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A Deterministic Mathematical Model for the Transmission Dynamics of Typhoid Fever Incorporating Asymptomatic Carriers

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Abstract

Typhoid fever is a highly infectious disease, and remains a major public health problem, especially in low- and middle-income countries, despite several interventions such as environmental sanitation, personal hygiene, vaccination, and treatment with antibiotic drugs. Evidently, asymptomatic typhoid carriers play a critical role in the transmission dynamics of typhoid fever. Therefore, this research work presents a deterministic mathematical model on the dynamics and spread of typhoid fever incorporating asymptomatic carriers. The basic reproduction number, R_0 of the proposed model is computed using the next generation matrix approach, and the stability analyses of the disease-free equilibrium were investigated. Results show that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, and globally asymptotically stable when $R_0 \leq 1$.

Keywords: Typhoid, Mathematical Model, Asymptomatic carrier, Deterministic, Stability Analysis.

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1 Introduction

Typhoid fever is a communicable disease caused by the bacterium *Salmonella Typhi* (*S. Typhi*), and it spreads either through direct transmission from humans to humans or indirect transmission from the environment (contaminated food, water, drinks, etc) to humans (WHO, 2023; Tijani, Madubueze and Gweryina, 2023). After a person ingests *S. Typhi* bacteria, they multiply in the digestive system and spread into the bloodstream (WHO, 2023). Incubation period

for this infection varies with an average of 14 days, and the illness lasts approximately 4 to 6 weeks (Baisa and Kotola, 2024).

The clinical presentation of typhoid fever includes: a prolonged fever, loss of appetite, vomiting, weight loss, severe headache, general body pain, constipation, diarrhea, and a slight dry cough. In severe cases, it is presented with multiple complications like intestinal perforation, hemorrhage, and hepatitis (Muche, Tesfaw, and Bayou, 2024). It is worth noting that human beings have been identified as the exclusive natural hosts for this disease (Baisa and Kotola, 2024). This life-threatening infectious disease is curable, and can be prevented in the infected population by public health policies such as providing portable water, proper personal hygiene, improved sanitation, and poverty alleviation programs. However, asymptomatic typhoid carrier, which describes a case where a person harbours the *S. Typhi* that can transmit the disease but not show clinical symptoms, have been reported as one of the challenges in the fight against typhoid fever (Uwanibe, Kayode, Oluniyi, Akano, Olawoye, Ugwu, Happi and Folarin, 2023).

Asymptomatic typhoid carriers, which often go undetected, play a critical role in typhoid fever burden and may serve as reservoirs for the bacteria, especially in the endemic area. Accordingly, asymptomatic typhoid carriers contribute to the spread of the bacteria within their local environment, resulting in the continuous transmission of typhoid in the human population. As pointed out by Uwanibe et al. (2023), this persistent transmission within the host population can significantly hinder the attainment of the Sustainable Development Goal (SDG) of healthy lives and promoting well-being for all. Typhoid fever remains a substantial cause of morbidity and mortality, especially in low- and middle-income countries, despite several interventions such as environmental sanitation, vaccination, and treatment with antibiotic drugs. According to the Centers for Disease Control and Prevention (2023), globally, an estimated 11–21 million cases of typhoid fever occur annually, resulting in an estimated 135,000 – 230,000 deaths. Asymptomatic carriers play a critical role in the transmission and persistence of typhoid fever. This continuous transmission of *Salmonelle Typhi* to the environment (food, water, drinks, etc) and human host hinders control measures and possible elimination of the disease.

Numerous mathematical models have been proposed to gain insights into the transmission dynamics of infectious diseases (Akerejola, 2026; Akerejola et al., 2024; Elakhe et al., 2023a; Elakhe et al., 2023b). Suhuyini and Seidu, (2023) formulated a mathematical model on the transmission dynamics of typhoid disease incorporating treatment and booster vaccination. The next-generation matrix approach was employed to derive the model's basic reproduction number R_0. Sensitivity analysis of the model parameters shows that the most sensitive parameters include transmission rate and recruitment rate. The numerical simulations reveal that increasing the rate of vaccine administration has the potential to reduce typhoid infections. The study, however, concludes that booster vaccination may not be beneficial in endemic regions. An optimal control of a mathematical model for the transmission dynamics of typhoid fever was developed by Lawal, Yusuf and Abidemi, (2024). The model incorporates medical treatment and environmental sanitation with personal hygiene practices as control interventions. The study examines the combined impact of these control measures on typhoid fever

prevention and control. Findings from the research show that a combination of treatment and environmental sanitation with personal hygiene is most efficient in curbing the spread of the disease.

Tsafack et al. (2025) proposed a mathematical model for typhoid fever disease that incorporates the use of modern and traditional medicines as modes of treatment in a health district in Cameroon. They proved the global asymptotic stability of the disease-free equilibrium (DFE) and the endemic equilibrium (EE) when the basic reproduction number (R_0) is less than one and greater than one, respectively. A sensitivity analysis with respect to the basic reproduction number was conducted. The study concludes that the environmental transmission rate and the decay rate of bacteria in the environment were the most sensitive parameters on R_0. To the best of our knowledge, no typhoid model has considered the impact of asymptomatic carriers on the transmission dynamics of typhoid fever. So, this paper proposes a novel deterministic mathematical model on the transmission dynamics and spread of typhoid fever, by subdividing the infected human population into two mutually disjoint sub-populations: the symptomatic infected human population, and the asymptomatic infected human population. The basic reproduction number, R_0 of the proposed model is computed using the next-generation matrix approach, and the stability analyses of the disease-free equilibrium were investigated.

