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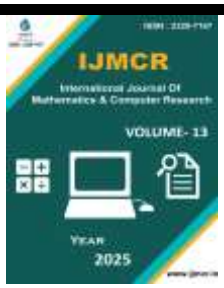

A Quantitative Analysis of the Joint Dynamics of the Interconnected Spread of Cholera and Typhoid Diseases

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ABSTRACT

In this paper, a mathematical model that captures the spread of Cholera and Typhoid is considered. The system of equations was solved using Laplace Adomian Decomposition Method (LADM) and was implemented using MATLAB. The analysis showed that an increase in the burden or cases of Cholera will result to an increase of Typhoid fever and vise-versa indicating that there is a symbiotic nature of the relationship between the typhoid disease and the cholera disease.

KEYWORDS: Laplace Adomian Decomposition Method (LADM), Cholera and Typhoid

1.0 IINTRODUCTION

Typhoid fever and cholera remain a huge public health problem on the African continent due to deteriorating infrastructure and declining funding for infrastructure development. The diseases are both caused by bacteria, and they are associated with poor hygiene and waste of disposal systems. Also, typhoid fever and cholera are potential life-threatening infectious diseases, which and are mainly transmitted through the consumption of food, drink or water that have been contaminated by the feces or urine of subjects excreting the pathogen.

Cholera, an acute gastro-intestinal water-borne infection, is caused by the bacterium *Vibrio Cholerae*, V. *cholerae* or O139. Some of the symptoms are vomiting and diarrhea. And if treatment is delayed, it can lead to severe dehydration and death within a few hours. The disease has two modes of transmission: direct and indirect transmission. Direct transmission (human-human) is very uncommon, while the indirect transmission (environment-human), which occurs through the ingestion of contaminated food or water, is more frequent. Known estimates of the incubation period for the cholera disease is (fourteen)14days. On the other hand, the *Salmonella Typhi* bacteria is responsible for causing the life-threatening typhoid fever disease. Cholera and typhoid fever have the same transmission modes. The reticuloendothelial system, the intestinal lymphoid, and the gall bladder are severely damaged by the typhoid fever disease. Once a susceptible individual is infected by the disease, roughly (nineteen) 19 days are required for the disease to incubate within the host. Mathematical models have been used for the past decades to give insights into the transmission dynamics of co-infections within the human population.

2.0 LITERATURE REVIEW

[12] in their work entitled mmathematical analysis of cholera and co-infection transmission dynamics, and cholera remain a huge public health problem on stated that typhoid fever the African continent due to deteriorating infrastructure and declining funding for infrastructure development. The diseases are both caused by bacteria, and**,** they are associated with poor hygiene and waste disposal systems. In this paper, they consider a nonlinear system of ordinary differential equations for the co-infection of typhoid and cholera in a homogeneously mixing population. The model's steady states are determined and analyzed in terms of the model's reproduction number. Impact analysis—how the diseases impact on each other—is carried out. Numerical simulations and sensitivity analysis are also given. The results show that the control of the diseases should be carried out in tandem for the greatest impact of disease control. The results have important implications in the management of the two diseases.

[20] in their work stated that Cholera, a waterborne gastro enteric infection, remains a significant threat to public health in sub-Saharan Africa, which is also the region most heavily affected by HIV. It is biologically plausible that immune suppression caused by HIV infection predisposes to cholera. In their study, a simple mathematical model was developed and comprehensively analyzed to assess whether HIV infection is associated with an increased risk for cholera or

not. Analytical results in the model showed that the quantities *R*c and *R*h, which represents the reproductive number for cholera and HIV infection, respectively, provided a threshold conditions that determine cumulative new single and dual infection cases. These threshold conditions can be used to gain important insights on the epidemiological consequences of HIV and cholera co-existence in the community. Numerical results were provided to support the analytical findings. The findings gave a suggestion that in a cholera-endemic area, HIV infection is associated with an increased risk for cholera.

[4], in their paper, described the two different stochastic differential equations representing cholera dynamics. The first stochastic differential equation is formulated by introducing the stochasticity to deterministic model by parametric perturbation technique which is a standard technique in stochastic modeling and the second stochastic differential equation is formulated with the use of transition probabilities. They also went ahead to analyse a stochastic model using suitable Lyapunov function and its formula. They stated and proved the conditions for global existence, uniqueness of positive solutions, stochastic boundedness, global stability in probability, moment exponential stability, and almost sure convergence. And also, carrying out numerical simulation using Euler-Maruyama scheme, they simulated the sample paths of stochastic differential equations. Their results showed that the sample paths are continuous but not differentiable (a property of Wiener process). Also, they compare the numerical simulation results for deterministic and stochastic models, and finding is that the sample path of SIsIaR-B stochastic differential equations model fluctuates within the solution of the SIsIaR-B ordinary differential equation model. Furthermore, they used extended Kalman filter to estimate the model compartments (states), and they found out that the state estimated fit the measurements.

[20] in their work entitled modeling the transmission dynamics of typhoid in malaria endemic setting demonstrated that a typhoid outbreak will inevitably lead to a spike in the malaria cases. That is typhoid and malaria co-infection is a major public health problem in many developing countries. In this paper, a deterministic model for malaria and typhoid co-infection was proposed and analyzed. It was established that the model exhibits a backward bifurcation phenomenon. Overall, the study revealed that a typhoid outbreak in malaria endemic settings may lead to higher cumulative cases of dually-infected individual displaying clinical symptoms of both infections than singly-infected individuals displaying clinical symptoms of either malaria or typhoid.

[26] in their paper presented a mathematical model for cholera epidemics which comprises seasonality, loss of host immunity, and control mechanisms, which are working towards reducing cholera transmission. A collection of data related to cholera disease allows us to show that outbreaks in endemic areas are subject to a resonant behavior, since the intrinsic oscillation period of the disease (1 year) is synchronized with the annual contact rate variation. Moreover, they argued that the short period of the host immunity may be associated to secondary peaks of incidence observed in some regions (a bimodal pattern). Finally, they went ahead to explore some possible mechanisms of cholera control, and analyze their efficiency. Conclusion made was that besides mass vaccination which may be impracticable, improvements in sanitation system and food/personal hygiene are the most effective ways to prevent an epidemic.

[11] in their work entitled mathematical model for the control of ccholera eepidemic wwithout natural recovery followed a slight modification as compared to previous cholera models for the Nigerian case. Their model incorporates treatment, water hygiene as well as environmental sanitation. the model employs a system of nonlinear ordinary differential equations, which is analyzed in detail for its stability properties. They computed the basic reproduction ratio for the various control parameters and discovered that with proper combination of control measures, the spread of cholera could be minimized.

[2] in their paper showed that whenever the basic reproduction number is lowered to below one, Then the malaria and the pneumonia cases will be reduced in a model of malaria pneumonia co-infection. They present and analyzed a cholera epidemiological model with control measures incorporated. This model is extended from the one proposed by including the effects of vaccination, therapeutic treatment, and water sanitation. Equilibrium analysis is conducted in the case with constant controls for both epidemic and endemic dynamics. Optimal control theory was applied to seek cost-effective solution of multiple time-dependent intervention strategies against cholera outbreaks.

[18], formulated a simple mathematical model to assess whether HIV infection is associated with an increased risk for cholera or not. They began first, by analyzing the sub-models for HIV only and Cholera only. Analysis of the steady states for the HIV sub model using Lyapunov functions shows that the model has globally asymptotically stable equilibrium, namely, the disease-free equilibrium (when the associated reproductive number is less than unity) and the endemic equilibrium (when the associated reproductive number is greater than unity). They observed, from the HIV sub-model that, HIV transmission and human recruitment have a positive impact on increasing the magnitude of HIV-induced reproductive number. Also, it was noted by them that, albeit natural mortality and AIDS related may reduce the magnitude of the HIV induced reproductive number. They stated clearly that do not have control over these parameters; hence there is need for HIV intervention strategies in order to reduce HIV transmission thereby reducing HIV prevalence. And also, important results from the sensitivity analysis of the cholera induced reproductive number suggests the need for cholera related intervention strategies which may reduce the

magnitude of bacteria ingestion and bacteria shedding, as well as increasing bacteria death rate.

