



Research article

Modelling the impact of dynamic hygiene behaviour on typhoid transmission dynamics with antibiotic resistance

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Abstract: In this paper, we develop a mathematical model to investigate the impact of dynamic hygiene behaviour on the transmission dynamics of typhoid fever in the presence of antibiotic resistance. The model extends existing modelling approaches by integrating behavioural responses with treatment and resistance dynamics. The model is calibrated using cumulative data on typhoid cases reported at the Mbandjock District Hospital in Cameroon. The calibrated parameter estimates give $\mathcal{R}_0 = 2.0754 > 1$ and $\mathcal{R}_{0e} = 1.9509 > 1$. These values indicate that the disease can persist in the population and remains endemic in the region. A sensitivity analysis of the basic reproduction number is performed to identify the parameters that have the greatest influence on disease transmission. The results show that the environmental transmission rate β_b and the bacterial decay rate μ_b in the environment are among the most influential parameters affecting the value of $\mathcal{R}_0$. The model shows the possibility of backward bifurcation, indicating that reducing the reproduction number below one may not be enough to eliminate the disease, particularly in settings with poor hygiene or high levels of antibiotic resistance. Numerical simulations demonstrate that improved hygiene practices can significantly reduce infections and environmental contamination. In general, the findings show the importance of combining behavioural interventions with effective treatment strategies to control typhoid fever.

Keywords: typhoid fever; antibiotic resistance; dynamic hygiene behaviour; bifurcation analysis; mathematical modelling

1. Introduction

Typhoid fever remains a significant public health concern in many parts of the world, particularly in regions where access to clean water and proper sanitation is limited. Typhoid fever is an infectious disease caused by *Salmonella typhi*, which is mainly transmitted by the faecal-oral route, typically through contaminated food and water [1]. Despite substantial public-health advances, recent global estimates indicate that typhoid fever causes millions of cases and tens of thousands of deaths annually, with the highest burden in South Asia and sub-Saharan Africa [1, 2].

Young children and populations living in poor socioeconomic conditions are particularly vulnerable to infection. Although the global burden of typhoid has declined in some regions, the disease remains persistent in areas with limited access to safe water, sanitation, and hygiene (WASH) infrastructure [3, 4]. Interventions that improve water quality, sanitation, and personal hygiene are widely recognized as essential strategies for controlling typhoid fever [5–7]. Studies have shown that household water treatment, safe storage, access to sanitation, and regular handwashing significantly reduce infection risk [5–7]. Evidence from meta-analyses also suggests that hygiene practices, particularly handwashing with soap, can be highly effective in preventing infection when adopted consistently within communities [6]. However, maintaining a high and sustained level of hygiene practices remains a major challenge. In many communities, the adoption of hygiene behaviours often changes over time and may depend on factors such as perceived disease risk, social norms, and availability of resources [8].

A major challenge in the control of typhoid fever is the emergence of antibiotic-resistant strains of *salmonella typhi* [9]. The misuse and overuse of antibiotics have contributed to the development of resistant infections, making treatment more difficult and increasing the risk of complications [9]. In addition to medical interventions, preventive measures such as improved hygiene and sanitation play a critical role in reducing transmission. Therefore, improved diagnostic methods and treatment strategies are essential for the early detection and effective management of resistant cases [10]. Behavioural responses, such as increased handwashing and improved awareness of hygiene during outbreaks, can influence how the disease spreads within a population.

Mathematical modelling has become an important tool for understanding the transmission dynamics of infectious diseases and for evaluating the potential impact of control strategies. In the context of typhoid fever, models have been used to examine the roles of environmental transmission, treatment, and vaccination [11, 12]. Compartmental models, in particular, capture key mechanisms such as human-to-human transmission, environmental contamination, and the effects of treatment on infectiousness [11, 13].

A study by [14] demonstrated the role of contaminated water and poor sanitation in sustaining disease transmission. Behavioural responses, particularly those related to hygiene practices, can significantly influence transmission by modifying contact patterns and exposure to contaminated environments [15, 16]. Behavioural responses within communities can play an important role in reducing disease transmission. For example, people can adopt improved hygiene practices, such as handwashing and safe food handling, when awareness of the risk of infection increases. These behavioural changes can influence how the disease spreads within a population, particularly in settings where environmental contamination contributes to transmission. However, the interaction between hygiene behaviour and antibiotic resistance in typhoid transmission remains less explored in

mathematical modelling studies.

More recent modelling studies have incorporated behavioural responses, demonstrating that changes in hygiene practices can significantly influence disease dynamics [17, 18]. These approaches account for risk-driven behaviour, behavioural fatigue, and adaptive responses that can alter transmission patterns. Models that integrate environmental bacterial dynamics with behaviourally driven contact or shedding rates are especially relevant for enteric diseases such as typhoid fever.

In addition, several studies have highlighted the growing impact of antibiotic resistance on typhoid transmission and control. The rise of multidrug-resistant strains has reduced the effectiveness of commonly used antibiotics and increased the complexity of treatment strategies [19]. Although some modelling studies have explored the dynamics and resistance of treatment [20], relatively few have considered the combined effects of behavioural responses and resistance to antibiotics within a unified framework.

A study by [17] developed a compartmental model to examine the interaction between cholera and typhoid fever, with particular emphasis on the role of hygiene behaviour in disease transmission. In their work, the contact rate was formulated as a function of hygiene level, allowing the model to capture how improved sanitation and behavioural changes can reduce the spread of infection. Their results show that better hygiene practices significantly lower transmission rates and can contribute to the control of both diseases. However, our study focuses on typhoid fever alone and extends the analysis by incorporating antibiotic resistance and treatment dynamics alongside behavioural responses.

In addition, a study by [8] emphasizes that poor water, sanitation, and hygiene conditions remain a major driver of disease transmission, particularly in many African settings. Furthermore, the study by [10] discusses modern detection methods, showing how improved diagnostic tools can help in the early identification of resistant strains and support better treatment decisions. Together, these studies indicate that effective control of typhoid fever depends on a combination of improved treatment strategies, better hygiene practices, and enhanced detection of drug-resistant pathogens.

Although several mathematical models have been developed to study typhoid transmission dynamics, many focus primarily on environmental transmission, vaccination, or treatment interventions. Only a limited number of studies, such as [17], incorporate behaviour-dependent transmission mechanisms, and even fewer account for the combined effects of behavioural responses and antibiotic resistance. This gap is particularly important in endemic settings, where behavioural changes, such as increased handwashing and hygiene awareness, can reduce exposure to contaminated environments, while antibiotic resistance may alter disease progression and transmission patterns, further complicating control efforts.

To address this gap, this study extends existing mathematical models of typhoid transmission by incorporating dynamic hygiene behaviour into a framework that also accounts for antibiotic resistance and treatment dynamics. Rather than proposing an entirely new modelling structure, the contribution lies in examining how behaviour-dependent hygiene and resistance interact within a unified setting. The model captures human-to-environment transmission, adaptive hygiene responses, and treatment-related resistance, and is used to provide new insights into how these factors jointly influence disease dynamics. In particular, the study explores the impact of behavioural changes on transmission, the role of resistance in shaping endemic outcomes, and the implications for control strategies in resource-limited settings.

The rest of the paper is organised as follows. Section 2 introduces the mathematical model and

explains how it is formulated. In Section 3, we examine the positivity and boundedness of solutions, along with a detailed analysis of the model. Sensitivity analysis and model calibration are discussed in Section 4, while numerical simulations are provided in Section 5. Section 6 concludes the paper.

2. Model formulation

We develop a deterministic compartmental model to describe the transmission dynamics of typhoid fever in the presence of antibiotic resistance, treatment switching, and behaviour-dependent hygiene interventions. The total human population at time t , denoted by $N(t)$, which is assumed to be bounded over time, is given by

$$N(t) = S(t) + I_s(t) + I_r(t) + T_{s1}(t) + T_{r1}(t) + T_{s2}(t) + T_{r2}(t) + R(t).$$

The model structure is illustrated in Figure 1. The population is divided into susceptible individuals $S(t)$, infected individuals with drug-sensitive strain $I_s(t)$, infected individuals with drug-resistant strain $I_r(t)$, people receiving first-line treatment ($T_{s1}(t), T_{r1}(t)$), people receiving second-line treatment ($T_{s2}(t), T_{r2}(t)$), and recovered individuals $R(t)$. The model also incorporates the environmental bacterial concentration $B_c(t)$ and a behaviour-dependent hygiene variable $H(t)$.

The hygiene variable $H(t)$ is modelled as a proxy for the behavioural response at the population-level driven by the perceived risk of infection, represented by the total number of infectious individuals ($I_s + I_r$). This assumption follows a common approach in behavioural epidemiology, where increasing the prevalence of diseases leads to increased awareness and the adoption of protective measures. Hygiene behaviour is influenced by multiple factors, including public health interventions, education, media influence, cultural practices, and access to sanitation resources. However, explicitly incorporating all of these factors would substantially increase the complexity of the model and the uncertainty of the parameters.

Therefore, this paper provides a simplified but analytically tractable representation of adaptive behaviour, which is sufficient for capturing its interaction with transmission and antibiotic resistance dynamics.

