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# Modeling and Simulation of Typhoid Fever Using a Fractional-Order Approach with the Generalized Adams–Bashforth–Moulton Method

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## Abstract:

We consider the epidemiological characteristics of Typhoid fever infection in this paper in the equation of a fractional-order mathematical model in Caputo derivative. The interventions that are employed in the model to control the disease include treatment and vaccination to investigate the impact of the controls on the dynamics of the disease. The existence and uniqueness of solutions under the frame of the fractional order and the stability of the endemic equilibrium point are defined and tested by the theory of Lyapunov functions. The model is numerically determined by using the fractional Adams-Bashforth-Moulton algorithm to point out the modification of the model parameters and the fractional orders of the model parameters into the influence of each of the above parameters on the disease progression. It has been demonstrated by the use of simulation that increased treatment and vaccination of the disease reduces the prevalence of Typhoid fever, and indicates the high degree of flexibility and realism of the fractional-order models compared to the classical integer order equations. The significance of fractional modeling in the description of the interactions between the effects of memory and nonlocal interaction between the biological systems is identified in the paper, and this improves the comprehension and management of infectious diseases. The model however presupposes that the population is homogeneous mixed and hypothetical values of the parameters therefore inhibits empirical validation. In order to render the model more predictive and applicable in practice in the development of effective control strategies on Typhoid fever, future investigations should be able to incorporate the spatial heterogeneity, stochastic effects.

## Keywords:

*Typhoid fever*, *Fractional*, *Adam-Bashforth-Moulton*, *Transmission*, *Control*, *strategies*

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## 1.0 Introduction

Typhoid fever, a serious illness caused by Salmonella Typhi, remains a major global health threat, especially in developing regions. Poor hygiene allows the bacteria to spread through contaminated food and water, leading to millions of infections and hundreds of thousands of deaths each year. The impact is starkly different from one community to another. For instance, while the Mekong Delta in Vietnam sees 198 cases per 100,000 people, Delhi, India, experiences a much higher rate of 980 per 100,000. History shows that improving basic living standards like access to clean water and sanitation is one of the most effective ways to reduce transmission. To fully combat this disease, we need a worldwide research effort that examines the problem from every angle, from the microscopic interaction between the pathogen and its host to the larger social, economic, and environmental factors that allow it to thrive.

Mathematical modeling has become a cornerstone of modern epidemiology, offering a way to simulate how diseases spread and to test the potential impact of interventions like vaccines. By translating biological processes into equations, these models help scientists pinpoint the most critical factors that drive an outbreak. A common and powerful approach uses systems of ordinary differential equations (ODEs). When built with the right assumptions and parameters, these equations can effectively represent the complex dynamics of disease transmission. For example, Fraser and colleagues (2007) used such a model to comprehensively assess how vaccination programs can alter the spread of typhoid fever.

To tackle public health crises like typhoid fever, scientists often turn to mathematical models. These models, built using differential equations, help us understand the core biological mechanisms that drive how a disease spreads. This entire field, known as mathematical epidemiology, has produced numerous studies on typhoid transmission, with foundational work from researchers like Ashcroft (1964) and Fraser et al. (2007), and continued by many others.

While traditional models are useful, fractional differential equations offer a more powerful way to simulate complex biological systems like disease spread. Their key advantage is a "memory effect," which allows the model to incorporate the past history of the disease such as previous infections and treatments into its current state.

In this paper, we use this advanced approach to model the transmission of typhoid fever, specifically including the effects of treatment and vaccination campaigns. By simulating different intervention scenarios, we can identify the most effective strategies to reduce the disease's prevalence. This method provides a more realistic picture, which is crucial for tackling persistent challenges like drug resistance, re-infection, and limited healthcare resources.

Fractional calculus is a powerful branch of mathematics that has evolved significantly over time. As highlighted by Atokolo et al. (2022), its true strength lies in modeling complex, real-world systems.

Unlike simpler "classical" models that only capture a snapshot in time, fractional-order models have a "memory." This means they can account for how past events influence the present, providing a more complete and realistic picture of a system's overall behavior.

This is especially valuable in understanding infectious diseases like typhoid fever. By more accurately depicting how the disease spreads over time, these models offer a stronger foundation for developing effective control strategies.

Think of modeling a disease like trying to understand a story. Traditional mathematical tools, known as Caputo and Riemann-Liouville derivatives, have been the go-to methods for years to write these "biological stories." More recently, scientists have started using newer, more advanced tools like the Mittag-Leffler and Atangana-Baleanu operators, which can often tell a smoother and more realistic results.

Recent years have seen a significant shift in mathematical epidemiology towards using fractional-order models to understand and combat infectious diseases. Unlike traditional models, these sophisticated tools can incorporate the "memory" of a system, leading to more realistic simulations of complex disease dynamics.

This approach has been successfully applied across a wide spectrum of public health threats. The work of Atokolo et al. (2022) on the Zika virus and Atokolo et al. (2023) on Lassa fever demonstrated how fractional-order models, solved using methods like the Laplace Adomian Decomposition Method (LADM), are effective for evaluating control strategies. Similarly, Yunus et al. (2023) found that a fractional COVID-19 model for Nigeria showed a better predicted recovery rate when vaccination and treatment were included, outperforming classic integer-order models. The flexibility of these models is further highlighted by their application to diverse pathogens. Omede et al. (2024) used a Caputo derivative-based model for soil-transmitted helminths, while Amos et al. (2024) and James et al. (2024) focused on Hepatitis C and HIV/AIDS, respectively. These studies, often employing the Adams-Bashforth-Moulton method, consistently found that fractional models were more adaptable and better at showing how reduced contact rates and effective treatment can curb transmission. This finding was reinforced by Abah et al. (2024)**,** who also used the Adams-Bashforth-Moulton method to capture the nuanced impact of public health interventions.

Finally, the power of fractional calculus extends to modeling complex co-infections. Ahmed et al. (2021) developed an ABC-fractional order model to control the co-epidemic dynamics of HIV and COVID-19, and the comprehensive review by Smith et al. (2023) synthesized the latest modeling approaches for Hepatitis C and COVID-19 co-infections, identifying key trends and future research directions.

Fractional-order models are gaining popularity because they offer a more adaptable and realistic way to model complex systems. Their key strength lies in capturing "non-local" and "memory" effects meaning they can account for how past conditions and distant interactions influence the present, something traditional models often miss.

This powerful ability to handle real-world complexity has inspired researchers to apply fractional calculus to increasingly challenging mathematical problems. For instance, building on foundational work like that of Ali et al. (2017), who pioneered stability analysis for fractional boundary value problems, others like Ullah et al. (2024) have developed innovative methods. Ullah's team, for example, combined Laplace transforms and decomposition to solve complex fuzzy integral equations, pushing the boundaries of dynamic systems theory.

The objectives that this paper is expected to accomplish are as follows:

- The proposed fractional-order model must have existence and uniqueness of solutions.
- Use Lyapunov function to perform a stability analysis of the endemic equilibrium point.
- Numerically computing solutions using the fractional Adams-Bashforth-Moulton method.
- Carry out numerical simulation so that the model behavior can be studied.

Our review of existing research on typhoid fever models revealed a gap: no previous study has used the Adams-Bashforth-Moulton method within a fractional calculus framework to simulate and analyze the typhoid fever transmission and control.

This paper is organized to address this gap. In Section 2, we present the mathematical model. Section 3 provides the analytical solutions, followed by the numerical results in Section 4. Finally, Section 5 offers a conclusion and discussion of our findings.

**1.1Preliminary**

This section covers the basics of fractional calculus. We will use the right and left Caputo fractional derivatives, following the work of Milici et al. (2018) and Bonyah et al. (2020). We'll also show how this powerful math is used to solve real-world problems in fields like physics, engineering, and biology.