The rest of this paper is organized as follows: Formulation of the deterministic model and analysis of the basic properties of the model are presented in Section 2. Section 3 covers a rigorous study of the local and global asymptotic stability of the disease-free equilibrium of the model, while Section 4 contains the conclusion.

2 Model Formulation

In this study, the total human population at time t, denoted by $N(t)$, is divided into five epidemiological compartments, namely: susceptible individuals $S(t)$, exposed individuals $E(t)$, symptomatic infectious individuals $I_s(t)$, asymptomatic infectious individuals $I_a(t)$, and recovered individuals $R(t)$. Thus, the total human population is given by

$$N(t) = S(t) + E(t) + I_s(t) + I_a(t) + R(t)$$

The environmental concentration of *Salmonella Typhi* bacteria at time t is represented by $B(t)$. Susceptible individuals become infected either through direct contact with infectious individuals or through indirect contact with contaminated environmental reservoirs such as food, water, and drinks. The force of infection due to environmental contamination is denoted by

$$\lambda_1 = \frac{\beta_3 B}{k + B}$$

while the force of infection arising from contact with symptomatic and asymptomatic infectious individuals is given by

$$\lambda_2 = \frac{\beta_1 I_s + \beta_2 I_a}{N}$$

Here, β_1 and β_2 represent the effective contact rates between susceptible individuals and symptomatic and asymptomatic infectious individuals, respectively, whereas β_3 denotes the transmission coefficient associated with environmental contamination. The parameter k represents the bacterial concentration that produces a 50% probability of infection.

Individuals enter the susceptible class through recruitment at a rate Λ. Susceptible individuals become infected at a rate $(\lambda_1 + \lambda_2)S$ and move into the exposed class $E(t)$. Recovered individuals lose their acquired immunity at a rate ψ and return to the susceptible class. Natural death occurs in all human compartments at a rate μ.

The exposed population consists of individuals who have acquired infection but are not yet infectious. Exposed individuals progress to the infectious stage at a rate θ. A proportion ρ, $(0 \leq \rho \leq 1)$ of exposed individuals develops symptomatic infection and enters the symptomatic infectious compartment $I_s(t)$, while the remaining proportion $(1-\rho)$ progresses to the asymptomatic infectious compartment $I_a(t)$.

Individuals in the symptomatic infectious class contribute to disease transmission and shed bacteria into the environment at a rate α_1. They recover at a rate τ and move to the recovered compartment. In addition to natural mortality, symptomatic infectious individuals may experience disease-induced mortality at a rate δ.

Similarly, asymptomatic infectious individuals contribute to disease transmission and contaminate the environment at a rate α_2. These individuals recover at a rate ε and move to the recovered compartment. The recovered population acquires temporary immunity, which wanes at a rate ψ, resulting in a return to the susceptible class.

The environmental bacterial population increases due to bacterial shedding from symptomatic and asymptomatic infectious individuals at rates $\alpha_1 I_s$ and $\alpha_2 I_a$, respectively. The bacteria undergo natural decay at a rate ϕB. Consequently, the environment serves as a reservoir for the persistence and spread of typhoid fever within the population.

Model Equations

Based on the transmission pathways described above, the dynamics of typhoid fever are governed by the following system of nonlinear ordinary differential equations:

Table 1: Description of Model Parameters

Parameter	Description
Λ	Recruitment rate of the human population
μ	Natural death rate of humans
β_1	Effective contact rate between susceptible and symptomatic infectious individuals
β_2	Effective contact rate between susceptible and asymptomatic infectious individuals
β_3	The effective contact rate between susceptible individuals and the contaminated environment
k	Bacterial concentration corresponding to 50% probability of infection
δ	Disease-induced death rate
ψ	Rate of loss of acquired immunity
θ	Progression rate from the exposed class to infectious classes
τ	Recovery rate of symptomatic infectious individuals
ε	Recovery rate of asymptomatic infectious individuals
α_1	Bacterial shedding rate from symptomatic infectious individuals
α_2	Bacterial shedding rate from asymptomatic infectious individuals
ϕ	Bacterial decay rate in the environment
ρ	Proportion of exposed individuals progressing to symptomatic infection

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \psi R - (\lambda_1 + \lambda_2 + \mu)S, \\
\frac{dE}{dt} &= (\lambda_1 + \lambda_2)S - (\theta + \mu)E, \\
\frac{dI_s}{dt} &= \rho\theta E - (\tau + \mu + \delta)I_s, \\
\frac{dI_a}{dt} &= (1-\rho)\theta E - (\varepsilon + \mu)I_a, \\
\frac{dR}{dt} &= \tau I_s + \varepsilon I_a - (\psi + \mu)R, \\
\frac{dB}{dt} &= \alpha_1 I_s + \alpha_2 I_a - \phi B.
\end{aligned} \tag{1}$$

with the following initial conditions;

$$S(0) > 0, \quad E(0) \geq 0, \quad I_s(0) > 0, \quad I_a(0) \geq 0, \quad R(0) \geq 0, \quad B(0) > 0 \tag{2}$$

Model equation (1) constitute the deterministic typhoid fever model with symptomatic and asymptomatic infectious classes and an environmental bacterial reservoir. The model captures both direct human-to-human transmission and indirect transmission through contaminated environmental sources, thereby providing a comprehensive framework for studying the transmission dynamics and control of typhoid fever.

2.1 Invariant Region and Positivity of Solutions

Theorem 2.1. The model system (1) has solutions which are contained in the feasible region

$$\Omega = \Omega_H \times \Omega_B,$$

where

$$\Omega_H = \left\{ (S, E, I_s, I_a, R) \in \mathbb{R}_+^5 : N(t) \leq \frac{\Lambda}{\mu} \right\},$$

and

$$\Omega_B = \left\{ B \in \mathbb{R}_+ : B(t) \leq \frac{\Lambda(\alpha_1 + \alpha_2)}{\mu\phi} \right\},$$

are respectively subset for the human and bacteria population, provided $\phi > r$.