Transmission is assumed to occur through exposure to contaminated environments such as unsafe water or food. The forces of infection for drug-sensitive and drug-resistant strains are defined as

$$\lambda_s = \omega \left(\frac{\beta_b B_c}{B_c + \kappa} \right) (1 - \xi H), \quad \lambda_r = (1 - \omega) \left(\frac{\beta_b B_c}{B_c + \kappa} \right) (1 - \xi H), \quad (2.1)$$

where β_b is the effective contact rate with contaminated environments, κ is the half-saturation constant, and $\omega \in [0, 1]$ represents the relative contribution of the drug-sensitive strain to transmission. The term $(1 - \xi H)$ captures the reduction in transmission due to hygiene behaviour, where $H \in [0, 1]$ denotes the level of hygiene adoption and $\xi \in (0, 1]$ measures the effectiveness of hygiene in reducing environmental contamination. The environmental contribution to infection is represented by the saturating function $\frac{\beta_b B_c}{B_c + \kappa}$, which describes the risk of infection from contact with contaminated water or food and approaches a maximum as the bacterial concentration increases. To account for the relative probability of infection with each strain, a weighting factor ω is introduced, where ω represents the probability of contracting the sensitive strain and $(1 - \omega)$ represents the probability of contracting the resistant strain.

Transmission is driven by environmental bacterial contamination and influenced by hygiene behaviour. Infection occurs when susceptible individuals (S) are exposed to a contaminated environment, after which they may become infected with the drug-sensitive strain (I_s) at rate λ_s or the drug-resistant strain (I_r) at rate λ_r . Following works by [20–22], treatment in this model is modelled using two lines of antibiotics: a first-line treatment (Antibiotic 1), which may be partially ineffective due to resistance or misdiagnosed, and second-line treatment (Antibiotic 2), which is assumed to be resistance-free.

Susceptible individuals are recruited into the population at rate Λ and become infected at rates λ_s and λ_r . Infected individuals in I_s and I_r may start first-line treatment at rates σ_s and σ_r , recover naturally at rates θ_s and θ_r or die due to the disease at rates δ_s and δ_r , respectively. Individuals receiving first-line treatment (T_{s1}, T_{r1}) may recover at rates ν_s and ν_r , move to second-line treatment (T_{s2}, T_{r2}) at rates ρ_s and ρ_r if treatment fails, or die due to the disease at rates δ_{s1} and δ_{r1} . Individuals in second-line treatment (T_{s2}, T_{r2}) recover at rates γ_s and γ_r , or die due to the disease at rates δ_{s2} and δ_{r2} . Recovered individuals lose immunity at rate ϵ and return to the susceptible class. All individuals are also subject to natural death at rate μ .

The environmental bacterial concentration $B_c(t)$ is driven by shedding from infectious individuals (I_s, I_r) and those receiving first-line treatment (T_{s1}, T_{r1}). Individuals in I_s and I_r shed bacteria at rates α_s and α_r , respectively, while those in (T_{s1}, T_{r1}) shed at reduced rates α_{s1} and α_{r1} . Shedding from individuals receiving second-line treatment is assumed negligible. The bacterial population grows at rate g and decays at rate μ_b , with $\mu_b > g$, ensuring that environmental bacteria remain bounded. We assume that first-line treatment (Antibiotic 1) is less effective, which justifies the need for second-line treatment (Antibiotic 2), thus it is assumed that individuals receiving first-line treatment in classes (T_{s1} and T_{r1}) can continue shedding bacteria due to treatment failure or resistance development. In addition, individuals in the infectious classes I_s and I_r shed bacteria at higher rates α_s and α_r respectively compared to those receiving first-line treatment in classes T_{s1} and T_{r1} , who shed at lower rates α_{s1} and α_{r1} . This reflects the assumption that first-line treatment is less effective, hence allowing continued shedding from individuals in T_{s1} and T_{r1} .

Bacterial shedding from individuals receiving second-line treatment (T_{s2}, T_{r2}) is assumed to be very low and therefore considered negligible. Although some bacteria may still be released during second-line treatment, the number is minimal because second-line antibiotics are typically more potent and effective at suppressing bacterial load than first-line drugs. As a result, individuals who receive second-line treatment contribute very little to environmental contamination compared to those who are infectious (I_s, I_r) or who undergo first-line treatment (T_{s1}, T_{r1}). This assumption simplifies the model without losing biological relevance, as the majority of bacterial shedding and transmission is expected to occur during earlier stages of infection and treatment when bacterial concentrations are highest.

As infection levels increase, individuals are more likely to adopt protective hygiene at a rate ψ_1 , in response to the increased risk. However, in the absence of sustained motivation, these practices decline at a rate ψ_2 . Higher levels of hygiene reduce environmental exposure to the environment, reducing the likelihood that susceptible individuals become infected. This effect also indirectly influences the flow of individuals into infected and treatment compartments. In this way, hygiene acts as a dynamic control measure that helps regulate the overall trajectory of the epidemic.

Shedding into the environment is assumed to be hygiene-dependent because the level of hygiene in a community strongly affects how much bacteria from infected individuals enters the surrounding

environment. In settings where hygiene practices are poor such as limited handwashing, inadequate waste disposal, or absence of proper sewage systems, infected individuals are more likely to contaminate water sources and food through faecal matter or improper sanitation. This leads to higher bacterial concentrations in the environment, which increases the risk of indirect transmission.

The term $\psi_1(I_s + I_r)(1 - H)$ represents the rate at which individuals adopt or improve hygiene practices in response to increasing infection levels. As the number of infected individuals rises, people are more likely to adopt protective behaviours such as frequent handwashing and improved sanitation. The term $\psi_2 H$ describes the decline in hygiene awareness over time. As infection levels decrease and the perceived risk becomes lower, individuals tend to relax these behaviours, leading to reduced compliance with recommended hygiene practices. Therefore, these terms capture the dynamic nature of human behaviour, in response to disease prevalence, suggesting that hygiene practices improve during outbreaks but may decline once the threat subsides.

Hygiene adoption is modelled as a population-level response driven by infection prevalence, represented by $I_s + I_r$, which is assumed to represent perceived risk in the community. Although individuals undergoing treatment (T_{s1} , T_{r1} , T_{s2} , T_{r2}) are influenced by hygiene awareness, as shown in the model diagram, this effect is not explicitly included in the equations. This is because treated individuals are assumed to follow prescribed medical guidelines, and the simplification avoids additional complexity and overlap with treatment effects.

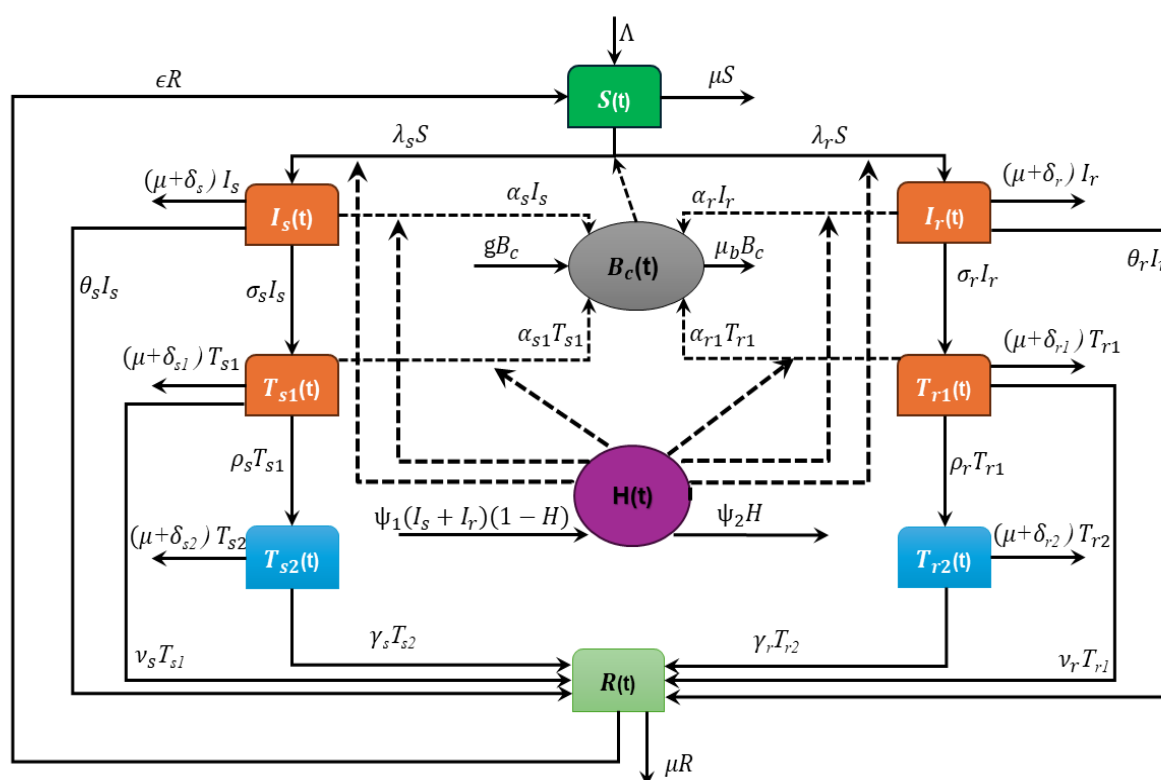


Figure 1. Schematic diagram of the typhoid fever model (2.2).