Definition 1: Let $f \in \Lambda^{\infty}(R)$ then the left and right Caputo fractional derivative of the function $f$ is given by:

$$ {}^{C}D_t^{\gamma} f(t) = \left( {}_t^{0}D_t^{-(n-\gamma)} \left( \frac{d}{dt} \right)^n f(t) \right) $$

$$ {}^{C}D_t^{\gamma} f(t) = \frac{1}{\Gamma(n-\gamma)} \int_0^t \left( (t-\lambda)^{n-\gamma-1} f^n(\lambda) \right) d\lambda \qquad (1) $$

The same way

$$ {}^{C}D_t^{\gamma} f(t) = \left( D_T^{-(n-\gamma)} \left( \frac{-d}{dt} \right)^n \right) f(t) $$

$$ {}^{C}D_T^{\gamma} f(t) = \frac{(-1)^n}{\Gamma(n-\gamma)} \int_t^T (\lambda - t)^{n-\gamma-1} f^n(\lambda) d\lambda $$

Definition 2: The generalized Mittag-Leffler function $E_{\gamma,\beta}(x)$ for $x \in R$ is given by

$$ E_{\gamma,\beta}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(\gamma n + \psi)}, \ \gamma, \psi > 0 \qquad (2) $$

which can also be represented as

$$E_{\gamma,\psi}\left(x\right)=xE_{\gamma,\gamma+\psi(x)}+\frac{1}{\Gamma\left(\psi\right)} \tag{3}$$

$$E_{\gamma,\psi}\left(x\right)=L\left[t^{\psi-1}E_{\gamma,\psi\left(\pm\omega t^{\eta}\right)}\right]=\frac{S^{\gamma-\psi}}{S^{\gamma}\pm\omega}\ . \tag{4}$$

Proposition 1.1

Let $f\in\Lambda^{\infty}\left(R\right)\cap C\left(R\right)$ and $\gamma\in R, n-1<\gamma<n,$

therefore, the conditions given below holds:

1. ${}_{t_0}^{C}D_t^{\gamma}I^{\gamma}f\left(t\right)=f\left(t\right)$

2. ${}_{t_0}^{C}D_t^{\gamma}I^{\gamma}f\left(t\right)=f\left(t\right)-\sum_{k-0}^{n-k}\frac{t^k}{K!}f^k\left(t_0\right)$

### 3.1 Model Formulation

In modeling the dynamics, the population is divided into seven groups: Susceptible human population $\left(S_h\right)$, Exposed human population $\left(E_h\right)$, Vaccinated human population $\left(V_h\right)$, infected human population $\left(I_h\right)$, humans on typhoid fever treatment $\left(T_h\right)$, Recovered human population $\left(R_h\right)$ and bacteria population.

The susceptible humans are recruited at the rate of $\Lambda_h$, while the susceptible bacteria population are recruited at the rate of $\Lambda_B$, Contact rate between the susceptible humans and infected humans population with typhoid fever, Contact rate between the susceptible humans and human population on typhoid fever treatment, Contact rate between the susceptible humans and bacteria population are $\beta_1$, $\beta_2$ *and* $\beta_3$ respectively. Natural death rate of human population and bacteria population are $\mu_h$ and $\mu_B$ respectively. Disease induced death rate of typhoid fever infected humans, Disease induced death rate of humans on typhoid fever treatment are $\delta_1$ *and* $\delta_2$ respectively. typhoid fever re-infection rate of recovered human population $\sigma$, Vaccination rate of susceptible human population against typhoid fever $\phi_1$, Waning rate vaccine $\phi_2$, Progression rate from Exposed human population to typhoid fever infected human population $\theta$, Treatment rate of typhoid fever infected human population $\eta$, Recovery rate due to treatment of typhoid fever $\alpha$.

### 3.2 Model Assumptions

1. We assume an imperfect vaccine in the human population
2. We assume exogenous re-infection in human population
3. We assume natural death in the population

4. We assume disease induced death in the population.

### 3.3 Model Flow Chart

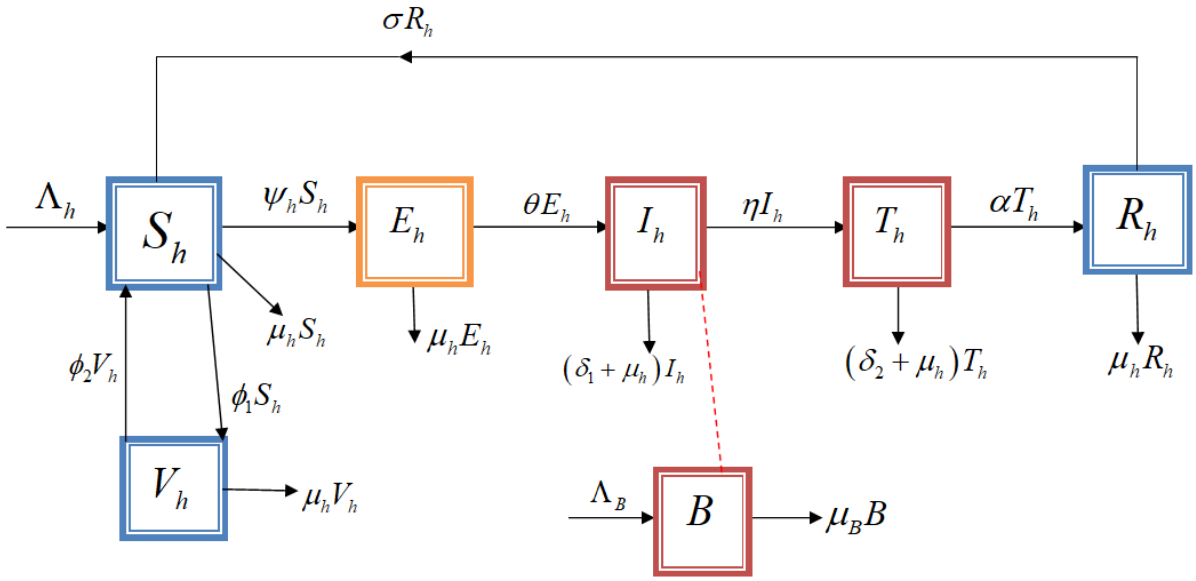


**Fig.1: Typhoid fever model flow Diagram**

### 3.4 Model Equations

$$\frac{dS_h}{dt} = \Lambda_h + \phi_2 V_h + \sigma R_h - \psi_h S_h - (\phi_1 + \mu_h) S_{h,}$$

$$\frac{dE_h}{dt} = \psi_h S_h - (\theta + \mu_h) E_{h,}$$

$$\frac{dV_h}{dt} = \phi_1 S_h - (\phi_2 + \mu_h) V_{h,}$$

$$\frac{dI_h}{dt} = \theta E_h - (\eta + \delta_1 + \mu_h) I_{h,}$$

$$\frac{dT_h}{dt} = \eta I_h - (\alpha + \delta_2 + \mu_h) T_{h,}$$

$$\frac{dR_h}{dt} = \alpha T_h - (\sigma + \mu_h) R_{h,}$$

$$\frac{dB}{dt} = \Lambda_B - \mu_B B.$$

Where $\psi_h = \dfrac{(\beta_1 I_h + \beta_2 T_h + \beta_3 B)}{N_h}$.

### 3.5 Model Variables and Parameters Descriptions

| Variables | Descriptions |
|---|---|
| $S_h$ | Susceptible human population to Typhoid fever |
| $E_h$ | Exposed human population to Typhoid fever |
| $V_h$ | Vaccinated human population against Typhoid fever |
| $I_h$ | Infected human population with Typhoid fever |
| $T_h$ | Human population on Typhoid fever treatment |
| $R_h$ | Recovered human population from Typhoid fever |
| $B$ | Bacteria population |
| **Parameters** | **Descriptions** |
| $\Lambda_h$ | Recruitment rate of human population |
| $\Lambda_B$ | Recruitment rate of the bacteria population |
| $\phi_1$ | Vaccination rate of human population |
| $\beta_1$ | **The frequency of contact between healthy individuals and people infected with typhoid.** |
| $\beta_2$ | **The frequency of contact between healthy individuals and people on typhoid fever treatment.** |
| $\phi_2$ | Waning rate of vaccine in the human population |
| $\mu_h$ | Natural death rate of human population |
| $\mu_V$ | Natural death rate of Bacteria population |
| $\theta$ | Progression rate from Exposed human population to infected human population |
| $\eta$ | Treatment rate of infected human population |
| $\alpha$ | Recovery due to treatment rate of human population |
| $\sigma$ | Rate at which recovered humans become susceptible again |
| $\delta_1$ | Disease induced death rate of infected humans with typhoid fever |
| $\delta_2$ | Disease induced death rate of humans on typhoid fever treatment |



### 4.0 Integer order Model Equation

$$\frac{dS_h}{dt} = \Lambda_h + \phi_2 V_h + \sigma R_h - \psi_h S_h - (\phi_1 + \mu_h) S_h,$$

$$\frac{dE_h}{dt} = \psi_h S_h - (\theta + \mu_h) E_h \qquad (5)$$

$$\frac{dV_h}{dt} = \phi_1 S_h - (\phi_2 + \mu_h) V_h$$

$$\frac{dI_h}{dt} = \theta E_h - (\eta + \delta_1 + \mu_h) I_h,$$

$$\frac{dT_h}{dt} = \eta I_h - (\alpha + \delta_2 + \mu_h) T_h,$$

$$\frac{dR_h}{dt} = \alpha T_h - (\sigma + \mu_h) R_h,$$

$$\frac{dB}{dt} = \Lambda_B - \mu_B B.$$

### 4.1 Fractional-order Mathematical Model

To developa more adaptable model of typhoid fever, we have reframed the original integer-order model (Eq. 5) using a Caputo fractional derivative. This key change allows the model to capture a broader spectrum of potential outbreak scenarios, offering a significant improvement in realism over the classical approach.

