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ORIGINAL RESEARCH

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 (R_*). 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.

1. 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).

2. Formulation of the model

We develop a model with Bacteria population, N_B denoted by B(t) and human population, N_H. The populations are subdivided into different epidemiological classes: Susceptible of the children (S_C), Susceptible of the adult (S_A), 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.

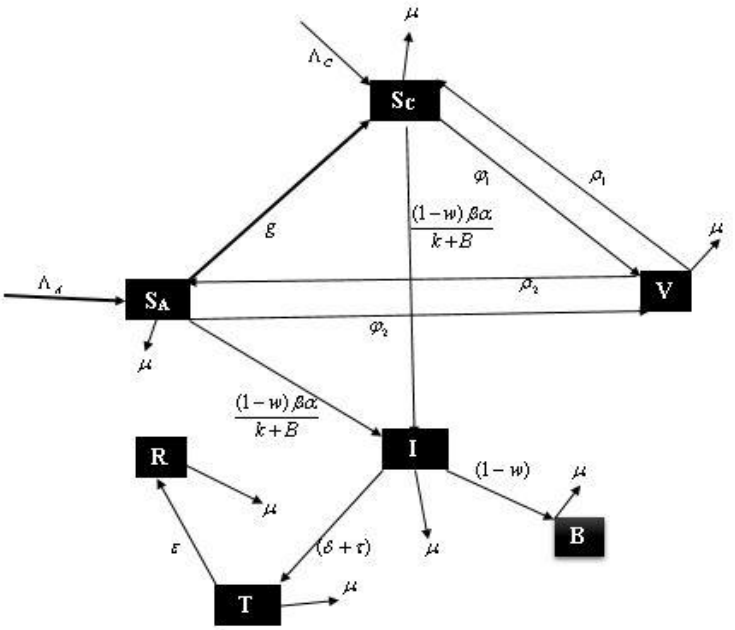


Figure 1. Schematic flow of the model formulation

Table 1. Description of parameters

Variables	Definitions
$S_C(t)$	The number of susceptible individual children
$S_A(t)$	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	Parameter descriptions	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	0.0186821	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	0.00174478	Muchmore *et al*. (2020)
ε	Recovery rate of treatment individuals	0.00331428	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 equations below.

Initial model

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \left[\frac{(1-\omega)\alpha B}{k+B} + \mu\right]S \\
\frac{dI}{dt} &= \frac{(1-\omega)\alpha BS}{k+B} - (\mu+\varepsilon+\delta)I \\
\frac{dR}{dt} &= \varepsilon I - \mu R \\
\frac{dB}{dt} &= (1-\omega)\eta I - \nu B
\end{aligned} \quad (1)$$

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 (2) inclusive of the above parameters. Hence we have:

$$\begin{aligned}
\frac{dS_c}{dt} &= \Lambda_c - (1-w)\lambda_c B[S_c+S_A] - (\mu+\psi_1)S_c + \rho_1 V - gS_c \\
\frac{dS_A}{dt} &= \Lambda_A - (1-w)\lambda_A B[S_c+S_A] - (\mu+\psi_2)S_A + \rho_2 V + gS_c \\
\frac{dV}{dt} &= \psi_1 S_c + S_A\psi_2 - (\rho_1+\rho_2)V - \mu V \\
\frac{dI}{dt} &= (1-w)\lambda_c B[S_c+S_A] + (1-w)\lambda_A B[S_c+S_A] - (\mu+\tau+\delta)I \\
\frac{dT}{dt} &= \tau I - \varepsilon T - \mu T \\
\frac{dR}{dt} &= \varepsilon T - \mu R \\
\frac{dB}{dt} &= (1-\omega)\eta I - \nu B
\end{aligned} \quad (2)$$

Where $\lambda_c = \lambda_A = \dfrac{B\alpha}{K+B}$

3. Analysis of the model

3.1. **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 (N_H) considered in above model are

$N_H = (S_c + S_A + V + I + T + R)$,

$$\frac{dN}{dt} = \Lambda_c - (1-w)\lambda_c B[S_c + S_A] - (\mu + \psi_1)S_c + \rho_1 \nu - gS_c + \Lambda_A - (1-w)\lambda_A B[S_c + S_A] - (\mu + \psi_2)S_A + \rho_2 \nu + gS_c + \psi_1 S_c + S_A \psi_2 - (\rho_1 + \rho_2)\nu - \mu\nu + (1-w)\lambda_c B[S_c + S_A] + (1-w)\lambda_A B[S_c + S_A] - (\mu + \tau + \delta)I + \tau I - \varepsilon T - \mu T + \varepsilon T - \mu R \quad (3)$$

$$\frac{dN}{dt} = \Lambda_c - \mu S_C - \psi_1 S_C + \rho_1 \nu + \rho_2 \nu + \Lambda_A - \mu S_A - \psi_2 S_A + \psi_1 S_C + \psi_2 S_A - \rho_1 \nu - \rho_2 \nu - \mu\nu - \mu I - \tau I + \tau I - \mu T - \mu R \quad (4)$$

$$\frac{dN}{dt} = \Lambda_C + \Lambda_A - \mu(S_C + S_A + \nu + I + R) \quad (5)$$

$$\frac{dN}{dt} = \Lambda_C + \Lambda_A - \mu N$$

At no outbreak of disease, $I = 0$

$$\frac{dN}{dt} = \Lambda_C + \Lambda_A - \mu N \quad (6)$$

$$\frac{dN}{dt} + \mu N = \Lambda_C + \Lambda_A$$

Let P= μ, $Q = \Lambda_C + \Lambda_A$

By method of integrating factor

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

I.F= $\ell^{\int P dt}$

I.F= $\ell^{\int \mu dt} = \ell^{\mu t}$

$$N.\ell^{\mu t} = \int \ell^{\mu t} \cdot (\Lambda_C + \Lambda_A) dt$$

$$N \cdot \ell^{\mu t} = (\Lambda_C + \Lambda_A) \int \ell^{\mu t} dt$$

$$N \cdot \ell^{\mu t} = (\frac{(\Lambda_C + \Lambda_A)\ell^{\mu t}}{\mu} + C$$

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

At t=0

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

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

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

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

$$N_H(t) \le \frac{\Lambda_C + \Lambda_A}{\mu} \quad (7)$$

Also the bacteria population is

$$N_B = (1-w)\eta I - \nu B$$

$$N_B(t) = \frac{(1-w)\eta}{\nu} \quad (8)$$

Thus, the feasible solution of the system equation of the model enters and remains in the region

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

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

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.

