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Original Research Article

Mathematical Modeling of Typhoid Dynamics with Age Structure, Vaccination, and Treatment through Homotopy Perturbation Method

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ABSTRACT

This research presents a mathematical model for understanding the dynamics of typhoid fever, incorporating age structure, vaccination, and treatment effects. The model captures the complexities of typhoid transmission by considering different age groups, which exhibit varying susceptibility and contact rates. The homotopy perturbation method is applied to solve the system of differential equations governing the disease dynamics. The model explores the impact of vaccination programs, treatment interventions, and age-specific factors on reducing transmission rates and controlling outbreaks. Sensitivity analysis is performed to identify key parameters that influence disease progression, including the basic reproduction number. The results highlight the importance of targeting vaccination and treatment strategies toward specific age groups to enhance intervention efficacy. Numerical simulations demonstrate that increasing vaccination coverage and treatment rates significantly reduce the spread of typhoid fever. The findings provide valuable insights for optimizing public health policies aimed at managing typhoid fever, particularly in regions with limited resources. This approach offers a robust framework for assessing the effectiveness of control measures and improving disease management.

Keywords: Typhoid outbreaks, Age-structure, Basic reproduction number. Homotopy perturbation method, Sensitivity analysis.

INTRODUCTION

Mathematical modeling is a crucial tool for understanding the transmission dynamics of infectious diseases like typhoid fever, a bacterial infection caused by salmonella typhi typhoid fever is a public health concern in areas with poor sanitation and contaminated water, with symptoms such as high fever, abdominal pain, and gastrointestinal distress. If left untreated, it can lead to severe complications or death (Keshav et al, 2019). Recent advancements in modeling have incorporated factors such as age structure, vaccination, and treatment effects. The homotopy perturbation method has been particularly effective in solving differential equations that describe

disease dynamics (Bwalya et al, 2022). Including age structure allows models to more accurately capture the transmission patterns and the effectiveness of interventions like vaccination in different demographic groups (Dougan and Baker 2014). Vaccination and treatment are key components in controlling typhoid fever. Vaccination reduces the susceptible population, while treatment helps alleviate symptoms and prevent complications. Modeling these interventions helps researchers evaluate the effectiveness of control measures and optimize strategies (Amouch and Karan 2023). Children are especially vulnerable to severe illness and mortality from typhoid fever, and they can serve as reservoirs for transmission. Population dynamics, including factors like migration and urbanization, also affect the spread of the disease. Models that incorporate these factors provide insights into the long-term trends of disease transmission (Kolawole et al, 2022, Kuehn et al, 2022). Overall, mathematical models combining epidemiological data, age-specific factors, and interventions provide valuable insights into typhoid fever dynamics. These models aid in evidence-based decision-making, contributing to the effective control of typhoid fever and other infectious diseases (Liu, 2023). Efforts to control typhoid outbreaks typically involve a combination of preventive and responsive measures aimed at interrupting the transmission of the bacterium and treating infected individuals. Water and sanitation interventions play a crucial role in preventing cholera transmission and reducing the burden of the disease. Recent control measures for Typhoid include (Masuet and Atouguia, 2021, Melnikov et al, 2023). Improved Water and Sanitation Infrastructure: Investing in infrastructure for clean water supply, sanitation facilities, and proper waste management is fundamental in preventing typhoid outbreaks. Providing access to safe drinking water and promoting hygienic practices, such as hand washing and proper food handling, can significantly reduce the risk of Typhoid transmission. Vaccination Campaigns oral typhis vaccines (OCVs) have been developed and deployed in Typhoid-endemic areas as part of targeted vaccination campaigns. OCVs can provide short to medium-term protection against Typhoid and are often used in outbreak response efforts and in areas with a high risk of Typhoid transmission (Muchmore et al., 2020, Muscat et al., 2022). Surveillance and early detection: Strengthening surveillance systems for Typhoid and enhancing early detection and response mechanisms are critical for containing outbreaks and preventing the spread of the disease. Rapid diagnostic tests, along with effective reporting and monitoring systems, enable health authorities to identify and respond to Typhoid cases promptly (Muthuirulandisethuvel et al., 2020). Health education and community engagement in promoting awareness about typhoid transmission, symptoms, and preventive measures through health education campaigns can empower communities to take proactive steps in preventing the disease. Engaging with community leaders and stakeholders fosters community participation and ownership of typhoid control efforts, leading to sustainable outcomes. Treatment and case management. Providing prompt and appropriate treatment for typhoid cases is essential for reducing morbidity and mortality associated with the disease, along with the administration of antibiotics in severe cases. Ensuring access to healthcare facilities equipped to manage Typhoid cases helps prevent complications and reduce the spread of the disease (Lawal et al., 2023). Integrated Approach: Implementing a multi-sectorial and integrated approach to Typhoid control, involving collaboration between health authorities, water and sanitation agencies, non-governmental organizations, and other stakeholders, is essential for addressing the complex determinants of cholera transmission. Coordinated efforts across various sectors can maximize the impact of control measures and contribute to sustainable Typhoid prevention and control strategies (Kolawole et al.2023).

Formulation of the model

We develop a model with Bacteria population, NB denoted by B(t) and human population, NH. The populations are subdivided into different epidemiological classes: Susceptible of the children (SC), Susceptible of the adult (SA), vaccination (V), Infected (I), Treatment (T), Recovered (R), and bacteria subclasses. The models assumes that human population will be recruited to susceptible compartment of the children at the rate Λ_C, and susceptible compartment of the adult at the rate Λ_A, and susceptible individuals are infected at the rate of $\frac{\alpha B}{k+B}$ where α is the rate of salmonella Typhi injections in foods and drinks $\frac{B}{k+B}$ is the probability of probability of individuals in consuming foods or drinks contaminated with typhoid causing bacteria. All human population have their natural death at the rate μ, and infected individuals die from typhoid at the rate δ. The treatment rate of infected individual infant is represented by τ, excretion of Salmonella Typhi bacteria by the infected children and adult to the environment at the rate η and salmonella Typhi will die to the environment at the rate ν, the parameter ω represent hygiene rate, ψ_1, ψ_2 denote vaccination rate of children and adult, while ρ_1, ρ_2 represents waning rate of immunity. The below diagram in figure (1) represent the model flow chart.