[19], formulated and qualitatively analysed the cholera model with and without public health interventions and carefully evaluated the impact of public health educational campaign, vaccination as possible control measures to curtail the spread of cholera epidemic in any community. Obviously, these assumptions are not complete; but the model is sophisticated enough and it captures some of the tenets of cholera epidemic and assumes basically that transmission of the disease is through ingestion of faecal matter contaminated with the V. cholerae bacterium, and our results are based on the model formulation and assumptions. The threshold and equilibria are obtained and stabilities examined.

They used a Lyapunov functional approach method to prove the global stability of the disease-free and endemic equilibria of the cholera only sub model, and to prove that the full model is ultimately uniformly persistent. Positivity and existence of solutions were presented. Their results showed that effective control of the epidemic can easily be achieved when vaccination, public health education and treatment are implemented. Otherwise in settings where education, vaccination or treatment coverage is not adequate (in this cases R_0 is large), cholera will not be fully controlled using only one control measure.

[3] the incubation period of cholera has important implication for clinical and public health decision-making, yet statements of the incubation period of cholera are often imprecise. Here we characterize the distribution of cholera's incubation period.

[9] Typhi bacteria, is associated with poor hygiene, poor waste disposal system and seasonal rains. Recently in Zimbabwe, the infection has been found to exist due to dilapidated infrastructure, The existence of the infection remains a huge public health problem. In the paper we study the typhoid fever transmission dynamics with fear in periodic environments. We formulate a nonlinear system of differential equations in which the infection rate is time dependent. The model's steady states are determined and the stability analysis carried out.

[15] In addition to true co-infection of malaria and typhoid, false diagnoses due to similar signs and symptoms and false positive results in testing methods leading to Improper controls, are the major challenges on managing these diseases. In this study we develop novel mathematical models describing the co-infection dynamics of malaria and typhoid. Through mathematical analyses of our models. We identify distinct features of typhoid and malaria infection dynamics as well as relationships associated to their co- infection. the global dynamics of typhoid can be determined by a single threshold.

[16] Infectious symptomatic and asymptomatic individuals are detected and quarantined. The impact of time dependent screening of infectious individuals is explored. Comprehensive analysis of the model suggest that time dependent screening can be effective to can be effective to control or eliminate new typhoid cases in the community. Further, we note that if there is a typhoid outbreak in the community then screening of both symptomatic and asymptomatic.

3.0 METHODS

Below are the step by step methods that shall be taken towards realization of our set objectives:

1) We shall carefully formulate the model that captures various features of the transmission dynamics of the disease.
2) Using appropriate techniques, we shall analyze the model for its properties.
3) Using next generation method, we shall compute the reproduction number for the disease and analyze it.
4) Using Blower's method, we shall conduct uncertainty and sensitivity analysis so as to determine the top rank parameters that drives the dynamic of the disease
5) By approaching authorized relevant authorities, we shall gather real life data pertaining to the disease.
6) Using the fmin con package in MATLAB, we shall fit the real-life data to our model for parameter estimation.
7) By adopting the parameter estimates, we shall do numerical simulation of the model towards validation of the theoretical results.
8) We shall do robust discussion of the plots from the simulation.
9) We shall make appropriate recommendations for the use of the policy makers in the health sector in combating the spread of the disease and procuring strategies that will help mitigating the disease burden.

3. 1 Model Formulation

The typhoid cholera co-infection model partitions, the human population $N(t)$, at time *t*, into a susceptible class $S(t)$, a cholera infection class $Ic(t)$ a cholera treatment class $Tc(t)$, a typhoid infection class $It(t)$, a typhoid treatment class $Tt(t)$, a co-infection class $Ict(t)$, a cholera recovery class $Rc(t)$, a typhoid recovery class $Rc(t)$, and a co-infection recovery class $Rct(t)$.

Thus,

$$N(t) = S(t) + Ic(t) + Tc(t) + Rc(t) + It(t) + Tt(t) + Rt(t) + Ict(t) + Rct(t)$$

The bacterial concentration of *Salmonella Typhi*, $Zt(t)$, and *Vibrio Cholerae*, $ZC(t)$, in the environment are incorporated into the model as well.

3.2 Assumptions Made in The Formulation of The Model

1. The natural death rates for humans are uniform.
2. Re-infection of the disease (Cholera) is possible (Endogenous re-infection) if an individual loses immunity.

3. There is bacteria reservoir class for the both diseases
4. All newly born individuals will join only susceptible class.

Below is the schematic diagram for the model.

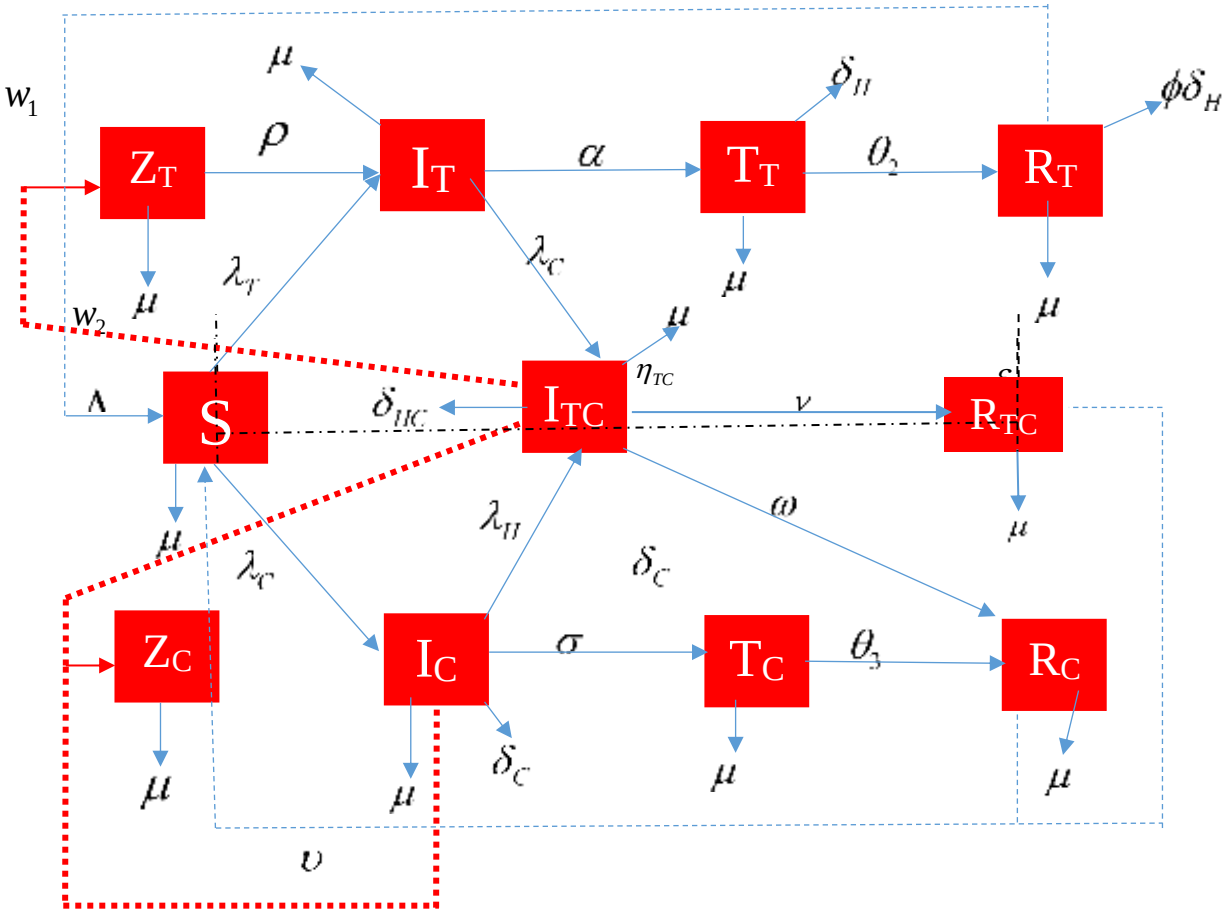

Fig. 1: Schematic diagram of the model

Where $\lambda c = \lambda c_1 + \lambda c_2$ **and** $\lambda_T = \lambda_{T1} + \lambda_{T2}$, $\lambda_1 = \lambda_{T3} + \lambda_{T4}$, $\lambda_2 = \lambda_{C3} + \lambda_{C4}$