Proof.

From the differential equations (1), we have

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I_s$$

In the absence of the disease ($\delta = 0$);

$$\frac{dN}{dt} \leq \Lambda - \mu N \tag{3}$$

Applying the method of integrating factors to equation (3) at $N(0) = N_0$, we have;

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu} \right) e^{-\mu t} \tag{4}$$

$$N(t) \leq \frac{\Lambda}{\mu} \quad \text{as } t \to \infty$$

in equation (4), suggesting that the feasible solutions of the human population are in the region,

$$\Omega_H = \left\{ (S, E, I_s, I_a, R) \in \mathbb{R}_+^5 : N(t) \leq \frac{\Lambda}{\mu} \right\}.$$

Observe that

$$I_s(t) + I_a(t) \leq N(t) \leq \frac{\Lambda}{\mu},$$

therefore, the last equation of model (1) can be written as;

$$\frac{dB(t)}{dt} \leq \frac{\Lambda}{\mu}(\alpha_1 + \alpha_2) - \phi B \tag{5}$$

Applying the method of integrating factor to equation (5) at $B(0) = B_0$ gives;

$$B(t) \leq \frac{\Lambda(\alpha_1+\alpha_2)}{\mu\phi} + \left(B_0 - \frac{\Lambda(\alpha_1+\alpha_2)}{\mu\phi}\right) e^{-\phi t} \tag{6}$$

as $t \to \infty$ in equation (6),

$$B(t) \leq \frac{\Lambda(\alpha_1+\alpha_2)}{\mu\phi}.$$

Hence, the feasible solution for the bacteria population enters the region,

$$\Omega_B = \left\{ B \in \mathbb{R}_+ : B(t) \leq \frac{\Lambda(\alpha_1+\alpha_2)}{\mu\phi} \right\}.$$

Therefore, all feasible solutions of model system (1) enter the region

$$\Omega = \Omega_H \times \Omega_B.$$

Hence, Ω is positively invariant and model system (1) is biologically meaningful and also mathematically well-posed in Ω.

Theorem 2.2. Let the initial conditions be

$$\{S(0) > 0,\ E(0) \geq 0,\ I_s(0) > 0,\ I_a(0) \geq 0,\ R(0) \geq 0,\ B(0) > 0\} \in \Omega,$$

then the solution set

$$\{S, E, I_s, I_a, R, B\}(t)$$

of the model equations (1) is positive for all $t \geq 0$.

Proof:

From the first equation of model system (1), we have;

$$\frac{dS}{dt} = \Lambda + \psi R - (\lambda_1 + \lambda_2 + \mu)S$$

where

$$\lambda_1 = \frac{\beta_3 B}{k+B}, \qquad \lambda_2 = \frac{\beta_1 I_s + \beta_2 I_a}{N}.$$

$$\frac{dS}{dt} \geq -(\lambda_1 + \lambda_2 + \mu)S \tag{7}$$

Solving equation (7) at $t = 0$ gives;

$$S(t) \geq S(0)e^{\left[-\int(\lambda_1+\lambda_2)\,dt+\mu t\right]} \geq 0, \qquad \text{since } e^x > 0,\ \forall x \in \mathbb{R} \tag{8}$$

Similarly, from the second equation of model system (1), that is;

$$\frac{dE}{dt} = (\lambda_1 + \lambda_2)S - (\theta + \mu)E,$$

which implies that;

$$\frac{dE}{dt} \geq -(\theta + \mu)E \tag{9}$$

Solving equation (9) at $t = 0$ yields;

$$E(t) \geq E(0)e^{-(\theta+\mu)t} \geq 0 \tag{10}$$

It can be shown, following the above procedure, that the solutions of the remaining equations of the model system (1) remain positive for all $t \geq 0$. Hence, this completes the proof.

2.2 Disease-Free Equilibrium (DFE) and Basic Reproduction Number, R_0

The disease-free equilibrium state is denoted by E_0 is when all infected and infectious states are equal to zero, and is obtained by setting the right-hand side of model system (1) to zero. Thus, the corresponding disease-free equilibrium point is:

$$E_0 = (S^*, E^*, I_s^*, I_a^*, R^*, B^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right) \tag{11}$$

The basic reproduction number given by R_0, is a threshold parameter used in epidemiology to study the behaviour of mathematical models. It represents the average number of secondary infections generated by one infectious individual in a completely susceptible population at any time t, and it is defined mathematically as

$$R_0 = \rho(A),$$

spectral radius of a matrix A. Here, matrix

$$A = FV^{-1},$$

where

$$F = \left(\frac{\partial F_i(E_0)}{\partial x_i}\right) \quad \text{and} \quad V = \left(\frac{\partial V_i(E_0)}{\partial x_i}\right)$$

are respectively the transmission and transition matrices obtained at the disease-free equilibrium, E_0. F_i denotes the appearance rate of new infections in compartments i while V_i represents the transfer rate of infections from and/or into the compartment i by all other means. Thus,

$$F = \begin{pmatrix} 0 & \beta_1 & \beta_2 & \dfrac{\beta_3\Lambda}{k\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} (\theta+\mu) & 0 & 0 & 0 \\ -\rho\theta & (\tau+\mu+\delta) & 0 & 0 \\ -(1-\rho)\theta & 0 & (\varepsilon+\mu) & 0 \\ 0 & -\alpha_1 & -\alpha_2 & \phi \end{pmatrix}.$$