Thus, based on the model assumption, formulation, and Figure 1, we have the corresponding system of ordinary differential equations:

$$\left\{ \begin{array}{l} \frac{dS}{dt} = \Lambda - (\lambda_s + \lambda_r + \mu)S + \epsilon R, \\ \frac{dI_s}{dt} = \lambda_s S - (\sigma_s + \theta_s + \delta_s + \mu)I_s, \\ \frac{dI_r}{dt} = \lambda_r S - (\sigma_r + \theta_r + \delta_r + \mu)I_r, \\ \frac{dT_{s1}}{dt} = \sigma_s I_s - (\rho_s + \nu_s + \delta_{s1} + \mu)T_{s1}, \\ \frac{dT_{r1}}{dt} = \sigma_r I_r - (\rho_r + \nu_r + \delta_{r1} + \mu)T_{r1}, \\ \frac{dT_{s2}}{dt} = \rho_s T_{s1} - (\gamma_s + \delta_{s2} + \mu)T_{s2}, \\ \frac{dT_{r2}}{dt} = \rho_r T_{r1} - (\gamma_r + \delta_{r2} + \mu)T_{r2}, \\ \frac{dR}{dt} = \theta_s I_s + \theta_r I_r + \nu_s T_{s1} + \nu_r T_{r1} + \gamma_s T_{s2} + \gamma_r T_{r2} - (\epsilon + \mu)R, \\ \frac{dB_c}{dt} = gB_c + (1 - \xi H)(\alpha_s I_s + \alpha_r I_r + \alpha_{s1} T_{s1} + \alpha_{r1} T_{r1}) - \mu_b B_c, \\ \frac{dH}{dt} = \psi_1(I_s + I_r)(1 - H) - \psi_2 H, \end{array} \right. \quad (2.2)$$

with the initial conditions be

$$\begin{aligned} S(0) > 0, \quad I_s(0) \geq 0, \quad I_r(0) \geq 0, \quad T_{s1}(0) \geq 0, \quad T_{r1}(0) \geq 0, \quad T_{s2}(0) \geq 0, \quad T_{r2}(0) \geq 0, \\ R(0) \geq 0, \quad B_c(0) \geq 0, \quad H(0) \geq 0. \end{aligned} \quad (2.3)$$

3. Model analysis

This section presents a detailed analysis of the formulated typhoid fever model to understand its qualitative and epidemiological behaviour. Furthermore, the basic reproduction number, denoted by $\mathfrak{R}_0$, is derived using the next-generation matrix method to determine the threshold condition that dictates whether the disease will die out or persist in the community. Local and global stability analyses of the disease-free equilibrium (DFE) are examined to understand the behaviour of the model around the equilibrium.

3.1. Positivity of solutions

We now analyse the positivity of the model system (2.2). We prove that all state variables remain non-negative and that every solution of the model system (2.2) with positive initial conditions remains positive for all $t > 0$. The following theorem is thus presented.

Theorem 1. *All solutions of the typhoid fever model (2.2) defined by the set*

$Y(t) = (S(t), I_s(t), I_r(t), T_{s1}(t), T_{r1}(t), T_{s2}(t), T_{r2}(t), R(t), B_c(t), H(t))$, *with positive initial conditions (2.3)*

$Y(0) = (S(0), I_s(0), I_r(0), T_{s1}(0), T_{r1}(0), T_{s2}(0), T_{r2}(0), R(0), B_c(0), H(0))$, *remain positive for all time $t > 0$.*

Proof. We need to show that the system solutions remain non-negative for all $t > 0$. Consider the first equation of model (2.2) given by

$$\frac{dS}{dt} = \Lambda + \epsilon R - (\mu + \lambda_s + \lambda_r) S = \Lambda + \epsilon R - (\mu + \lambda) S, \text{ where } \lambda = \lambda_s + \lambda_r. \quad (3.1)$$

Solving (3.1), we have

$$\frac{d}{dt} \left(S(t) \exp \left(\mu t + \int_0^t \lambda(s) ds \right) \right) = (\Lambda + \epsilon R(t)) \exp \left(\mu t + \int_0^t \lambda(s) ds \right).$$

So,

$$S(\hat{t}) \exp \left(\mu \hat{t} + \int_0^{\hat{t}} \lambda(s) ds \right) - S(0) = \int_0^{\hat{t}} (\Lambda + \epsilon R(t)) \exp \left(\mu \hat{t} + \int_0^{\hat{t}} \lambda(v) dv \right) d\hat{t},$$

giving

$$\begin{aligned} S(\hat{t}) &= S(0) \exp \left(- \left(\mu \hat{t} + \int_0^{\hat{t}} \lambda(s) ds \right) \right) \\ &+ \exp \left(- \left(\mu \hat{t} + \int_0^{\hat{t}} \lambda(s) ds \right) \right) \int_0^{\hat{t}} \left[(\Lambda + \epsilon R(t)) \exp \left(\mu \hat{t} + \int_0^{\hat{t}} \lambda(v) dv \right) d\hat{t} \right] > 0. \end{aligned}$$

From the second equation of model system equations (2.2), we obtain

$$\begin{aligned} \frac{dI_s(t)}{dt} &= \lambda_s S - (\sigma_s + \theta_s + \delta_s + \mu) I_s \geq -(\sigma_s + \theta_s + \delta_s + \mu) I_s \\ &\Rightarrow I(\hat{t}) \geq I_{s0} \exp [-(\sigma_s + \theta_s + \delta_s + \mu) \hat{t}] > 0. \end{aligned}$$

Similarly, using the same method for the other state variables, it can be shown that the solution set $I_r(t), T_{s1}(t), T_{r1}(t), T_{s2}(t), T_{r2}(t), R(t), B_c(t)$, and $H(t)$ remains positive for all $t > 0$, therefore, the model (2.2) is positively invariant in $\mathbb{R}_+^{10}$, and this completes the proof.

3.2. Boundedness of solutions

To ensure that the model is mathematically and epidemiologically well posed, it is necessary to establish that all state variables remain non-negative and bounded for all time $t \geq 0$.

Theorem 2. *The feasible region Υ of the typhoid fever model (2.2), as defined in (3.2), is positively invariant and attracting:*

$$\Upsilon = \left\{ (S, I_s, I_r, T_{s1}, T_{r1}, T_{s2}, T_{r2}, R, B_c, H) \in \mathbb{R}_+^{10} : H \in [0, 1), N \leq \frac{\Lambda}{\mu}, B_c \leq M \right\}, \quad (3.2)$$

where

$$M = \max \left\{ B_c(0), \frac{(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})\Lambda}{\mu(\mu_b - g)} \right\}.$$

Proof. Summing the first order of the linear equation for the rate of change in the total human population $N(t)$, we have

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - (\delta_s I_s + \delta_r I_r + \delta_{s1} I_{s1} + \delta_{r1} I_{r1} + \delta_{s2} I_{s2} + \delta_{r2} I_{r2}) \leq \Lambda - \mu N(t), \quad (3.3)$$

since the disease-induced death rates are non-negative. Thus, the linear differential inequality has the solution,

$$N(t) \leq N(0) \exp^{-\mu t} + \frac{\Lambda}{\mu} (1 - \exp^{-\mu t}),$$

which implies that $N(t)$ is uniformly bounded and that

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

This shows that the total human population is uniformly bounded above by $\frac{\Lambda}{\mu}$ for all $t > 0$.

For the environmental bacteria B_c , we have

$$\begin{aligned} \frac{dB_c}{dt} &= (g - \mu_b)B_c + (1 - \xi H)(\alpha_s I_s + \alpha_r I_r + \alpha_{s1} T_{s1} + \alpha_{r1} T_{r1}) \\ &\leq (g - \mu_b)B_c + (1 - \xi H)(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})N(t). \end{aligned}$$

Since $0 \leq (1 - \xi H) \leq 1$ and all state variables are non-negative, it follows that

$$\frac{dB_c}{dt} \leq (g - \mu_b)B_c + \frac{(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})\Lambda}{\mu}.$$

Since $\mu_b > g$, then we have:

$$\begin{aligned} B_c(t) &\leq B_c(0) \exp^{-(\mu_b - g)t} + \frac{(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})\Lambda}{\mu(\mu_b - g)} (1 - \exp^{-(\mu_b - g)t}) \\ &\leq \max \left\{ B_c(0), \frac{(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})\Lambda}{\mu(\mu_b - g)} \right\}. \end{aligned}$$

Thus, $B_c(t)$ is uniformly bounded and

$$\limsup_{t \rightarrow \infty} B_c(t) \leq \frac{(\alpha_s + \alpha_r + \alpha_{s1} + \alpha_{r1})\Lambda}{\mu(\mu_b - g)}.$$

For the hygiene variable $H(t)$, we have

$$\frac{dH}{dt} = \psi_1(I_s + I_r)(1 - H) - \psi_2 H.$$

Given that $H \in [0, 1)$, at $H = 0$, $\frac{dH}{dt} = \psi_1(I_s + I_r) \geq 0$. Therefore, $H(t)$ remains within the biologically meaningful interval $[0, 1)$ for all $t \geq 0$.

Combining these results, the feasible (positively invariant) region for the system is defined as

$$\Upsilon = \left\{ (S, I_s, I_r, T_{s1}, T_{r1}, T_{s2}, T_{r2}, R, B_c, H) \in \mathbb{R}_+^{10} : H \in [0, 1), N \leq \frac{\Lambda}{\mu}, B_c \leq M \right\}.$$

Hence, all solutions of the model with non-negative initial conditions remain in Υ for all $t \geq 0$, ensuring that the model is biologically and mathematically well-posed.

3.3. Typhoid-free equilibrium and the basic reproduction number

In this section, we establish the typhoid DFE of the model (2.2) and derive the corresponding basic reproduction number, denoted by $\mathcal{R}_0$. The DFE represents a steady state in which no infection persists in the population.