3.2. Positivity and boundedness of model solution

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

Theorem 1

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

Then the solution of $\{S_C, S_A, \nu, I, T, R, B\}$ are positive for t ≥ 0

Proof

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

First Equation;

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

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

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

$$\frac{dS_C}{S_c(t)} + (\mu + \psi_1 + g)dt \geq 0$$

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

$$S_C(t)\ell^{(\mu+\psi_1+g)t} \geq \int \ell^{(\mu+\psi_1+g)t} \cdot 0dt$$

$$S_C(t)\ell^{(\mu+\psi_1+g)t} \geq 0 + C$$

$$S_C(t)\ell^{(\mu+\psi_1+g)t} \geq C$$

$$S_C(t) \geq \ell^{-(\mu+\psi_1+g)t} \cdot C$$

$$S_C(t) \geq S_{C0} \cdot \ell^{-(\mu+\psi_1+g)t} \geq 0 \qquad (9)$$

Then solving the second equation,

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

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

$$\frac{dS_A}{dt} + (\mu + \psi_2)S_A(t) \geq 0$$

Similarly using integrating factor and applying conditions, it gives

$$S_A(t) \cdot \ell^{(\mu+\psi_2)t} \geq \int \ell^{(\mu+\psi_2)t} \cdot 0dt$$

$$\Rightarrow \ S_A(t) \geq S_{A_0}(t) \cdot \ell^{(\mu+\psi_2)t} \geq 0 \qquad (10)$$

Also we took the third equation of (3.2)

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

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

$$\frac{dV}{dt} + (\rho_1 + \rho_2 + \mu)\nu(t) \geq 0$$

Also using integrating factor and applying the condition,

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

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

$$V(t) \geq V_0 \cdot \ell^{(\rho_1+\rho_2+\mu)t} \geq 0 \qquad (11)$$

then taking the fourth equation of

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

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

$$\frac{dI}{dt}+(\mu+\tau+\delta)I(t)\geq 0$$

$$I(t)\cdot \ell^{(\mu+\tau+\delta)t} \geq \int \ell^{(\mu+\tau+\delta)t}\cdot 0\,dt$$

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

$$I(t)\geq I_0\cdot I(t)\cdot \ell^{(\mu+\tau+\delta)t}\geq 0 \qquad (12)$$

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

$$\frac{dT}{dt}\geq -(\varepsilon+\mu)T(t)$$

$$\frac{dT}{dt}+(\varepsilon+\mu)T(t)\geq 0$$

$$\frac{dT}{dt}+(\varepsilon+\mu)T(t)\geq 0$$

$$T(t)\cdot \ell^{(\varepsilon+\mu)t} \geq \int \ell^{(\varepsilon+\mu)t}\cdot 0\,dt$$

$$T(t)\geq T_0\cdot \ell^{-(\varepsilon+\mu)t}\geq 0 \qquad (13)$$

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

$$\frac{dR}{dt}\geq -\mu R(t)$$

$$\frac{dR}{dt}+\mu R(t)\geq 0$$

$$R(t)\cdot \ell^{(\mu)t} \geq \int \ell^{(\mu)t}\cdot 0\,dt$$

$$R(t)\cdot \ell^{(\mu)t} \geq 0+C$$

$$R(t)\geq R_0\cdot \ell^{(\mu)t}\geq 0$$

$$\frac{dB}{dt}=(1-w)\eta I-\nu B \qquad (14)$$

$$\frac{dB}{dt}\geq -\nu B(t)$$

$$\frac{dB}{dt}+\nu B(t)\geq 0$$

$$B(t)\cdot \ell^{(V)t} \geq \int \ell^{(V)t}\cdot 0\,dt$$

$$B(t)\cdot \ell^{(V)t} \geq 0+C$$

$$B(t)\geq B_0\ell^{(V)t} \qquad (15)$$

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

3.3. Existence of disease free equilibrium state

To analyse the disease free-equilibrium we let the right hand side of the model (2) 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.

$$S_{A_0}=\frac{\Lambda_A+\rho_2 V+gS_C}{(\mu+\psi_2)} \qquad (16)$$

$$S_{A_0}=\Lambda_A+\frac{\rho_2 V+g\Lambda_C}{\frac{(\mu+\psi_1+g)}{(\mu+\psi_2)}} \qquad (17)$$

$$S_{A_0}=\frac{\Lambda_A(\mu+\psi_1+g)+\rho_2 V+g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}$$

$$\psi_1 S_C+\psi_2 S_A-(\rho_1+\rho_2)V-\mu V=0$$

$$\psi_1 S_C+\psi_2 S_A=(\rho_1+\rho_2+\mu)V$$

$$V=\frac{\psi_1 S_C+\psi_2 S_A}{(\rho_1+\rho_2+\mu)} \qquad (18)$$

$$E_0=(S_{C_0},S_{A_0},V_0,I_0,T_0,R_0,B_0)=\left(\frac{\Lambda_C+\rho_1 V}{(\mu+\psi_1+g)},\ \frac{\Lambda_A(\mu+\psi_1+g)+\rho_2 V+g\Lambda_C}{((\mu+\psi_1+g)(\mu+\psi_2)},\ \frac{\psi_1 S_C+\psi_2 S_A}{(\rho_1+\rho_2+\mu)},0,0,0,0\right)$$

3.4. 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 prove this theorem, we equate the right-hand side of the system to zero and substitute S_C, S_A, V, I, T, R, B with $S_C^*, S_A^*, V^*, I^*, T^*, R^*, B^*$, respectively, to get

$$\left.\begin{aligned}
0 &= \Lambda_C - (1-w)\lambda_C B^*(S_C^* + S_A^*) - (\mu+\psi_1)S_C^* + \rho_1 V^* - gS_C^* \\
0 &= \Lambda_A - (1-w)\lambda_A B^*(S_C^* + S_A^*) - (\mu+\psi_2)S_A^* + \rho_2 V^* + gS_C^* \\
0 &= \psi_1 S_C^* + \psi_2 S_A^* - (\rho_1+\rho_2)V^* - \mu V^* \\
0 &= (1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*) - (\mu+\tau+\delta)I^* \\
0 &= \tau I^* - \varepsilon T^* - \mu T^* \\
0 &= \varepsilon T^* - \mu R^* \\
0 &= (1-\omega)\eta I^* - \nu B^*
\end{aligned}\right\} \qquad (19)$$

From the last equation of system (19) we have

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

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

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

$$V^* = \frac{\psi_1 S_C^* + \psi_2 S_A^*}{(\rho_1+\rho_2+\mu)}$$

$$T^* = \frac{\tau(1-w)\lambda_C B^*(S_C^* + S_A^*) + (1-w)\lambda_A B^*(S_C^* + S_A^*)}{(\varepsilon+\mu)(\mu+\tau+\delta)} \qquad (20)$$

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

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

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

$$F_i = \left[\frac{\partial f_i(x_0)}{\partial x_j}\right] \quad V_i = \left[\frac{\partial v_i(x_0)}{\partial x_j}\right] \qquad (21)$$

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

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial B} \\ \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial B} \\ \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial B} \end{bmatrix}$$

Let

$f_1 = (1-w)\lambda_C B(S_C + S_A) + (1-w)\lambda_A B(S_C + S_A)$,

$f_2=0$, $f_3=0$

$$V^T = adjV = \begin{bmatrix} \nu(\varepsilon+\mu) & 0 & 0 \\ [\nu\tau & \nu(\mu+\tau+\delta) & 0] \\ [[(\varepsilon+\mu)(1-\omega)\eta & 0 & (\varepsilon+\mu)(\mu+\tau+\delta)]] \end{bmatrix}$$

Now let

$A = (1-w)\lambda_C(S_C + S_A)$, $B = (1-w)\lambda_A(S_C + S_A)$

$$F = \begin{bmatrix} 0 & 0 & A+B \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \frac{\partial V_1}{\partial I} & \frac{\partial V_1}{\partial T} & \frac{\partial V_1}{\partial B} \\ \frac{\partial V_2}{\partial I} & \frac{\partial V_2}{\partial T} & \frac{\partial V_2}{\partial B} \\ \frac{\partial V_3}{\partial I} & \frac{\partial V_3}{\partial T} & \frac{\partial V_3}{\partial B} \end{bmatrix}$$