The fractional Typhoid fever method is therefore presented as follows;

$$\begin{aligned}
{}^{c}D_t^{\gamma} S &= \Lambda_h + \phi_2 V_h + \sigma R_h - \psi_h S_h - (\phi_1 + \mu_h) S_h, \\
{}^{c}D_t^{\gamma} E_h &= \psi_h S_h - (\theta + \mu_h) E_h \\
{}^{c}D_t^{\gamma} V_h &= \phi_1 S_h - (\phi_2 + \mu_h) V_h \\
{}^{c}D_t^{\gamma} I_h &= \theta E_h - (\eta + \delta_1 + \mu_h) I_h, \\
{}^{c}D_t^{\gamma} T_h &= \eta I_h - (\alpha + \delta_2 + \mu_h) T_h, \\
{}^{c}D_t^{\gamma} R_h &= \alpha T_h - (\sigma + \mu_h) R_h, \\
{}^{c}D_t^{\gamma} B &= \Lambda_B - \mu_B B.
\end{aligned} \tag{6}$$

Where $L_1 = (\phi_1 + \mu_h)$ , $\mathrm{L}_2 = (\theta + \mu_h)$ , $\mathrm{L}_3 = (\phi_2 + \mu_h)$ , $\mathrm{L}_4 = (\eta + \delta_1 + \mu_h)$ , $\mathrm{L}_5 = (\alpha + \delta_2 + \mu_h)$ , $\mathrm{L}_6 = (\sigma + \mu_h)$. (7)

Subject to the positive initial conditions

$$S_h(0) = S_{h0},\ E_h(0) = E_{h0}, V_h(0) = V_{h0}, \mathrm{I}_h(0) = I_{h0}, T_h(0) = T_{h0}, \mathrm{R}_h(0) = R_{h0}, B(0) = B_0. \tag{8}$$

### 4.2 Positivity of Model Equation

We considered the non-negativity of the initial values

$$\lim Sup.N_h(t) \le \frac{\Lambda_h}{\mu_h},$$

Secondly, If $\lim Sup.N_{h0}(t) \le \frac{\Lambda_h}{\mu_h}$, then our model feasible domain is given by:

$$\Omega = \left\{ \left(S_h, \mathrm{E}_h, \mathrm{V}_h, \mathrm{I}_h, \mathrm{T}_h, \mathrm{R}_h, B\right) \subset R_+^7 : S_h + \mathrm{E}_h + \mathrm{V}_h + \mathrm{I}_h + \mathrm{T}_h + \mathrm{R}_h + B \le \frac{\Lambda_h}{\mu_h}, \right\}, \text{ so that}$$

$$\Omega = \Omega_T \subset R_+^7,$$

hence $\Omega$ is positively invariant.

If $\left(S_{h0}, \mathrm{E}_{h0}, \mathrm{V}_{h0}, \mathrm{I}_{h0}, \mathrm{T}_{h0}, \mathrm{R}_{h0}, B_0\right)$ are non-negative, then the solution of model (6) will be non-negative for $t > 0$. From Eq. (6), picking the first equation, we have that:

$$\begin{aligned}
&{}^cD_t^\gamma S = \Lambda_h + \phi_2 V_h + \sigma R_h - \psi_h S_h - \left(\phi_1 + \mu_h\right) S_h, \\
&{}^cD_t^\gamma S + \psi_h S_h + \left(\phi_1 + \mu_h\right) S_h = \Lambda_h + \phi_2 V_h + \sigma R_h, \\
&\Lambda_h + \phi_2 V_h + \sigma R_h \ge 0, \text{ Then,} \\
&{}^cD_t^\gamma S + \psi_h S_h + \left(\phi_1 + \mu_h\right) S_h \ge 0
\end{aligned}$$

Applying the Laplace transform, we have:

$$\begin{aligned}
&L\left[{}^cD_t^\gamma S\right] + L\left[\psi_h S_h + \left(\phi_1 + \mu_h\right) S_h\right] \ge 0 \\
&S_h^\gamma S_h(s) - S_h^{\gamma-1} S_h(0) + \left[\psi_h + \left(\phi_1 + \mu_h\right)\right] S_h(s) \ge 0, \\
&S_h(s) \ge \frac{S_h^{\gamma-1}}{S_h^\gamma + \left[\psi_h + \left(\phi_1 + \mu_h\right)\right]} S_h(0).
\end{aligned}$$

By taking the inverse Laplace transform, we obtained ;

$$S_h(t) \ge E_{r,1}\left(-\left[\psi_h + \left(\phi_1 + \mu_h\right)\right] t^\gamma\right) \mathrm{S}_{h0}.$$

Now since the term on the right-hand side of Eq. (9) is positive, we conclude that $S \ge 0$ for $t \ge 0$. In similar way, we also have that $\left(S_h \ge 0, \mathrm{E}_h \ge 0, \mathrm{V}_h \ge 0, \mathrm{I}_h \ge 0, \mathrm{T}_h \ge 0, \mathrm{R}_h \ge 0, B \ge 0\right)$, that is positives; therefore, the solution will remain in $R_+^7$ for all $t \ge 0$ with positive initial conditions.

### 4.3 Boundedness of fractional Model Equation

The total population of individuals from our model is given by;

$$N_h(t) = S_h(t) + \mathrm{E}_h(t) + \mathrm{V}_h(t) + \mathrm{I}_h(t) + \mathrm{T}_h(t) + \mathrm{R}_h(t).$$

So from our fractional model (6), we now obtain:

$$
\begin{aligned}
&{}^{c}D_t^{\gamma}N_h(t) = {}^{c}D_t^{\gamma}S_h(t) + {}^{c}D_t^{\gamma}\mathrm{E}_h(t) + {}^{c}D_t^{\gamma}\mathrm{V}_h(t) + {}^{c}D_t^{\gamma}\mathrm{I}_h(t) + {}^{c}D_t^{\gamma}\mathrm{T}_h(t) + {}^{c}D_t^{\gamma}\mathrm{R}_h(t).\\
&{}^{c}D_t^{\gamma}N_h(t) = \Lambda_h - \mu_h N_h(t)
\end{aligned}
\tag{10}
$$

We the take the Laplace transformation of (10) to get:

$$
\begin{aligned}
&L\left[{}^{c}D_t^{\gamma}N_h(t)\right] \le L\left[\Lambda_h - \mu_h N_h(t)\right]\\
&S_h^{\gamma}N_h(\mathrm{s}) - S_h^{\gamma-1}N_h(0) + \mu_h N_h(s) \le \frac{\Lambda_h}{\mu_h},\\
&N_h(\mathrm{s}) \le \frac{S_h^{\gamma-1}}{\left(S_h^{\gamma}+\mu\right)}N_h(0) + \frac{\Lambda_h}{S_h\left(S_h^{\gamma}+\mu_h\right)}
\end{aligned}
\tag{11}
$$

We take the inverse Laplace transform of Eq. (11), we obtained:

$$
N_h(\mathrm{t}) \le E_{r,1}\left(-\mu_h t^{\gamma}\right)N_h(0) + \psi_h E_{r,\mathrm{r}+1}\left(-\mu_h t^{\gamma}\right) \tag{12}
$$

At $t \to \infty$, the limit of Eq. (12) becomes

$$
\lim_{t\to\infty} Sup N_h(t) = \frac{\Lambda_h}{\mu_h}.
$$

This means that, if $N_{h0} \le \frac{\Lambda_h}{\mu_h}.$ then $N_h \le \frac{\Lambda_h}{\mu_h}.$ which implies that, $N_h(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_h$ , is well posed and equally feasible epidemiologically.