3.3 Model Equations

$$S' = \Lambda - (\lambda_{C1} + \lambda_C + \lambda_{T1} + \lambda_{T2})S - \mu S + \eta_C R_C + \eta_T R_T + \eta_{CT} R_{CT}$$

$$I_C' = (\lambda_{C1} + \lambda_{C2})S - (\lambda_{T3} + \lambda_{T4})I_C - (\mu + \delta_C + \xi_C + \tau_C)I_C$$

$$T_C' = \tau_C - (\mu - \xi_C)T_C$$

$$I_T' = (\lambda_{T1} + \lambda_{T2})S - (\lambda_{C3} + \lambda_{C4})I_T - (\mu + \delta_T + \xi_T + \tau_{T_t})I_T$$

$$T_T' = \tau_T - (\mu - \xi_T)T_T$$

$$I_{TC}' = (\lambda_{T3} + \lambda_{T4})I_C + (\lambda_{C3} + \lambda_{C4}) - (\mu - \theta - \delta_{CT})I_{TC}$$

$$T_C' = \theta I_{CT} - (\tau_{CT} - \mu)T_C$$

$$R_C' = \delta_C I_C - (\mu + \eta_C)R_C$$

$$R_T' = \delta_T I_T - (\mu + \eta_T)R_T$$

$$R_{TC}' = \delta_{CT} I_{CT} - (\mu + \eta_{CT})R_{TC}$$

$$Z_C' = d_C B_C \left(1 - \frac{B_C}{k_C}\right) + \gamma_C I_C + \phi_C I_{TC} + \rho_C I_C - \mu Z_C$$

$$Z_t' = d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}+\phi_T I_{TC}+\rho_T I_T-\mu Z_T$$

Where $S' = dS/dt$, $I' = dI/dt$, e.t.c

3.4 Model Description

Susceptible Class(S): The class for the susceptible individuals are increased by the recruitment of individuals into the class by birth or immigration at the rate Λ. The class reduces following the infection acquired by contact between the susceptible and infected class.

Infected Typhoid Class (I_T): This class grows by the inflow of those who are susceptible to cholera progresses to the infected class of cholera at the rate (λ), disease induced occur at rate (λ_C) and disease induced death at rate (δ_C).

Typhoid Treatment Class (T_T): This class grow due to the population of infected typhoid that progress to treatment class of typhoid at rate (α) and natural death occur at (μ) and treatment induced recovery at rate $\phi\delta_T$.

Infected Cholera and Typhoid Class (I_{TC}): This class grows due to the population that flows from Infected typhoid to Infected typhoid and Cholera at the rate λ_1 and λ_2 and further diminishes by natural death at rate (μ), Disease induced death at rate (δ_{TC}).

Infected cholera Class (I_C): This class increase as a result of susceptible individual who are fully infected and infectious at rate (λ_C) and further diminishes into natural death at rate (μ) and disease induce death at rate (δ_C).

Treatment cholera class (T_C): this class increase due to the follow of cholera infected individual to the treatment class at rate σ and natural death occurs at rate (μ).

Recovered Class of cholera: This class is populated due to progression from treatment class of cholera at rate (θ_3) and natural death occurs at rate (μ).

Recovered class of typhoid (R_C): This class is populated because of the inflow of individual who progressed from treatment class of cholera at rate (θ_2) to recovered class of typhoid, it further diminishes to natural death at rate (μ) and treatment induced recovery occur at rate $(\phi\delta_T)$.

Bacterial Class of Cholera (Zc): bacteria reservoir class.

Bacterial class of typhoid (ZT) : bacterial reservoir class.

3.5 Table 1: Variable and Parameter description

Variable	Description
S	Population of individuals who are Susceptible to typhoid –cholera
I_T	Population of individuals who are Infected with typhoid
I_C	Population of individuals who are Infected With cholera.
T_T	Population of individuals who are in treatment class of typhoid.
T_C	Population of individuals who are in treatment class of cholera
I*Tc*	Individual infected with both typhoid and cholera
z_T	Bacterial reservoir or population for typhoid
z_C	Bacterial reservoir or population for Cholera
RT	Recovered individual from typhoid
RC	Population of individuals recovered from cholera
RTC	Recovered individual from both typhoid and cholera
Λ	Recruitment rate
λ_T	Progression rate from susceptible to infected typhoid
λ_C	Progression rate from susceptible to infected cholera
α	Progression rate from infected typhoid to treatment class of typhoid
δ_T	Typhoid induced death rate

θ_2	Progression from treatment class of cholera to recovered class of cholera
υ	Transition rate from recovered class of cholera to susceptible class
ω	Progression from infected typhoid and cholera to recovered cholera
δ	Disease induced death rate
μ	Natural death rate
θ_3	Progression from treatment class of cholera to recovered class of cholera.
ε	Rate at which the bacterial transferred disease to susceptible human.
ν	Progression rate from infected typhoid and cholera to recovered class of typhoid and cholera

3.6 Model Analysis

3.6.1 Fractional Order of Typhoid/Cholera Model

The Caputo derivative is measured as a differential operator in our model. We present in this segment some well-known definitions and effects that we shall be using throughout this research.

Definition: The Caputo fractional order derivative of a function (f) on the interval [O,T] is defined by:

$$\left[{}^{C}D_0^{\beta} f(t)\right] = \frac{1}{\Gamma(n-\beta)}\int_0^t (t-s)^{n-\beta-1} f^{(n)}(s)ds, \tag{1}$$

Where $n=[\beta]+1$ and $[\beta]$ represents the integer part of β. In particular, for $0<\beta<1,$ the Caputo derivative becomes:

$$\left[{}^{C}D_0^{\beta} f(t)\right] = \frac{1}{\Gamma(1-\beta)}\int_0^t \frac{f(s)}{(t-s)^{\beta}} ds, \tag{2}$$

Definition: Laplace transform of Caputo derivatives is defined as

$$\mathcal{L}[{}^{C}D^{\beta}q(t)] = S^{\beta}h(S) - \sum_{K=0}^{n} S^{\beta-i-1}y^{k}(0), \quad n-1<\beta<n,\ n\in N, \tag{3}$$

For arbitrary $c_i \in R, i=0,1,2,...n-1, n=[\beta]+1$ and $[\beta]$ represents the non-integer part of β.

Lemma : The following results hold for fractional differentiation equations

$$I^{\beta}[{}^{c}D^{\beta}h](t) = h(t) + \sum_{i=0}^{n-1} \frac{h^{(t)}(0)}{i!} t^{i}, \tag{4}$$

For arbitrary $\beta>0, i=0,1,2,...,n-1,$ where $n=[\beta]+1$ and $[\beta]$ represents the integer part of β.