Where

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

$$V = \begin{bmatrix} (\mu+\tau+\delta) & 0 & 0 \\ -\tau & \varepsilon+\mu & 0 \\ -(1-\omega)\eta & 0 & \nu \end{bmatrix} \quad (22)$$

$$|V| = (\mu+\tau+\delta)\nu(\varepsilon+\mu)$$

$$V^T = adjV = \begin{bmatrix} \nu(\varepsilon+\mu) & 0 & 0 \\ \nu\tau & \nu(\mu+\tau+\delta) & 0 \\ (\varepsilon+\mu)(1-\omega)\eta & 0 & (\varepsilon+\mu)(\mu+\tau+\delta) \end{bmatrix} \quad (23)$$

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

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

$$\left[R_* = \frac{2(1-\omega)^2\eta\lambda_C(\mu+\psi_2)(\Lambda_C+\rho_1 V)+\Lambda_A(\mu+\psi_1+g)+\rho_2 V+g\Lambda_C)}{\nu(\mu+\tau+\delta)(\mu+\psi_1+g)(\mu+\tau+\delta)} \right] \quad (24)$$

3.6. 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 (2), where $|J_{E_i} - \lambda_i I| = 0$ as λ_i *and* I are the Eigen-values and identity matrix respectively. Where i=1, 2,…

$$J_{E_0} = \begin{vmatrix} -[(1-w)\lambda_C BS_A + (\mu+\psi_1+g)], -(1-w)\lambda_C BS_C, & \rho_1, & 0 & 0 & 0 & -[(1-w)\lambda_C(S_C+S_A)] \\ -[(1-w)\lambda_A BS_A - g], -[(1-w)\lambda_A BS_C + (\mu+\psi_2)], & \rho_2, & 0 & 0 & 0 & 0 & -[(1-w)\lambda_A(S_C+S_A)] \\ \psi_1 & \psi_2, & -(\rho_1+\rho_2+\mu), & 0 & 0 & 0 & 0 & \\ [(1-w)\lambda_C BS_A + (1-w)\lambda_A BS_A], [(1-w)\lambda_C BS_C + (1-w)\lambda_A BS_C], & 0, & (\mu+\tau+\delta), & 0, & 0, & H & & \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu), & 0, & 0 & \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 & \\ 0 & 0 & 0 & (1-w)\eta & 0 & 0 & -\nu & \end{vmatrix}$$

Where $H = [(1-w)\lambda_C(S_C + S_A) + (1-w)\lambda_A(S_C + S_A)]$

$$J_{E_0}=\begin{vmatrix} -(\mu+\psi_1+g) & 0 & \rho_1 & 0 & 0 & 0 & 0 \\ g & -(\mu+\psi_2) & \rho_2 & 0 & 0 & 0 & 0 \\ \psi_1 & \psi_2 & -(\rho_1+\rho_2+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\mu+\tau+\delta) & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 \\ 0 & 0 & 0 & (1-w)\eta & 0 & 0 & -\nu \end{vmatrix} \quad (25)$$

Then $\left|J_{E_0}-\lambda I\right|=0$, (25) will becomes

$$\begin{vmatrix} -(\mu+\psi_1+g)-\lambda_1 & 0 & \rho_1 & 0 & 0 & 0 & 0 \\ g & -(\mu+\psi_2)-\lambda_2 & \rho_2 & 0 & 0 & 0 & 0 \\ \psi_1 & \psi_2 & -(\rho_1+\rho_2+\mu)-\lambda_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\mu+\tau+\delta)-\lambda_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu)-\lambda_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu-\lambda_6 & 0 \\ 0 & 0 & 0 & (1-w)\eta & 0 & 0 & -\nu-\lambda_7 \end{vmatrix}=0$$

By using Atangana Belame invariance principle by lower triangular matrix.

We obtain, therefore,

$$\lambda_1=-(\mu+\psi_1+g),\lambda_2=-(\mu+\psi_2),\ \lambda_3=-(\rho_1+\rho_2+\mu),$$
$$\lambda_4=-(\mu+\tau+\delta),\lambda_5=-(\varepsilon+\mu),\lambda_6=-\mu,\ \lambda_7=-\nu \quad (26)$$

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

3.7. 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 compartment by let

$$S_C=a+S_C^*,S_A=b+S_A^*,V=c+V^*,I=e+I^*,T=x+T^*,$$
$$R=y+R^*,B=z+B^* \quad (27)$$

From the system of equation (27), we obtain

$$\frac{da}{dt} = \Lambda_C - [(1-w)\lambda_C za] - [(1-\omega)\lambda_C zb] - (\mu+\psi_1)a + \rho_1 c - ga + higher\ order + non-linear\ terms$$

$$\frac{db}{dt} = \Lambda_A - [(1-w)\lambda_A za] - [(1-\omega)\lambda_A zb] - (\mu+\psi_2)b + \rho_2 c - ga + higher\ order + non-linear\ terms$$

$$\frac{dc}{dt} = \psi_1 a + \psi_2 b - (\rho_1+\rho_2)c - \mu c + higher\ order + non-linear\ terms \qquad (28)$$

$$\frac{de}{dt} = [(1-w)\lambda_C za] + [(1-\omega)\lambda_C zb] + [(1-w)\lambda_A zb] - (\mu+\tau+\delta)e + higher\ order + non-inear\ terms$$

$$\frac{dx}{dt} = \tau e - (\varepsilon+\mu)x + higher\ order + non-inear\ terms$$

$$\frac{dy}{dt} = \varepsilon x - \mu y + higher\ order + non-inear\ terms$$

$$\frac{dz}{dt} = (1-\omega)\eta e - \nu z + higher\ order + non-inear\ terms$$

Then we differentiate each compartment one by one and take the Jacobian-Matrix.

$$J_{EE} = \begin{vmatrix} -[(1-w)\lambda_C z + (\mu+\psi_1+g)], & -[(1-\omega)\lambda_C z], & \rho_1, & 0 & 0 & 0 & -[(1-w)\lambda_C(a+b)] \\ -[(1-w)\lambda_A z - g], & -[(1-w)\lambda_A z + (\mu+\psi_2)], & \rho_2, & 0 & 0 & 0 & -[(1-w)\lambda_A(a+b)] \\ \psi_1 & \psi_2 & -(\rho_1+\rho_2+\mu), & 0 & 0 & 0 & 0 \\ [(1-\omega)\lambda_C z + (1-\omega)\lambda_A z], & [(1-w)\lambda_A z + (1-w)\lambda_A z], & 0, & -(\mu+\tau+\delta) & 0 & 0 & L \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 \\ 0 & 0 & 0 & (1-w)\eta & 0 & 0 & -\nu \end{vmatrix} \qquad (29)$$

Where L= $\{[(1-w)\Lambda_C a] + [(1-w)\Lambda_C b] + [(1-w)\Lambda_A a] + [(1-w)\Lambda_A b]\}$