The DFE of (2.2) is denoted by $\mathcal{D}_0 = (S^*, I_s^*, I_r^*, T_{s1}^*, T_{r1}^*, T_{s2}^*, T_{r2}^*, R^*, B_c^*, H^*)$, obtained by setting all infection-related compartments to zero. That is,

$$I_s^* = I_r^* = T_{s1}^* = T_{r1}^* = T_{s2}^* = T_{r2}^* = R^* = B_c^* = 0.$$

The hygiene equation at equilibrium becomes

$$0 = \psi_1(0)(1 - H^*) - \psi_2 H^* \implies H^* = 0.$$

The susceptible equation reduces to

$$0 = \Lambda - (\mu)S^* \implies S^* = \frac{\Lambda}{\mu}.$$

Thus, the DFE is

$$\mathcal{D}_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0 \right).$$

The basic reproduction number $\mathcal{R}_0$, is derived using the next-generation matrix approach [23]. This threshold quantity measures the expected number of secondary cases produced by a single infected individual in a susceptible population. The value of $\mathcal{R}_0$ serves as a key determinant of disease dynamics: If $\mathcal{R}_0 < 1$, the typhoid infection will eventually die out, while $\mathcal{R}_0 > 1$ implies that the disease can invade and persist in the community. Therefore, computing $\mathcal{R}_0$ is crucial for identifying the conditions under which typhoid transmission can be controlled or eliminated.

To derive the basic reproduction number, we follow the standard next-generation matrix approach (van den Driessche and Watmough) [24]. The method involves identifying the new infection terms F and the transition terms V for all infected compartments. The infected compartments of the system are I_s , I_r , T_{s1} , T_{r1} , T_{s2} , T_{r2} , and B_c . New infections occur in compartments I_s and I_r , driven by the force of infection. The transition terms account for recovery, treatment, switching, resistance development, and natural mortality.

$$\mathcal{F} = \begin{pmatrix} \lambda_s S \\ \lambda_r S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\mu + \sigma_s + \theta_s + \delta_s)I_s \\ (\mu + \sigma_r + \theta_r + \delta_r)I_r \\ (\mu + \rho_s + \nu_s + \delta_{s1})T_{s1} - \sigma_s I_s \\ (\mu + \rho_r + \nu_r + \delta_{r1})T_{r1} - \sigma_r I_r \\ (\mu + \gamma_s + \delta_{s2})T_{s2} - \rho_s T_{s1} \\ (\mu + \gamma_r + \delta_{r2})T_{r2} - \rho_r T_{r1} \\ (\mu_b - g)B_c - (1 - \xi H)(\alpha_s I_s + \alpha_r I_r + \alpha_{s1} T_{s1} + \alpha_{r1} T_{r1}) \end{pmatrix}.$$

Let

$$\begin{aligned} \mathcal{Z}_1 &= \mu + \sigma_s + \theta_s + \delta_s, \quad \mathcal{Z}_2 = \mu + \sigma_r + \theta_r + \delta_r, \quad \mathcal{Z}_3 = \mu + \rho_s + \nu_s + \delta_{s1}, \quad \mathcal{Z}_8 = \mu_b - g, \\ \mathcal{Z}_4 &= \mu + \rho_r + \nu_r + \delta_{r1}, \quad \mathcal{Z}_5 = \mu + \gamma_s + \delta_{s2}, \quad \mathcal{Z}_6 = \mu + \gamma_r + \delta_{r2}, \quad \mathcal{Z}_7 = \epsilon + \mu. \end{aligned} \quad (3.4)$$

Then

$$FV^{-1} = \begin{pmatrix} Q_1 & \mathcal{K}_6 \frac{\omega\beta_b\Lambda}{\mu\kappa} & Q_2 & \frac{\omega\beta_b\Lambda\alpha_{r1}}{\mu\kappa\mathcal{Z}_4\mathcal{Z}_8} & 0 & 0 & \frac{\omega\beta_b\Lambda}{\mu\kappa\mathcal{Z}_8} \\ \mathcal{K}_5 \frac{(1-\omega)\beta_b\Lambda}{\mu\kappa} & Q_3 & \frac{(1-\omega)\beta_b\Lambda\alpha_{s1}}{\mu\kappa\mathcal{Z}_3\mathcal{Z}_8} & Q_4 & 0 & 0 & \frac{(1-\omega)\beta_b\Lambda}{\mu\kappa\mathcal{Z}_8} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$Q_1 = \mathcal{K}_5 \frac{\omega\beta_b\Lambda}{\mu\kappa}, \quad Q_2 = \frac{\omega\beta_b\Lambda\alpha_{s1}}{\mu\kappa\mathcal{Z}_3\mathcal{Z}_8}, \quad Q_3 = \mathcal{K}_6 \frac{(1-\omega)\beta_b\Lambda}{\mu\kappa}, \quad Q_4 = \frac{(1-\omega)\beta_b\Lambda\alpha_{r1}}{\mu\kappa\mathcal{Z}_4\mathcal{Z}_8}, \quad (3.5)$$

with

$$\mathcal{K}_5 = \frac{\mathcal{Z}_3\alpha_s + \alpha_{s1}\sigma_s}{\mathcal{Z}_1\mathcal{Z}_3\mathcal{Z}_8}, \quad \mathcal{K}_6 = \frac{\mathcal{Z}_4\alpha_r + \alpha_{r1}\sigma_r}{\mathcal{Z}_2\mathcal{Z}_4\mathcal{Z}_8}.$$

The matrix product FV^{-1} captures the expected number of new infections produced in each infection class by a single infected individual introduced into the DFE. The eigenvalues of FV^{-1} , are given as

$$\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 = \lambda_6 = 0, \quad \lambda_7 = \frac{(Q_1 + Q_3) - \sqrt{\Psi}}{2}, \quad \lambda_8 = \frac{(Q_1 + Q_3) + \sqrt{\Psi}}{2},$$

where

$$\Psi = (Q_1 - Q_3)^2 + 4 \frac{\Lambda}{\mu} \omega\beta_b\mathcal{K}_5 \frac{\Lambda}{\mu} (1-\omega)\beta_b\mathcal{K}_6.$$

Since $\Psi > 0$, then $\lambda_8 > 0$ and λ_7 will be positive if $(Q_1 + Q_3) > \sqrt{\Psi}$. Given that $\Psi > 0$, then $\lambda_8 > \lambda_7$, so λ_8 is the dominant eigenvalue of FV^{-1} . Hence, the basic reproduction number of the model (2.2), obtained by $\mathcal{R}_0 = \rho(FV^{-1})$, where ρ is the spectral radius of the product FV^{-1} , is given by

$$\mathcal{R}_0 = \frac{(Q_1 + Q_3) + \sqrt{\Psi}}{2}.$$

Let

$$\mathcal{R}_s = \mathcal{K}_5 \frac{\omega\beta_b\Lambda}{\mu\kappa}, \quad \mathcal{R}_r = \mathcal{K}_6 \frac{(1-\omega)\beta_b\Lambda}{\mu\kappa}, \quad \mathcal{R}_{sr} = \frac{\Lambda}{\mu} \omega\beta_b\mathcal{K}_5 \frac{\Lambda}{\mu} (1-\omega)\beta_b\mathcal{K}_6, \quad (3.6)$$

with $\mathcal{R}_s$ and $\mathcal{R}_r$ representing the contributions of environmental transmission pathways for sensitive and resistant strains, respectively. The interaction term $\mathcal{R}_{sr}$ captures the combined effect of sensitive and resistance strains through shared environment.

The basic reproduction number can be rewritten in terms of $\mathcal{R}_s$, $\mathcal{R}_r$, and $\mathcal{R}_{sr}$ as:

$$\mathcal{R}_0 = \frac{\mathcal{R}_s + \mathcal{R}_r + \sqrt{(\mathcal{R}_s - \mathcal{R}_r)^2 + 4\mathcal{R}_{sr}}}{2}. \quad (3.7)$$

It is seen that the basic reproduction number is expressed in terms of contributions from the sensitive and resistant strains. This decomposition assumes that the two strains are epidemiologically separable, but still interact through the shared environmental bacterial compartment. $\mathcal{R}_{sr}$ represents this interaction, capturing the indirect link between the strains through the shared environment. This means that the presence of one strain can increase environmental contamination, which in turn enhances the transmission potential of the other strain, leading to co-circulation of both strains.

In the absence of hygiene behaviour, $\mathcal{R}_0$ is assumed to represent the intrinsic transmission capacity of typhoid fever under natural conditions. In the absence of resistant strains ($I_r = 0$), the transmission dynamics of the model are driven solely by the sensitive strain. Under this condition, the interaction term $\mathcal{R}_{sr} = 0$, and the basic reproduction number $\mathcal{R}_0$ in Eq (3.7) reduces to the component associated with the sensitive strain, that is,

$$\mathcal{R}_s = \mathcal{K}_5 \frac{\omega\beta_b\Lambda}{\mu\kappa}.$$

This quantity represents the expected number of secondary infections caused by a single infectious individual in a completely susceptible population when only the sensitive strain is circulating. Similarly, in the absence of the sensitive strain ($I_s = 0$), transmission is driven entirely by the resistant strain, leading to

$$\mathcal{R}_r = \mathcal{K}_6 \frac{(1 - \omega)\beta_b\Lambda}{\mu\kappa}.$$

This is assumed to represent the contribution of the resistant strain to new infections through environmental transmission. In the presence of hygiene behaviour, the effective reproduction number is reduced due to a decrease in environmental transmission. Specifically, hygiene awareness modifies the terms of transmission in the force of infection through the factor $(1 - \xi H)$, and $H \in [0, 1)$ represents the level of hygiene awareness in the population. We thus have the following Remark 1.