Hence, the basic reproduction number R_0 for model system (1) is given as;

$$R_0 = \rho(FV^{-1}) = \frac{\beta_1\rho\theta k\mu}{(\theta+\mu)(\tau+\mu+\delta)} + \frac{\beta_2(1-\rho)\theta k\mu}{(\theta+\mu)(\varepsilon+\mu)} + \frac{\beta_3\Lambda\left[\rho\theta\alpha_1(\varepsilon+\mu)+(1-\rho)\theta\alpha_2(\tau+\mu+\delta)\right]}{k\mu\phi(\theta+\mu)(\tau+\mu+\delta)(\varepsilon+\mu)}$$

$$R_0 = \frac{\beta_1\rho\theta k\mu\phi(\varepsilon+\mu) + \beta_2(1-\rho)\theta k\mu\phi(\tau+\mu+\delta) + \beta_3\Lambda\left[\rho\theta\alpha_1(\varepsilon+\mu)+(1-\rho)\theta\alpha_2(\tau+\mu+\delta)\right]}{k\mu\phi(\theta+\mu)(\tau+\mu+\delta)(\varepsilon+\mu)} \tag{12}$$

Let $(R_0)'$ denotes a case where asymptomatic carriers are excluded from the population, then in the absence of asymptomatic population, $\rho = 1$; hence,

$$(R_0)' = \frac{\beta_1\theta k\mu\phi(\varepsilon+\mu) + \beta_3\Lambda\theta\alpha_1(\varepsilon+\mu)}{k\mu\phi(\theta+\mu)(\tau+\mu+\delta)(\varepsilon+\mu)} \tag{13}$$

which follows that

$$R_0 > (R_0)'.$$

Hence, it can be seen that R_0 increases when there are asymptomatic individuals capable of transmitting the disease.

3 Stability of the Disease-Free Equilibrium

Theorem 3.1. The disease-free equilibrium, E_0 for model system (1) is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable otherwise.

Proof:

The Jacobian matrix of the model system (1) evaluated at the disease-free equilibrium, E_0 denoted by $J_0[E_0]$ is given as:

$$J_0[E_0] = \begin{pmatrix} -\mu & 0 & -\beta_1 & -\beta_2 & \psi & -\dfrac{\beta_3\Lambda}{k\mu} \\ 0 & -(\theta+\mu) & \beta_1 & \beta_2 & 0 & \dfrac{\beta_3\Lambda}{k\mu} \\ 0 & \rho\theta & -(\tau+\mu+\delta) & 0 & 0 & 0 \\ 0 & (1-\rho)\theta & 0 & -(\varepsilon+\mu) & 0 & 0 \\ 0 & 0 & \tau & \varepsilon & -(\psi+\mu) & 0 \\ 0 & 0 & \alpha_1 & \alpha_2 & 0 & -\phi \end{pmatrix}$$

We need to show that all the eigenvalues of $J_0[E_0]$ have negative real parts. The first column of $J_0[E_0]$ contains only the diagonal term, $-\mu$, which form the first eigenvalue. Therefore,

$$J_1[E_0] = \begin{pmatrix} -(\theta+\mu) & \beta_1 & \beta_2 & 0 & \dfrac{\beta_3\Lambda}{k\mu} \\ \rho\theta & -(\tau+\mu+\delta) & 0 & 0 & 0 \\ (1-\rho)\theta & 0 & -(\varepsilon+\mu) & 0 & 0 \\ 0 & \tau & \varepsilon & -(\psi+\mu) & 0 \\ 0 & \alpha_1 & \alpha_2 & 0 & -\phi \end{pmatrix}$$

Similarly, the fourth column of $J_1[E_0]$ contains only the diagonal term, $-(\psi+\mu)$, which forms a negative eigenvalue. Hence;

$$J_2[E_0] = \begin{pmatrix} -(\theta+\mu) & \beta_1 & \beta_2 & \dfrac{\beta_3\Lambda}{k\mu} \\ \rho\theta & -(\tau+\mu+\delta) & 0 & 0 \\ (1-\rho)\theta & 0 & -(\varepsilon+\mu) & 0 \\ 0 & \alpha_1 & \alpha_2 & -\phi \end{pmatrix}$$

The remaining eigenvalues are obtained from the sub–matrix $J_2[E_0]$. Obviously, the eigenvalues of the matrix $J_2[E_0]$ are the roots of the characteristic equation:

$$|J_2[E_0] - \lambda I| = 0 \tag{14}$$

That is;

$$\begin{vmatrix} -(\theta+\mu)-\lambda & \beta_1 & \beta_2 & \dfrac{\beta_3\Lambda}{k\mu} \\ \rho\theta & -(\tau+\mu+\delta)-\lambda & 0 & 0 \\ (1-\rho)\theta & 0 & -(\varepsilon+\mu)-\lambda & 0 \\ 0 & \alpha_1 & \alpha_2 & -\phi-\lambda \end{vmatrix} = 0 \tag{15}$$