The Jacobian matrix of the system of (3.83) were obtained, where $\left|J_{E_i} - \lambda_i I\right| = 0$

$$\begin{vmatrix} -[(1-w)\Lambda_C z + (\mu+\psi_1+g)] - \lambda_1^* & -[(1-w)\Lambda_C z], & \rho_1, & 0 & 0 & 0 & -(1-w)\Lambda_C(a+b) \\ -[(1-w)\Lambda_A zb - g], & -[(1-w)\Lambda_A z + \mu + \psi_2] - \lambda_2^* \rho_2, & 0 & 0 & 0 & -(1-w)\Lambda_A(a+b) & \\ \psi_1 & \psi_2 & -(\rho_1+\rho_2+\mu) - \lambda_3^* & 0 & 0 & 0 & 0 \\ [(1-w)\Lambda_C z + (1-w)\Lambda_A z], & [(1-w)\Lambda_A z + (1-w)\Lambda_A z], & 0, & -(\mu+\tau+\delta) - \lambda_4^* & 0 & 0 & L \\ 0 & 0 & 0 & \tau & -(\varepsilon+\mu) - \lambda_5^* & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & -\mu - \lambda_6^* & 0 \\ 0 & 0 & 0 & (1-w)\eta & 0 & 0 & -\nu - \lambda_7^* \end{vmatrix} = 0$$

$$A = -[(1-w)\lambda_C z + (\mu + \psi_1 + g)],\ B = -[(1-w)\lambda_A z + \mu + \psi_2],$$
$$C = -(\rho_1 + \rho_2 + \mu),\ D = -(\mu + \tau + \delta), E = -(\varepsilon + \mu), F = -\mu, G = -\nu \tag{30}$$

$$a = S_C - S_C^*,\ b = S_A - S_A^*,\ c = V - V^*, e = I - I^*, x = T - T^*,$$
$$y = R - R^*, z = B - B^* \tag{31}$$

$$(A - \lambda^*)(B - \lambda^*)(C - \lambda^*)(D - \lambda^*)(E - \lambda^*)(F - \lambda^*)(G - \lambda^*) = 0$$

The Characteristics polynomial is given by

$$\lambda_*^{\,7} + a_1\lambda_*^{\,6} + a_2\lambda_*^{\,5} + a_3\lambda_*^{\,4} + a_4\lambda_*^{\,3} + a_5\lambda_*^{\,2} + a_6\lambda_*^{\,1} + a_7 \tag{32}$$

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.

3.8. 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;

$$V(S_1, S_2, I_1, I_2, R, P, t) = C_1 I_1 + C_2 I_2 + C_3 I_3 \tag{33}$$

$$\frac{dV}{dt} = C_1 I_1^{\,1} + C_2 I_2^{\,1} + C_3 I_3 \ as\ I_1 = I_1 \Rightarrow I_2 = I_2\ and\ I_3 = P \tag{34}$$

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, S_C, S_A, 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

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

$$\frac{dV}{dt} = C_1[(1-w)\lambda_C I_3(S_C + S_A) + (1-w)\lambda_A I_3(S_C + S_A) - (\mu + \tau + \delta)I_1] +$$
$$C_2[(\tau I_1 - (\varepsilon + \mu)I_2 + C_3[(1-w)\eta I_1 - \nu I_3] \tag{35}$$

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

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

$$\left.\begin{aligned}\frac{dV}{dt} \le [C_2\tau + C_3(1-w)\eta - C_1(\mu+\tau+\delta)]I_1 - C_2(\varepsilon+\mu)I_2 + [C_1(1-w)\lambda_C S_C \\ + C_1(1-w)\lambda_C S_A + C_1(1-w)\lambda_A S_C] + C_1(1-w)\lambda_A S_A]I_3\end{aligned}\right\}$$

Since $C_3 \ge 0$

$$\left.\begin{aligned}\frac{dV}{dt} \le [C_2\tau - C_1(\mu+\tau+\delta)]I_1 - C_2(\varepsilon+\mu)I_2 \\ + \left(\begin{aligned}&[C_1(1-w)\lambda_C \frac{\Lambda_C + \rho_1 V}{(\mu+\psi_1+g)}] + [C_1(1-w)\lambda_C \frac{\Lambda_A(\mu+\psi_1+g) + \rho_2 V + g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}] + \\ &[C_1(1-w)\lambda_A \frac{\Lambda_C}{(\mu+\psi_1+g)}] + [C_1(1-w)\lambda_A \frac{\Lambda_A(\mu+\psi_1+g) + g\Lambda_C}{(\mu+\psi_1+g)(\mu+\psi_2)}]\end{aligned}\right) I_3\end{aligned}\right\} \quad (36)$$

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

Recall that $C_1 < C_2 < C_3$, $C_3 \ge 0$, *and let* $C_1 = \dfrac{1}{(\mu+\tau+\delta)}$

$$\frac{dV}{dt} \le \left(\begin{aligned}&\frac{(1-w)\lambda_C(\mu+\psi_2)(\Lambda_C + \rho_1 V) + (1-w)\lambda_C[\Lambda_A(\mu+\psi_1+g) + \rho_2 V + g\Lambda_C] + }{(\mu+\psi_1+g)(\mu+\psi_2)(\mu+\tau+\delta)} \\ &\frac{(1-w)\lambda_A(\mu+\psi_2) + (\Lambda_C + \rho_1 V)(1-w)\lambda_A[\Lambda_A(\mu+\psi_1+g)] + (\rho_2 V + g\Lambda_C)}{(\mu+\psi_1+g)(\mu+\psi_2)(\mu+\tau+\delta)} - 1\end{aligned}\right) \quad (37)$$

It is important to note that V=0, only when I=0, the substitution of I=0 into the model system of equation (3) shows that $S_{C_0} = \dfrac{\Lambda_C + \rho_1 \nu}{(\mu+\psi_1+g)}$, $S_{A_0} = \dfrac{\Lambda_A(\mu+\psi_1+g) + \rho_2 \nu + g\Lambda_C}{((\mu+\psi_1+g)(\mu+\psi_2)}$ at t→∞, w<1 and based on Lasalle's invariance principle, Hence $E_0 = 0$ is Globally Asymptotically stable whenever $R_* < 1$

3.9. 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= (S_C, S_A, V, I, T, R, B), where $G(X) = \dfrac{1}{S_C S_A}$ (38)

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

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

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

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

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

$$G(\frac{dI}{dt}) = \frac{1}{S_C S_A}\left[(1-w)\lambda_C B(S_C + S_A) + (1-w)\lambda_A B(S_C + S_A) - (\mu + \tau + \delta)I\right] \quad (39)$$