Introducing fractional-order into the model, we now present a new model described by the following Introducing fractional order derivative into the model we present new mathematical model describe by set of fractional difference of order β for $0<\beta<1$

$$D^{\beta}(S) = \Lambda - \left(\lambda_{C1}+\lambda_{C}+\lambda_{T1}+\lambda_{T2}\right)S - \mu S + \eta_C R_C + \eta_T R_T + \eta_{CT} R_{CT}$$

$$D^{\beta}(I_c) = \left(\lambda_{C1}+\lambda_{C2}\right)S - \left(\lambda_{T3}+\lambda_{T4}\right)I_C - \left(\mu+\delta_C+\xi_C+\tau_C\right)I_C$$

$$D^{\beta}(T_C) = \tau_C - \left(\mu-\xi_C\right)T_C$$

$$D^{\beta}\left(I_T\right) = \left(\lambda_{T1}+\lambda_{T2}\right)S - \left(\lambda_{C3}+\lambda_{C4}\right)I_T - \left(\mu+\delta_T+\xi_T+\tau_{T_t}\right)I_T$$

$$
\begin{aligned}
D^{\beta}\left(T_T\right) &= \tau_T - \left(\mu - \xi_T\right)T_T \\
D^{\beta}\left(I_{TC}\right) &= \left(\lambda_{T3} + \lambda_{T4}\right)I_C + \left(\lambda_{C3} + \lambda_{C4}\right) - \left(\mu - \theta - \delta_{CT}\right)I_{TC} \\
D^{\beta}\left(T_C\right) &= \theta I_{CT} - \left(\tau_{CT} - \mu\right)T_C \\
D^{\beta}\left(R_C\right) &= \delta_C I_C - \left(\mu + \eta_C\right)R_C \\
D^{\beta}\left(R_T\right) &= \delta_T I_T - \left(\mu + \eta_T\right)R_T \\
D^{\beta}\left(R_{TC}\right) &= \delta_{CT} I_{CT} - \left(\mu + \eta_{CT}\right)R_{TC} \\
D^{\beta}\left(Z_C\right) &= d_C B_C\left(1 - \frac{B_C}{k_C}\right) + \gamma_C I_C + \varphi_C I_{TC} + \rho_C I_C - \mu Z_C \\
D^{\beta}\left(Z_T\right) &= d_T B_T\left(1 - \frac{B_T}{k_T}\right) + \gamma_T I_{TC} + \varphi_T I_{TC} + \rho_T I_T - \mu Z_T
\end{aligned}
\tag{5}
$$

From the impact analysis section, we found that an increase in cholera cases may be associated with an increased risk of typhoid and that an increase in typhoid cases may be associated with an increased risk of cholera. The result proves the symbiotic nature of the relationship between the typhoid disease and the cholera disease.

3.6.2 The Laplace Adomian Decomposition Method (LADM) Implementation

In this section, we deliberate the general procedure of this method with the given initial conditions. Applying Laplace transforms to both sides of the equation (1), and then we obtain:

$$
\begin{aligned}
S^{\beta}\mathcal{L}(S) - S^{\beta-1}S(0) &= \mathcal{L}\left[\Lambda - \left(\lambda_{C1} + \lambda_C + \lambda_{T1} + \lambda_{T2}\right)S - \mu S + \eta_C R_C + \eta_T R_T + \eta_{CT} R_{CT}\right] \\
S^{\beta}\mathcal{L}(I_C) - S^{\beta-1}I_C(0) &= \mathcal{L}\left[\left(\lambda_{C1} + \lambda_{C2}\right)S - \left(\lambda_{T3} + \lambda_{T4}\right)I_C - \left(\mu + \delta_C + \xi_C + \tau_C\right)I_C\right] \\
S^{\beta}\mathcal{L}(T_C) - S^{\beta-1}T_C(0) &= \mathcal{L}\left[\tau_C - \left(\mu - \xi_C\right)T_C\right] \\
S^{\beta}\mathcal{L}(I_T) - S^{\beta-1}S(0) &= \mathcal{L}\left[\left(\lambda_{T1} + \lambda_{T2}\right)S - \left(\lambda_{C3} + \lambda_{C4}\right)I_T - \left(\mu + \delta_T + \xi_T + \tau_{T_T}\right)I_T\right] \\
&\vdots \\
S^{\beta}\mathcal{L}(Z_T) - S^{\beta-1}Z_T(0) &= \mathcal{L}\left[d_T B_T\left(1 - \frac{B_T}{k_T}\right) + \gamma_T I_{TC} + \varphi_T I_{TC} + \rho_T I_T - \mu Z_T\right]
\end{aligned}
\tag{6}
$$

With initial conditions

$S(0) = n_1,\ \ I_C(0) = n_2\ ,\ T_C(0) = n_3,\ \ I_T(0) = n_4,\ \ T_T(0) = n_5,\ \ I_{TC}(0) = n_6,\ T_C(0) = n_7,\ R_C(0) = n_8,$

$R_T(0) = n_9,\ \ R_{TC}(0) = n_{10},\ Z_C(0) = n_{11},\ Z_T(0) = n_{12}$

Dividing equation (2) by (S^{β}) we obtain:

$$
\begin{aligned}
\mathcal{L}(S) &= \frac{n_1}{S} + \frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda - \left(\lambda_{C1} + \lambda_C + \lambda_{T1} + \lambda_{T2}\right)S - \mu S + \eta_C R_C + \eta_T R_T + \eta_{CT} R_{CT}\right] \\
\mathcal{L}(I_C) &= \frac{n_2}{S} + \frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{C1} + \lambda_{C2}\right)S - \left(\lambda_{T3} + \lambda_{T4}\right)I_C - \left(\mu + \delta_C + \xi_C + \tau_C\right)I_C\right] \\
\mathcal{L}(T_C) &= \frac{n_3}{S} + \frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C - \left(\mu - \xi_C\right)T_C\right] \\
\mathcal{L}(I_T) &= \frac{n_4}{S} + \frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{T1} + \lambda_{T2}\right)S - \left(\lambda_{C3} + \lambda_{C4}\right)I_T - \left(\mu + \delta_T + \xi_T + \tau_{T_T}\right)I_T\right] \\
&\vdots \\
\mathcal{L}(Z_T) &= \frac{n_{12}}{S} + \frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1 - \frac{B_T}{k_T}\right) + \gamma_T I_{TC} + \varphi_T I_{TC} + \rho_T I_T - \mu Z_T\right]
\end{aligned}
\tag{7}
$$

By disintegrating the non-linear term of equation (3) whereby we assume the solution of $S(t)$, $I_C(t)$, $T_C(t)$, $I_T(t)$, $T_T(t)$, $I_{TC}(t)$, $T_C(t)$ - - - $Z_T(t)$ are in the form of infinite series given by:

$$S(t)=\sum_{n=0}^{\infty}S(n),\quad I_C(t)=\sum_{n=0}^{\infty}I_C(n),\quad T_C(t)=\sum_{n=0}^{\infty}T_C(n),\quad I_T(t)=\sum_{n=0}^{\infty}I_T(n),\quad T_T(t)=\sum_{n=0}^{\infty}T_T(n),$$
$$I_{TC}(t)=\sum_{n=0}^{\infty}I_{TC}(n),\quad T_C(t)=\sum_{n=0}^{\infty}T_C(n),\quad R_C(t)=\sum_{n=0}^{\infty}R_C(n),\quad R_T(t)=\sum_{n=0}^{\infty}R_T(n),\quad R_{TC}(t)=\sum_{n=0}^{\infty}R_{TC}(n), \tag{8}$$
$$Z_C(t)=\sum_{n=0}^{\infty}Z_C(n),\quad Z_T(t)=\sum_{n=0}^{\infty}Z_T(n),$$