### 4.4 Existence and Uniqueness of our Model Equation

Let the real non-negative be P, we consider $Q = [0, \mathrm{P}].$

The set of all continuous function that is defined on M is represented by $N_e^0(Q)$ with norm as;

$$
\|X\| = Sup\left\{\left|X(t)\right|, \mathrm{t} \in \mathrm{Q}\right\}.
$$

Considering model (6) with initial conditions presented in (8) which can be denoted as an initial value problem (IVP) in (13).

$$
\begin{aligned}
&{}^{c}D_t^{\gamma}(t) = Z\left(t, X(t)\right), 0 < t < P < \infty,\\
&X(0) = X_0.
\end{aligned}
$$

Where $Y(t)=\left(S_h(t),\mathrm{E}_h(t),\mathrm{V}_h(t),\mathrm{I}_h(t),\mathrm{T}_h(t),\mathrm{R}_h(t),B(t)\right)$ represents the classes and Z be a continuous function defined as follows:

$$Z\left(t,X\left(t\right)\right)=\begin{pmatrix} Z_1\left(t,S_h\left(t\right)\right)\\ Z_2\left(t,\mathrm{E}_h\left(t\right)\right)\\ Z_3\left(t,\mathrm{V}_h\left(t\right)\right)\\ Z_4\left(t,\mathrm{I}_h\left(t\right)\right)\\ Z_5\left(t,T_h\left(t\right)\right)\\ Z_6\left(t,\mathrm{R}_h\left(t\right)\right)\\ Z_7\left(t,B\left(t\right)\right)\end{pmatrix}=\begin{pmatrix} \Lambda_h+\phi_2V_h+\sigma R_h-\left(\dfrac{\beta_1I_h+\beta_2T_h+\beta_3B}{N_h}\right)S_h-\left(\phi_1+\mu_h\right)S_h\\ \left(\dfrac{\beta_1I_h+\beta_2T_h+\beta_3B}{N_h}\right)S_h-\left(\theta+\mu_h\right)E_h\\ \phi_1S_h-\left(\phi_2+\mu_h\right)V_h\\ \theta E_h-\left(\eta+\delta_1+\mu_h\right)I_h\\ \eta I_h-\left(\alpha+\delta_2+\mu_h\right)T_h\\ \alpha T_h-\left(\sigma+\mu_h\right)R_h\\ \Lambda_B-\mu_BB\end{pmatrix} \quad ...(14)$$

Using proposition (2.1), we have that,

$$\begin{aligned} S_h\left(t\right)&=S_{h0}+I_t^{\gamma}\left[\Lambda_h+\phi_2V_h+\sigma R_h-\left(\frac{\beta_1I_h+\beta_2T_h+\beta_3B}{N_h}\right)S_h-\left(\phi_1+\mu_h\right)S_h\right],\\ E_h\left(t\right)&=E_{h0}+I_t^{\gamma}\left[\left(\frac{\beta_1I_h+\beta_2T_h+\beta_3B}{N_h}\right)S_h-\left(\theta+\mu_h\right)E_h\right],\\ V_h\left(t\right)&=V_{h0}+I_t^{\gamma}\left[\phi_1S_h-\left(\phi_2+\mu_h\right)V_h\right],\\ I_h\left(t\right)&=I_{h0}+I_t^{\gamma}\left[\theta E_h-\left(\eta+\delta_1+\mu_h\right)I_h\right],\\ T_h\left(t\right)&=T_{h0}+I_t^{\gamma}\left[\eta I_h-\left(\alpha+\delta_2+\mu_h\right)T_h\right],\\ R_h\left(t\right)&=R_{h0}+I_t^{\gamma}\left[\alpha T_h-\left(\sigma+\mu_h\right)R_h\right],\\ B\left(t\right)&=B_0+I_t^{\gamma}\left[\Lambda_B-\mu_BB\right]. \end{aligned} \quad (15)$$

We obtain the Picard iteration of (15) as follows:

$$\begin{aligned} S_{hn}\left(t\right)&=S_{h0}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-\lambda\right)^{\gamma-1}Z_1\left(\lambda,S_{h(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ E_{hn}\left(t\right)&=E_{h0}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-\lambda\right)^{\gamma-1}Z_2\left(\lambda,\mathrm{E}_{h(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ V_{hn}\left(t\right)&=V_{h0}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-\lambda\right)^{\gamma-1}Z_3\left(\lambda,\mathrm{V}_{h(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ I_{hn}\left(t\right)&=I_{h0}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-\lambda\right)^{\gamma-1}Z_4\left(\lambda,\mathrm{I}_{h(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda,\\ T_{hn}\left(t\right)&=T_{h0}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-\lambda\right)^{\gamma-1}Z_5\left(\lambda,\mathrm{T}_{h(n-1)}\left(\lambda\right)\right)\mathrm{d}\lambda, \end{aligned} \quad (16)$$

$$R_{hn}(t) = R_{h0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda)^{\gamma-1} Z_6\left(\lambda, \mathrm{R}_{h(n-1)}(\lambda)\right)\mathrm{d}\lambda,$$

$$B_n(t) = B_{n0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda)^{\gamma-1} Z_7\left(\lambda, B_{(n-1)}(\lambda)\right)\mathrm{d}\lambda.$$

We transformed the initial value problem of Eq. (13) to obtain:

$$X(t) = X(0) + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda)^{\gamma-1} Z\left(\lambda, X(\lambda)\right)\mathrm{d}\lambda. \tag{17}$$

Lemma 1, The Lipchitz condition described from Eq. (14) is satisfied by vector $Z\left(\mathrm{t}, X(t)\right)$ on a set $[0,P]\times R_+^7$ with the Lipchitz constant given as;

$$\beta = \max\left(\left(\beta_1^* + \beta_2^* + \beta_3^* + \phi_1 + \mu\right), (\theta + \mu_h), (\phi_2 + \mu_h), , (\eta + \delta_1 + \mu_h), (\alpha + \delta_2 + \mu_h), (\sigma + \mu_h)(\sigma + \mu_h), \mu\right.$$

Proof.

$$\left\|Z_1(t, S_h) - Z_1(t, S_{h1})\right\|$$

$$= \left\| \begin{array}{l} \Lambda_h + \phi_2 V_h + \sigma R_h - \left(\dfrac{\beta_1 I_h + \beta_2 T_h + \beta_3 B}{N_h}\right) S_h - (\phi_1 + \mu_h) S_h \\ -\Lambda_h - \phi_2 V_h - \sigma R_h - \left(\dfrac{\beta_1 I_h + \beta_2 T_h + \beta_3 B}{N_h}\right) S_h - (\phi_1 + \mu_h) S_{h1} \end{array} \right\|,$$

$$= \left\| -\left(\frac{\beta_1 I_h + \beta_2 T_h + \beta_3 B}{N_h}\right) - (\phi_1 + \mu_h)(S_h - S_{h1}) + \mu_h (S_h - S_{h1}) \right\| \le \left(\beta_1^* + \beta_2^* + \beta_3^*\right)\|S_h - S_{h1}\| + \mu_h \|S_h - S_{h1}\|,$$

$$\therefore \left\|Z_1(t, S_h) - Z_1(t, S_{h1})\right\| \le \left(\beta_1^* + \beta_2^* + \beta_3^* + \phi_1 + \mu_h\right)\|S_h - S_{h1}\|.$$

Similarly we obtained the following;

$$\begin{aligned} &\left\|Z_2(t, \mathrm{E}_h) - Z_2(t, \mathrm{E}_{h1})\right\| \le (\theta + \mu_h)\|\mathrm{E}_h - \mathrm{E}_{h1}\|, \\ &\left\|Z_3(t, \mathrm{V}_h) - Z_3(t, \mathrm{V}_{h1})\right\| \le (\phi_2 + \mu_h)\|V_h - V_{h1}\|, \\ &\left\|Z_4(t, \mathrm{I}_h) - Z_4(t, \mathrm{I}_{h1})\right\| \le (\eta + \delta_1 + \mu_h)\|I_h - I_{h1}\|, \\ &\left\|Z_5(t, \mathrm{T}_h) - Z_5(t, \mathrm{T}_{h1})\right\| \le (\alpha + \delta_2 + \mu_h)\|T_h - T_{h1}\|, \\ &\left\|Z_6(t, \mathrm{R}_h) - Z_5(t, \mathrm{R}_{h1})\right\| \le (\sigma + \mu_h)\|R_h - R_{h1}\|, \\ &\left\|Z_7(t, B) - Z_7(t, B_1)\right\| \le (\mu_B)\|B - B_1\|. \end{aligned} \tag{18}$$

Where we obtained:

$$\left\|Z(t, \mathrm{X}_1(t)) - Z(t, \mathrm{X}_2(t))\right\| \le \beta\|\mathrm{X}_1 - \mathrm{X}_2\|,$$

$$\beta = \max\left(\left(\beta_1^* + \beta_2^* + \beta_3^* + \phi_1 + \mu\right), \left(\theta + \mu_h\right), \left(\phi_2 + \mu_h\right), , \left(\eta + \delta_1 + \mu_h\right), \left(\alpha + \delta_2 + \mu_h\right), \left(\sigma + \mu_h\right)\left(\sigma + \mu_h\right), \mu_h\right) \quad (19)$$

Lemma 2. The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution.