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

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

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

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

Then we obtain

$$\frac{d}{dt}(GX) = \left(G\frac{dS_C}{dt} \right) + \frac{\partial}{\partial S_A}\left(G\frac{dS_A}{dt} \right) + \frac{\partial}{\partial V}\left(G\frac{dV}{dt} \right) + \frac{\partial}{\partial I}\left(G\frac{dI}{dt} \right) + \left[\frac{\partial}{\partial T}\left(G\frac{dT}{dt} \right) + \frac{\partial}{\partial R}\left(G\frac{dR}{dt} \right) + \frac{\partial}{\partial B}\left(G\frac{dB}{dt} \right) \right]$$

$$\begin{aligned}\frac{d}{dt}(GX)=&\frac{\partial}{\partial S_C}\left(\frac{\Lambda_C}{S_CS_A}-\frac{(1-w)\lambda_C B}{S_A}-\frac{(1-w)\lambda_C B}{S_C}-\frac{(\mu+\psi_1)}{S_A}+\frac{\rho_1\nu}{S_CS_A}-\frac{g}{S_C}\right)+\\&\frac{\partial}{\partial S_A}\left(\frac{\Lambda_A}{S_CS_A}-\frac{(1-w)\lambda_A B}{S_A}-\frac{(1-w)\lambda_A B}{S_C}-\frac{(\mu+\psi_2)}{S_C}+\frac{\rho_2\nu}{S_AS_C}+\frac{g}{S_C}\right)+\\&\frac{\partial}{\partial V}\left(\frac{\psi_1}{S_AS_C}+\frac{\psi_2}{S_CS_A}-\frac{(\rho_1+\rho_2)\nu}{S_CS_A}-\frac{\mu\nu}{S_CS_A}\right)+\\&\frac{\partial}{\partial I}\left(\frac{(1-w)\lambda_C B}{S_A}+\frac{(1-w)\lambda_C B}{S_C}+\frac{(1-w)\lambda_A B}{S_A}+\frac{(1-w)\lambda_A B}{S_C}-\frac{(\mu+\tau+\delta)}{S_CS_A}\right)+\\&\frac{\partial}{\partial T}\left(\frac{\tau I}{S_CS_A}-\frac{\varepsilon I}{S_CS_A}-\frac{\mu T}{S_CS_A}\right)+\frac{\partial}{\partial R}\left(\frac{\varepsilon T}{S_CS_A}-\frac{\mu R}{S_CS_A}\right)+\\&\frac{\partial}{\partial B}\left(\frac{(1-w)\eta}{S_CS_A}-\frac{\nu B}{S_CS_A}\right)\end{aligned}\tag{40}$$

Then we obtain

$$\begin{aligned}\frac{d}{dt}(GX)=&\frac{\partial}{\partial S_C}\left(\frac{\Lambda_C}{S_CS_A}-\frac{(\mu+\psi_1)}{S_A}+\frac{\rho_1\nu}{S_CS_A}-\frac{g}{S_A}\right)+\\&\frac{\partial}{\partial S_A}\left(\frac{\Lambda_A}{S_CS_A}-\frac{(\mu+\psi_2)}{S_CS_A}+\frac{\rho_2\nu}{S_CS_A}+\frac{g}{S_A}\right)+\\&\frac{\partial}{\partial V}\left(\frac{\psi_1}{S_A}+\frac{\psi_2}{S_C}-\frac{(\rho_1+\rho_2)\nu}{S_CS_A}-\frac{\mu\nu}{S_CS_A}\right)+\frac{\partial}{\partial I}\left(-\frac{(\mu+\tau+\delta)}{S_CS_A}\right)+\\&\frac{\partial}{\partial T}\left(\frac{\tau I}{S_CS_A}-\frac{\varepsilon I}{S_CS_A}-\frac{\mu T}{S_CS_A}\right)+\frac{\partial}{\partial R}\left(\frac{\varepsilon T}{S_CS_A}-\frac{\mu R}{S_CS_A}\right)+\\&\frac{\partial}{\partial B}\left(\frac{(1-w)\eta}{S_CS_A}\right)\end{aligned}\tag{41}$$

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_CS_A

$$\begin{aligned}\frac{d}{dt}(GX)=&\frac{\partial}{\partial S_C}\left(-\frac{\Lambda_C}{S_CS_A}-\frac{(\mu+\psi_1)}{S_A}-\frac{g}{S_A}\right)+\frac{\partial}{\partial S_A}\left(-\frac{(\mu+\psi_2)}{S_CS_A}\right)+\\&\frac{\partial}{\partial V}\left(-\frac{(\rho_1+\rho_2)\nu}{S_CS_A}-\frac{\mu\nu}{S_CS_A}\right)+\frac{\partial}{\partial I}\left(-\frac{(\mu+\tau+\delta)}{S_CS_A}\right)\end{aligned}$$

$$\frac{d}{dt}(GX)=-\frac{1}{S_CS_A}\left\{\Lambda_C+(\mu+\psi_1)+g+(\mu+\psi_2)+(\rho_1+\rho_2)\nu+\mu\nu+(\mu+\tau+\delta)\right\}<1\tag{42}$$

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

3.9.1. Sensitivity analysis of R_0

The robustness of the model predictions with respect to parameter values and also to detect parameters that mostly impact on R_0 . To study the behaviour of a relative change in a variable with respect to changes in a parameter. In order to measure the impact of the model parameters. The sensitivity index analysis using normalized sensitivity is obtained as;

$$\left.\begin{aligned}
&\frac{\partial R_0}{\partial \omega} = \frac{\partial R_0}{\partial \omega}\cdot\frac{\omega}{R_0} = -1.278688525 && \frac{\partial R_0}{\partial \delta} = \frac{\partial R_0}{\partial \delta}\cdot\frac{\delta}{R_0} = 0.01799667\\
&\frac{\partial R_0}{\partial \eta} = \frac{\partial R_0}{\partial \eta}\cdot\frac{\eta}{R_0} = 1.000000000 && \frac{\partial R_0}{\partial \mu} = \frac{\partial R_0}{\partial \mu}\cdot\frac{\mu}{R_0} = 1.003122405\times10^{-15}\\
&\frac{\partial R_0}{\partial \psi_1} = \frac{\partial R_0}{\partial \psi_1}\cdot\frac{\psi_1}{R_0} = 3.738474319\times10^{-3} && \frac{\partial R_0}{\partial \rho_1} = \frac{\partial R_0}{\partial \rho_1}\cdot\frac{\rho_1}{R_0} = 2.00004182\times10^{-4}\\
&\frac{\partial R_0}{\partial \psi_2} = \frac{\partial R_0}{\partial \psi_2}\cdot\frac{\psi_2}{R_0} = 2.610142447\times10^{-3} && \frac{\partial R_0}{\partial \rho_2} = \frac{\partial R_0}{\partial \rho_2}\cdot\frac{\rho_2}{R_0} = 2.193040109\times10^{-4}\\
&\frac{\partial R_0}{\partial g} = \frac{\partial R_0}{\partial g}\cdot\frac{g}{R_0} = 9.2234591679\times10^{-3}\\
&\frac{\partial R_0}{\partial \nu} = \frac{\partial R_0}{\partial \nu}\cdot\frac{\nu}{R_0} = 1\times10^{-6}\\
&\frac{\partial R_0}{\partial \tau} = \frac{\partial R_0}{\partial \tau}\cdot\frac{\tau}{R_0} = 0.089585635
\end{aligned}\right\} \quad (43)$$

Table 2. Sensitivity index of R_0

Description	Parameter	Sensitivity indices
Waning rate of immunity	ρ_1, ρ_2	0.63663
Excretion rate of salmonella typhoid	η	1.82363
Concentration of salmonella bacteria infection in fluids and water	ψ_1, ψ_2	0.06253
Rate at which children becomes adult	G	-1.71212
Death rate	μ	0.08273
Rate at which salmonella typhi will die to the environment	ν	0.00263
Disease induced death	δ	0.01263
Hygiene rate	w	1.20826
Treatment rate	τ	1.23627

The table above shows the sensitivity index value of model parameters. The positive values in the above table describes or show the non-prevalence of the virus increases. They contribute in decreasing the value of basic reproduction number R_*