Remark 1. Role of hygiene in the reproduction number

In this model, hygiene reduces both disease transmission and bacterial shedding. The forces of infection for the drug-sensitive and drug-resistant strains include the factor $(1 - \xi H)$, which represents how increased hygiene lowers the likelihood of infection.

At the DFE, all infected compartments are zero, and hygiene awareness naturally decays to its minimum level, $H^* = 0$, because no infections exist to stimulate increased hygiene behaviour. Consequently, when the basic reproduction number $\mathcal{R}_0$ is evaluated at the DFE, hygiene parameters do not appear explicitly. Importantly, this is an emergent property of the model and not an imposed assumption. In the absence of infections, hygiene awareness naturally decays to zero.

To account for pre-existing hygiene in the population, the model introduces a hygiene-adjusted reproduction number, $\mathcal{R}_{0e}$, where $H_0 \in [0, 1)$ represents a baseline hygiene level. This baseline reduces disease transmission through the factor $(1 - \xi H_0)$, reflecting both the effectiveness of hygiene ξ and the proportion of the population practising hygiene (H_0). Because this factor scales all infection terms equally, the next-generation matrix is proportionally scaled, giving

$$\mathcal{R}_{0e} = (1 - \xi H_0)\mathcal{R}_0,$$

where $\mathcal{R}_0$, given in (3.7), is the basic reproduction number without hygiene effects.

Thus, as H_0 increases, the effective reproduction number decreases proportionally, capturing the important role of hygiene practices in reducing typhoid transmission via environmental contamination. When $\xi H = 0$ (either no hygiene awareness or ineffective hygiene), the factor $(1 - \xi H) = 1$, so hygiene has no effect on infection spread.

3.4. Stability of disease-free equilibrium

In this section, the local and global asymptotic stabilities of the DFE of the model (2.2) are investigated.

Theorem 3. *The bacteria-free equilibrium point $\mathcal{D}_0$ of system (2.2) is locally asymptotically stable in Υ if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

Proof. The basic reproduction number $\mathcal{R}_0$ is derived using the next-generation matrix approach [23].

When $\mathcal{R}_0 < 1$, the matrix $F - V$ is Metzler, and $V - F$ is a nonsingular M -matrix. This ensures that all eigenvalues of the linearized system have negative real parts, and hence the typhoid-free equilibrium $\mathcal{D}_0$ is locally asymptotically stable. Conversely, if $\mathcal{R}_0 > 1$, the stability condition is not satisfied and $\mathcal{D}_0$ becomes unstable. Further details of this approach can be found in [23, 25].

Theorem 4. *The bacteria-free equilibrium point $\mathcal{D}_0$ of system (2.2) is locally asymptotically stable in Υ when $\mathcal{R}_s < 1$ and $\mathcal{R}_r < 1$ and unstable when $\mathcal{R}_s > 1$ or $\mathcal{R}_r > 1$.*

Theorem 4 is sufficient, but not necessary, for stability of the DFE. $\mathcal{R}_0 < 1$ is not equivalent to requiring both $\mathcal{R}_s < 1$ and $\mathcal{R}_r < 1$ due to the interaction term $\mathcal{R}_{sr}$.

3.5. Global stability of the disease-free equilibrium

The following theorem establishes the global asymptotic stability of the DFE, demonstrating that the disease will be eliminated regardless of the initial number of infected individuals whenever $\mathcal{R}_0 < 1$. We follow the famous approach of Castillo-Chavez et al. [26], as demonstrated in [27]. To analyse the global stability of the system (2.2), we rewrite it in the following form. We list two conditions that, if met, guarantee the global asymptotic stability of the disease-free state.

Define the uninfected class by

$$\mathcal{X} = (S, R, H)^T \in \mathbb{R}^3$$

and the infected class by

$$\mathcal{Y} = (I_s, I_r, T_{s1}, T_{r1}, T_{s2}, T_{r2}, B_c)^T \in \mathbb{R}^7,$$

and the DFE is written in the form $\Omega_1 = (\mathcal{X}^*, \mathbf{0})$, $\mathcal{X}^* = (\frac{\Lambda}{\mu}, \mathbf{0})$. Thus, Eq (2.2) becomes:

$$\begin{aligned} \frac{d\mathcal{X}}{dt} &= F(\mathcal{X}, \mathcal{Y}), \\ \frac{d\mathcal{Y}}{dt} &= G(\mathcal{X}, \mathcal{Y}), \quad G(\mathcal{X}^*, \mathbf{0}) = 0. \end{aligned}$$

The two conditions that must be satisfied in order for the system to be globally asymptotically stable are:

H1: For $\frac{dX}{dt} = F(X^*, \mathbf{0})$, X^* is globally asymptotically stable;

H2: $G(X, \mathcal{Y}) = A\mathcal{Y} - \widehat{G}(X, \mathcal{Y})$, such that $\widehat{G}(X, \mathcal{Y}) \geq 0$ for $(X, \mathcal{Y}) \in \Upsilon$,

whereby the Jacobian of $A = G(X^*, \mathbf{0})$ forms a Metzler matrix M , and Υ is the region of biological significance. Then the DFE is globally asymptotically stable provided that $\mathcal{R}_0 < 1$ and the conditions *H1* and *H2* are satisfied.

Theorem 5. *The DFE, Ω_1 of the model system (2.2), is globally asymptotically stable in the positive invariant region Υ if $\mathcal{R}_0 < 1$.*

Proof. For the DFE, Ω_1 of the system (2.2), to be globally asymptotically stable within the positively invariant region Υ , the system must satisfy the two conditions proposed in the Castillo–Chavez framework [26]. In this study, we establish that conditions *H1* and *H2* are satisfied.

From *H1*, we have the following system:

$$\frac{dX}{dt} = F(X^*, \mathbf{0}) = \begin{pmatrix} \Lambda - \mu S + \epsilon R \\ -Z_7 R \\ -\psi_2 H \end{pmatrix}. \quad (3.8)$$

The Jacobian of matrix (3.8) is $D_X F(X^*, \mathbf{0}) = \begin{pmatrix} -\mu & \epsilon & 0 \\ 0 & -Z_7 & 0 \\ 0 & 0 & -\psi_2 \end{pmatrix}$.

We conclude that $(X^*, \mathbf{0})$ is a global asymptotically stable equilibrium point of the system (2.2) since the system is linear, and all the eigenvalues of the Jacobian matrix are negative and real. Thus, the result holds because $\lim_{t \rightarrow \infty} \|X(t)\| = X^*$.

Applying condition *H2* to the system, the Metzler matrix M and the column vector $\widehat{G}(X, \mathcal{Y})$ are, respectively, defined as,

$$A = \begin{pmatrix} -Z_1 & 0 & 0 & 0 & 0 & 0 & \frac{\Lambda\omega\beta_b}{\kappa\mu} \\ 0 & -Z_2 & 0 & 0 & 0 & 0 & \frac{\Lambda\beta_b(1-\omega)}{\kappa\mu} \\ \sigma_s & 0 & -Z_3 & 0 & 0 & 0 & 0 \\ 0 & \sigma_r & \tau_1 & -Z_4 & 0 & 0 & 0 \\ 0 & 0 & \rho_s & 0 & -Z_5 & 0 & 0 \\ 0 & 0 & 0 & \rho_r & 0 & -Z_6 & 0 \\ \alpha_s & \alpha_r & \alpha_{s1} & \alpha_{r1} & 0 & 0 & -Z_8 \end{pmatrix}, \widehat{G}(X, \mathcal{Y}) = \begin{pmatrix} \omega\beta_b B_c \left(\frac{\Lambda}{\kappa\mu} - \frac{(1-\xi H)S}{B_c + \kappa} \right) \\ (1-\omega)\beta_b B_c \left(\frac{\Lambda}{\kappa\mu} - \frac{(1-\xi H)S}{B_c + \kappa} \right) \\ 0 \\ 0 \\ 0 \\ 0 \\ Q \end{pmatrix},$$

where $Q = \xi H(\alpha_s I_s + \alpha_r I_r + \alpha_{s1} T_{s1} + \alpha_{r1} T_{r1})$. Since $\frac{B_c}{B_c + \kappa} \leq \frac{1}{\kappa}$, because $B_c > 0$, $0 \leq (1 - \xi H) \leq 1$, $H \in [0, 1)$, and $S \leq \frac{\Lambda}{\mu}$, it follows that $\frac{(1 - \xi H)S}{B_c + \kappa} \leq \frac{S}{B_c + \kappa} \leq \frac{\Lambda}{\kappa\mu}$, that is, $\frac{\Lambda}{\kappa\mu} - \frac{(1 - \xi H)S}{B_c + \kappa} \geq 0$. We conclude that $\widehat{G}(X, \mathcal{Y}) \geq 0$ in the biologically feasible region Υ . Since the model system (2.2) satisfies conditions *H1* and *H2*, it follows from the corresponding global stability theorem that if $\mathcal{R}_0 < 1$, the DFE of (2.2) is globally asymptotically stable. This result guarantees that, under the threshold $\mathcal{R}_0 < 1$, the infection will ultimately be eradicated in the population.