Let

$$a_1 = (\theta + \mu), \qquad a_2 = (\tau + \mu + \delta), \qquad a_3 = (\varepsilon + \mu), \qquad a_4 = \phi,$$

$$a_5 = a_6 = \beta_2, \qquad a_7 = \frac{\beta\Lambda}{k\mu}, \qquad a_8 = \rho\theta, \qquad a_9 = (1-\rho)\theta,$$

$$a_{10} = \alpha_1, \qquad a_{11} = \alpha_2.$$

Equation (15) reduces to;

$$A_0\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 = 0 \tag{16}$$

where;

$$A_0 = 1$$

$$A_1 = a_1 + a_2 + a_3 + a_4$$

$$A_2 = a_1a_2 + a_1a_3 + a_1a_4 + a_2a_3 + a_2a_4 + a_3a_4 - a_5a_8 - a_6a_9$$

$$A_3 = a_1a_2a_3 + a_1a_2a_4 + a_1a_3a_4 + a_2a_3a_4 - (a_2a_6a_9 + a_3a_5a_8) - (a_4a_5a_8 + a_7a_8a_{10}) - (a_4a_6a_9 + a_7a_9a_{11})$$

$$A_4 = a_1a_2a_3a_4 - a_2a_4a_6a_9 - a_3a_4a_5a_8 - a_3a_7a_8a_{10} - a_2a_7a_9a_{11}$$

$$A_4 = a_1a_2a_3a_4\left[1 - \frac{a_2a_4a_6a_9 + a_3a_4a_5a_8 + a_3a_7a_8a_{10} + a_2a_7a_9a_{11}}{a_1a_2a_3a_4}\right]$$

In terms of R_0, further simplification of A_4 yields;

$$A_4 = a_1a_2a_3a_4(1 - R_0) \tag{17}$$

The expression for R_0 in terms of a_i's can be written as;

$$R_0 = \frac{a_2a_4a_6a_9 + a_3a_4a_5a_8 + a_3a_7a_8a_{10} + a_2a_7a_9a_{11}}{a_1a_2a_3a_4}$$

Obviously, $A_4 > 0$ if and only if $R_0 < 1$, which implies that;

$$a_1a_2a_3a_4 > a_2a_4a_6a_9 + a_3a_4a_5a_8 + a_3a_7a_8a_{10} + a_2a_7a_9a_{11} \tag{18}$$

From (18), we have;

$$\left.\begin{aligned} a_1a_2 &> a_5a_8, \\ a_1a_3 &> a_6a_9, \\ a_1a_2a_3 &> a_2a_6a_9 + a_3a_5a_8, \\ a_1a_2a_4 &> a_4a_5a_8 + a_7a_8a_{10}, \\ a_1a_3a_4 &> a_4a_6a_9 + a_7a_9a_{11} \end{aligned}\right\} \tag{19}$$

By the inequalities in equation (19), it follows that $A_2, A_3 > 0$, hence $A_i > 0$ for $i = 0, 1, 2, 3, 4$ since all a_i's are positive.

To prove that all roots of equation (16) have negative real parts, we employ the Routh-Hurwitz criterion, which states that all roots of the polynomial (16) have negative real parts if and only if the coefficients A_i, $i = 0, 1, 2, 3, 4$ and the determinant of the matrices, H_i, $i = 0, 1, 2, 3, 4$ are positive.

Using Lienard-Chipart criteria, we only need to show that $|H_3| > 0$ (see Daud M.A.A, 2021), and it can be shown that for $R_0 < 1$,

$$|H_3| = a_1a_2a_3 - a_3^2 + a_1^2a_4 > 0.$$

Hence, all the roots of the characteristic polynomial equation (16) have negative real parts, which implies that the disease–free equilibrium point is locally asymptotically stable when $R_0 < 1$. However, if $R_0 > 1$ and by Descartes' rule of signs, we observed there is a positive eigenvalue, which indicates that the disease–free equilibrium point will be unstable.

Theorem 3.2. The disease-free equilibrium, E_0 for model system (1) is globally asymptotically stable (GAS) if $R_0 \leq 1$, and unstable otherwise.

Proof:

We prove the global asymptotic stability for a special case ($\delta_h = 0$ and $\psi = 0$) of the disease-free equilibrium of model (1) following the method in Castillo-Chavez et al., (2002), Collins and Duffy, (2022). The model system (1) is transformed as follows:

$$\frac{dP_1}{dt} = Y_1(P_1, P_2) \tag{20}$$

$$\frac{dP_2}{dt} = Y_2(P_1, P_2), \qquad Y_2(P_1, 0) = 0 \tag{21}$$

where the components of $P_1 \in \mathbb{R}^2$ denote the uninfected population and the components of $P_2 \in \mathbb{R}^4$ denote the infected population.

The disease-free equilibrium point of the model system (1) is given by

$$E_0 = (P_1^0, 0).$$

The fixed point $E_0 = (P_1^0, 0)$ is a globally asymptotically stable equilibrium for system (1) provided $R_0 \leq 1$ and the below criteria are satisfied.

$$(H1) \quad \frac{dP_1}{dt} = Y_1(P_1, 0), \qquad P_1^0 \text{ is globally asymptotically stable}$$

$$(H2) \quad Y_2(P_1, P_2) = MP_2 - \hat{Y}_2(P_1, P_2), \qquad \hat{Y}_2(P_1, P_2) \geq 0$$

for $(P_1, P_2) \in \Omega$, where the Jacobian

$$M = \frac{\partial Y_2}{\partial P_2}(P_1, 0)$$

is an M-matrix (the off diagonal elements of M are non-negative) and Ω is a positively invariant attracting domain.

From the model system (1), we have that;

$$\frac{dP_1}{dt} = Y_1(P_1, 0) = \begin{pmatrix} \Lambda - \mu S \\ -\mu R \end{pmatrix} \tag{22}$$

From equation (22), observe that $Y_1(P_1, 0)$ is a system of linear ordinary differential equations and solving it gives;

$$\left.\begin{aligned} S(t) &= \frac{\Lambda}{\mu} + C_1 e^{-\mu t}, \\ R(t) &= C_2 e^{-\mu t} \end{aligned}\right\} \tag{23}$$

where C_1 and C_2 are constants, thus,

$$(S(t), R(t)) \to \left(\frac{\Lambda}{\mu}, 0\right) \quad \text{as } t \to \infty.$$

Hence P_1^0 is globally asymptotically stable, which verifies the first condition (H1).