The non-linear term in the model is as follows:

$$I_CS,\quad I_TS,\quad I_{TC}S, \tag{9}$$

We have three (3) non-linear terms. The non-linear term in equation (5) are decomposed by Adomian polynomial as follows:

$$I_C(t)S(t)=\sum_{n=0}^{\infty}A(n),\quad I_T(t)S(t)=\sum_{n=0}^{\infty}C(n),\quad I_{TC}(t)S(t)=\sum_{n=0}^{\infty}D(n) \tag{10}$$

Where A_n, C_n and D_n are Adomian polynomials given by

$$A(n)=\frac{1}{\Gamma(n+1)}\frac{d^n}{d\lambda^n}\left[\sum_{k=0}^{n}\lambda^k I_{C_k}(t)\sum_{k=0}^{n}\lambda^k S_k(t)\right]_{\lambda=0}$$

$$C(n)=\frac{1}{\Gamma(n+1)}\frac{d^n}{d\lambda^n}\left[\sum_{k=0}^{n}\lambda^k I_{T_k}(t)\sum_{k=0}^{n}\lambda^k S_k(t)\right]_{\lambda=0} \tag{11}$$

$$D(n)=\frac{1}{\Gamma(n+1)}\frac{d^n}{d\lambda^n}\left[\sum_{k=0}^{n}\lambda^k (t)I_{TC_k}\sum_{k=0}^{n}\lambda^k S_k(t)\right]_{\lambda=0}$$

The polynomials are given by

$$A_0=I_{C_0}(t)S_0(t),$$
$$A_1=I_{C_0}(t)S_1(t)+I_{C_1}(t)S_0(t),$$
$$A_2=I_{C_0}(t)S_2(t)+I_{C_1}(t)S_1(t)+I_{C_2}(t)S_0(t),$$
$$C_0=I_{T_0}(t)S_0(t),$$
$$C_1=I_{T_0}(t)S_1(t)+I_{T_1}(t)S_0(t),$$
$$C_2=I_{T_0}(t)S_2(t)+I_{T_1}(t)S_1(t)+I_{T_2}(t)S_0(t),$$
$$D_0=I_{TC_0}(t)S_0(t),$$
$$D_1=I_{TC_0}(t)S_1(t)+I_{TC_1}(t)S_0(t),$$
$$D_2=I_{TC_0}(t)S_2(t)+I_{TC_1}(t)S_1(t)+I_{TC_2}(t)S_0(t),$$

Substituting equation (4) and (7) into equation (3) we obtain:

$$\mathcal{L}\left\{\sum_{n=0}^{\infty}S_n\right\}=\frac{n_1}{S}+\left[\frac{1}{S^{\beta}}\mathcal{L}\left\{\Lambda-\left(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2}\right)S-\mu S+\eta_C R_C+\eta_T R_T+\eta_{CT}R_{CT}\right\}\right]$$
$$\mathcal{L}\left\{\sum_{n=0}^{\infty}I_C\right\}=\frac{n_2}{S}+\left[\frac{1}{S^{\beta}}\mathcal{L}\left\{\left(\lambda_{C1}+\lambda_{C2}\right)S-\left(\lambda_{T3}+\lambda_{T4}\right)\sum_{n=0}^{\infty}A(n)-\left(\mu+\delta_C+\xi_C+\tau_C\right)\sum_{n=0}^{\infty}A(n)\right\}\right]$$

$$\mathcal{L}\left\{\sum_{n=0}^{\infty}T_C\right\}=\frac{n_3}{S}+\left[\frac{1}{S^{\beta}}\mathcal{L}\left\{\tau_C-(\mu-\xi_C)\sum_{n=0}^{\infty}D(n)\right\}\right]$$

$$\mathcal{L}\left\{\sum_{n=0}^{\infty}I_T\right\}=\frac{n_5}{S}+\left[\frac{1}{S^{\beta}}\mathcal{L}\left\{(\lambda_{T1}+\lambda_{T2})S-(\lambda_{C3}+\lambda_{C4})\sum_{n=0}^{\infty}C(n)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)\sum_{n=0}^{\infty}C(n)\right\}\right]$$

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$$\mathcal{L}\left\{\sum_{n=0}^{\infty}Z_T\right\}=\frac{n_{12}}{S}+\left[\frac{1}{S^{\beta}}\mathcal{L}\left\{d_TB_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T\sum_{n=0}^{\infty}D(n)+\varphi_T\sum_{n=0}^{\infty}D(n)+\rho_T\sum_{n=0}^{\infty}C(n)-\mu Z_T\right\}\right]$$

From equation (7), we compute the Adomian polynomials when n = 0, as

$$A(0)=\frac{1}{\Gamma(n+1)}\frac{d^0}{d\lambda^0}\left\{\left[\lambda^0I_C(0)\right]\left[\lambda^0S(0)\right]\right\}\big|_{\lambda}=I_C(0)S(0)$$

$$C(0)=\frac{1}{\Gamma(n+1)}\frac{d^0}{d\lambda^0}\left\{\left[\lambda^0I_T(0)\right]\left[\lambda^0S(0)\right]\right\}\big|_{\lambda}=I_T(0)S(0) \qquad (12)$$

$$D(0)=\frac{1}{\Gamma(n+1)}\frac{d^0}{d\lambda^0}\left\{\left[\lambda^0I_{TC}(0)\right]\left[\lambda^0S(0)\right]\right\}\big|_{\lambda}=I_{TC}(0)S(0)$$

Similarly, when n = 1 the Adomian polynomials are as:

$$A(1)=\frac{1}{\Gamma(2)}\frac{d^1}{d\lambda^1}\left\{\left[I_C(0)+\lambda I_C(1)\right]\left[S(0)+\lambda S(1)\right]\right\}\big|=I_C(0)I_C(1)+S(1)S(0)$$

$$C(1)=\frac{1}{\Gamma(2)}\frac{d^1}{d\lambda^1}\left\{\left[I_T(0)+\lambda I_T(1)\right]\left[S(0)+\lambda S(1)\right]\right\}\big|=I_T(0)I_T(1)+S(1)S(0) \qquad (13)$$

$$D(1)=\frac{1}{\Gamma(2)}\frac{d^1}{d\lambda^1}\left\{\left[I_{TC}(0)+\lambda I_{TC}(1)\right]\left[S(0)+\lambda S(1)\right]\right\}\big|=I_{TC}(0)I_{TC}(1)+S(1)S(0)$$

Taking the Laplace transform of the initial conditions we have that;

$$\mathcal{L}S(0)=\frac{n_1}{s},\quad \mathcal{L}I_C(0)=\frac{n_2}{s},\quad \mathcal{L}T_C(0)=\frac{n_3}{s},\quad \mathcal{L}I_T(0)=\frac{n_4}{s},\quad \mathcal{L}T_T(0)=\frac{n_5}{s},$$

$$\mathcal{L}I_{TC}(0)=\frac{n_6}{s},\quad \mathcal{L}T_C(0)=\frac{n_7}{s}\quad \mathcal{L}R_C(0)=\frac{n_8}{s},\quad \mathcal{L}R_T(0)=\frac{n_9}{s},\quad \mathcal{L}R_{TC}(0)=\frac{n_{10}}{s},$$

$$\mathcal{L}Z_C(0)=\frac{n_{11}}{s},\quad \mathcal{L}Z_T(0)=\frac{n_{12}}{s}$$

Substituting these initial conditions and the Adomian polynomials presented in equations (7), (9) into (3), and then we obtain:

$$\mathcal{L}(S)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})S(0)-\mu S(0)+\eta_CR_C(0)+\eta_TR_T(0)+\eta_{CT}R_{CT}(0)\right]$$

$$\mathcal{L}(I_C)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})S(0)-(\lambda_{T3}+\lambda_{T4})I_C(0)-(\mu+\delta_C+\xi_C+\tau_C)I_C(0)\right] \qquad (14)$$

$$\mathcal{L}(T_C)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)T_C(0)\right]$$

$$\mathcal{L}(I_T)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(0)-(\lambda_{C3}+\lambda_{C4})I_T(0)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(0)\right]$$