3.6. Typhoid-persistent equilibria

We compute the possible typhoid-persistent equilibrium points for the system (2.2). There are three possible endemic equilibria, namely: (1) typhoid-sensitive-only equilibrium, (2) typhoid-resistant-only equilibrium, and (3) the endemic equilibrium where both typhoid strains exist.

3.6.1. Typhoid-sensitive strain only

We compute the sensitive-strain - only equilibrium, denoted by $\mathcal{D}_s = (S^*, I_s^*, T_{s1}^*, T_{s2}^*, R^*, B_c^*, H^*)$. This is obtained by setting $I_r = 0$ in system (2.2) and solving the following system of equations:

$$0 = \Lambda - (\lambda_s^* + \mu)S^* + \epsilon R^*, \quad (3.9)$$

$$0 = \lambda_s^* S^* - \mathcal{Z}_1 I_s^*, \quad (3.10)$$

$$0 = \sigma_s I_s^* - \mathcal{Z}_3 T_{s1}^*, \quad (3.11)$$

$$0 = \rho_s T_{s1}^* - \mathcal{Z}_5 T_{s2}^*, \quad (3.12)$$

$$0 = \theta_s I_s^* + \nu_s T_{s1}^* + \gamma_s T_{s2}^* - \mathcal{Z}_7 R^*, \quad (3.13)$$

$$0 = (1 - \xi H^*)(\alpha_s I_s^* + \alpha_{s1} T_{s1}^*) - \mathcal{Z}_8 B_c^*, \quad (3.14)$$

$$0 = \psi_1 I_s^* (1 - H^*) - \psi_2 H^*. \quad (3.15)$$

From Eqs (3.11), (3.12), and (3.14), we have

$$T_{s1}^* = \phi_1 I_s^*, \quad T_{s2}^* = \phi_2 I_s^*, \quad \text{and} \quad H^* = \frac{\psi_1 I_s^*}{\psi_1 I_s^* + \psi_2}, \quad \text{with} \quad \phi_1 = \frac{\sigma_s}{\mathcal{Z}_3}, \quad \phi_2 = \frac{\rho_s}{\mathcal{Z}_5} \phi_1. \quad (3.16)$$

Substituting expressions given in (3.16) into Eq (3.14), we have that:

$$R^* = \phi_5 I_s^*, \quad \text{and} \quad B_c^* = (1 - \xi H^*) \phi_7 I_s^*, \quad \text{with} \quad \phi_5 = \frac{\theta_s + \nu_s \phi_1 + \gamma_s \phi_2}{\mathcal{Z}_7}, \quad \phi_7 = \frac{\alpha_s + \alpha_{s1} \phi_1}{\mathcal{Z}_8}.$$

From (3.9), we have

$$S^* = \frac{\Lambda + \epsilon \phi_5 I_s^*}{\lambda_s^* + \mu},$$

where,

$$\lambda_s^* = \omega \frac{\psi_2 + (1 - \xi) \psi_1 I_s^*}{\psi_1 I_s^* + \psi_2} \left[\beta_b \frac{(\psi_2 + (1 - \xi) \psi_1 I_s^*) \phi_7 I_s^*}{(\psi_2 + (1 - \xi) \psi_1 I_s^*) \phi_7 I_s^* + \kappa (\psi_1 I_s^* + \psi_2)} \right]. \quad (3.17)$$

Substituting expression (3.17) into Eq (3.10) leads to the following fourth-order polynomial equation:

$$I_s^* (m_3 I_s^{*3} + m_2 I_s^{*2} + m_1 I_s^* + m_0) = 0. \quad (3.18)$$

Solving (3.18) gives $I_s^* = 0$, which corresponds to the DFE, or

$$m_3 I_s^{*3} + m_2 I_s^{*2} + m_1 I_s^* + m_0 = 0, \quad (3.19)$$

where the coefficients m_i for $i = 0, 1, 2, 3$ are

$$\begin{aligned} m_3 &= (1 - \xi)\psi_1^2\phi_7((1 - \xi)\omega\beta_b(\epsilon\phi_5 - \mathcal{Z}_1) - \mathcal{Z}_1\mu), \\ m_2 &= \psi_1(1 - \xi)\omega\phi_7\beta_b(\Lambda\psi_1(1 - \xi) + 2\psi_2\epsilon\phi_5) - \psi_1\mathcal{Z}_1\psi_2\phi_7(2\omega\beta_b(1 - \xi) + \mu(2 - \xi)) - \psi_1^2\mathcal{Z}_1\kappa\mu, \\ m_1 &= \psi_2\omega\phi_7\beta_b(2\Lambda(1 - \xi)\psi_1 + \psi_2\epsilon\phi_5) - \psi_2\mathcal{Z}_1(\psi_2\phi_7(\omega\beta_b + \mu) + 2\kappa\mu\psi_1), \\ m_0 &= \psi_2^2\kappa\mu\mathcal{Z}_1(\mathcal{R}_s - 1). \end{aligned}$$

We clearly note that, $m_0 > 0$ if and only if $\mathcal{R}_s > 1$, and $m_0 < 0$ if and only if $\mathcal{R}_s < 1$. We now apply Descartes' rule of signs to determine the number of possible positive real roots of the cubic Eq (3.19). The possibilities can be determined in Table 1, indicating the potential for multiple endemic equilibria.

The application of Descartes' rule of signs to the polynomial obtained for the sensitive strain (see Table 1) indicates that the equation may admit more than one positive real root. Since each positive root represents a biologically feasible endemic equilibrium, this result suggests the possible existence of multiple endemic equilibria for the sensitive strain. This implies that the disease dynamics may allow the persistence of the sensitive infection at different endemic levels under certain parameter conditions.

We show the existence of a backward bifurcation through numerical examples by creating bifurcation diagram around $\mathcal{R}_s = 1$ (Figure 2).

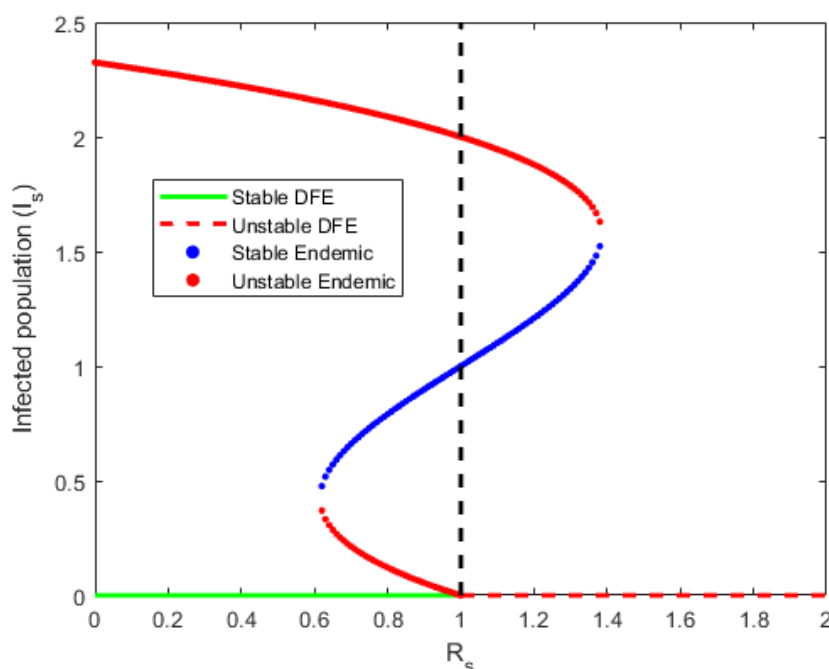


Figure 2. Backward bifurcation diagram for the sensitive-strain-only sub-model, using the parameter values as given in Table 2.

Table 1. Number of positive roots of the polynomial (3.19). Here, I_s^* denotes the number of possible roots for the three combination, $m_i^+ \Leftrightarrow m_i > 0$ for $i = 0, 1, 2, 3$ and $m_i^- \Leftrightarrow m_i < 0$ for $i = 0, 1, 2, 3$.

Coefficients of the Polynomial (m_3, m_2, m_1, m_0)				I_s^*
m_3^+	m_2^+	m_1^+	m_0^+	0
			m_0^-	1
		m_1^-	m_0^+	2
			m_0^-	1
	m_2^-	m_1^+	m_0^+	2
			m_0^-	3
		m_1^-	m_0^+	2
			m_0^-	1
m_3^-	m_2^+	m_1^+	m_0^+	1
			m_0^-	2
		m_1^-	m_0^+	3
			m_0^-	2
	m_2^-	m_1^+	m_0^+	1
			m_1^-	2
		m_1^-	m_0^+	1
			m_0^-	0

Figure 2 shows the bifurcation diagram of I_s^* as a function of $\mathcal{R}_s$, obtained from the cubic equilibrium equation using the parameter values listed in Table 2. The figure shows the presence of multiple equilibria for a given reproduction number, including two stable endemic equilibria separated by an unstable threshold. This means that even when the reproduction number is less than one, the disease can persist at a low endemic level if the infection is above the unstable threshold.

Table 2. Parameter values used in the numerical simulations and their descriptions.