Next, we show that the second condition (H2) holds. From model system (1), we have that;

$$Y_2(P_1, P_2) = \begin{pmatrix} \left(\frac{\beta_3 B}{k+B} + \frac{\beta_1 I_s + \beta_2 I_a}{N}\right) S - (\theta + \mu)E \\ \rho\theta E - (\tau + \mu + \delta) I_s \\ (1-\rho)\theta E - (\varepsilon + \mu) I_a \\ \alpha_1 I_s + \alpha_2 I_a - \phi B \end{pmatrix} = MP_2 - \hat{Y}_2(P_1, P_2) \tag{24}$$

where

$$M = \begin{pmatrix} -(\theta + \mu) & \beta_1 & \beta_2 & \dfrac{\beta_3 \Lambda}{k\mu} \\ \rho\theta & -(\tau + \mu + \delta) & 0 & 0 \\ (1-\rho)\theta & 0 & -(\varepsilon + \mu) & 0 \\ 0 & \alpha_1 & \alpha_2 & -\phi \end{pmatrix},$$

$$P_2 = \begin{pmatrix} E \\ I_s \\ I_a \\ B \end{pmatrix}$$

and

$$\hat{Y}_2(P_1, P_2) = \begin{pmatrix} \frac{\beta_3 \Lambda B}{k\mu}\left(1 - \frac{k}{k+B}\right) + \beta_1 I_s\left(1 - \frac{S}{N}\right) + \beta_2 I_a\left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Since,

$$\frac{k}{k+B} \leq 1 \qquad \text{and} \qquad \frac{S}{N} \leq 1,$$

it follows that

$$\hat{Y}_2(P_1, P_2) \geq 0.$$

Clearly, matrix M is an M-matrix with the off-diagonal entries positive, which satisfies the second condition (H2). Hence, the disease-free equilibrium E_0 is globally asymptotically stable if $R_0 \leq 1$ and unstable otherwise.

3.1 Bifurcation Analysis

We consider model system (1), and use the center manifold theory approach introduced by Castillo-Chavez & Song (2004) to investigate the nature of the bifurcation involving the disease-free equilibrium point E_0 at $R_0 = 1$ for the special case $\psi = 0$.

From the model (1) above, let

$$S = x_1, \qquad E = x_2, \qquad I_s = x_3, \qquad I_a = x_4, \qquad R = x_5, \qquad B = x_6$$

and scaling the transmission parameters, we have;

$$\begin{cases} h_1 = \dfrac{dx_1}{dt} = \Lambda - (\lambda_1 + \lambda_2 + \mu)x_1, \\ h_2 = \dfrac{dx_2}{dt} = (\lambda_1 + \lambda_2)x_1 - (\theta + \mu)x_2, \\ h_3 = \dfrac{dx_3}{dt} = \rho\theta x_2 - (\tau + \mu + \delta)x_3, \\ h_4 = \dfrac{dx_4}{dt} = (1-\rho)\theta x_2 - (\varepsilon + \mu)x_4, \\ h_5 = \dfrac{dx_5}{dt} = \tau x_3 + \varepsilon x_4 - \mu x_5, \\ h_6 = \dfrac{dx_6}{dt} = \alpha_1 x_3 + \alpha_2 x_4 - \phi x_6. \end{cases} \tag{25}$$

where

$$\lambda_1 = \frac{\lambda\beta_3 x_6}{k + x_6}, \qquad \lambda_2 = \frac{\lambda\beta_1 x_3 + \lambda\beta_2 x_4}{N}.$$

$$R_0(\lambda) = \frac{\lambda\left[\beta_1\rho\theta k\mu\phi(\varepsilon+\mu) + \beta_2(1-\rho)\theta k\mu\phi(\tau+\mu+\delta) + \beta_3\Lambda\Big(\rho\theta\alpha_1(\varepsilon+\mu) + (1-\rho)\theta\alpha_2(\tau+\mu+\delta)\Big)\right]}{k\mu\phi(\theta+\mu)(\tau+\mu+\delta)(\varepsilon+\mu)}$$

Let

$$Z_1 = (\theta+\mu), \qquad Z_2 = (\tau+\mu+\delta), \qquad Z_3 = (\varepsilon+\mu),$$

then;

$$R_0(\lambda) = \frac{\lambda\left[\beta_1\rho\theta k\mu\phi Z_3 + \beta_2(1-\rho)\theta k\mu\phi Z_2 + \beta_3\Lambda\left(\rho\theta\alpha_1 Z_3 + (1-\rho)\theta\alpha_2 Z_2\right)\right]}{k\mu\phi Z_1 Z_2 Z_3}.$$

Let $\lambda = \lambda^*$ be the bifurcation parameter, at $R_0 = 1$, we have;

$$\lambda^* = \frac{k\mu\phi Z_1 Z_2 Z_3}{\beta_1\rho\theta k\mu\phi Z_3 + \beta_2(1-\rho)\theta k\mu\phi Z_2 + \beta_3\Lambda\left[\rho\theta\alpha_1 Z_3 + (1-\rho)\theta\alpha_2 Z_2\right]}.$$

The Jacobian matrix of model system (1) at the disease-free equilibrium is given by;

$$J[E_0,\lambda^*]=\begin{pmatrix} -\mu & 0 & -\lambda^*\beta_1 & -\lambda^*\beta_2 & 0 & -\dfrac{\lambda^*\beta_3\Lambda}{k\mu} \\ 0 & -Z_1 & \lambda^*\beta_1 & \lambda^*\beta_2 & 0 & \dfrac{\lambda^*\beta_3\Lambda}{k\mu} \\ 0 & \rho\theta & -Z_2 & 0 & 0 & 0 \\ 0 & (1-\rho)\theta & 0 & -Z_3 & 0 & 0 \\ 0 & 0 & \tau & \varepsilon & -\mu & 0 \\ 0 & 0 & \alpha_1 & \alpha_2 & 0 & -\phi \end{pmatrix}.$$