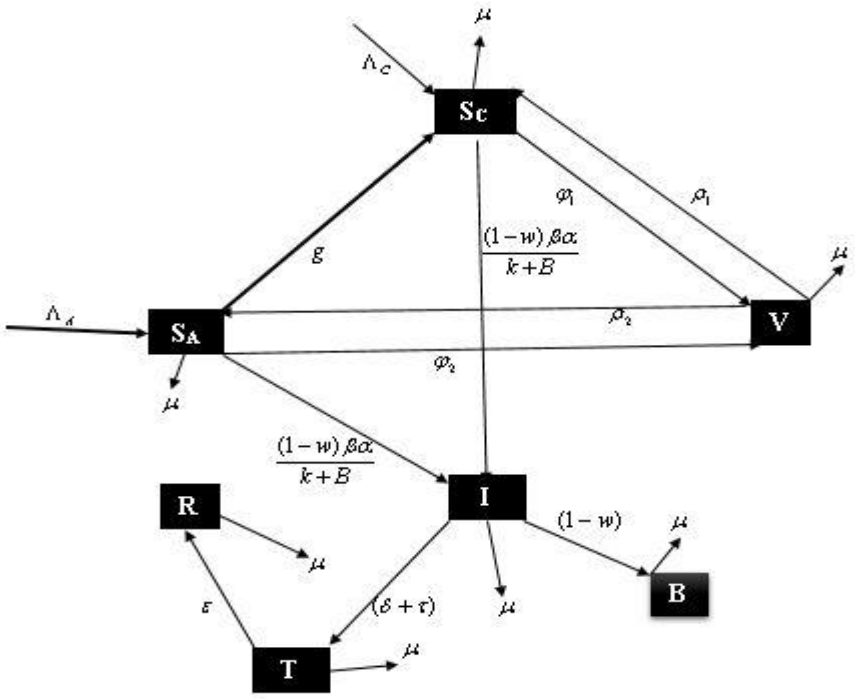


Figure 1: Schematic flow of the model formulation

Table 1: Description of parameters

Parameter	Parameter descriptions
SC(t)	The number of susceptible individual children
SA(t)	The number of susceptible adult individual
V(t)	The number of vaccinated individual
I(t)	The number of infected individual
T(t)	The number of Treated individual
R(t)	The number of Recovered individual
B(t)	The number of Bacteria in the population

parameter	Description	value	references
W	Hygiene rate	0.3	Kuehn et al.(2022)
δ	Disease induced death rate	0.015	Kolawole et al.(2023)
ρ_1, ρ_2	Waning rate of immunity	0.018682	Lawal et al.(2023)
μ	Natural death rate	1/60,000	Keshav et al.(2019)
η	Excretion rate of salmonella Typhoid	0.003	Kolawole et al.(2022)
τ	Treatment rate of individuals	0.001744	Muchmore et al.(2020)
ε	Recovery rate of treatment individuals	0.0033142	Bwalya et al.(2022)
ψ_1, ψ_2	Vaccination rate of Children and Adult	0.0002	Komarovkaya et al.(2023)
G	Rate at which children become adult	0.01	Assumed
K	Concentration of salmonella bacteria in foods and water.	50,000	Assumed
α	The rate of salmonella Typhi injections in foods and drinks	10	Dougan and Baker, 2014
Λ_C, Λ_A	Recruitment rate of children and adult	150,000.1854	Muscat et al.(2022)
ν	The rate at which salmonella Typhi will die to the environment	0.001	Assumed

A compartmental based model for analysing the treatment of typhoid fever capturing age structure and vaccine. The govern model is given by the system of non-linear ordinary differential equation (1) below.

Formulated model

The equation (1) of Halson et, al (2021) was extended by incorporating the following novelty, age structure, Vaccine and Treatment. The model equation is therefore given in equation (1) inclusive of the above parameters. Hence we have

$$\frac{dS_c}{dt} = \Lambda_c - (1-w)\lambda_c B[S_c + S_A] - (\mu + \psi_1)S_c + \rho_1 \nu - gS_c$$

$$\frac{dS_A}{dt} = \Lambda_A - (1-w)\lambda_A B[S_c + S_A] - (\mu + \psi_2)S_A + \rho_2 \nu + gS_c$$

$$\frac{dV}{dt} = \psi_1 S_c + S_A \psi_2 - (\rho_1 + \rho_2)\nu - \mu\nu$$

$$\frac{dI}{dt} = (1-w)\lambda_c B[S_c + S_A] + (1-w)\lambda_A B[S_c + S_A] - (\mu + \tau + \delta)I \quad (1)$$

$$\frac{dT}{dt} = \tau I - \varepsilon T - \mu T$$

$$\frac{dR}{dt} = \varepsilon T - \mu R$$

$$\frac{dB}{dt} = (1-\omega)\eta I - \nu B$$

$$\lambda = \frac{B\alpha}{K+B} \text{ Where } S_c(0) = S_a(0) = s_0, \ V(0) = v_0, \ I(0) = i_0, \ T(0) = t_0, \ R(0) = r_0,$$
$$B(0) = b_0 \geq 0$$

ANALYSIS OF THE MODEL

Existence and Uniqueness of Model Solution

Feasible Region: The analysis of the feasible was done, in which the model solution is bounded. The total human population (NH) considered in above model are

$$N = S_A + S_c + V + I + R$$

$$\frac{dN}{dt} = \Lambda_c - \mu S_C - \psi_1 S_C + \rho_1 \nu + \rho_2 \nu + \Lambda_A - \mu S_A - \psi_2 S_A + \psi_1 S_C + \psi_2 S_A - \rho_1 \nu$$
$$- \rho_2 \nu - \mu\nu - \mu I - \tau I + \tau I - \mu T - \mu R \quad (2)$$

By method of integrating factor

$$N \cdot IF = \int IF \cdot Q dt$$

$$N(t) = \frac{(\Lambda_C + \Lambda_A)}{\mu} + \ell^{-\mu t} C \quad \text{at } t=0$$

$$N(0) = \frac{\Lambda_C + \Lambda_A}{\mu} + C, \quad C = N(0) - \frac{\Lambda_C + \Lambda_A}{\mu}$$

$$N(t) \le \left[\frac{\Lambda_C + \Lambda_A}{\mu} + (N(0) - \frac{\Lambda_C + \Lambda_A}{\mu})\ell^{-\mu t} \right] \lim_{t\to\infty} N(t) \le \lim_{t\to\infty} \left[\frac{\Lambda_C + \Lambda_A}{\mu} + (N(0) - \frac{\Lambda_C + \Lambda_A}{\mu})\ell^{-\mu t} \right]$$

$$N_H(t) \le \frac{\Lambda_C + \Lambda_A}{\mu} \qquad (3)$$

Also the bacteria population is. Thus, the feasible solution of the system equation of the model enters and remains in the region.

$$\Gamma_H = (S_C, S_A, V, I, T, R) \in \Re_+^{\,6}; S_C, S_A. > 0, V, I, T, R \ge 0; N_H \le \frac{\Lambda_C + \Lambda_A}{\mu}$$

$$\Gamma_B = B \in \Re_+ : B \ge 0 : N_B \le \frac{(\Lambda_C + \Lambda_A)(1-w)\eta}{\mu\nu} \qquad (4)$$

is positive invariant. Therefore, the model is well posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the basic model in Γ.

Positivity and boundedness of model solution

The initial condition of the model was assumed to be nonnegative and now, we also proof that the solution of the model is positive.

Theorem 1

$$let, \Gamma = \left\{ (S_C, S_A, \nu, I, T, R, B) \in R^7; S_{C0} \ge 0, S_{A0} \ge 0, \nu_0 \ge 0, I_0 \ge 0, T_0 \ge 0, R_0 \ge 0, B_0 \ge 0 \right\}$$

Then the solution of $\{S_C, S_A, \nu, I, T, R, B\}$ are positive for t ≥ 0 in the region of $\Gamma \in [0,1] \in \Re_7^+$

Proof

From the system of differential equation, we solve the equation one after the other.