$$\mathrm{X}(t) \in A_c^0(f).$$

We use the Picard-Lindelof and fixed point theory to consider the solution of

$$\mathrm{X}(t) = S_h\left(\mathrm{X}(t)\right),$$

We defined the Picard operator expressed as S:

$$S : A_c^0\left(f, R_+^7\right) \to A_c^0\left(f, R_+^7\right).$$

Therefore

$$S_h\left(X(t)\right) = X(0) + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda)^{\gamma-1} Z\left(\lambda, \mathrm{X}(\lambda)\right) \mathrm{d}\lambda.$$

which becomes:

$$\left\| S_h\left(\mathrm{X}_1(t)\right) - S_h\left(\mathrm{X}_2(t)\right) \right\|$$

$$= \left\| \frac{1}{\Gamma(\gamma)} \left[ \int_0^t (t-\lambda)^{\gamma-1} Z\left(\lambda, \mathrm{X}_1(\lambda)\right) - Z\left(\lambda, \mathrm{X}_2(\lambda)\right) \mathrm{d}\lambda \right] \right\|$$

$$\leq \frac{1}{\Gamma(\gamma)} \int_0^t (t-\lambda)^{\gamma-1} \left\| Z\left(\lambda, \mathrm{X}_1(\lambda)\right) - Z\left(\lambda, \mathrm{X}_2(\lambda)\right) \mathrm{d}\lambda \right\|.$$

$$\leq \frac{\beta}{\Gamma(\gamma)} \int_0^t (t-\lambda)^{\gamma-1} \left\| \mathrm{X}_1 - \mathrm{X}_2 \right\| d\lambda.$$

$$\left\| S_h\left(\mathrm{X}_1(t)\right) - S_h\left(\mathrm{X}_2(t)\right) \right\| \leq \frac{\beta}{\Gamma(\gamma+1) S_h}.$$

When $\frac{\beta}{\Gamma(\gamma+1)} S_h \leq 1$, then the Picard operator gives a contradiction, so Eq.(6) , (7) solution is unique.

### 4.5 Disease Free Equilibrium Point

It is a state where there is no disease, this implies

$S_h \neq 0, \mathrm{E}_h = 0, \mathrm{V}_h \neq 0, \mathrm{I}_h = 0, \mathrm{T}_h = 0, \mathrm{R}_h = 0, B \neq 0.$

The state is represented by $E_0 = \left\{ S_h^0, E_h^0, \mathrm{V}_h^0, I_h^0, \mathrm{T}_h^0, R_h^0, B^0 \right\}$.

$$\left(S_h^0, \mathrm{E}_h^0, \mathrm{V}_h^0, \mathrm{I}_h^0, \mathrm{T}_h^0, \mathrm{R}_h^0, B\right) = \left(\frac{\Lambda_h\left(\phi_2+\mu_h\right)}{\mu_h\left(\phi_2+\phi_1+\mu_h\right)}, 0, \frac{\phi_1\Lambda_h}{\mu_h\left(\phi_2+\phi_1+\mu_h\right)}, 0, 0, 0, \frac{\Lambda_B}{\mu_B}\right).$$

### 4.6 Basic Reproduction Number

In epidemiology, the basic reproduction number was identified as a major measure of potential in an outbreak. In the case of Typhoid fever, it is the mean number of secondary cases produced by one infected individual within a fully susceptible community. In order to calculate this value to use in our model, we used the already proven next-generation operator technique on the system of equations.

Hence, it follows that:

$R_0 = \rho\left(FV^{-1}\right)$ where $\rho$ is the dominant Eigen value of $FV^{-1}$

To find the basic reproduction number $\mathrm{R}_0$ for the Typhoid fever model, we use the formula: $\mathrm{R}_0 = \rho(FV^{-1})$ where $\rho$ denotes the spectral radius, $F$ is the matrix of new infections, and $V$ is the matrix of transitions.

Given:

$$F = \begin{pmatrix} 0 & \frac{\beta_1\left(\phi_2+\mu_h\right)}{\phi_2+\phi_1+\mu_h} & \frac{\beta_2\left(\phi_2+\mu_h\right)}{\phi_2+\phi_1+\mu_h} & \frac{\beta_3\left(\phi_2+\mu_h\right)}{\phi_2+\phi_1+\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \textbf{ and } V = \begin{pmatrix} L_2 & 0 & 0 & 0 \\ -\theta & L_4 & 0 & 0 \\ 0 & -\eta & L_5 & 0 \\ 0 & 0 & 0 & L_7 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{L_2} & 0 & 0 & 0 \\ \frac{\theta}{L_2L_4} & \frac{1}{L_4} & 0 & 0 \\ \frac{\eta\theta}{L_2L_4L_5} & \frac{\eta}{L_4L_5} & \frac{1}{L_5} & 0 \\ 0 & 0 & 0 & \frac{1}{L_7} \end{pmatrix}$$

$$FV^{-1}=\begin{pmatrix} \frac{\beta_1(\phi_2+\mu_h)\theta}{(\phi_2+\phi_1+\mu_h)L_2L_4}+\frac{\beta_2(\phi_2+\mu_h)\eta\theta}{(\phi_2+\phi_1+\mu_h)L_4L_2L_5} & \frac{\beta_1(\phi_2+\mu_h)}{(\phi_2+\phi_1+\mu_h)L_4}+\frac{\beta_2(\phi_2+\mu_h)\eta}{(\phi_2+\phi_1+\mu_h)L_4L_5} & \frac{\beta_2(\phi_2+\mu_h)}{(\phi_2+\phi_1+\mu_h)L_5} & \frac{\beta_3(\phi_2+\mu_h)}{(\phi_2+\phi_1+\mu_h)L_7} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$Eigen\ values=\begin{pmatrix} 0 \\ 0 \\ 0 \\ \frac{\theta(\eta\beta_2\mu_h+\eta\beta_2\phi_2+L_5\beta_1\mu_h+L_5\beta_1\phi_2)}{L_2L_4L_5(\phi_2+\phi_1+\mu_h)} \end{pmatrix}$$

$$R_0=\frac{\theta(\eta\beta_2\mu_h+\eta\beta_2\phi_2 l+L_5\beta_1\mu_h+L_5\beta_1\phi_2)}{L_2L_4L_5(\phi_2+\phi_1+\mu_h)}.$$

This is the largest Eigen value.

### 4.7 Endemic Equilibrium Point of Typhoid fever

Endemic equilibrium point refers to the point of a limited disease within the population in which the disease stays constant.

At endemic equilibrium point $S_h\neq 0, \mathrm{E}_h\neq 0, \mathrm{V}_h\neq 0, \mathrm{I}_h\neq 0, \mathrm{T}_h\neq 0, \mathrm{R}_h\neq 0, B\neq 0.$

$$S_h^*=-\frac{\Lambda_h L_2L_3L_4L_5L_6}{L_5L_4L_6\left(\left(-\psi_h-L_1\right)L_3+\phi_1\phi_2\right)L_2+\alpha\eta\sigma\theta L_3\psi_h},$$

$$E_h^*=-\frac{\Lambda_h L_3L_4L_5L_6\psi_h}{L_5L_4L_6\left(\left(-\psi_h-L_1\right)L_3+\phi_1\phi_2\right)L_2+\alpha\eta\sigma\theta L_3\psi_h},$$

$$V_h^*=-\frac{\Lambda_h L_2L_4L_5L_6\phi_1}{L_5L_4L_6\left(\left(-\psi_h-L_1\right)L_3+\phi_1\phi_2\right)L_2+\alpha\eta\sigma\theta L_3\psi_h},$$

$$I_h^*=-\frac{\psi_h\Lambda_h L_5L_6L_3\theta}{L_5L_4L_6\left(\left(-\psi_h-L_1\right)L_3+\phi_1\phi_2\right)L_2+\alpha\eta\sigma\theta L_3\psi_h},$$

$$T_h^*=-\frac{\psi_h\Lambda_h L_6L_3\theta\eta}{L_5L_4L_6\left(\left(-\psi_h-L_1\right)L_3+\phi_1\phi_2\right)L_2+\alpha\eta\sigma\theta L_3\psi_h},$$

$$R_h^*=-\frac{\alpha\psi_h\Lambda_h L_3\theta\eta}{\alpha\eta\sigma\theta L_3\psi_h-L_1L_2L_3L_4L_5L_6-L_2L_3L_4L_5L_6\psi_h+L_2L_4L_5L_6\phi_1\phi_2},$$

$$B^*=\frac{\Lambda_B}{\mu_B}.$$

Substituting into the force of infection $\psi_h = \frac{\left(\beta_1 I_h + \beta_2 T_h + \beta_3 B\right)}{N_h}$ we have:

$Q_1 \psi_h + Q_2 = 0$ .

$$Q_1 = \left(\alpha\eta\theta L_3 L_7 \Lambda_h + \eta\theta L_3 L_6 L_7 \Lambda_h + \theta L_3 L_5 L_6 L_7 \Lambda_h + L_3 L_4 L_5 L_6 L_7 \Lambda_h\right),$$

$$Q_2 = L_2 L_3 L_4 L_5 L_6 L_7 \Lambda_h \left(1 - \frac{\theta\left(\eta\beta_2\mu_h + \eta\beta_2\phi_2 l + L_5\beta_1\mu_h + L_5\beta_1\phi_2\right)}{L_2 L_4 L_5\left(\phi_2 + \phi_1 + \mu_h\right)}\right),$$

$$Q_2 = L_2 L_3 L_4 L_5 L_6 L_7 \Lambda_h \left(1 - R_0\right).$$

This implies that the above model has a stable endemic equilibrium point if $R_0 > 0$.