4. 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

$$\begin{aligned}
&(1-p)\frac{dS_c(t)}{dt}+p\left(\frac{dS_c(t)}{dt}-\left(\Lambda_c-(1-w)\lambda_c B(t)[S_c(t)+S_A(t)]-(\mu+\psi_1)S_c(t)-gS_c(t)+\rho_1 V(t)\right)\right)=0\\
&(1-p)\frac{dS_A(t)}{dt}+p\left(\frac{dS_A(t)}{dt}-\left(\Lambda_A-(1-w)\lambda_A B(t)[S_c(t)+S_A(t)]-(\mu+\psi_2)S_A(t)+gS_c(t)+\rho_2 V(t)\right)\right)=0\\
&(1-p)\frac{dV(t)}{dt}+p\left(\frac{dV(t)}{dt}-\left(\psi_1 S_c(t)+\psi_2 S_A(t)-(\rho_1+\rho_2)V(t)-\mu V(t)\right)\right)=0\\
&(1-p)\frac{dI(t)}{dt}+p\left(\frac{dI(t)}{dt}-\left((1-w)B(t)[S_c(t)+S_A(t)](\lambda_c+\lambda_A)-(\mu+\tau+\delta)I\right)\right)=0\\
&(1-p)\frac{dT(t)}{dt}+p\left(\frac{dT(t)}{dt}-\left(\tau I-(\varepsilon+\mu)T\right)\right)=0\\
&(1-p)\frac{dR(t)}{dt}+p\left(\frac{dR(t)}{dt}-\left(\varepsilon T-\mu R\right)\right)=0\\
&(1-p)\frac{dB(t)}{dt}+p\left(\frac{dB(t)}{dt}-\left(\eta(1-w)I(t)-\nu B(t)\right)\right)=0
\end{aligned} \tag{44}$$

We can assume the following power series of p as solution for the model variables in (44) such that

$$S_c(t)=\sum_{n=0}^{\infty}p^n s_{cn}(t),\ S_a(t)=\sum_{n=0}^{\infty}p^n s_{an}(t),\ V(t)=\sum_{n=0}^{\infty}p^n v_n(t)\ I(t)=\sum_{n=0}^{\infty}p^n i_n(t)$$

$$T(t)=\sum_{n=0}^{\infty}p^n \xi_n(t)\ R(t)=\sum_{n=0}^{\infty}p^n r_n(t)\quad B(t)=\sum_{n=0}^{\infty}p^n b_n(t) \tag{45}$$

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:

$$\begin{aligned}
&\frac{ds_{c0}(t)}{dt}=0,\ \frac{ds_{A0}(t)}{dt}=0,\ \frac{dv_0(t)}{dt}=0,\frac{di_0(t)}{dt}=0,\frac{d\xi_0(t)}{dt}=0\\
&\frac{dr_0(t)}{dt}=0,\ \frac{db_0(t)}{dt}=0
\end{aligned} \tag{46}$$

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

$$\left[\begin{array}{l}
\frac{ds_{c1}(t)}{dt}=(\Lambda_c-(1-w)\lambda_c b_0(t)[s_{c0}(t)+s_{A0}(t)]-(\mu+\psi_1)s_{c0}(t)-gs_{c0}(t)+\rho_1 v_0(t)) \\
\frac{ds_{A1}(t)}{dt}=(\Lambda_c-(1-w)\lambda_a b_0(t)[s_{c0}(t)+s_{A0}(t)]-(\mu+\psi_2)s_{a0}(t)-gs_{a0}(t)+\rho_2 v_0(t)) \\
\frac{dv_1(t)}{dt}=(\psi_1 s_{c0}(t)+\psi_2 s_{A0}(t)-(\rho_1+\rho_2)v_0(t)-\mu v_0(t)) \\
\frac{di_1(t)}{dt}=((1-w)b_0(t)[s_{c0}(t)+s_{A0}(t)](\lambda_c+\lambda_A)-(\mu+\tau+\delta)i_0(t)) \\
\frac{d\tau_1(t)}{dt}=(\tau i_0(t)-(\varepsilon+\mu)\xi_0(t)) \\
\frac{dr_1(t)}{dt}=\varepsilon\xi_0(t)-\mu r_0(t) \\
\frac{db_1(t)}{dt}=\eta(1-w)i_0(t)-\nu b_0(t)
\end{array}\right] \quad (47)$$

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

$$\left[\begin{array}{l}
\frac{ds_{c2}(t)}{dt}=\left(\begin{array}{l}\Lambda_c-(1-w)\lambda_c(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)])- \\ (\mu+\psi_1)s_{c1}(t)-gs_{c1}(t)+\rho_1 v_1(t)\end{array}\right) \\
\frac{ds_{A2}(t)}{dt}=\left(\begin{array}{l}\Lambda_a-(1-w)\lambda_a(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)])- \\ (\mu+\psi_2)s_{a1}(t)+gs_{c1}(t)+\rho_2 v_1(t)\end{array}\right) \\
\frac{dv_2(t)}{dt}=(\psi_1 s_{c1}(t)+\psi_2 s_{A1}(t)-(\rho_1+\rho_2)v_1(t)-\mu v_1(t)) \\
\frac{di_2(t)}{dt}=\left(\begin{array}{l}(1-w)(b_1(t)[s_{c0}(t)+s_{A0}(t)]+b_0(t)[s_{c1}(t)+s_{A1}(t)])(\lambda_c+\lambda_A)- \\ (\mu+\tau+\delta)i_1(t)\end{array}\right) \\
\frac{d\tau_2(t)}{dt}=(\tau i_1(t)-(\varepsilon+\mu)\xi_1(t)) \\
\frac{dr_2(t)}{dt}=(\varepsilon\xi_1(t)-\mu r_1(t)) \\
\frac{db_2(t)}{dt}=\eta(1-w)i_1(t)-\nu b_1(t)
\end{array}\right] \quad (48)$$

And so on. Solving system (3) 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

$$
\begin{aligned}
s_{c1}(t) &= \left(\Lambda_c - (1-w)\lambda_c b_0\left[s_{c0}+s_{A0}\right] - (\mu+\psi_1)s_{c0} - gs_{c0} + \rho_1 v_0\right)t \\
s_{A1}(t) &= \left(\Lambda_a - (1-w)\lambda_a b_0\left[s_{c0}+s_{A0}\right] - (\mu+\psi_2)s_{a0} + gs_{c0} + \rho_2 v_0\right)t \\
v_1(t) &= \left(\psi_1 s_{c0} + \psi_2 s_{A0} - (\rho_1+\rho_2)v_0 - \mu v_0\right)t \\
i_1(t) &= \left((1-w)b_0\left[s_{c0}+s_{A0}\right](\lambda_c+\lambda_A) - (\mu+\tau+\delta)i_0\right)t \\
\xi(t) &= \left(\tau i_0 - (\varepsilon+\mu)\xi_0\right)t \\
r_1(t) &= \left(\varepsilon\xi_0 - \mu r_0\right)t \\
b_1(t) &= \left(\eta(1-w)i_0 - \nu b_0\right)t
\end{aligned}
\tag{49}
$$

the second approximate solution is obtained:

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

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

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

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

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

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

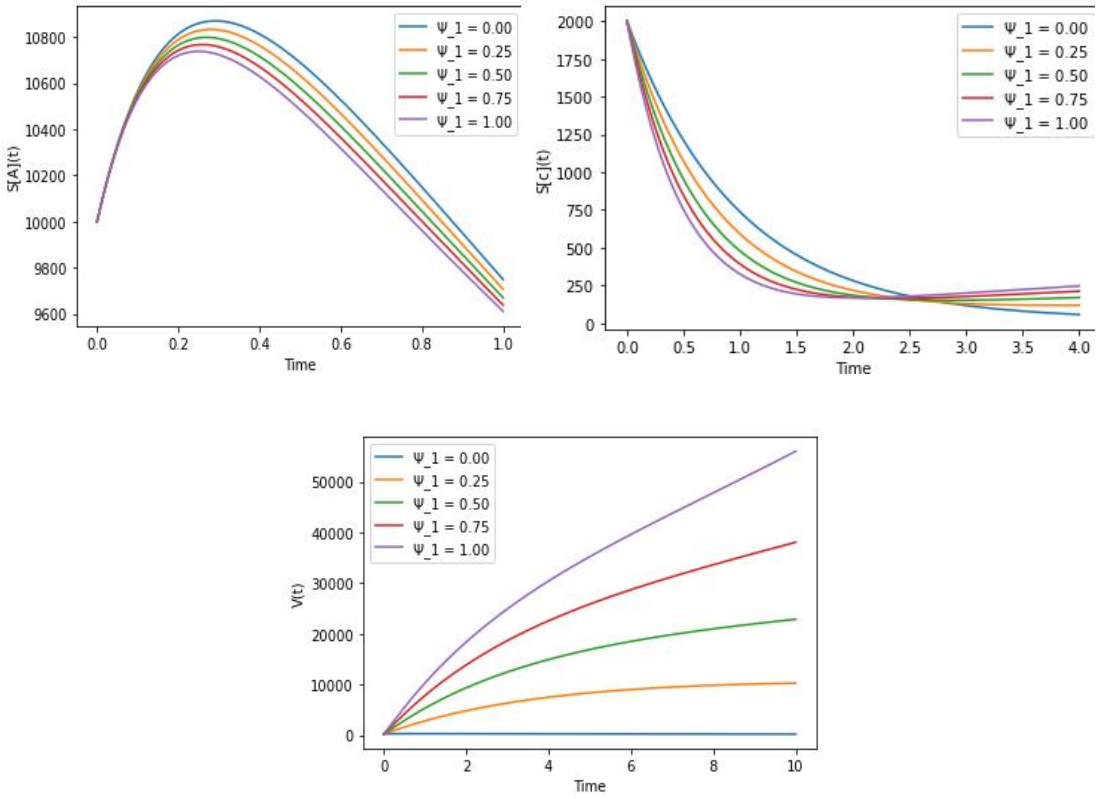
Summing these results up gives the approximate series solution of the system given by

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

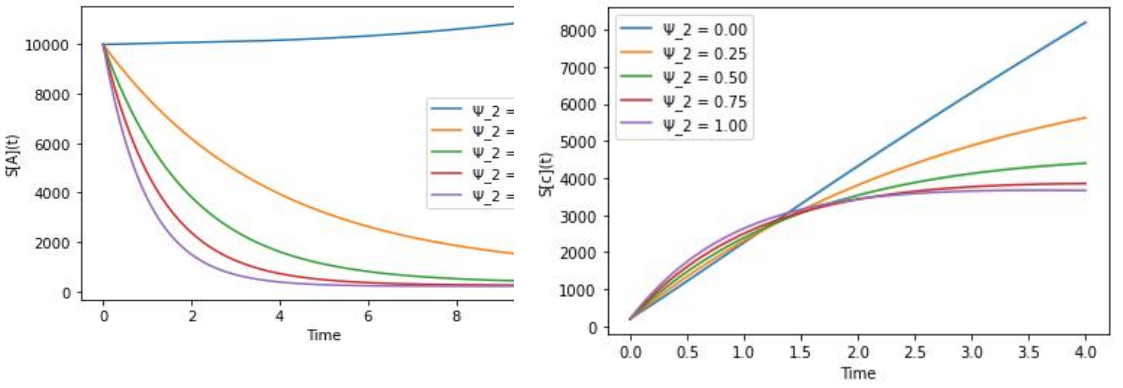
$$
T(t) = \sum_{n=0}^{2}\xi_n(t) \; R(t) = \sum_{n=0}^{2} r_n(t) \; B(t) = \sum_{n=0}^{2} b_n(t)
$$