First Equation;

$$\frac{dS_c}{dt} = \Lambda_c - (1-w)\lambda_c B[S_c + S_A] - (\mu + \psi_1)S_c + \rho_1 \nu - gS_c \qquad (5)$$

$$\frac{dS_C}{dt} \ge -(\mu + \psi_1 + g)S_c(t)$$

$$\frac{dS_C}{S_c(t)} \ge -(\mu + \psi_1 + g)dt$$

Then solving using method of integrating factor and applying condition, we obtained

$$S_C(t) \ge S_{C0} \cdot \ell^{-(\mu+\psi_1+g)t} \ge 0 \qquad (6)$$

Then solving the second equation,

$$\frac{dS_A}{dt} = \Lambda_A - (1-w)\lambda_A B[S_C + S_A] - (\mu + \psi_2)S_A + \rho_2 \nu + gS_C$$

$$\frac{dS_A}{S_A(t)} \ge -(\mu + \psi_2)dt$$

Similarly using integrating factor and applying conditions, it gives

$$S_A(t)\cdot \ell^{(\mu+\psi_2)t} \geq \int \ell^{(\mu+\psi_2)t} dt$$

$$\Rightarrow \quad S_A(t) \geq S_{A_0}(t)\cdot \ell^{(\mu+\psi_2)t} \geq 0 \tag{7}$$

Also we took the third equation of

$$\frac{dV}{dt} = \psi_1 S_C + \psi_2 S_A - (\rho_1+\rho_2)\nu - \mu\nu$$

$$\frac{dV}{dt} \geq -(\rho_1+\rho_2+\mu)\nu$$

$$\frac{dV}{\nu(t)} \geq -(\rho_1+\rho_2+\mu)dt$$

Also using integrating factor and applying the condition as discussed above it is obtained that,

$$V(t)\cdot \ell^{(\rho_1+\rho_2+\mu)t} \geq \int \ell^{(\rho_1+\rho_2+\mu)t}\cdot 0\, dt$$

$$V(t)\cdot \ell^{(\rho_1+\rho_2+\mu)t} \geq C+0$$

$$V(t) \geq V_0 \cdot \ell^{(\rho_1+\rho_2+\mu)t} \geq 0 \tag{8}$$

Taking the fourth equation from (1) to obtain its positivity, it is obtained that

$$\frac{dI}{dt} = (1-w)\lambda_C B(S_C+S_A) + (1-w)\lambda_A B(S_C+S_A) - (\mu+\tau+\delta)I$$

$$\frac{dI}{I(t)} \geq -(\mu+\tau+\delta)dt$$

$$I(t)\cdot \ell^{(\mu+\tau+\delta)t} \geq \int \ell^{(\mu+\tau+\delta)t} dt$$

$$I(t) \geq I_0 \cdot I(t)\cdot \ell^{(\mu+\tau+\delta)t} \geq 0 \tag{9}$$

This completes the proof of the theorem, and it shows that the solution of the model is positive.

Existence of disease free equilibrium state

To analyse the disease free-equilibrium. Let equation (1) be subjected to zero, evaluating it at $S'_C = S'_A = V' = I' = T' = R' = B' = 0$ and solving for the non-infected and non-carrier state variables.

$$S_{A_0} = \frac{\Lambda_A + \rho_2 V + gS_C}{(\mu+\psi_2)}, \quad S_{A_0} = \Lambda_A + \frac{\rho_2 V + g\Lambda_C}{(\mu+\psi_2)},$$

$$S_{A_0} = \frac{\Lambda_A(\mu+\psi_1+g) + \rho_2 V + g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}, \quad \psi_1 S_C + \psi_2 S_A - (\rho_1+\rho_2)V - \mu V = 0,$$

$$\psi_1 S_C + \psi_2 S_A = (\rho_1+\rho_2+\mu)V \quad V = \frac{\psi_1 S_C + \psi_2 S_A}{(\rho_1+\rho_2+\mu)} \tag{10}$$

$$S_c, S_A, V_0, I_0, T_0, R_0, B_0 = \left(\frac{\Lambda_C + \rho_1 V}{(\mu + \psi_1 + g)}, \frac{\Lambda_A(\mu + \psi_1 + g) + \rho_2 V + g\Lambda_C}{((\mu + \psi_1 + g)(\mu + \psi_2)}, \frac{\psi_1 S_C + \psi_2 S_A}{(\rho_1 + \rho_2 + \mu)}, 0,0,0,0 \right)$$

Endemic equilibrium point

We represent our endemic equilibrium point as $E^*(S_C^*, S_A^*, V^*, I^*, T^*, R^*, B^*)$,

Theorem 2

There exists a unique equilibrium of system of when $R_0 > 1$

Proof:

To establish this theorem, equate $(S_c, S_A, V, I, T, R, B) = 0$ with ($(S_C^*, S_A^*, V^*, I^*, T^*, R^*, B^*)$ representing the respectively compartments of the model equations to obtain in equation (11) below

$$\begin{aligned}
0 &= \Lambda_C - (1-w)\lambda_C B^*(S_C^* + S_A^*) - (\mu + \psi_1)S_C^* + \rho_1 V^* - gS_C^* \\
0 &= \Lambda_A - (1-w)\lambda_A B^*(S_C^* + S_A^*) - (\mu + \psi_2)S_A^* + \rho_2 V^* + gS_C^* \\
0 &= \psi_1 S_C^* + \psi_2 S_A^* - (\rho_1 + \rho_2)V^* - \mu V^* \\
0 &= (1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*) - (\mu + \tau + \delta)I^* \\
0 &= \tau I^* - \varepsilon T^* - \mu T^* \\
0 &= \varepsilon T^* - \mu R^* \\
0 &= (1-\omega)\eta I^* - \nu B^*
\end{aligned} \tag{11}$$

From the last equation of system (11) we have

$$B^* = \frac{(1-w)\eta(1-\omega)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*)}{\nu(\mu + \tau + \delta)}$$

$$I^* = \frac{(1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*)}{(\mu + \tau + \delta)}$$

$$R^* = \frac{\varepsilon\tau(1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*)}{\mu(\varepsilon + \mu)(\mu + \tau + \delta)}, \quad V^* = \frac{\psi_1 S_C^* + \psi_2 S_A^*}{(\rho_1 + \rho_2 + \mu)} \tag{12}$$

$$T^* = \frac{\tau(1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*)}{(\varepsilon + \mu)(\mu + \tau + \delta)}$$

$$S_C^* = \frac{\Lambda_C - (1-w)\lambda_C B^* S_A^* + \rho_1 V^*}{\{(1-w)\lambda_C B^* + (\mu + \psi_1) + g\}}$$

$$S_A^* = \frac{\Lambda_A - \{(1-w)\lambda_A B^* + g\}S_C^* + \rho_2 V^*}{\{(1-w)\lambda_A B^* + (\mu + \psi_2)\}}$$

Basic reproduction number R_*

There are two diseases state but only one way to create new infections. Hence exposed and infected compartments of the model are involved in the calculation of R_*.

Where $R_* = \rho(G - \lambda I)$

$$F_i = \left[\frac{\partial f_i(x_0)}{\partial x_j}\right] \quad V_i = \left[\frac{\partial v_i(x_0)}{\partial x_j}\right] \tag{13}$$

F is the new infections, while the V are transfers of infections from one compartment to another.