Par.	Description	Range	Baseline	Unit	Source
γ_s	Recovery rate for individuals receiving second-line treatment (T_{s2})	0–1	0.4	month ⁻¹	[20]
γ_r	Recovery rate for individuals receiving second-line treatment (T_{r2})	0–1	0.35	month ⁻¹	[20]
ρ_s	Switching rate from first-line to second-line treatment ($T_{s1} \rightarrow T_{s2}$)	0–1	0.25	month ⁻¹	[20]
ρ_r	Switching rate from first-line to second-line treatment ($T_{r1} \rightarrow T_{r2}$)	0–1	0.25	month ⁻¹	[20]
κ	Half-saturation constant for environmental transmission	$0 \leq \kappa \leq 1$	0.62	bacteria concentration	[25]
θ_s	Natural recovery rate of individuals in I_s	0–1	0.6	month ⁻¹	[25, 30]
θ_r	Natural recovery rate of individuals in I_r	0–1	0.3	month ⁻¹	[25, 30]
δ_{s1}	Disease-induced death rate for individuals in T_{s1}	0–0.001	0.0001	month ⁻¹	[25, 30]
δ_{r1}	Disease-induced death rate for individuals in T_{r1}	0–0.001	0.0002	month ⁻¹	[25, 30]
ω	Proportion of infections caused by sensitive strains	$0 \leq \omega \leq 1$	0.8	dimensionless	[31]
Λ	Recruitment rate into the susceptible population	1–6	2	individuals month ⁻¹	[32]
μ	Natural death rate of humans	0.0001–0.021	0.001658	month ⁻¹	[32]
ϵ	Rate at which recovered individuals lose immunity and become susceptible	0.0833–0.666	0.0834	month ⁻¹	[32]
δ_s	Disease-induced death rate for I_s individuals	0.001–0.15	0.001	month ⁻¹	[32]
δ_r	Disease-induced death rate for I_r individuals	0.0001–0.01	0.0089	month ⁻¹	[32]
ν_s	Recovery rate of individuals receiving first-line treatment (T_{s1})	0–0.60012	0.002485	month ⁻¹	[32]
ν_r	Recovery rate of individuals receiving first-line treatment (T_{r1})	0–0.60012	0.002485	month ⁻¹	[32]
δ_{s2}	Disease-induced death rate for individuals in T_{s2}	0–0.00001	0.0001	month ⁻¹	Assumed
δ_{r2}	Disease-induced death rate for individuals in T_{r2}	0–0.00001	0.0003	month ⁻¹	Assumed
α_s	Bacterial shedding rate from infected individuals (I_s)	0–0.15	0.03	bacteria month ⁻¹	[33]
α_r	Bacterial shedding rate from infected individuals (I_r)	0–0.15	0.02	bacteria month ⁻¹	[33]
α_{s1}	Bacterial shedding rate from individuals in T_{s1}	0–0.15	0.003	bacteria month ⁻¹	[33]
α_{r1}	Bacterial shedding rate from individuals in T_{r1}	0–0.15	0.002	bacteria month ⁻¹	[33]
μ_b	Natural death rate of bacteria in the environment	0.001–0.15	0.05	month ⁻¹	[32]
ψ_1	Rate at which hygiene awareness increases in response to infection	0–1	0.4	month ⁻¹	Assumed
ψ_2	Rate at which hygiene awareness decays over time	0–1	0.1	month ⁻¹	Assumed

The conditions for backward bifurcation are analysed using the center manifold theory described in Theorem 4.1, as established by Castillo-Chavez and Song [28]. Rather than restating the theorem, we focus on its application. This approach has been used in several studies, including [25, 29].

For the sensitive-strain sub-model, $I_r = 0$, we thus introduce the following change of variables:

$$S = x_1, \quad I_s = x_2, \quad T_{s1} = x_3, \quad T_{s2} = x_4, \quad R = x_5, \quad B_c = x_6, \quad H = x_7,$$

so that the total sensitive population is defined as $N_s = \sum_{i=1}^5 x_i$. We now define the state vector

$\mathbf{X} = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$. With this notation, system (2.2) can be written compactly as a system of differential equations:

$$\frac{d\mathbf{X}}{dt} = \mathbf{F}(t, \mathbf{X}(t)) = (f_1^s, f_2^s, f_3^s, f_4^s, f_5^s, f_6^s, f_7^s)^T,$$

where the components f_i^s for $i = 1, 2, 3, 4, 5, 6, 7$ correspond to the right-hand sides of the respective

equations in system (2.2). Explicitly:

$$\left\{ \begin{array}{l} \frac{dx_1}{dt} = \Lambda - \omega \left(\frac{\beta_b x_6}{x_6 + \kappa} \right) x_1 (1 - \xi x_7) - \mu x_1 + \epsilon x_5 = f_1^s, \\ \frac{dx_2}{dt} = \omega \left(\frac{\beta_b x_6}{x_6 + \kappa} \right) x_1 (1 - \xi x_7) - \mathcal{Z}_1 x_2 = f_2^s, \\ \frac{dx_3}{dt} = \sigma_s x_2 - \mathcal{Z}_3 x_3 = f_3^s, \\ \frac{dx_4}{dt} = \rho_s x_3 - \mathcal{Z}_5 x_4 = f_4^s, \\ \frac{dx_5}{dt} = \theta_s x_2 + \nu_s x_3 + \gamma_s x_4 - \mathcal{Z}_7 x_5 = f_5^s, \\ \frac{dx_6}{dt} = (1 - \xi x_7)(\alpha_s x_2 + \alpha_{s1} x_3) - \mathcal{Z}_8 x_6 = f_6^s, \\ \frac{dx_7}{dt} = \psi_1 x_2 (1 - x_7) - \psi_2 x_7 = f_7^s. \end{array} \right. \quad (3.20)$$

In our analysis, we select β_b as the bifurcation parameter and investigate the system dynamics near the threshold $\mathcal{R}_s = 1$. The condition for the sub-reproduction number $\mathcal{R}_s = 1$ yields a critical threshold value β_b^* , given by:

$$\beta_b = \beta_b^{*s} = \frac{\mu \kappa \mathcal{Z}_1 \mathcal{Z}_3 \mathcal{Z}_8}{\Lambda \omega (\mathcal{Z}_3 \alpha_s + \alpha_{s1} \sigma_s)}.$$

Taking the Jacobian matrix of the system (3.20) in the DFE, denoted by $\mathcal{D}_{s0} = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0 \right)$, and substituting $\beta_b = \beta_b^{*s}$, we obtain:

$$J(\mathcal{D}_{s0}) = \begin{pmatrix} -\mu & 0 & 0 & 0 & \epsilon & -\omega \frac{\beta_b^{*s} \Lambda}{\mu \kappa} & 0 \\ 0 & -\mathcal{Z}_1 & 0 & 0 & 0 & \omega \frac{\beta_b^{*s} \Lambda}{\mu \kappa} & 0 \\ 0 & \sigma_s & -\mathcal{Z}_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \rho_s & -\mathcal{Z}_5 & 0 & 0 & 0 \\ 0 & \theta_s & \nu_s & \gamma_s & -\mathcal{Z}_7 & 0 & 0 \\ 0 & \alpha_s & \alpha_{s1} & 0 & 0 & -\mathcal{Z}_8 & 0 \\ 0 & \psi_1 & 0 & 0 & 0 & 0 & -\psi_2 \end{pmatrix},$$

where $\mathcal{Z}_i$, for $i = 1, 3, 5, 7, 8$ are as defined in (3.4).

The eigenvalues of $J(\mathcal{D}_{s0})$ are:

$$\mathcal{H}_1 = 0, \quad \mathcal{H}_2 = -\mu, \quad \mathcal{H}_3 = -\mathcal{Z}_5, \quad \mathcal{H}_4 = -\mathcal{Z}_7, \quad \mathcal{H}_7 = -\psi_2,$$

$$\mathcal{H}_{5,6} = -\frac{1}{2\mathcal{W}_1} \left(\mathcal{W}_2 + \sqrt{\mathcal{W}_1} \sqrt{\mathcal{Z}_1^2 \mathcal{W}_1 + (\mathcal{Z}_3 - \mathcal{Z}_8)^2 \mathcal{W}_1 - 2\mathcal{Z}_1 (\mathcal{Z}_3 \alpha_s (\mathcal{Z}_3 - \mathcal{Z}_8) + \alpha_{s1} \sigma_s (\mathcal{Z}_8 + \mathcal{Z}_3))} \right),$$

where

$$\mathcal{W}_1 = \mathcal{Z}_3 \alpha_s + \alpha_{s1} \sigma_s, \quad \mathcal{W}_2 = (\mathcal{Z}_3 \alpha_s + \alpha_{s1} \sigma_s) (\mathcal{Z}_1 + \mathcal{Z}_3 + \mathcal{Z}_8).$$

It can be seen that the model system (3.20), with $\beta_b = \beta_b^{*s}$, has a simple eigenvalue, hence the center manifold theory can be used to analyse the dynamics of the sensitive sub-model near $\beta_b = \beta_b^{*s}$. It can be shown that $J(\mathcal{D}_{s0})$, has a right eigenvector given by $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$, where,

$$w_1 = \frac{\psi_2(\mathcal{Z}_3\mathcal{Z}_5\theta_s\epsilon + \mathcal{Z}_5\epsilon\nu_s\sigma_s + \epsilon\gamma_s\rho_s\sigma_s - \mathcal{Z}_1\mathcal{Z}_3\mathcal{Z}_5\mathcal{Z}_7)}{\mathcal{Z}_3\mathcal{Z}_5\mathcal{Z}_7\psi_1\mu}, \quad w_2 = \frac{\psi_2}{\psi_1}, \quad w_3 = \frac{\sigma_s\psi_2}{\mathcal{Z}_3\psi_1},$$

$$w_4 = \frac{\rho_s\sigma_s\psi_2}{\mathcal{Z}_3\mathcal{Z}_5\psi_1}, \quad w_5 = \frac{\psi_2(\mathcal{Z}_3\mathcal{Z}_5\theta_s + \mathcal{Z}_5\nu_s\sigma_s + \gamma_s\rho_s\sigma_s)}{\mathcal{Z}_3\mathcal{Z}_5\mathcal{Z}_7\psi_1}, \quad w_6 = \frac{\psi_2(\mathcal{Z}_3\alpha_s + \alpha_{s1}\sigma_s)}{\mathcal{Z}_3\mathcal{Z}_8\psi_1}, \quad w_7 = 1.$$