### 4.8 Global Asymptomatic Stability of the Disease-Free Equilibrium Point

The global stability of the equilibrium point was studied using the direct Lyapunov method. The global stability of the endemic equilibrium (a situation, which occurs when $R_0 > 1$ ) implies that the disease will not go away initially in the population regardless of the number of people, who were initially infected. We verified that this conclusion is true in our fractional model (6).

Where $\psi_h = \left(\frac{\beta_1 I_h + \beta_2 T_h + \beta_3 B}{N_h}\right)$

Where $P = \left(S_h^*, \mathrm{E}_h^*, V_h^*, I_h^*, \mathrm{T}_h^*, , \mathrm{R}_h^*, B^*\right) \in R_+^7$,

then $\psi_h = \left(\frac{\beta_1 I_h + \beta_2 T_h + \beta_3 B}{N_h}\right)$

We expressed our fractional model as:

$$\begin{aligned}
{}^{c}D_t^{\gamma} S &= \Lambda_h + \phi_2 V_h + \sigma R_h - \psi_h S_h - \left(\phi_1 + \mu_h\right) S_h, \\
{}^{c}D_t^{\gamma} E_h &= \psi_h S_h - \left(\theta + \mu_h\right) E_h \\
{}^{c}D_t^{\gamma} V_h &= \phi_1 S_h - \left(\phi_2 + \mu_h\right) V_h \\
{}^{c}D_t^{\gamma} I_h &= \theta E_h - \left(\eta + \delta_1 + \mu_h\right) I_h, \\
{}^{c}D_t^{\gamma} T_h &= \eta I_h - \left(\alpha + \delta_2 + \mu_h\right) T_h, \\
{}^{c}D_t^{\gamma} R_h &= \alpha T_h - \left(\sigma + \mu_h\right) R_h, \\
{}^{c}D_t^{\gamma} B &= \Lambda_B - \mu_B B.
\end{aligned} \tag{6}$$

We obtained the following results at equilibrium point Eq. (24):

$$\psi_h S_h + (\phi_1 + \mu_h) S_h = \Lambda_h + \phi_2 V_h + \sigma R_h, \qquad (\theta + \mu_h) E_h = \psi_h S_h,$$
$$(\phi_2 + \mu_h) V_h = \phi_1 S_h, \; (\eta + \delta_1 + \mu_h) I_h = \theta E_h, \; (\alpha + \delta_2 + \mu_h) T_h = \eta I_h,$$
$$(\sigma + \mu_h) R_h = \alpha T_h, \; \mu_B B = \Lambda_B.$$

Theorem 1. Demonstrate that the system Model (1) is globally asymptotically stable at disease free equilibrium, furthermore, at $R_0 < 1$.

Proof

We construct the lyapunor function to prove the results,

$$L^1 = \Lambda_h u_1 + \Lambda_B S_h E_h (u_2 - u_1) + (1 - \Lambda_B)(u_3 - u_1)$$
$$+\theta(u_3 - u_2) + \Lambda_B (u_4 - u_2) + \eta(u_4 - u_3)$$
$$\Lambda_r (u_5 - u_4) + \theta(u_6 - u_4) + \sigma(u_7 - u_4).$$

where $u_{1,} u_{2,} u_{3,} u_{4,} u_{5,} u_6$ *and* $u_7$ *are positive constant*

We take the derivative of the Lyapunov, we have:

$R_0 < 1.$

Let the positive constants be:

$$u_{1,} = u_{2,} = u_{3,} = u_{4,} = u_{5,} = u_6 = u_7 = 1$$

*and* $N_h > \frac{\Lambda_h}{\mu_h}$ ,then we have

$$L^1 = \Lambda_h - U_h N_h$$

$$L^1 = (-U_h N_h + \Lambda_h) < 0,$$

The system (5) is, therefore, globally asymptotically stable with respect to the disease-free equilibrium and at $R_0 < 1$.

## 4.9 Numerical Results of the Fractional-Order Model

To simulate the behavior of our typhoid fever model, we used a numerical technique called the generalized fractional Adams-Bashforth-Moulton method, following the approach of Amos et al. (2024). We ran these simulations using the parameter values listed in Table 1, testing different fractional orders to see how they affected the outcome $(\gamma)$.

### 4.10. Implementation of the Fractional Adams-Bashforth-Moulton Method

In this paper we use a fractional Adams-Bashforth-Moulton algorithm, as in the study of Diethelm, Freed, and Baskonus et al. (2015), to estimate the solution of our fractional typhoid fever model (6). The presentation of this model is modified after Amos et al. (2024) and it is provided as follows:

$${}^{c}D_{t}^{\gamma}M\left(t\right)=N\left(t,\mathrm{m}\left(t\right)\right),\ 0<t<\psi,\ \ldots(27)$$

$$M^{(n)}\left(0\right)=M_{0}^{(n)},\ \ n=1,0,\ldots,\mathrm{m},\mathrm{m}=\left[\gamma\right].$$

Where $M=\left(S_h^*,\mathrm{E}_h^*,\mathrm{V}_h^*,\mathrm{I}_h^*,\mathrm{T}_h^*,\mathrm{R}_h^*,B^*\right)\in R_+^7$ and $Q\left(t,m\left(t\right)\right)$ is a real valued function that is continous.

Eq. (27) can be consequently be denoted using the notion of fractional integral as follows:

$$M\left(t\right)=\sum_{n=0}^{m-1}M_0^{(n)}\frac{t^n}{n!}+\frac{1}{\Gamma\left(\gamma\right)}\int_0^t\left(t-y\right)^{\gamma-1}R\left(y,m\left(y\right)\right)dy\ \ldots(28)$$

We apply the method described by Amos et al.(2024), let consider the step size $g=\frac{\psi}{N}$, $N\in\mathrm{N}$ with a grid that is uniform on $\left[0,\psi\right]$. Where $t_c=cr$, $c=0,1,1,\ldots N$. This implies that, the fractional order model of Typhoid fever model presented in (6) can approximately be expressed as:

$$S_{h(k+1)}\left(t\right)=S_{h0}+\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\left\{\Lambda_h+\phi_2V_h^n+\sigma R_h^n-\left(\frac{\beta_1I_h^n+\beta_2T_h^n+\beta_3B^n}{N_h^n}\right)S_h^n-\left(\phi_1+\mu_h\right)S_h^n\right\}+$$
$$\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\sum_{y=0}^{k}dy,k+1\left\{\Lambda_h+\phi_2V_{hy}+\sigma R_{hy}-\left(\frac{\beta_1I_{hy}+\beta_2T_{hy}+\beta_3B_y}{N_h}\right)S_{hy}-\left(\phi_1+\mu_h\right)S_{hy}\right\},$$

$$E_{h(k+1)}\left(t\right)=E_{h0}+\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\left\{\left(\frac{\beta_1I_h^n+\beta_2T_h^n+\beta_3B^n}{N_h^n}\right)S_h^n-\left(\theta+\mu_h\right)E_h^n\right\}+$$
$$\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\sum_{y=0}^{k}dy,k+1\left\{\left(\frac{\beta_1I_{hy}+\beta_2T_{hy}+\beta_3B_y}{N_{hy}}\right)S_h-\left(\theta+\mu_h\right)E_{hy}\right\},\qquad(29)$$

$$V_{h(k+1)}\left(t\right)=V_0+\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\left\{\phi_1S_h^n-\left(\phi_2+\mu_h\right)V_h^n\right\}+$$
$$\frac{g^{\gamma}}{\Gamma\left(\gamma+2\right)}\sum_{y=0}^{k}dy,k+1\left\{\phi_1S_{hy}-\left(\phi_2+\mu_h\right)V_{hy}\right\},$$

$$I_{h(k+1)}(t) = I_{h0} + \frac{g^{\gamma}}{\Gamma(\gamma+2)}\left\{\theta E_h^n - (\eta+\delta_1+\mu_h) I_h^n\right\} +$$

$$\frac{g^{\gamma}}{\Gamma(\gamma+2)}\sum_{y=0}^{k} dy, k+1\left\{\theta E_{hy} - (\eta+\delta_1+\mu_h) I_{hy}\right\},$$