Where $V_1 = -(\mu+\tau+\delta)I$, $V_2 = \tau I - (\varepsilon+\mu)T$, $V_3 = (1-\omega)\eta I - \nu B$

$$V = \begin{pmatrix} (\mu+\tau+\delta) & 0 & 0 \\ -\tau & (\varepsilon+\mu) & 00 \\ -(1-\omega)\eta & 0 & \nu \end{pmatrix}$$

$$V^{-1} = \frac{1}{\nu(\mu+\tau+\delta)(\varepsilon+\mu)} \cdot \begin{pmatrix} \nu(\varepsilon+\mu) & 0 & 0 \\ \nu\tau & \nu(\mu+\tau+\delta) & 0 \\ -(\varepsilon+\mu)(1-\omega)\eta & 0 & (\varepsilon+\mu)(\mu+\tau+\delta) \end{pmatrix}$$

$$R_* = \frac{2(1-\omega)^2\eta\lambda_C(\mu+\psi_2)(\Lambda_C+\rho_1 V)+\Lambda_A(\mu+\psi_1+g)+\rho_2 V+g\Lambda_C)}{\nu(\mu+\tau+\delta)(\mu+\psi_1+g)(\mu+\tau+\delta)} \tag{14}$$

Local stability of disease free equilibrium

The disease-free equilibrium is locally asymptotically stable if the basic reproduction number. $R_* < 1$.

The characteristic polynomial of the Jacobian matrix of disease-free equilibrium is given by $|J_E - \lambda_i I| = 0$ where is the Eigen value and I is the identity matrix. The Stability criterion of Disease Free Equilibrium, the general Jacobian matrix has been calculated as obtained. The local stability of the disease free equilibrium of the Jacobian matrix of the system of (1), whereas λ_i are the eigen-values, I the identity matrix and $i = 1,2,3,\ldots 7$ respectively. At disease free equilibrium of equation (1), respectively the Jacobian matrix is obtained as;

$$J_e = \begin{pmatrix} -(\mu+\psi_1+g) & 0 & \rho_1 & 0 & 0 & 0 & 0 \\ g & -(\mu+\psi_2) & 0 & 0 & 0 & 0 & 0 \\ \psi_1 & \psi_2 & -(\rho_1+\rho_2+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\mu+\tau+\delta) & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 \\ 0 & 0 & 0 & (1-\omega)\eta & 0 & 0 & -\nu \end{pmatrix}$$

As discussed above that $\left|J_{E_0} - \lambda I\right| = 0$, this results to,

$$\begin{vmatrix} a-\lambda & 0 & \rho_1 & 0 & 0 & 0 & 0 \\ g & b-\lambda & 0 & 0 & 0 & 0 & 0 \\ \psi_1 & \psi_2 & c-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & d-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & e-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu-\lambda & 0 \\ 0 & 0 & 0 & (1-\omega)\eta & 0 & 0 & -\nu-\lambda \end{vmatrix} = 0 \quad (15)$$

By using Atangana Belamus's invariance principle by lower triangular matrix. We obtain, therefore, $a-(\mu+\psi_1+g)$, $b-=(\mu+\psi_2)$, $c=-(\rho_1+\rho_2+\mu)$, $d=-(\mu+\tau+\delta)$, $e=-(\varepsilon+\mu)$,

$$\lambda_1=-(\mu+\psi_1+g)<0, \lambda_2=-(\mu+\psi_2)<0, \lambda_3=-(\rho_1+\rho_2+\mu)<0, \lambda_4=-(\mu+\tau+\delta)<0$$
$$\lambda_5=-(\varepsilon+\mu)<0, \lambda_6=-\mu<0, \lambda_7=-\nu \quad (16)$$

Hence, since all the eigen values are all negative, hence the disease free equilibrium is locally asymptotically stable.

Local stability of endemic equilibrium

The Endemic equilibrium of the proposed Epidemic model is locally asymptotically stable if $R_*<1$ and unstable otherwise if $R_*>1$

We linearized each of the compartments by let

$$S_C=a+S_C^*, S_A=b+S_A^*, V=c+V^*, I=e+I^*, T=x+T^*, R=y+R^*, B=z+B^* \quad (17)$$

From the system of equation (17), we obtain

$$\frac{da}{dt}=\Lambda_C-[(1-w)\lambda_C za]-[(1-\omega)\lambda_C zb]-(\mu+\psi_1)a+\rho_1 c-ga+higherorder+non+linearterms$$
$$\frac{db}{dt}=\Lambda_A-[(1-w)\lambda_A za]-[(1-\omega)\lambda_A zb]-(\mu+\psi_2)b+\rho_2 c-ga+higherorder+non+linearterms$$
$$\frac{dc}{dt}=\psi_1 a+\psi_2 b-(\rho_1+\rho_2)c-\mu c+higherorder+non+linearterms \quad (18)$$
$$\frac{de}{dt}=[(1-w)\lambda_C za]+[(1-\omega)\lambda_C zb]+[(1-w)\lambda_A zb]-(\mu+\tau+\delta)e+higherorder+non+linearterms$$
$$\frac{dx}{dt}=\tau e-(\varepsilon+\mu)x+higherorder+non+linearterms$$
$$\frac{dy}{dt}=\varepsilon x-\mu y+higherorder+non+inearterms$$
$$\frac{dz}{dt}=(1-\omega)\eta e-\nu z+higherorder+non+inearterms$$

The Jacobian matrix of the system of were obtained, where $\left|J_{E_i}-\lambda_i I\right|=0$, from respective values of their endemic equilibrium points as obtained from (12)

$$\begin{vmatrix} A & -[(1-w)\Lambda_C z] & \rho_1 & 0 & 0 & 0 & -(1-w)\Lambda_C(a+b) \\ -[(1-w)\Lambda_A zb-g] & B & \rho_2 & 0 & 0 & 0 & -(1-w)\Lambda_a(a+b) \\ \psi_1 & \psi_2 & C & 0 & 0 & 0 & 0 \\ -(1-w)\Lambda_c z+(1-w)\Lambda_a z & 0 & 0 & D & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & E & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 \\ 0 & 0 & 0 & (1-\omega)\eta & 0 & 0 & -v \end{vmatrix} = 0$$

Where it is obtained that $A=-[(1-w)\Lambda_C z+(\mu+\psi_1+g)]$, $B=-[(1-w)\Lambda_A z+\mu+\psi_2]$, $C=-(\rho_1+\rho_2+\mu)$, $D=-(\tau+\delta+\mu)$, $E=-(\varepsilon+\mu)$

(19)

$$\begin{vmatrix} A-\lambda & -[(1-w)\Lambda_C z] & \rho_1 & 0 & 0 & 0 & -(1-w)\Lambda_C(a+b) \\ -[(1-w)\Lambda_A zb-g] & B-\lambda & 0 & 0 & 0 & 0 & -(1-w)\Lambda_a(a+b) \\ \psi_1 & \psi_2 & C-\lambda & 0 & 0 & 0 & 0 \\ -(1-w)\Lambda_c z+(1-w)\Lambda_a z & 0 & 0 & D-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & E-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu-\lambda & 0 \\ 0 & 0 & 0 & (1-\omega)\eta & 0 & 0 & -v-\lambda \end{vmatrix} = 0$$

$(A-\lambda^*)(B-\lambda^*)(C-\lambda^*)(D-\lambda^*)(E-\lambda^*)(F-\lambda^*)(G-\lambda^*)=0$ .The Characteristics polynomial is given by

$$\lambda_*^{\,7}+a_1\lambda_*^{\,6}+a_2\lambda_*^{\,5}+a_3\lambda_*^{\,4}+a_4\lambda_*^{\,3}+a_5\lambda_*^{\,2}+a_6\lambda_*^{\,1}+a_7 \quad (20)$$

Applying the Routh-Hurwitz criterion, it can be seen that all the eigen values of the characteristics equation above have negative real part. Then the endemic equilibrium is locally asymptotically stable.

