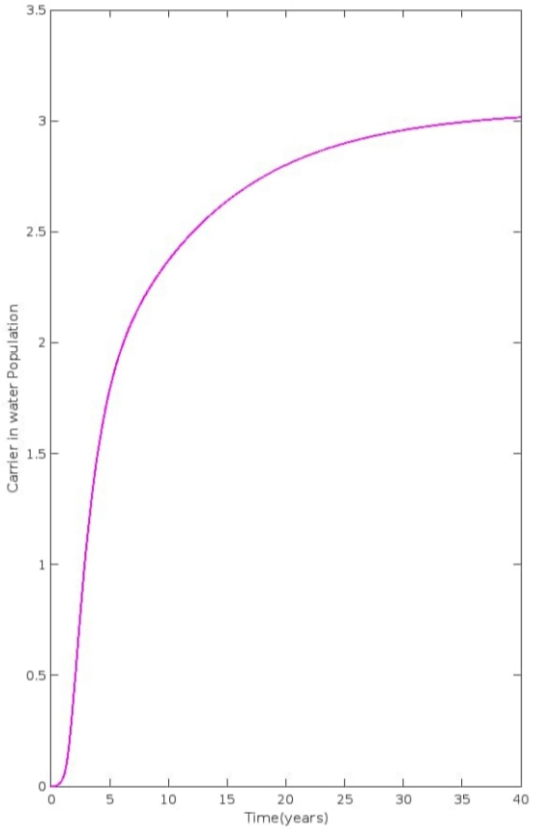

Figure 6: Graph of Carrier in water Population

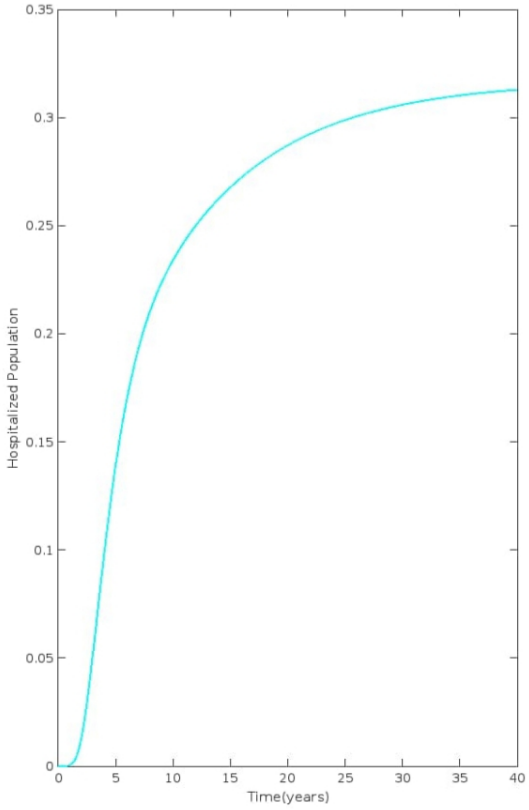

Figure 7: Graph of Hospitalized Population

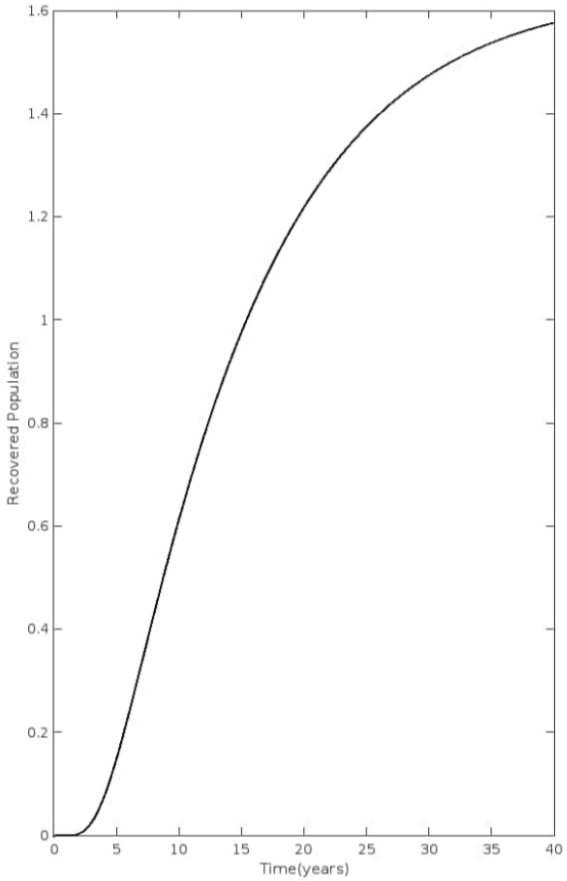

Figure 8: Graph of Recovered Population 5

5. Discussion

We have achieved a significant milestone by successfully extending a mathematical model, building upon the existing typhoid fever model by incorporating two new compartments: the chronic compartment and the hospital lockdown compartment.