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$$\mathcal{L}(Z_T)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_TB_T\left(1-\frac{B_T}{k_T}\right)+\gamma_TI_{TC}(0)+\varphi_TI_{TC}(0)+\rho_TI_T(0)-\mu Z_T(0)\right]$$

Similarly:

$$\mathcal{L}(S)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-\left(\lambda_{C1}+\lambda_{C}+\lambda_{T1}+\lambda_{T2}\right)S(1)-\mu S(1)+\eta_{C}R_{C}(1)+\eta_{T}R_{T}(1)+\eta_{CT}R_{CT}(1)\right]$$

$$\mathcal{L}(I_{C})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{C1}+\lambda_{C2}\right)S(1)-\left(\lambda_{T3}+\lambda_{T4}\right)I_{C}(1)-\left(\mu+\delta_{C}+\xi_{C}+\tau_{C}\right)I_{C}(1)\right]$$

$$\mathcal{L}(T_{C})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_{C}-\left(\mu-\xi_{C}\right)T_{C}(1)\right]$$

$$\mathcal{L}(I_{T})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{T1}+\lambda_{T2}\right)S(1)-\left(\lambda_{C3}+\lambda_{C4}\right)I_{T}(1)-\left(\mu+\delta_{T}+\xi_{T}+\tau_{T_{T}}\right)I_{T}(1)\right]$$

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$$\mathcal{L}(Z_{T})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_{T}B_{T}\left(1-\frac{B_{T}}{k_{T}}\right)+\gamma_{T}I_{TC}(1)+\varphi_{T}I_{TC}(1)+\rho_{T}I_{T}(1)-\mu Z_{T}(1)\right]$$

$$\vdots \qquad = \qquad \vdots$$

$$\mathcal{L}(S)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-\left(\lambda_{C1}+\lambda_{C}+\lambda_{T1}+\lambda_{T2}\right)S(n)-\mu S(n)+\eta_{C}R_{C}(n)+\eta_{T}R_{T}(n)+\eta_{CT}R_{CT}(n)\right]$$

$$\mathcal{L}(I_{C})(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{C1}+\lambda_{C2}\right)S(n)-\left(\lambda_{T3}+\lambda_{T4}\right)I_{C}(n)-\left(\mu+\delta_{C}+\xi_{C}+\tau_{C}\right)I_{C}(n)\right]$$

$$\mathcal{L}(T_{C})(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_{C}-\left(\mu-\xi_{C}\right)T_{C}(n)\right] \qquad (15)$$

$$\mathcal{L}(I_{T})(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{T1}+\lambda_{T2}\right)S(n)-\left(\lambda_{C3}+\lambda_{C4}\right)I_{T}(n)-\left(\mu+\delta_{T}+\xi_{T}+\tau_{T_{T}}\right)I_{T}(n)\right]$$

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$$\mathcal{L}(Z_{T})(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_{T}B_{T}\left(1-\frac{B_{T}}{k_{T}}\right)+\gamma_{T}I_{TC}(n)+\varphi_{T}I_{TC}(n)+\rho_{T}I_{T}(n)-\mu Z_{T}(n)\right]$$

Evaluating the Laplace transforms of the right-hand sides of equations (9) and (10), we obtain:

$$\mathcal{L}(S)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-\left(\lambda_{C1}+\lambda_{C}+\lambda_{T1}+\lambda_{T2}\right)S(0)-\mu S(n)+\eta_{C}R_{C}(0)+\eta_{T}R_{T}(0)+\eta_{CT}R_{CT}(0)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(I_{C})(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{C1}+\lambda_{C2}\right)S(0)-\left(\lambda_{T3}+\lambda_{T4}\right)I_{C}(0)-\left(\mu+\delta_{C}+\xi_{C}+\tau_{C}\right)I_{C}(0)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(T_{C})(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_{C}-\left(\mu-\xi_{C}\right)T_{C}(0)\right]\frac{1}{S^{\beta+1}} \qquad (16)$$

$$\mathcal{L}(I_{T})(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{T1}+\lambda_{T2}\right)S(0)-\left(\lambda_{C3}+\lambda_{C4}\right)I_{T}(0)-\left(\mu+\delta_{T}+\xi_{T}+\tau_{T_{T}}\right)I_{T}(0)\right]\frac{1}{S^{\beta+1}}$$

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$$\mathcal{L}(Z_{T})(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_{T}B_{T}\left(1-\frac{B_{T}}{k_{T}}\right)+\gamma_{T}I_{TC}(0)+\varphi_{T}I_{TC}(0)+\rho_{T}I_{T}(0)-\mu Z_{T}(0)\right]$$

Similarly:

$$\mathcal{L}(S)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-\left(\lambda_{C1}+\lambda_{C}+\lambda_{T1}+\lambda_{T2}\right)S(1)-\mu S(1)+\eta_{C}R_{C}(1)+\eta_{T}R_{T}(1)+\eta_{CT}R_{CT}(1)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(I_{C})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\left(\lambda_{C1}+\lambda_{C2}\right)S(1)-\left(\lambda_{T3}+\lambda_{T4}\right)I_{C}(1)-\left(\mu+\delta_{C}+\xi_{C}+\tau_{C}\right)I_{C}(1)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(T_{C})(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_{C}-\left(\mu-\xi_{C}\right)T_{C}(1)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(I_T)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(1)-(\lambda_{C3}+\lambda_{C4})I_T(1)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(1)\right]\frac{1}{S^{\beta+1}}$$

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$$\mathcal{L}(Z_T)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}(1)+\varphi_T I_{TC}(1)+\rho_T I_T(1)-\mu Z_T(1)\right]$$

$$\vdots \quad = \quad \vdots$$

$$\mathcal{L}(S)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})S(n)-\mu S(n)+\eta_C R_C(n)+\eta_T R_T(n)+\eta_{CT}R_{CT}(n)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(I_C)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})S(n)-(\lambda_{T3}+\lambda_{T4})I_C(n)-(\mu+\delta_C+\xi_C+\tau_C)I_C(n)\right]\frac{1}{S^{\beta+1}}$$

$$\mathcal{L}(T_C)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)T_C(n)\right]\frac{1}{S^{\beta+1}} \qquad (17)$$

$$\mathcal{L}(I_T)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(n)-(\lambda_{C3}+\lambda_{C4})I_T(n)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(n)\right]\frac{1}{S^{\beta+1}}$$

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$$\mathcal{L}(Z_T)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}(n)+\varphi_T I_{TC}(n)+\rho_T I_T(1)-\mu Z_T(n)\right]\frac{1}{S^{\beta+1}}$$

Taking the inverse Laplace transform of (12) and (13) we obtain:

$$\mathcal{L}(S)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})S(0)-\mu S(n)+\eta_C R_C(0)+\eta_T R_T(0)+\eta_{CT}R_{CT}(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(I_C)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})S(0)-(\lambda_{T3}+\lambda_{T4})I_C(0)-(\mu+\delta_C+\xi_C+\tau_C)I_C(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(T_C)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)T_C(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)} \qquad (18)$$

$$\mathcal{L}(I_T)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(0)-(\lambda_{C3}+\lambda_{C4})I_T(0)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

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$$\mathcal{L}(Z_T)(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}(0)+\varphi_T I_{TC}(0)+\rho_T I_T(0)-\mu Z_T(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

Similarly:

$$\mathcal{L}(S)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})S(1)-\mu S(1)+\eta_C R_C(1)+\eta_T R_T(1)+\eta_{CT}R_{CT}(1)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(I_C)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})S(1)-(\lambda_{T3}+\lambda_{T4})I_C(1)-(\mu+\delta_C+\xi_C+\tau_C)I_C(1)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(T_C)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)T_C(1)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(I_T)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(1)-(\lambda_{C3}+\lambda_{C4})I_T(1)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(1)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