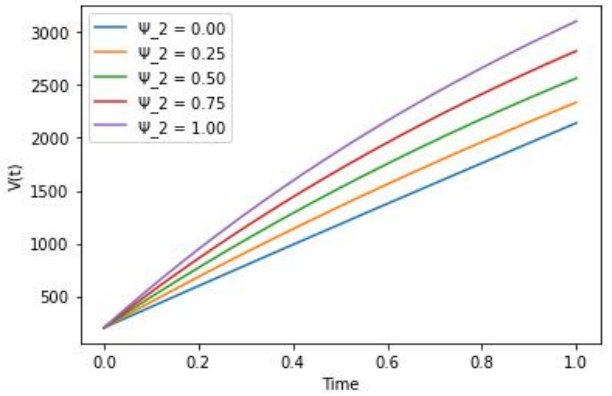
5. Result

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

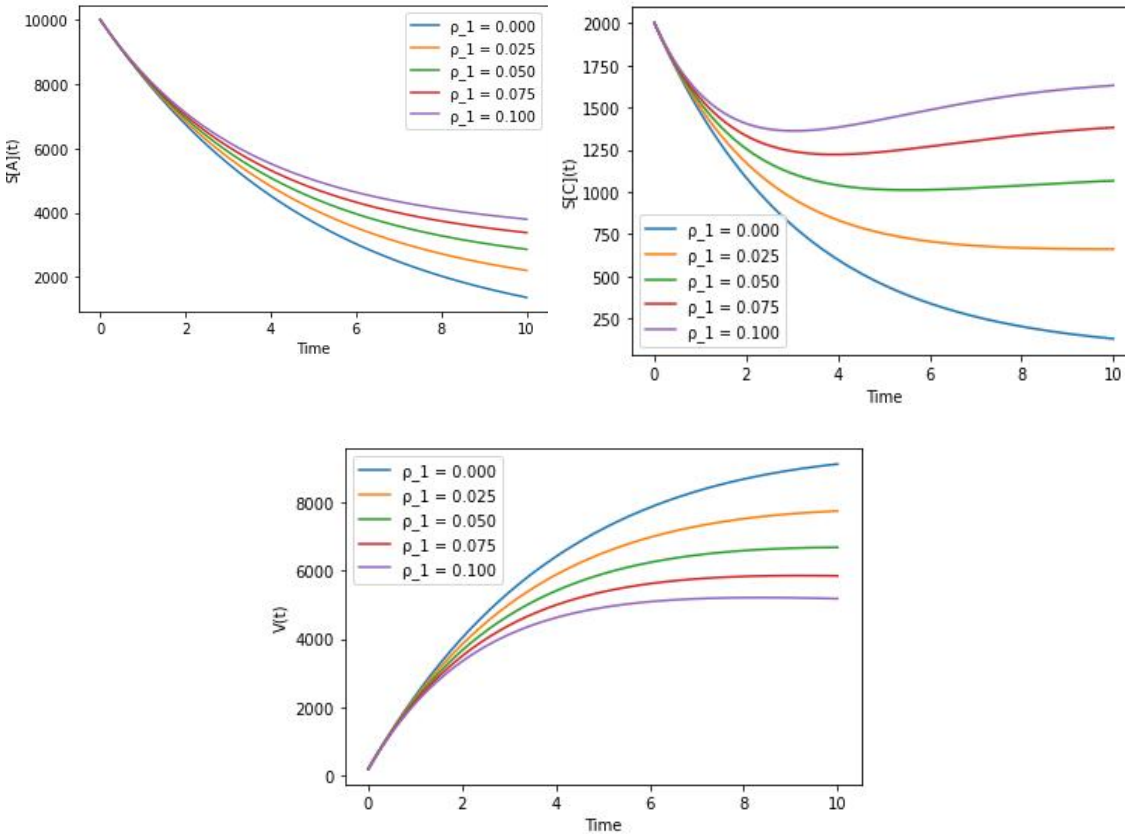


Figures 2. Impact of children vaccination on model variables

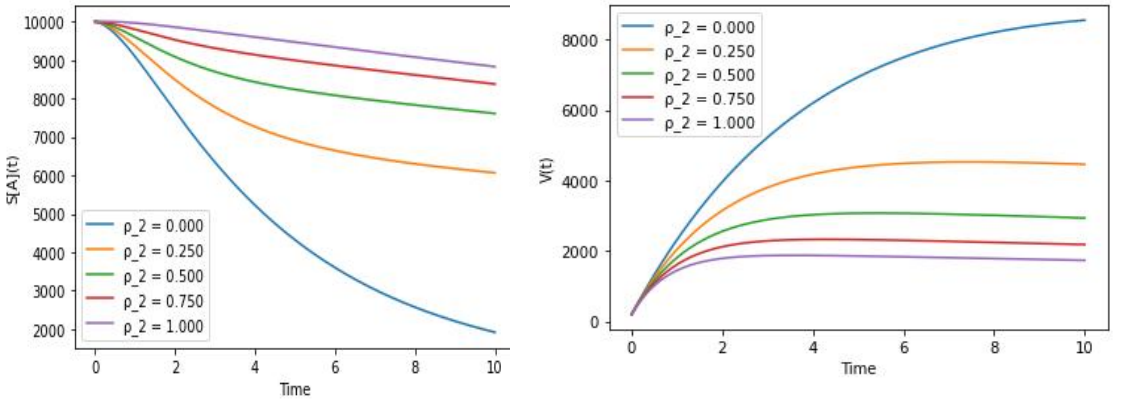




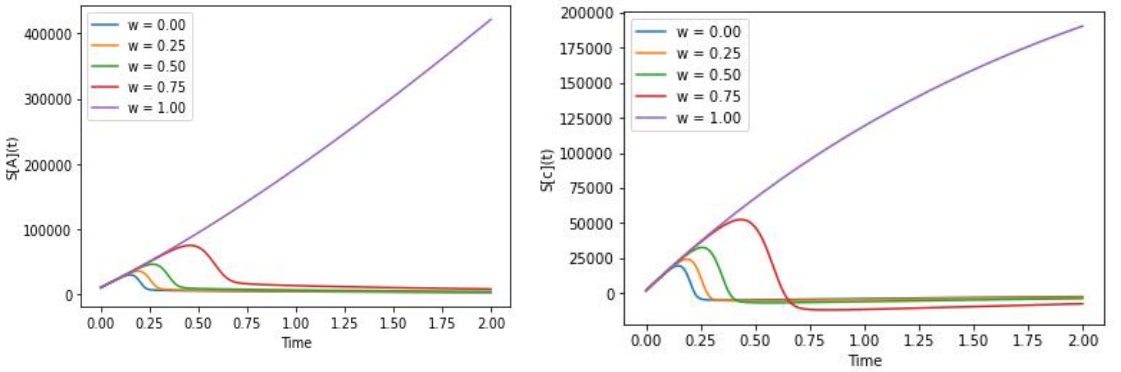
Figures 3. Impact of children vaccination on model variables



Figures 4. Impact of children vaccination on model variables



Figures 5: Impact of children vaccination on model variables



Figures 6. Impact of children vaccination on model variables

6. Discussion

The presented Figs. 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 (Figs. 2 and 3): Figs. 2 and 3 highlight the influence of vaccination rates on the various population groups. In both cases, the susceptible adult and children populations increase as the vaccination rate of children ψ_1 rises. This suggests that increasing the vaccination rate for children has a positive correlation with the susceptibility of both adult and child populations. Moreover, Figs. 2 demonstrate that the rise in the vaccination rate of adults (ψ_2) results in a faster increase in susceptibility for both adult and child populations compared to the effects of ψ_1. This underscores the importance of considering the different vaccination rates for distinct age groups when modeling disease dynamics.

Waning Immunity and Vaccine Boosters (Figs. 4 and 5): Figs. 3 and 4 explore the impact of waning immunity rates on the model variables. An increase in the waning immunity rate of children (ρ_1) leads to a rise in susceptible adult and child populations while concurrently reducing the vaccinated population. This emphasizes the necessity of vaccine boosters to counteract fast immunity waning, especially in

children. Similar trends are observed in Figs. 4, emphasizing the relevance of waning immunity rates for adults (ρ_2). The findings suggest that booster vaccinations may be crucial in maintaining effective immunity over time for both age groups.

Hygiene Dynamics (Fig. 6): Fig. 5 explores into the role of hygiene (hygienic rate "w") in the Typhoid model. As the hygiene rate increases, susceptibility levels for both adult and child populations (S_A and S_C) grow rapidly. Notably, the linear growth of susceptibility when "w" equals 1 suggests that heightened hygiene does not provide significant protection, and a considerable portion of the population remains exposed. This underscores the need for comprehensive hygiene practices to effectively mitigate the spread of the disease. In conclusion, the Figs. contribute valuable insights into the complex dynamics of the Typhoid model, emphasizing the importance of vaccination rates, waning immunity, and hygiene in shaping population susceptibility and the need for targeted interventions based on age groups and hygiene practices. The findings provide a basis for further exploration and refinement of public health strategies to control and prevent the spread of Typhoid.

7. Conclusion

In conclusion, the mathematical modeling of typhoid fever dynamics, incorporating age structure, vaccine, and treatment effects through the Homotopy Perturbation Method, offers valuable insights into the transmission and control of this infectious disease. By integrating these critical factors, the model provides a deeper understanding of disease spread and the effectiveness of intervention strategies, including vaccination and treatment. This comprehensive approach aids in making informed decisions for public health interventions, helping to reduce the burden of typhoid fever and improve population health. The ongoing refinement of such models, along with their validation using real-world data, is essential for optimizing their predictive accuracy and ensuring their relevance to real-life scenarios. Effective collaboration between mathematicians, epidemiologists, and public health professionals will further enhance the translation of these findings into actionable policies and interventions aimed at controlling typhoid fever and improving overall health outcomes.

8. Recommandation

This research underscores the importance of age-specific vaccination and treatment strategies in controlling typhoid fever. Public health policies should prioritize targeted interventions to enhance their effectiveness, especially in resource-constrained settings. Future research should focus on refining the model to account for environmental and socioeconomic factors influencing typhoid transmission. Additionally, incorporating real-world data on vaccine efficacy, treatment adherence, and behavioral changes could improve the model's predictive accuracy. Expanding the study to include co-infections or drug-resistant strains may further enhance its utility in guiding comprehensive disease management strategies.

Conflict of Interest

The authors declare that there is no conflicts of interest.

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