Let the right and left components of the eigenvectors of the matrix $J[E_0,\lambda^*]$ associated with the zero eigenvalue, be respectively given as

$$P=(p_1,p_2,p_3,p_4,p_5,p_6)^T$$

and

$$Q=(q_1,q_2,q_3,q_4,q_5,q_6),$$

then considering the equation

$$J[E_0,\lambda^*]\cdot P=0,$$

we have;

$$\begin{cases} -\mu p_1-\lambda^*\beta_1 p_3-\lambda^*\beta_2 p_4-\dfrac{\lambda^*\beta_3\Lambda}{k\mu}p_6=0, \\ -Z_1p_2+\lambda^*\beta_1 p_3+\lambda^*\beta_2 p_4+\dfrac{\lambda^*\beta_3\Lambda}{k\mu}p_6=0, \\ \rho\theta p_2-Z_2p_3=0, \\ (1-\rho)\theta p_2-Z_3p_4=0, \\ \tau p_3+\varepsilon p_4-\mu p_5=0, \\ \alpha_1p_3+\alpha_2p_4-\phi p_6=0. \end{cases} \tag{26}$$

From equation (26), we have the right eigenvectors corresponding to the zero eigenvalue as;

$$\begin{cases} p_1 = -\dfrac{1}{\mu}\left[v_6 + \dfrac{\beta_3\Lambda}{k\mu}v_5\right]p_2 < 0, \\ p_2 = p_2 > 0, \\ p_3 = \dfrac{\rho\theta}{Z_2}p_2 > 0, \\ p_4 = \dfrac{(1-\rho)\theta}{Z_3}p_2 > 0, \\ p_5 = \left[\dfrac{\tau\rho\theta Z_3 + (1-\rho)\theta\varepsilon Z_2}{\mu Z_2 Z_3}\right]p_2 > 0, \\ p_6 = v_5 p_2 > 0. \end{cases} \tag{27}$$

where;

$$v_5 = \frac{\rho\theta\alpha_1 Z_3 + (1-\rho)\theta\alpha_2 Z_2}{\phi Z_2 Z_3},$$

and

$$v_6 = \frac{\beta_1\rho\theta Z_3 + \beta_2(1-\rho)\theta Z_2}{Z_2 Z_3}.$$

Similarly, by considering the equation

$$Q \cdot J[E_0, \lambda^*] = 0,$$

we have;

$$\begin{cases} -\mu q_1 = 0, \\ -Z_1 q_2 + \rho\theta q_3 + (1-\rho)\theta q_4 = 0, \\ -\lambda^*\beta_1 q_1 + \lambda^*\beta_1 q_2 - Z_2 q_3 + \tau q_5 + \alpha_1 q_6 = 0, \\ -\lambda^*\beta_2 q_1 + \lambda^*\beta_2 q_2 - Z_3 q_4 + \varepsilon q_5 + \alpha_2 q_6 = 0, \\ -\mu q_5 = 0, \\ -\dfrac{\lambda^*\beta_3\Lambda}{k\mu}q_1 + \dfrac{\lambda^*\beta_3\Lambda}{k\mu}q_2 - \phi q_6 = 0. \end{cases} \tag{28}$$

From equation (28), we have the left eigenvectors corresponding to zero eigenvalue as;

$$\begin{cases} q_1 = q_5 = 0, \\ q_2 = q_2 > 0, \\ q_3 = \dfrac{\lambda^*}{Z_2}\left(\dfrac{\beta_3\Lambda\alpha_1}{k\mu\phi} + \beta_1\right) q_2 > 0, \\ q_4 = \dfrac{\lambda^*}{Z_3}\left(\dfrac{\beta_3\Lambda\alpha_1}{k\mu\phi} + \beta_1\right) q_2 > 0, \\ q_6 = \dfrac{\lambda^*\beta_3\Lambda}{k\mu\phi} q_2 > 0. \end{cases} \tag{29}$$

For model system (25), the associated non-zero partial derivatives of the right hand side functions (h_i) are given by;

$$\begin{cases} \dfrac{\partial^2 h_2}{\partial x_3 \partial x_2}(x_{E_0}, \lambda^*) = \dfrac{\partial^2 h_2}{\partial x_3 \partial x_5}(x_{E_0}, \lambda^*) = -\dfrac{\lambda^*\beta_1\mu}{\Lambda}, \\ \dfrac{\partial^2 h_2}{\partial x_4 \partial x_2}(x_{E_0}, \lambda^*) = \dfrac{\partial^2 h_2}{\partial x_4 \partial x_5}(x_{E_0}, \lambda^*) = -\dfrac{\lambda^*\beta_2\mu}{\Lambda}, \\ \dfrac{\partial^2 h_2}{\partial x_3 \partial x_4}(x_{E_0}, \lambda^*) = \dfrac{\partial^2 h_2}{\partial x_4 \partial x_3}(x_{E_0}, \lambda^*) = -\dfrac{\lambda^*(\beta_1+\beta_2)\mu}{\Lambda}, \\ \dfrac{\partial^2 h_2}{\partial x_3^2}(x_{E_0}, \lambda^*) = -\dfrac{2\lambda^*\beta_1\mu}{\Lambda}, \\ \dfrac{\partial^2 h_2}{\partial x_4^2}(x_{E_0}, \lambda^*) = -\dfrac{2\lambda^*\beta_2\mu}{\Lambda}, \\ \dfrac{\partial^2 h_2}{\partial x_6^2}(x_{E_0}, \lambda^*) = -\dfrac{2\lambda^*\beta_3\Lambda}{\mu k^2}, \\ \dfrac{\partial^2 h_2}{\partial x_6 \partial x_1}(x_{E_0}, \lambda^*) = \dfrac{\lambda^*\beta_3}{k}, \\ \dfrac{\partial^2 h_2}{\partial x_6 \partial \lambda^*}(x_{E_0}, \lambda^*) = \dfrac{\beta_3\Lambda}{\mu k}, \\ \dfrac{\partial^2 h_2}{\partial x_3 \partial \lambda^*}(x_{E_0}, \lambda^*) = \beta_1, \\ \dfrac{\partial^2 h_2}{\partial x_4 \partial \lambda^*}(x_{E_0}, \lambda^*) = \beta_2. \end{cases} \tag{30}$$