Global stability of disease free equilibrium

At equilibrium where C_1, C_2, C_3 are constants, the global stability for the disease free equilibrium is stable if $R_* < 1$ unless otherwise. Consider the Lyapunov approach on the disease class deduced as;

$$V(S_1,S_2,I_1,I_2,R,P,t)=C_1I_1+C_2I_2+C_3I_3$$

$$\frac{dV}{dt}=C_1I_1^{\,1}+C_2I_2^{\,1}+C_3I_3 \; as \; I_1=I_1 \Rightarrow I_2=I_2 \; and \; I_3=P \quad (21)$$

Using Lyapunov function approach to proceed for the result for global asymptotic stability of the proposed model at disease free equilibrium state. Let V (t, SC, SA, V, I, Q, R, B), on the disease state, the derivatives of the respective state variables is deduced as:

V (t, I, T, B) = $C_1 I_1 + C_2 I_2 + C_3 I_3$, as

$$\frac{dV}{dt} = C_1 I_1^{'} + C_2 I_2^{'} + C_3 I_3^{'}$$, where C1 < C2 < C3

$$\frac{dV}{dt} = C_2 \tau I_1 + C_3(1-w)\eta I_1 - C_1(\mu+\tau+\delta)I_1 - C_2(\varepsilon+\mu)I_2 + C_1[(1-w)\lambda_C I_3 S_C] + C_1[(1-w)\lambda_C I_3 S_A] + C_1[(1-w)\lambda_A I_3 S_C] + C_1[(1-w)\lambda_A I_3 S_A]$$

$$\frac{dV}{dt} \le [C_2\tau + C_3(1-w)\eta - C_1(\mu+\tau+\delta)]I_1 - C_2(\varepsilon+\mu)I_2 + [C_1(1-w)\lambda_C S_C + C_1(1-w)\lambda_C S_A + C_1(1-w)\lambda_A S_C] + C_1(1-w)\lambda_A S_A]I_3 \quad (22)$$

$$\frac{dV}{dt} \le [C_2\tau - C_1(\mu+\tau+\delta)]I_1 - C_2(\varepsilon+\mu)I_2 + C_3\begin{pmatrix}[C_1(1-w)\lambda_C \frac{\Lambda_C + \rho_1 V}{(\mu+\psi_1+g)}] + [C_1(1-w)\lambda_C \frac{\Lambda_A(\mu+\psi_1+g)+\rho_2 V + g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}] + \\ [C_1(1-w)\lambda_A \frac{\Lambda_C}{(\mu+\psi_1+g)}] + [C_1(1-w)\lambda_A \frac{\Lambda_A(\mu+\psi_1+g)+g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}]\end{pmatrix} I_3$$

Recall that $C_1 < C_2 < C_3,\ C_3 \ge 0 C_1 = \frac{1}{(\mu+\tau+\delta)}$ at equilibrium

$$\frac{dV}{dt} \le \left(\frac{(1-w)\lambda_C(\mu+\psi_2)(\Lambda_C+\rho_1 V) + (1-w)\lambda_C[\Lambda_A(\mu+\psi_1+g)+\rho_2 V + g\Lambda_C] + (1-w)\lambda_A(\mu+\psi_2) + (\Lambda_C+\rho_1 V)(1-w)\lambda_A[\Lambda_A(\mu+\psi_1+g)] + (\rho_2 V + g\Lambda_C)}{(\mu+\psi_1+g)(\mu+\psi_2)(\mu+\tau+\delta)} - 1\right) \quad (23)$$

It is important to note that V=0, only when I=0, the substitution of I=0 into the model system of equation (23) shows that

$S_{C_0} = \frac{\Lambda_C + \rho_1 \nu}{(\mu+\psi_1+g)}$, $S_{A_0} = \frac{\Lambda_A(\mu+\psi_1+g)+\rho_2\nu+g\Lambda_C}{((\mu+\psi_1+g)(\mu+\psi_2)}$ at t→∞, w<1 and based on Lasalle's invariance principle, Hence $E_0 = 0$ is Globally Asymptotically stable whenever $R_* < 1$

Global stability analysis for endemic equilibrium point

By employing Dulac criterion to proceed for the result for global asymptotic stability of modified model.

Let X= (SC, SA, V, I, T, R, B), where $G(X) = \frac{1}{S_C S_A}$ (24)

$$G(\frac{dS_C}{dt}) = \frac{1}{S_C S_A}[\Lambda_C - (1-w)\lambda_C B(S_C + S_A) - (\mu + \psi_1)S_C + \rho_1 \nu - gS_C$$

$$= \frac{\Lambda_C}{S_C S_A} - \frac{(1-w)\lambda_C B}{S_A} - \frac{(1-w)\lambda_C B}{S_C} - \frac{(\mu+\psi_1)}{S_C S_A} + \frac{\rho_1 \nu}{S_C S_A} - \frac{g}{S_A}$$

$$G(\frac{dS_A}{dt}) = \frac{1}{S_C S_A}[\Lambda_A - (1-w)\lambda_A B(S_C + S_A) - (\mu + \psi_2)S_A + \rho_2 \nu + gS_C$$

$$= \frac{\Lambda_A}{S_C S_A} - \frac{(1-w)\lambda_A B}{S_A} - \frac{(1-w)\lambda_A B}{S_C} - \frac{(\mu+\psi_2)}{S_C} + \frac{\rho_2 \nu}{S_A S_C} + \frac{g}{S_A}$$

$$G(\frac{dV}{dt}) = \frac{1}{S_C S_A}[\psi_1 S_C + \psi_2 S_A - (\rho_1 + \rho_2)\nu - \mu\nu] = \frac{\psi_1}{S_C S_A} + \frac{\psi_2}{S_C S_A} - \frac{(\rho_1+\rho_2)\nu}{S_C S_A} - \frac{\mu\nu}{S_C S_A}$$

$$G(\frac{dI}{dt}) = \frac{1}{S_C S_A}\left[(1-w)\lambda_C B(S_C + S_A) + (1-w)\lambda_A B(S_C + S_A) - (\mu + \tau + \delta)I\right]$$

$$= \frac{(1-w)\lambda_C B}{S_C S_A} + \frac{(1-w)\lambda_C B}{S_A} + \frac{(1-w)\lambda_A B}{S_A S_C} + \frac{(1-w)\lambda_A B}{S_C S_A} - \frac{(\mu+\tau+\delta)}{S_C S_A} \quad (25)$$

$$G(\frac{dT}{dt}) = \frac{1}{S_C S_A}[\tau I - \varepsilon I - \mu T] = \frac{\tau I}{S_C S_A} - \frac{\varepsilon I}{S_C S_A} - \frac{\mu T}{S_C S_A}$$

$$G(\frac{dR}{dt}) = \frac{1}{S_C S_A}[\varepsilon T - \mu R] = \frac{\varepsilon T}{S_C S_A} - \frac{\mu R}{S_C S_A}$$

$$G(\frac{dB}{dt}) = \frac{1}{S_C S_A}[(1-\omega)\eta I - \nu B] = \frac{(1-\omega)\eta I}{S_C S_A} - \frac{\nu B}{S_C S_A}$$