$$T_{h(k+1)}(t) = T_{h0} + \frac{g^{\gamma}}{\Gamma(\gamma+2)}\left\{\eta I_h^n - (\alpha+\delta_2+\mu_h) T_h^n\right\} +$$

$$\frac{g^{\gamma}}{\Gamma(\gamma+2)}\sum_{y=0}^{k} dy, k+1\left\{\eta I_{hy} - (\alpha+\delta_2+\mu_h) T_{hy}\right\},$$

$$R_{h(k+1)}(t) = R_{h0} + \frac{g^{\gamma}}{\Gamma(\gamma+2)}\left\{\alpha T_h^n - (\sigma+\mu_h) R_h^n\right\} +$$

$$\frac{g^{\gamma}}{\Gamma(\gamma+2)}\sum_{y=0}^{k} dy, k+1\left\{\alpha T_{hy} - (\sigma+\mu_h) R_{hy}\right\},$$

$$B_{(k+1)}(t) = B_0 + \frac{g^{\gamma}}{\Gamma(\gamma+2)}\left\{\Lambda_B - \mu_B B^n\right\} +$$

$$\frac{g^{\gamma}}{\Gamma(\gamma+2)}\sum_{y=0}^{k} dy, k+1\left\{\Lambda_B - \mu_B B_y\right\}.$$

Where

$$S_{h(k+1)}(t) = S_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_h + \phi_2 V_{hy} + \sigma R_{hy} - \left(\frac{\beta_1 I_{hy} + \beta_2 T_{hy} + \beta_3 B_y}{N_h}\right) S_{hy} - (\phi_1+\mu_h) S_{hy}\right\},$$

$$E_{h(k+1)}(t) = E_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\left(\frac{\beta_1 I_{hy} + \beta_2 T_{hy} + \beta_3 B_y}{N_{hy}}\right) S_h - (\theta+\mu_h) E_{hy}\right\},$$

$$V_{h(k+1)}(t) = V_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\phi_1 S_{hy} - (\phi_2+\mu_h) V_{hy}\right\},$$

$$I_{h(k+1)}(t) = I_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\theta E_{hy} - (\eta+\delta_1+\mu_h) I_{hy}\right\}, \tag{30}$$

$$T_{h(k+1)}(t) = T_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\eta I_{hy} - (\alpha+\delta_2+\mu_h) T_{hy}\right\},$$

$$R_{h(k+1)}(t) = R_{h0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\alpha T_{hy} - (\sigma+\mu_h) R_{hy}\right\},$$

$$B_{(k+1)}(t) = B_0 + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_B - \mu_B B_y\right\}.$$

We obtained the result below from (29) and (30).

$$dy,_{K+1} = K^{\gamma+1} - (k-\gamma)(k+\gamma)^{\gamma},\ y = 0$$

$$(k-y+2)^{\gamma+1} + (k-\gamma)^{\gamma+1} - 2(k-y+1)^{\gamma+1},\ 1 \le y \le k$$

$$1, y = k+1$$

and

$$f_{y,k+1} = \frac{g^{\gamma}}{\gamma}\left[(k-y+1)^{\gamma}(k-y)^{\gamma}\right],\ 0 \le y \le k.$$

## 5.0 Numerical Simulation

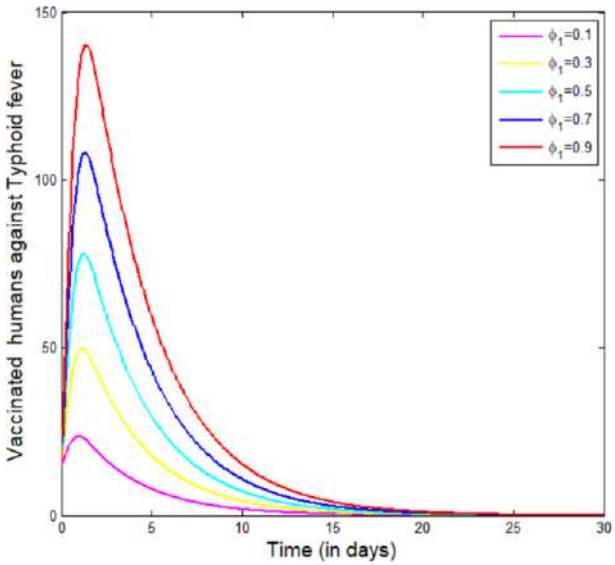


Fig.2a: Simulation of the effect $\phi_1$ on Vaccinated humans against Typhoid fever

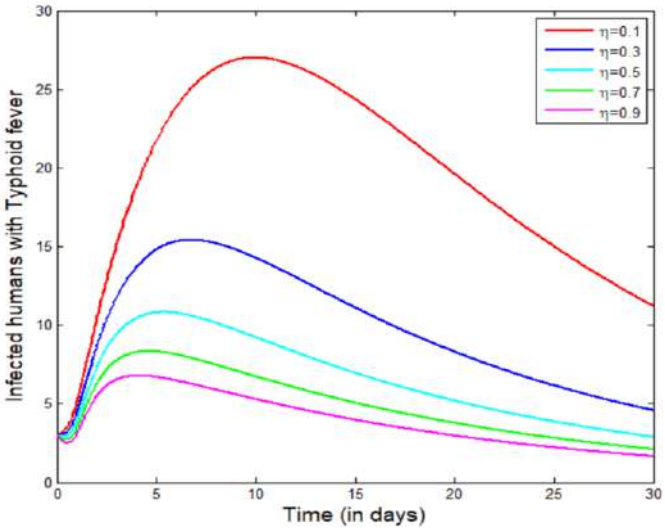


Fig.2b: Simulation of the effect of $\eta$ on infected humans with Typhoid fever

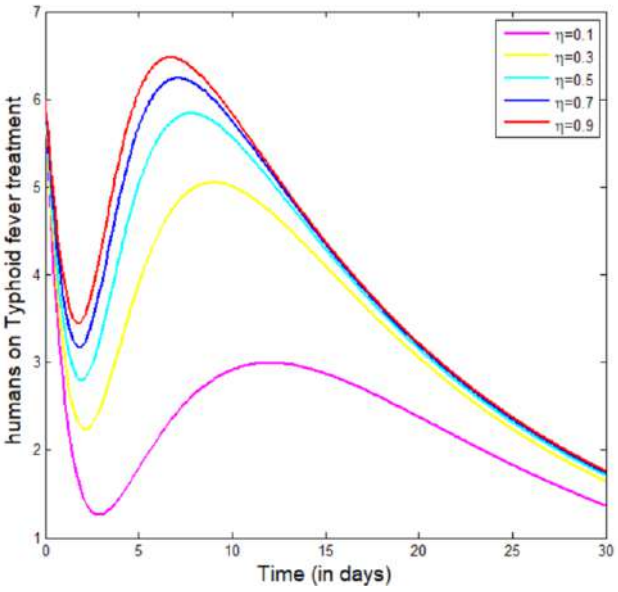


Fig.2c: Simulation of the effect of $\eta$ on humans on Typhoid fever treatment

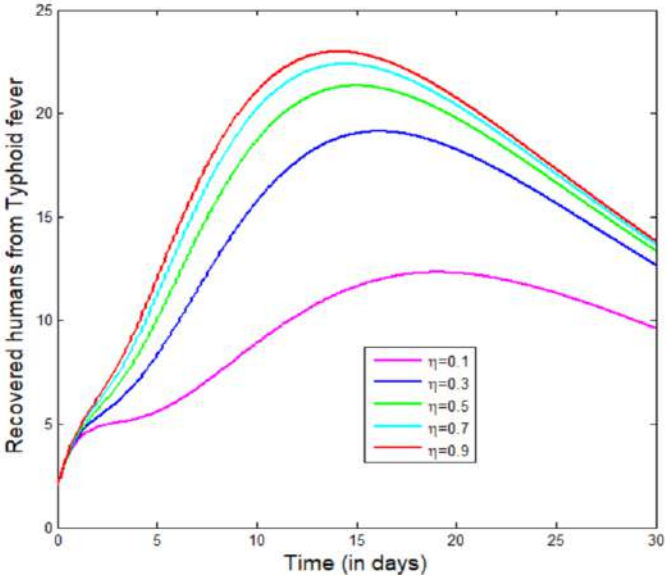


Fig.2d: Simulation of the effect of $\eta$ on Recovered humans from Typhoid fever