According to Castillo-Chavez & Song (2004), the bifurcation parameter a is given as;

$$a = \sum_{k,i,j=1}^{n} q_k p_i p_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(x_{E_0}, \lambda^*).$$

Since $q_k = 0$ for $k = 1, 5$ it follows that;

$$
\begin{aligned}
a = {} & 2q_2p_1p_6\frac{\partial^2 h_2}{\partial x_1\partial x_6}(x_{E_0},\lambda^*) + q_2p_6^2\frac{\partial^2 h_2}{\partial x_6^2}(x_{E_0},\lambda^*) \\
& + 2q_2p_3p_2\frac{\partial^2 h_2}{\partial x_3\partial x_2}(x_{E_0},\lambda^*) + 2q_2p_3p_4\frac{\partial^2 h_2}{\partial x_3\partial x_4}(x_{E_0},\lambda^*) \\
& + 2q_2p_3p_5\frac{\partial^2 h_2}{\partial x_3\partial x_5}(x_{E_0},\lambda^*) + 2q_2p_4p_2\frac{\partial^2 h_2}{\partial x_4\partial x_2}(x_{E_0},\lambda^*) \\
& + 2q_2p_4p_5\frac{\partial^2 h_2}{\partial x_4\partial x_5}(x_{E_0},\lambda^*) + q_2p_3^2\frac{\partial^2 h_2}{\partial x_3^2}(x_{E_0},\lambda^*) \\
& + q_2p_4^2\frac{\partial^2 h_2}{\partial x_4^2}(x_{E_0},\lambda^*)
\end{aligned} \tag{31}
$$

Using equation (27), (29) and (30) then equation (31) simplifies to;

$$
\begin{aligned}
a = -2q_2p_2^2\lambda^*\Bigg[& \frac{\beta_3 v_5}{\mu k}\left(v_6 + \frac{\beta_3\Lambda}{k\mu}v_5\right) + \frac{\beta_3\Lambda v_5^2}{\mu k^2} + \frac{\beta_1\mu\rho\theta}{\Lambda Z_2} \\
& + \frac{(\beta_1+\beta_2)\mu\rho(1-\rho)\theta^2}{\Lambda Z_2 Z_3} + \frac{\beta_1\rho\theta^2\mu}{\Lambda Z_2}\left(\frac{\tau\rho Z_3 + (1-\rho)\varepsilon Z_2}{\mu Z_2 Z_3}\right) \\
& + \frac{\beta_2\mu(1-\rho)\theta}{\Lambda Z_3} + \frac{\beta_2(1-\rho)\theta^2\mu}{\Lambda Z_3}\left(\frac{\tau\rho Z_3 + (1-\rho)\varepsilon Z_2}{\mu Z_2 Z_3}\right) \\
& + \frac{\beta_1\mu(\rho\theta)^2}{\Lambda Z_2^2} + \frac{\beta_2\mu[(1-\rho)\theta]^2}{\Lambda Z_3^2}\Bigg]
\end{aligned} \tag{32}
$$

Similarly, the bifurcation parameter b is given as;

$$
b = \sum_{k,i=1}^{n} q_k p_i \frac{\partial^2 f_k}{\partial X_i \partial \beta^*}(X_{E_0},\lambda^*).
$$

Recall that $q_1 = 0$, therefore;

$$
b = q_2p_3\frac{\partial^2 h_2}{\partial X_3\partial\lambda^*}(X_{E_0},\lambda^*) + q_2p_4\frac{\partial^2 h_2}{\partial X_4\partial\lambda^*}(X_{E_0},\lambda^*) + q_2p_6\frac{\partial^2 h_2}{\partial X_6\partial\lambda^*}(X_{E_0},\lambda^*).
$$

Using equation (27), (29), and (30), we have;

$$
b = q_2p_2\left[\frac{\beta_1\rho\theta}{Z_2} + \frac{\beta_2(1-\rho)\theta}{Z_3} + \frac{v_5\Lambda}{\mu k}\right] > 0 \tag{33}
$$

Hence, the model system (1) will undergo a forward bifurcation at $R_0 = 1$ since $a < 0$ and $b > 0$.

4 Conclusion

In this research work, we formulated and studied a deterministic continuous-time model of typhoid fever with asymptomatic carriers. The epidemiologically feasible region where the model is mathematically well–posed was established, and the basic reproduction number R_0 was explicitly derived using the next-generation matrix approach. We assessed the impact of asymptomatic carriers on the basic reproduction R_0, our analysis revealed that R_0 increases when there are asymptomatic individuals capable of transmitting the disease. Local stability analysis of the disease-free equilibrium of the model was investigated, and our investigation showed that the disease–free equilibrium of the model is locally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$. We also proved that the disease-free equilibrium of our model is globally asymptotically stable if $R_0 \leq 1$ and unstable otherwise.

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