Then we obtain

$$\frac{d}{dt}(GX) = \frac{\partial}{\partial S_C}\left(G\frac{dS_C}{dt}\right) + \frac{\partial}{\partial S_A}\left(G\frac{dS_A}{dt}\right) + \frac{\partial}{\partial V}\left(G\frac{dV}{dt}\right) + \frac{\partial}{\partial I}\left(G\frac{dI}{dt}\right) + \frac{\partial}{\partial T}\left(G\frac{dT}{dt}\right)$$

$$+ \frac{\partial}{\partial R}\left(G\frac{dR}{dt}\right) + \frac{\partial}{\partial B}\left(G\frac{dB}{dt}\right)$$

$$\frac{d}{dt}(GX) = \frac{\partial}{\partial S_C}\left(\frac{\Lambda_C}{S_C S_A} - \frac{(1-w)\lambda_C B}{S_A} - \frac{(1-w)\lambda_C B}{S_C} - \frac{(\mu+\psi_1)}{S_A} + \frac{\rho_1\nu}{S_C S_A} - \frac{g}{S_C}\right) +$$

$$\frac{\partial}{\partial S_A}\left(\frac{\Lambda_A}{S_C S_A} - \frac{(1-w)\lambda_A B}{S_A} - \frac{(1-w)\lambda_A B}{S_C} - \frac{(\mu+\psi_2)}{S_C} + \frac{\rho_2\nu}{S_A S_C} + \frac{g}{S_C}\right) +$$

$$\frac{\partial}{\partial V}\left(\frac{\psi_1}{S_A S_C} + \frac{\psi_2}{S_C S_A} - \frac{(\rho_1+\rho_2)\nu}{S_C S_A} - \frac{\mu\nu}{S_C S_A}\right) +$$

$$\frac{\partial}{\partial I}\left(\frac{(1-w)\lambda_C B}{S_A} + \frac{(1-w)\lambda_C B}{S_C} + \frac{(1-w)\lambda_A B}{S_A} + \frac{(1-w)\lambda_A B}{S_C} - \frac{(\mu+\tau+\delta)}{S_C S_A}\right) +$$

$$\frac{\partial}{\partial T}\left(\frac{\tau I}{S_C S_A} - \frac{\varepsilon I}{S_C S_A} - \frac{\mu T}{S_C S_A}\right) + \frac{\partial}{\partial R}\left(\frac{\varepsilon T}{S_C S_A} - \frac{\mu R}{S_C S_A}\right) +$$

$$\frac{\partial}{\partial B}\left(\frac{(1-w)\eta}{S_C S_A} - \frac{\nu B}{S_C S_A}\right) \quad (26)$$

Then we obtain

Now we consider the parameter with and without state variables i.e those parameter without are negative invariant as those with states variables are neglected not relevance to $S_C S_A$

$$\frac{d}{dt}(GX)=\frac{\partial}{\partial S_C}\left(-\frac{\Lambda_C}{S_C S_A}-\frac{(\mu+\psi_1)}{S_A}-\frac{g}{S_A}\right)+\frac{\partial}{\partial S_A}\left(-\frac{(\mu+\psi_2)}{S_C S_A}\right)+$$
$$\frac{\partial}{\partial V}\left(-\frac{(\rho_1+\rho_2)\nu}{S_C S_A}-\frac{\mu\nu}{S_C S_A}\right)+\frac{\partial}{\partial I}\left(-\frac{(\mu+\tau+\delta)}{S_C S_A}\right)$$
$$\frac{d}{dt}(GX)=-\frac{1}{S_C S_A}\{\Lambda_C+(\mu+\psi_1)+g+(\mu+\psi_2)+(\rho_1+\rho_2)\nu+\mu\nu+(\mu+\tau+\delta)\}<1 \quad (27)$$

Hence the orbit of the region is epidemiologically stable at $R_* > 1$ such that perseverance of the disease reduces and controlled at $t > 1$.

Numerical simulation

In this section, we apply the homotopy perturbation method to obtain an approximate solution for the Typhoid model (1) by constructing the following correctional functional

$$(1-p)\frac{dS_c(t)}{dt}+p\left(\frac{dS_c(t)}{dt}-(\Lambda_c-(1-w)\lambda_c B(t)[S_c(t)+S_A(t)]-(\mu+\psi_1)S_c(t)-gS_c(t)+\rho_1 V(t))\right)=0$$

$$(1-p)\frac{dS_A(t)}{dt}+p\left(\frac{dS_A(t)}{dt}-(\Lambda_A-(1-w)\lambda_A B(t)[S_c(t)+S_A(t)]-(\mu+\psi_2)S_A(t)+gS_c(t)+\rho_2 V(t))\right)=0$$
$$(1-p)\frac{dV(t)}{dt}+p\left(\frac{dV(t)}{dt}-(\psi_1 S_c(t)+\psi_2 S_A(t)-(\rho_1+\rho_2)V(t)-\mu V(t))\right)=0$$
(28)
$$(1-p)\frac{dI(t)}{dt}+p\left(\frac{dI(t)}{dt}-((1-w)B(t)[S_c(t)+S_A(t)](\lambda_c+\lambda_A)-(\mu+\tau+\delta)I)\right)=0$$
$$(1-p)\frac{dT(t)}{dt}+p\left(\frac{dT(t)}{dt}-(\tau I-(\varepsilon+\mu)T)\right)=0$$
$$(1-p)\frac{dR(t)}{dt}+p\left(\frac{dR(t)}{dt}-(\varepsilon T-\mu R)\right)=0$$
$$(1-p)\frac{dB(t)}{dt}+p\left(\frac{dB(t)}{dt}-(\eta(1-w)I(t)-\nu B(t))\right)=0$$

We can assume the following power series of p as solution for the model variables in (44) such that $S_c(t)=\sum_{n=0}^{\infty}p^n s_{cn}(t)$, $S_a(t)=\sum_{n=0}^{\infty}p^n s_{an}(t)$, $V(t)=\sum_{n=0}^{\infty}p^n v_n(t)$ $I(t)=\sum_{n=0}^{\infty}p^n i_n(t)$, $T(t)=\sum_{n=0}^{\infty}p^n \xi_n(t)$ $R(t)=\sum_{n=0}^{\infty}p^n r_n(t)$

Evaluating (44) using (45) and subsequently collecting coefficients of powers of p, for $n \geq 1$ yields the following system