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$$\mathcal{L}(Z_T)(2)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}(1)+\varphi_T I_{TC}(1)+\rho_T I_T(1)-\mu Z_T(1)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\vdots \quad = \quad \vdots$$

$$\mathcal{L}(S)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})S(0)-\mu S(0)+\eta_C R_C(0)+\eta_T R_T(0)+\eta_{CT}R_{CT}(0)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(I_C)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})S(n)-(\lambda_{T3}+\lambda_{T4})I_C(n)-(\mu+\delta_C+\xi_C+\tau_C)I_C(n)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

$$\mathcal{L}(T_C)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)T_C(n)\right]\frac{t^{\beta}}{\Gamma(\beta+1)} \qquad (19)$$

$$\mathcal{L}(I_T)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})S(n)-(\lambda_{C3}+\lambda_{C4})I_T(n)-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)I_T(n)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

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$$\mathcal{L}(Z_T)(n+1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T I_{TC}(n)+\varphi_T I_{TC}(n)+\rho_T I_T(n)-\mu Z_T(n)\right]\frac{t^{\beta}}{\Gamma(\beta+1)}$$

We recall the stated initial conditions and we substitute it into (18) then we obtain equation (21) as:

$$S=n_1,\quad I_C=n_2,\quad T_C=n_3,\quad I_T=n_4,\quad T_T=n_5,\ I_{TC}=n_6,\quad T_C=n_7,\quad R_C=n_8,\quad R_T=n_9,$$
$$R_{TC}=n_{10},\quad Z_C=n_{11},\quad Z_T=n_{12}.$$

$$S(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\Lambda-(\lambda_{C1}+\lambda_C+\lambda_{T1}+\lambda_{T2})n_1-\mu n_1+\eta_C n_8+\eta_T n_9+\eta_{CT}n_{10}(n)\right]\frac{1}{S^{\beta+1}}$$

$$I_C(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{C1}+\lambda_{C2})n_1-(\lambda_{T3}+\lambda_{T4})n_2-(\mu+\delta_C+\xi_C+\tau_C)n_2\right]\frac{1}{S^{\beta+1}}\ T_C(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[\tau_C-(\mu-\xi_C)n_3\right]\frac{1}{S^{\beta+1}}$$

(20)

$$I_T(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[(\lambda_{T1}+\lambda_{T2})n_1-(\lambda_{C3}+\lambda_{C4})n_4-\left(\mu+\delta_T+\xi_T+\tau_{T_T}\right)n_n\right]\frac{1}{S^{\beta+1}}$$

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$$Z_T(1)=\frac{1}{S^{\beta}}\mathcal{L}\left[d_T B_T\left(1-\frac{B_T}{k_T}\right)+\gamma_T n_6+\varphi_T n_6+\rho_T n_5-\mu n_{12}\right]\frac{1}{S^{\beta+1}}$$

The series for each compartment can be expressed as follows:

$$S(t)=S(0)+S(1)+S(2)...$$
$$I_C(t)=I_C(0)+I_C(1)+I_C(2)...$$
$$T_C(t)=T_C(0)+T_C(1)+T_C(2)... \qquad (21)$$
$$I_T(t)=I_T(0)+I_T(1)+I_T(2)...$$
$$T_T(t)=T_T(0)+T_T(1)+T_T(2)...$$
$$I_{TC}(t)=I_{TC}(0)+I_{TC}(1)+I_{TC}(2)...$$
$$R_C(t)=R_C(0)+R_C(1)+R_C(2)...$$
$$R_T(t)=R_T(0)+R_T(1)+R_T(2)...$$
$$R_{TC}(t)=R_{TC}(0)+R_{TC}(1)+R_{TC}(2)...$$
$$Z_C(t)=Z_C(0)+Z_C(1)+Z_C(2)...$$
$$Z_T(t)=Z_T(0)+Z_T(1)+Z_T(2)...$$

Numerical Solution of Laplace Adomian Decomposition Method (LADM)

In this section, we will see the numerical solution of the model. Using the initial conditions, the Laplace Adomian Decomposition Method (LADM) gives us an approximate solution in in terms of an infinite series presented as:

$$S(t) = 1900 + 52100.2\frac{t^{\beta}}{\Gamma(\beta+1)} - 27054.2\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$I_C(t) = 900 + 250\frac{t^{\beta}}{\Gamma(\beta+1)} + 1876.32\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$T_C(t) = 110 + 25.10\frac{t^{\beta}}{\Gamma(\beta+1)} + 1076.32\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$I_T(t) = 120 + 67.33\frac{t^{\beta}}{\Gamma(\beta+1)} - 176.32\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$I_{TC}(t) = 130 + 167.33\frac{t^{\beta}}{\Gamma(\beta+1)} - 16.42\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$R_C(t) = 70 - 167.33\frac{t^{\beta}}{\Gamma(\beta+1)} + 9.31\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$R_T(t) = 1000 + 67.330\frac{t^{\beta}}{\Gamma(\beta+1)} + 39.32\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$R_{TC}(t) = 70 - 167.33\frac{t^{\beta}}{\Gamma(\beta+1)} + 9.31\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$Z_C(t) = 120 + 67.33\frac{t^{\beta}}{\Gamma(\beta+1)} - 176.32\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

$$Z_T(t) = 130 + 167.33\frac{t^{\beta}}{\Gamma(\beta+1)} - 16.42\frac{t^{2\beta}}{\Gamma(2\beta+1)}\ldots$$

Numerical Simulation

The numerical simulation of our fractional order typhoid/cholera model as to compare with the classical order.

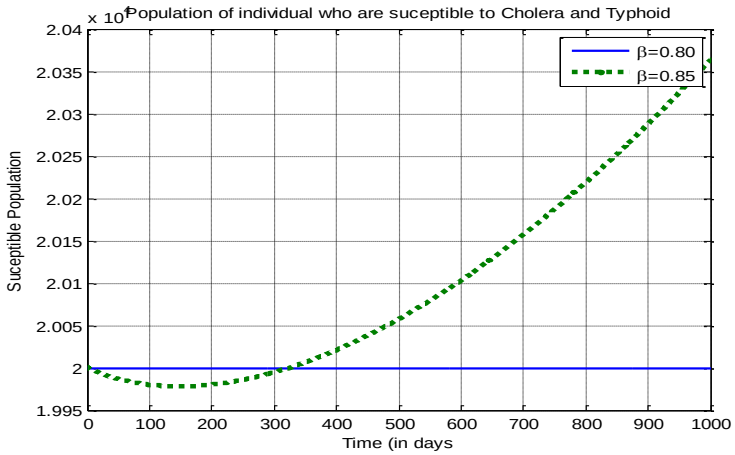

Figure 1: The above figure shows the population of individuals who are susceptible to cholera and typhoid disease with different values of β .

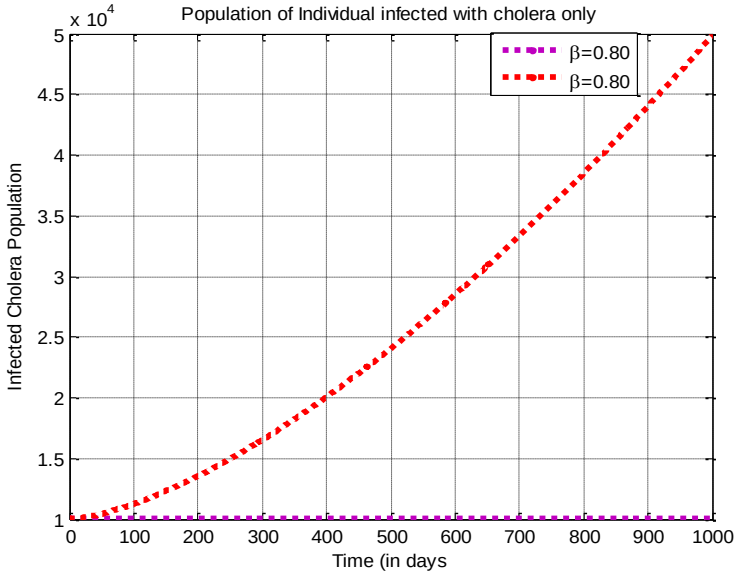

Figure 2: The above figure shows the population of individuals who are infected with cholera disease only with different values of β .