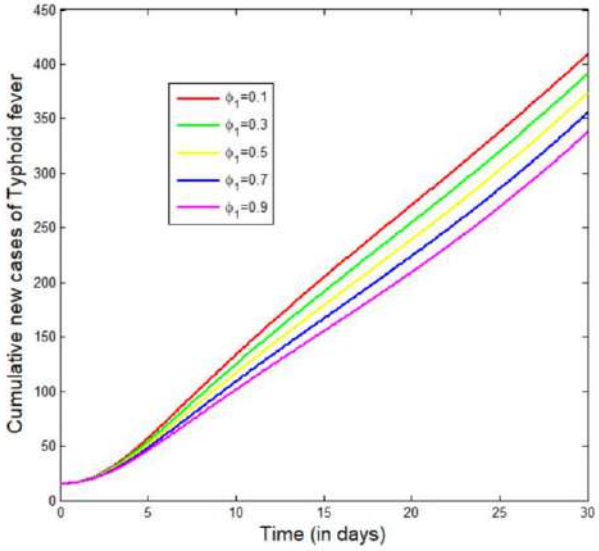


Fig.2e: Simulation of the effect of $\phi_1$ on cumulative new cases of Typhoid fever

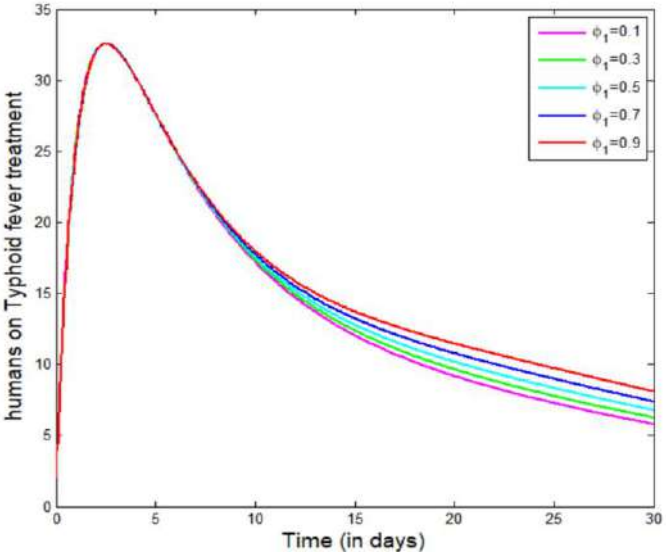


Fig.2f: Simulation of the effect $\phi_1$ on humans on Typhoid fever treatment

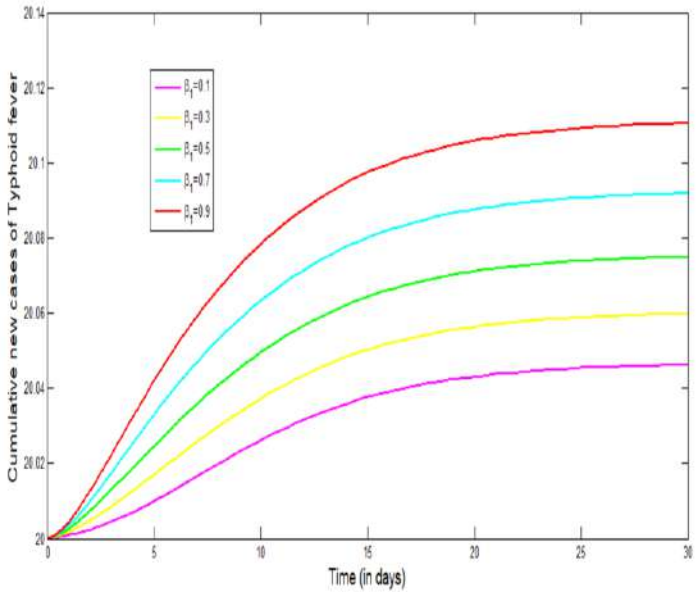


Fig.2h: Simulation of the effect of $\beta_1$ on cumulative new cases of Typhoid fever

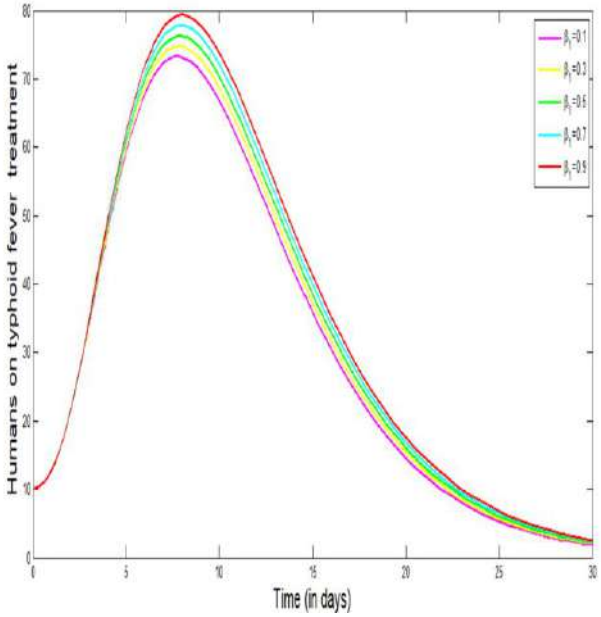


Fig.2h: Simulation of the effect of $\beta_1$ on humans on Typhoid fever treatment

(2a) illustrates the simulation of the outcome of vaccination rate $(\phi_1)$ on vaccinated human population against Typhoid fever. It is detected that, as the vaccination rate $(\phi_1)$ increases, the number of vaccinated human population against Typhoid fever increases. (2b) shows the simulation of the outcome of treatment rate $(\eta)$ on infected human population with Typhoid fever. It is detected that, as the treatment rate $(\eta)$ increases, the number of infected human population with Typhoid fever decreases.

(2c) represents the simulation of the outcome of treatment rate $(\eta)$ on human population on Typhoid fever treatment. It is practical that, as the treatment rate $(\eta)$ increases, the number of humans with Typhoid fever treatment increases.

(2d) represents the simulation of the effect of treatment rate $(\eta)$ on recovered human population from Typhoid fever. It is practical that, as the treatment rate $(\eta)$ increases, the number of recovered human population from Typhoid fever increases.

(2e) shows the simulation of the outcome of vaccination rate $(\phi_1)$ on cumulative new cases of Typhoid fever. It is revealed that, as the vaccination rate $(\phi_1)$ increases, the cumulative new cases of Typhoid fever decreases.

(2f) denotes the simulation of the outcome of vaccination rate $(\phi_1)$ on humans on Typhoid fever treatment. It is observed that, as the vaccination rate $(\phi_1)$ increases, the number of humans on Typhoid fever treatment increases.

(2g) shows the simulation of the effect of contact rate $(\beta_1)$ on cumulative new cases of typhoid fever. It is observed that, as the contact rate $(\beta_1)$ increases, the cumulative new cases of typhoid fever increases.

(2h) represents the simulation of the effect of contact rate $(\beta_1)$ on humans on treatment of typhoid fever. It is observed that, as the contact rate $(\beta_1)$ increases, the number of humans on treatment of typhoid fever increases.

## 5.1 Conclusions

In this study, we developed a detailed mathematical model using fractional calculus to understand how vaccination and treatment influence the spread of typhoid fever. We used a specific numerical technique, the fractional Adams–Bashforth–Moulton method, to simulate the model's behavior. This method was key because it allowed us to capture the "memory" and hereditary properties that are fundamental to how a real disease moves through a population. The outcomes from our simulations provide a clear and compelling results. We observed that increasing the vaccination rate directly shrinks the number of people who are susceptible to the disease. This reduction effectively lowers the overall infection burden and acts as a strong brake on further transmission. In a similar vein, increasing the treatment rate proves to be highly effective. It speeds up the recovery of those already infected, which shortens the period they are contagious and, as a result, leads to a noticeable decline in the number of active cases. The powerful part is how these two actions work together. The combination of vaccination and treatment creates a synergistic control mechanism. This dual strategy doesn't just prevent new infections from taking root; it also actively mitigates the spread from infections that already exist. In summary, our findings underscore that integrating thorough vaccination campaigns with prompt and effective treatment strategies offers a robust and reliable pathway toward reducing the prevalence of typhoid fever. Our fractional modeling approach further confirms its value by capturing these complex epidemiological patterns, ultimately offering valuable insights for designing more effective and sustainable public health interventions against this disease.

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## Credit authorship contribution statement

Hawah Oyiza Rabiu: Writing – original draft, Formal analysis. Jeremiah Amos: Writing – review & editing,Formal analysis. William Atokolo: Writing – original draft, Formal analysis, David Omale: Writing – original draft, Formal analysis, Bolarinwa Bolaji: Writing – original draft, Formal analysis.

## Declaration of competing interest

According to the authors, there are no financial conflicts or personal relationships that could have influenced the findings of the research of this paper.

## Data availability

Values of parameters used are adequately cited and referenced.

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