At $n=0$, coefficients of p^0 are

At $n=1$, coefficients of p^1 are: Also at $n=2$, coefficients of p^2 are:

$$\frac{ds_{c2}(t)}{dt} = (\Lambda_c - (1-w)\lambda_c(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)]) - (\mu+\psi_1)s_{c1}(t) - gs_{c1}(t)+\rho_1 v_1(t))$$
$$\frac{ds_{A2}(t)}{dt} = (\Lambda_a - (1-w)\lambda_a(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)]) - (\mu+\psi_2)s_{a1}(t)+gs_{c1}(t)+\rho_2 v_1(t))$$
$$\frac{dv_2(t)}{dt} = (\psi_1 s_{c1}(t)+\psi_2 s_{A1}(t) - (\rho_1+\rho_2)v_1(t) - \mu v_1(t))$$
$$\frac{di_2(t)}{dt} = ((1-w)(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)])(\lambda_c+\lambda_A) - (\mu+\tau+\delta)i_1(t))$$
$$\frac{d\tau_2(t)}{dt} = (\tau i_1(t) - (\varepsilon+\mu)\xi_1(t))$$
$$\frac{dr_2(t)}{dt} = (\varepsilon\xi_1(t) - \mu r_1(t))$$
$$\frac{db_2(t)}{dt} = \eta(1-w)i_1(t) - \nu b_1(t)$$

(29)

And so on. Solving system (28) using the initial conditions
$s_c(0)=s_{c0}$, $s_a(0)=s_{a0}$, $v(0)=v_0$, $i(0)=i_0$, $\xi(0)=\xi_0$, $r(0)=r_0$, $b(0)=b_0$,

Also, evaluating (48) using the initial conditions the following results are obtained for the first approximations

$$s_{c1}(t) = (\Lambda_c - (1-w)\lambda_c b_0[s_{c0}+s_{A0}] - (\mu+\psi_1)s_{c0} - gs_{c0}+\rho_1 v_0)t$$
$$s_{A1}(t) = (\Lambda_a - (1-w)\lambda_a b_0[s_{c0}+s_{A0}] - (\mu+\psi_2)s_{a0} + gs_{c0}+\rho_2 v_0)t$$
$$v_1(t) = (\psi_1 s_{c0}+\psi_2 s_{A0} - (\rho_1+\rho_2)v_0 - \mu v_0)t$$
$$i_1(t) = ((1-w)b_0[s_{c0}+s_{A0}](\lambda_c+\lambda_A) - (\mu+\tau+\delta)i_0)t$$
$$\xi(t) = (\tau i_0 - (\varepsilon+\mu)\xi_0)t$$
$$r_1(t) = (\varepsilon\xi_0 - \mu r_0)t$$
$$b_1(t) = (\eta(1-w)i_0 - \nu b_0)t$$

(30)

the second approximate solution is obtained:

$$s_{c2}(t) = \begin{pmatrix} \Lambda_c - (1-w)\lambda_c\left(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)\begin{bmatrix}(\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0)\\ +(\Lambda_a-(1-w)\lambda_a b_0[s_{c0}+s_{A0}]-(\mu+\psi_2)s_{a0}+gs_{c0}+\rho_2 v_0)\end{bmatrix}\right) \\ -(\mu+\psi_1+s_{c1})(\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0)+\rho_1(\psi_1 s_{c0}+\psi_2 s_{A0}-(\rho_1+\rho_2)v_0-\mu v_0) \end{pmatrix}\frac{t^2}{2}$$

$$s_{A2}(t) = \begin{pmatrix} \Lambda_a - (1-w)\lambda_a\left(b_0(t)\begin{bmatrix}(\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0)\\ +(\Lambda_a-(1-w)\lambda_a b_0[s_{c0}+s_{A0}]-(\mu+\psi_2)s_{a0}+gs_{c0}+\rho_2 v_0)\end{bmatrix}+(\eta(1-w)i_0-\nu b_0)[s_{c0}+s_{A0}]\right) \\ -(\mu+\psi_2+g)(\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0)+\rho_2(\psi_1 s_{c0}+\psi_2 s_{A0}-(\rho_1+\rho_2)v_0-\mu v_0) \end{pmatrix}\frac{t^2}{2}$$

$$v_2(t) = \begin{pmatrix} \psi_1(\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0) \\ +\psi_2(\Lambda_a-(1-w)\lambda_a b_0[s_{c0}+s_{A0}]-(\mu+\psi_2)s_{a0}+gs_{c0}+\rho_2 v_0)-(\rho_1+\rho_2+\mu)(\psi_1 s_{c0}+\psi_2 s_{A0}-(\rho_1+\rho_2)v_0-\mu v_0) \end{pmatrix}\frac{t^2}{2}$$

$$i_2(t)=\left(\begin{array}{l}\left(\begin{array}{l}(1-w)\left(\begin{array}{l}(\eta(1-w)i_0-vb_0)[s_{c0}+s_{A0}]+b_0(t)\left[\begin{array}{l}\left(\begin{array}{l}\Lambda_c-(1-w)\lambda_c b_0[s_{c0}+s_{A0}]\\-(\mu+\psi_1)s_{c0}-gs_{c0}+\rho_1 v_0\end{array}\right)\\+\left(\begin{array}{l}\Lambda_a-(1-w)\lambda_a b_0[s_{c0}+s_{A0}]\\-(\mu+\psi_2)s_{a0}+gs_{c0}+\rho_2 v_0\end{array}\right)\end{array}\right]\end{array}\right)(\lambda_c+\lambda_A)\\-(\mu+\tau+\delta)((1-w)b_0[s_{c0}+s_{A0}](\lambda_c+\lambda_A)-(\mu+\tau+\delta)i_0)\end{array}\right)\frac{t^2}{2}$$

$$r_2(t)=((\varepsilon(\tau i_0-(\varepsilon+\mu)\xi_0)-\mu(\varepsilon\xi_0-\mu r_0)))\frac{t^2}{2}$$

$$b_2(t)=(\eta(1-w)((1-w)b_0[s_{c0}+s_{A0}](\lambda_c+\lambda_A)-(\mu+\tau+\delta)i_0)-v(\eta(1-w)i_0-vb_0))\frac{t^2}{2} \quad (31)$$

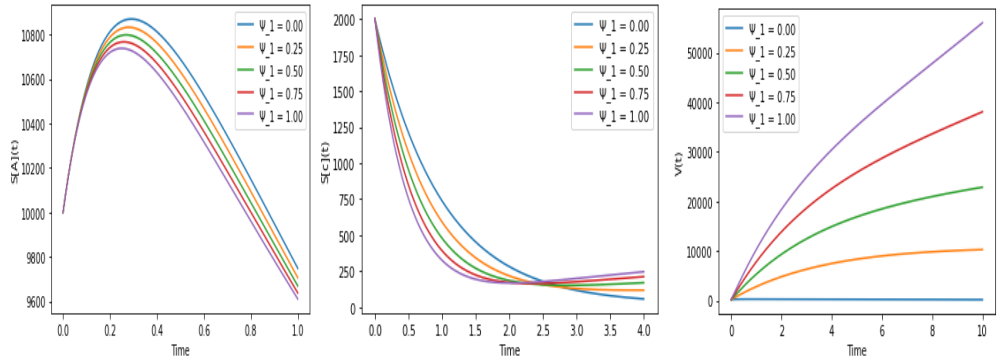
Summing these results up gives the approximate series solution of the system given by

$$S_c(t)=\sum_{n=0}^{2}s_{cn}(t),\quad S_a(t)=\sum_{n=0}^{2}s_{an}(t),\quad V(t)=\sum_{n=0}^{2}v_n(t)\ I(t)=\sum_{n=0}^{2}i_n(t),\quad T(t)=\sum_{n=0}^{2}\xi_n(t)\ R(t)=\sum_{n=0}^{2}r_n(t)$$