Figure 3: The above figure shows the population of individuals who are treated of cholera disease only with different values of β .

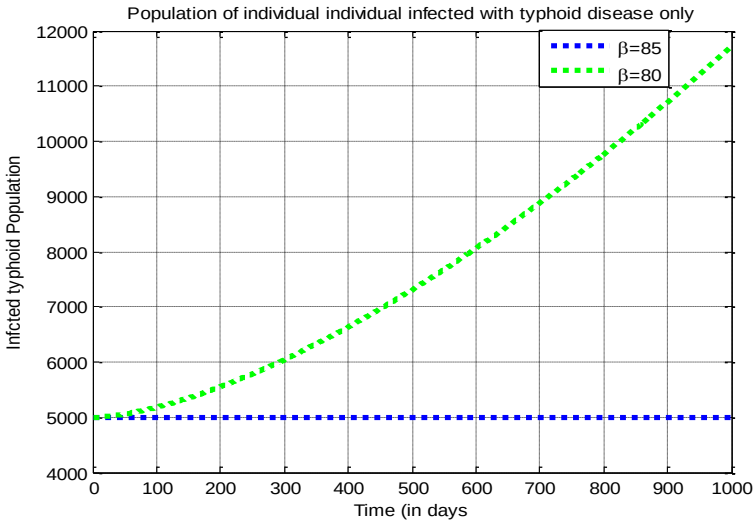

Figure 4: The above figure shows the population of individuals who are infected with typhoid disease only with different values of β .

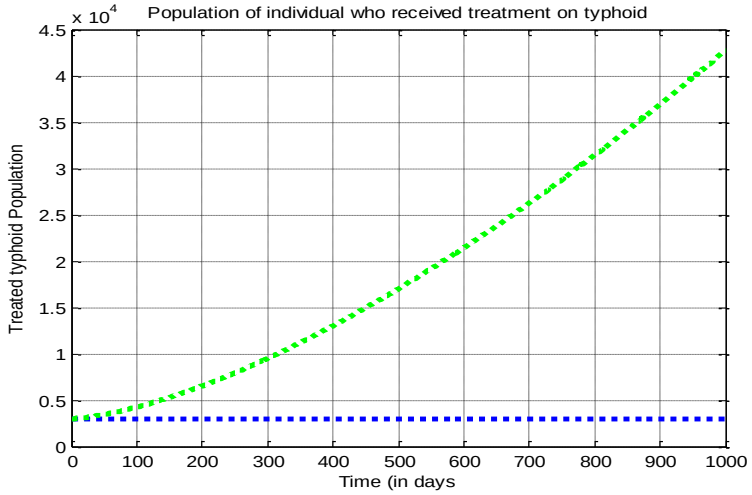

Figure 5: The above figure shows the population of individuals who receive treatment on typhoid disease only with different values of β .

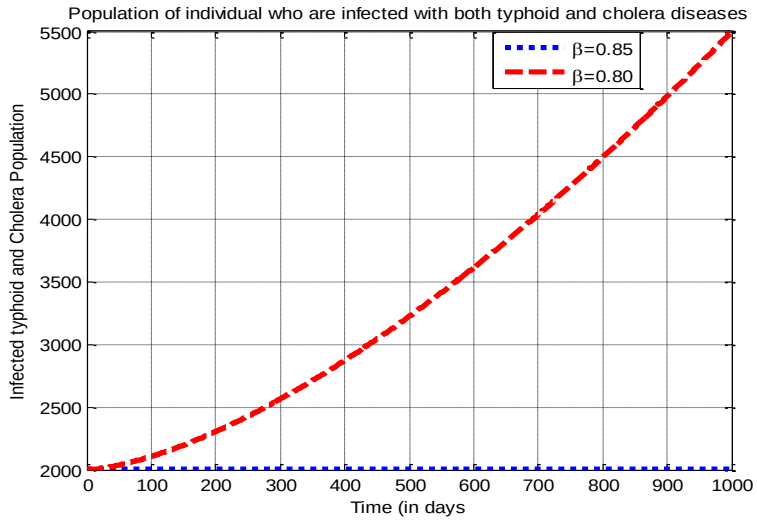

Figure 6: The above figure shows the population of individuals who are infected with both typhoid and cholera disease only with different values of β .

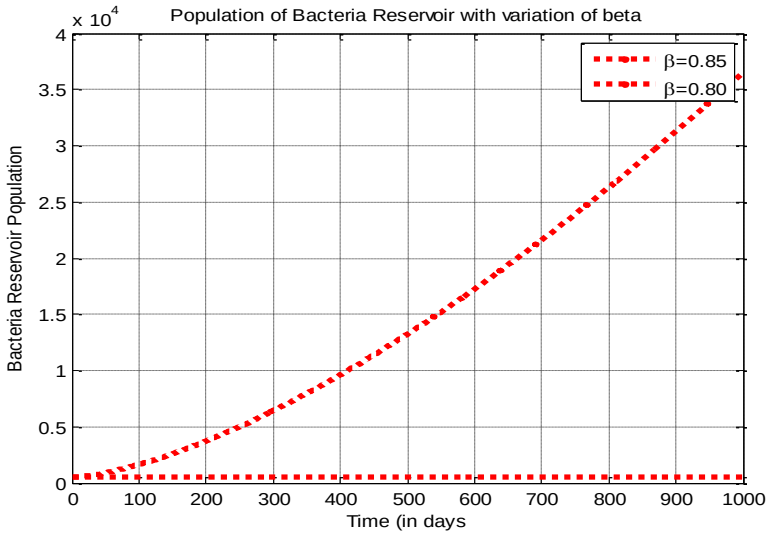

Figure 7: The above figure shows the population of bacteria reservoir with different values of β .

4.4 Results Model Variables/Parameters Values Used

Table 2: parameters and their estimated values used in the model

Variable/Parameters	Values	Sources
S	10000	[4]
I_T	8000	[4]
I_C	7000	Assume
T_T	5000	Assume
T_C	3000	Estimated
I_{ct}	1000	Assume
Z_t	500	Estimated
Z_c	10000	[4]
R_t	8000	[4]
R_c	7000	Assume
R_{tc}	5000	Assume
Λ	0.5	[4]
λ_T	0.5	[4]
λ_C	0.2	[4]
α	0.4	Estimated
δ_T	0.7	Assume
θ_2	0.003	Assume
υ	0.7	Assume
ω	0.05	Estimated
δ	0.0005	Estimated
μ	0.005	Estimated
θ_3	0.005	[4]
ε	0.5	[4]
ν	0.5	[4]

5.0 CONCLUSION

In this paper, we utilized a mathematical model to examine the dynamics of the co-infection of cholera and typhoid fever while incorporating treatment as a preventative measure. The schematic diagram of the model was drawn, and from it, the system of non-linear differential equations governing the dynamics of disease transmission were derived. The variables and parameters used to develop the model's equations were carefully interpreted. Then an explanation of the creation of the model equations was provided. The model was analyzed using Laplace Adomian Decomposition Method (LADM). The analysis was done by the help of MATLAB and the

results show us that the spread of typhoid and cholera depends on the contact rate between the infected population and susceptible population.

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