Our findings, as illustrated in Figure 4.1, indicate that over time, the susceptible population decreases as individuals become infected with typhoid fever and progress to the exposed and infectious classes. A closer examination of the exposed (Figure 4.2) and infectious (Figure 4.3) compartments reveals an initial increase, followed by a decline as individuals are hospitalized and eventually move into the recovered compartment (R). This transition is evident in the significant growth of the hospital lockdown (H) and recovered (R) populations, as shown in Figures 4.5 and 4.6, respectively. These results demonstrate the dynamic interplay between the various compartments and highlight the importance of hospitalization and recovery in mitigating the spread of typhoid fever. Our extended model provides a more comprehensive understanding of the disease's progression and underscores the value of integrated interventions in combating infectious diseases. Our model analysis reveals a crucial finding: the existence of local stability for both the disease-free equilibrium and the endemic equilibrium, contingent upon the basic reproduction number R_0 being less than 1. However, when R0 exceeds 1, the equilibrium becomes unstable, as determined through the Jacobian matrix approach. This highlights the critical threshold of R0 in determining the disease's spread. Sensitivity analysis identifies key parameters that drive the epidemiology of typhoid fever, with positive sensitivity signs indicating a direct proportional relationship with R_0 and negative signs indicating an inverse proportional relationship (Table 4.1). These findings underscore the need for concerted efforts to combat the disease. To effectively mitigate the spread of typhoid fever, the government must implement comprehensive awareness programs and ensure access to adequate medical facilities to meet the demands of the epidemic.

6. Conclusion

Our comprehensive analysis has elucidated the significant impact of various parameter values on the spread of typhoid fever within the population. Specifically, our findings indicate that increasing parameters with a positive sensitivity index will substantially exacerbate the burden of typhoid fever, while increasing parameters with a negative sensitivity index will have a profound mitigating effect. The basic reproductive number R_0 plays a pivotal role in determining the dynamics of typhoid fever, with a direct correlation between increases

in R_0 and the disease's burden. Notably, our model has demonstrated the efficacy of integrated interventions, including vaccination, hospitalization, and quarantine measures, in controlling the spread of typhoid fever. As more individuals receive vaccination and appropriate treatment, the recovered population grows over time, highlighting the effectiveness of these strategies in mitigating the disease's impact. By elucidating the impact of these parameters and implementing targeted strategies to reduce the burden of typhoid fever, we can work towards alleviating its challenges and safeguarding public health. Our findings underscore the importance of a multifaceted approach, incorporating both medical interventions and public health measures, to effectively combat the spread of typhoid fever and protect vulnerable populations.

The integrated model, bolstered by its sensitivity analysis, offers a profound understanding of the complex dynamics of typhoid fever, serving as an indispensable tool in the quest to combat this disease. To effectively mitigate the spread of typhoid fever, it is imperative to prioritize awareness and education, ensuring that the public is well-informed about the risks and consequences of this disease. This knowledge empowerment will foster a culture of prevention and early intervention.

Furthermore, maximum vaccination efforts should be implemented, ideally commencing at birth, to provide early protection against typhoid fever. Vaccination should be complemented by social distancing measures to minimize contact rates and reduce the likelihood of transmission. In the event of a failed vaccination or infection, prompt hospitalization or quarantine is crucial to prevent further spread and ensure proper treatment. The synergistic implementation of these measures will significantly contribute to controlling the spread of typhoid fever, improving the quality of life for individuals, and ultimately, eradicating this disease from the population. By acknowledging the importance of a multi-faceted approach, we can work towards a future where the threat of typhoid fever is significantly diminished, and public health is safeguarded. Through this comprehensive strategy, we can create a society where individuals are empowered to take control of their health, and where the burden of typhoid fever is significantly alleviated [8-23].

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