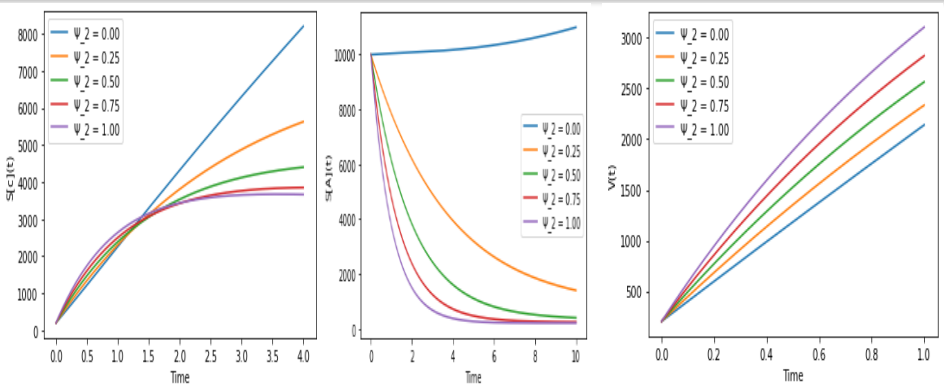
$$B(t)=\sum_{n=0}^{2}b_n(t)$$

RESULTS AND DISCUSSION

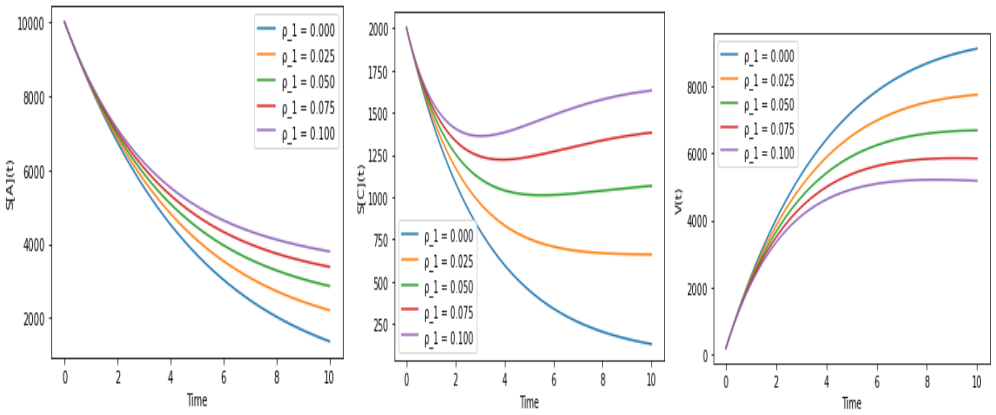
In this section, we conduct a numerical evaluation of the model results and discuss the convergence of the obtained solution. Utilizing the base line parameter values outlined below



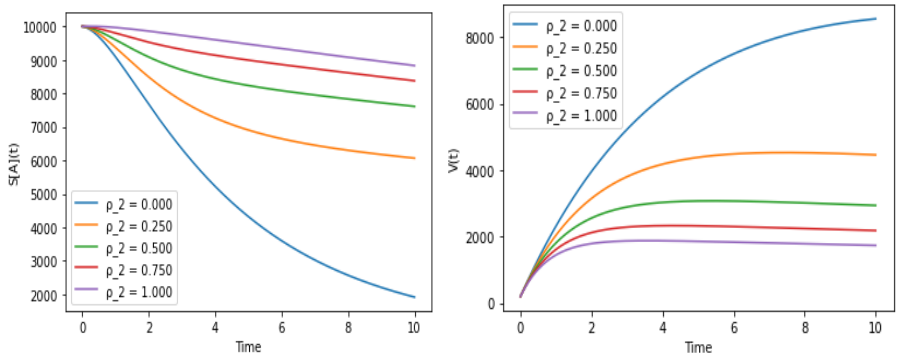
Figures 2: Impact of children vaccination on model variables



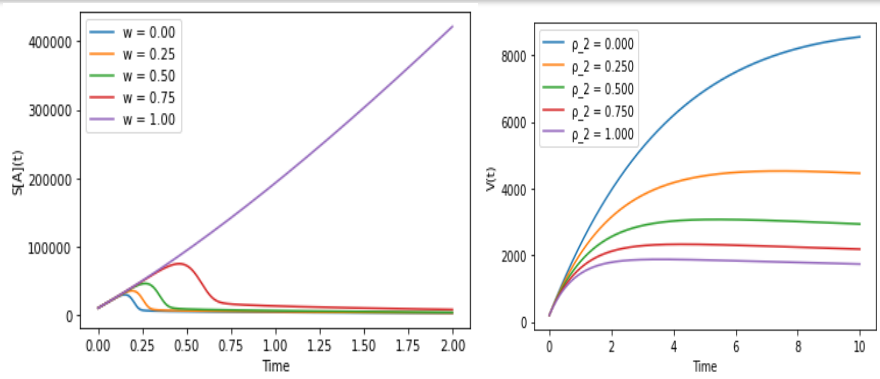
Figures 3: Impact of adult vaccination on model variables



Figures 4: Impact of waning rate of adult vaccination on model variables



Figures 5: Impact of waning rate of adult vaccination on model variables



Figures 6: Impact of hygienic on model variables

The presented figures provide a comprehensive insight into the dynamics of a Typhoid model, focusing on the impact of vaccination rates, waning immunity, and hygiene on the susceptible adult population $S_A(t)$, susceptible children population $S_C(t)$, and the vaccinated population $V(t)$.

Vaccination Rates and Population Dynamics

Figures 2 and 3 show that increasing vaccination rates for children positively correlates with the susceptibility of both adult and child populations. Adult vaccination rates also increase susceptibility faster, emphasizing the importance of considering different vaccination rates for different age groups.

Waning Immunity and Vaccine Boosters

Figures 3 and 4 show that waning immunity rates in children and adults lead to increased susceptible populations and reduced vaccinated ones, emphasizing the need for vaccine boosters to counteract fast immunity waning.

Hygiene Dynamics

Figure 5 reveals that hygiene rates increase susceptibility levels for both adult and child populations, but this does not provide significant protection. The study emphasizes the need for comprehensive hygiene practices to mitigate the spread of Typhoid, emphasizing vaccination rates, waning immunity, and hygiene in shaping population susceptibility. This provides a basis for further public health strategies.

Conclusion

The mathematical modeling of typhoid fever dynamics, using the Homotopy Perturbation Method, provides insights into disease transmission and control. It helps make informed decisions for public health interventions, reducing typhoid fever burden and improving population health. Regular refinement and validation with real-world data are crucial for improving predictive accuracy.

Recommendation

The research emphasizes the significance of age-specific vaccination and treatment strategies in controlling typhoid fever, emphasizing the need for targeted interventions in resource-constrained settings, and suggests future research to account for environmental and socioeconomic factors.

Declaration of Competing Interest

The authors declare that there are no conflicts of interest.

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Data Availability Statement

Data set generated during this research are readily available from the corresponding author on reasonable request.

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