Furthermore, the left eigenvectors of $J(\mathcal{D}_{s0})$, associated with the zero eigenvalue at $\beta_b = \beta_b^{*s}$, are given by $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)^T$, where

$$v_1 = v_4 = v_5 = v_7 = 0, \quad v_2 = \frac{\mathcal{Z}_3\alpha_s + \alpha_{s1}\sigma_s}{\mathcal{Z}_1\mathcal{Z}_3}, \quad v_3 = \frac{\alpha_{s1}}{\mathcal{Z}_3}, \quad v_6 = 1.$$

The sign of a and b determines the type of bifurcation.

$$a = \sum_{k,i,j=1}^7 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{D}_{s0}, \beta_b^{*s}) \quad \text{and} \quad b = \sum_{k,i=1}^7 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_b}(\mathcal{D}_{s0}, \beta_b^{*s}).$$

For system (3.20), the associated non-zero second-order partial derivatives of $\mathbf{F}$ at DFE equilibrium are

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_6} = -\frac{\omega\beta_b^{*s}}{\kappa}, \quad \frac{\partial^2 f_1}{\partial x_6 \partial x_6} = \frac{2\omega\Lambda\beta_b^{*s}}{\mu\kappa^2}, \quad \frac{\partial^2 f_1}{\partial x_6 \partial x_7} = \frac{\omega\xi\Lambda\beta_b^{*s}}{\mu\kappa}, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_6} = \frac{\omega\beta_b^{*s}}{\kappa}, \quad \frac{\partial^2 f_2}{\partial x_6 \partial x_6} = -\frac{2\omega\Lambda\beta_b^{*s}}{\mu\kappa^2},$$

$$\frac{\partial^2 f_2}{\partial x_6 \partial x_7} = -\omega\xi\beta_b^{*s}\frac{\Lambda}{\mu\kappa}, \quad \frac{\partial^2 f_6}{\partial x_2 \partial x_7} = -\xi\alpha_s, \quad \frac{\partial^2 f_6}{\partial x_3 \partial x_7} = -\xi\alpha_{s1}, \quad \frac{\partial^2 f_7}{\partial x_2 \partial x_7} = -\psi_1, \quad \frac{\partial^2 f_1}{\partial x_6 \partial \beta_b^{*s}} = -\omega\frac{\Lambda}{\mu\kappa},$$

$$\frac{\partial^2 f_2}{\partial x_6 \partial \beta_b^{*s}} = \omega\frac{\Lambda}{\mu\kappa}.$$

Clearly, since $v_1 = v_4 = v_5 = v_7 = 0$, it thus follows that

$$a = \sum_{i,j=1}^7 v_6 w_i w_j \frac{\partial^2 f_6}{\partial x_i \partial x_j} + \sum_{i,j=1}^7 v_2 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + \sum_{i,j=1}^7 v_3 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}$$

$$= 2\left(\omega\frac{\beta_b^{*s}}{\kappa} v_2 w_1 w_6 - \left(\xi w_7(\alpha_s w_2 + \alpha_{s1} w_3) + \frac{\omega\beta_b^{*s}\Lambda v_2 w_6}{\mu\kappa}\left(\frac{w_6}{\kappa} + \xi w_7\right)\right)\right)$$

$$= 2(\mathcal{M}_1^s - \mathcal{M}_2^s),$$

where

$$\mathcal{M}_1^s = \omega\frac{\beta_b^{*s}}{\kappa} v_2 w_1 w_6 \quad \text{and} \quad \mathcal{M}_2^s = \xi w_7(\alpha_s w_2 + \alpha_{s1} w_3) + \frac{\omega\beta_b^{*s}\Lambda v_2 w_6}{\mu\kappa}\left(\frac{w_6}{\kappa} + \xi w_7\right),$$

and,

$$b = \sum_{k,i=1}^7 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_b}(\mathcal{D}_{s0}, \beta_b^{*s}) = \frac{\omega\Lambda w_6}{\mu\kappa}(v_2 - v_1) = \frac{\Lambda\omega}{\mu\kappa} v_2 w_6 > 0.$$

We therefore have the following results:

Theorem 6. If $\mathcal{M}_1^s > \mathcal{M}_2^s$, then the model (3.20) exhibits a backward bifurcation at $\mathcal{R}_s = 1$. Otherwise, if $\mathcal{M}_1^s < \mathcal{M}_2^s$, the endemic equilibrium exists and is locally asymptotically stable for $\mathcal{R}_s > 1$ near one.

In this case, backward bifurcation implies the existence of a locally asymptotically stable endemic equilibrium for $\mathcal{R}_s < 1$.

Remark 2. The condition $\mathcal{M}_1^s > \mathcal{M}_2^s$ indicates that the combined effects of transmission and persistence outweigh the effects of recovery and control. In this case, the disease can persist even when the basic reproduction number $\mathcal{R}_s$ is less than one, leading to backward bifurcation, where both the disease-free and endemic equilibria coexist (see Figure 2).

From a public health perspective, the inequality $\mathcal{M}_1^s > \mathcal{M}_2^s$ implies that reducing $\mathcal{R}_s$ below one is not sufficient to eliminate the disease. Additional interventions are required, such as improving hygiene effectiveness, reducing environmental contamination, and enhancing treatment outcomes.

On the other hand, if $\mathcal{M}_1^s < \mathcal{M}_2^s$, then control measures dominate transmission, and the standard threshold result holds: When $\mathcal{R}_s < 1$, the disease will eventually die out.

3.6.2. Typhoid-resistant strain only

We compute the resistant-strain- only equilibrium, denoted by $\mathcal{D}_r = (S^\circ, I_r^\circ, T_{r1}^\circ, T_{r2}^\circ, R^\circ, B_c^\circ, H^\circ)$. This is obtained by setting $I_s = 0$ in system (2.2) and solving the system of equations. We obtain the following:

$$\begin{cases} T_{r1}^\circ = \phi_3 I_r^\circ, & T_{r2}^\circ = \phi_4 I_r^\circ, & H^\circ = \frac{\psi_1 I_r^\circ}{\psi_1 I_r^\circ + \psi_2}, & R^\circ = \phi_6 I_r^\circ, \\ B_c^\circ = (1 - \xi H^\circ) \phi_8 I_r^\circ, & S^\circ = \frac{\Lambda + \epsilon \phi_6 I_r^\circ}{\lambda_r^\circ + \mu}, & I_r^\circ = \frac{1}{\mathcal{Z}_2} \frac{\lambda_r^\circ (\Lambda + \epsilon \phi_6 I_r^\circ)}{\lambda_r^\circ + \mu}, \end{cases} \quad (3.21)$$

with

$$\lambda_r^\circ = (1 - \omega) \frac{\psi_2 + (1 - \xi) \psi_1 I_r^\circ}{\psi_1 I_r^\circ + \psi_2} \left[\beta_b \frac{(\psi_2 + (1 - \xi) \psi_1 I_r^\circ) \phi_8 I_r^\circ}{(\psi_2 + (1 - \xi) \psi_1 I_r^\circ) \phi_8 I_r^\circ + \kappa (\psi_1 I_r^\circ + \psi_2)} \right].$$

Simplifying I_r° in Eq (3.21), we have

$$I_r^\circ (n_3 I_r^{\circ 3} + n_2 I_r^{\circ 2} + n_1 I_r^\circ + n_0) = 0. \quad (3.22)$$

Solving (3.22) gives $I_r^\circ = 0$, which corresponds to the typhoid-resistant- free equilibrium or

$$n_3 I_r^{\circ 3} + n_2 I_r^{\circ 2} + n_1 I_r^\circ + n_0 = 0,$$

where the coefficients n_i for $i = 0, 1, 2, 3$ are given (3.23) as

$$\begin{aligned} n_3 &= \phi_8 \psi_1^2 (1 - \xi) \left((1 - \xi)(1 - \omega) \beta_b (\epsilon \phi_6 - \mathcal{Z}_2) - \mu \mathcal{Z}_2 \right), \\ n_2 &= \psi_1 (1 - \xi) (1 - \omega) \beta_b \phi_8 \left(\psi_1 (1 - \xi) \Lambda + 2 \psi_2 \epsilon \phi_6 \right) - \mathcal{Z}_2 \psi_1 \psi_2 \phi_8 \left(2(1 - \omega) \beta_b (1 - \xi) + \mu (2 - \xi) \right) - \psi_1^2 \mathcal{Z}_2 \kappa \mu, \\ n_1 &= \psi_2 \beta_b \phi_8 (1 - \omega) \left(2 \Lambda (1 - \xi) \psi_1 + \epsilon \phi_6 \psi_2 \right) - \mathcal{Z}_2 \psi_2 \left(2 \kappa \mu \psi_1 + \phi_8 \psi_2 (\mu + (1 - \omega) \beta_b) \right), \\ n_0 &= \kappa \mu \mathcal{Z}_2 \psi_2^2 (\mathcal{R}_r - 1). \end{aligned} \quad (3.23)$$

We clearly note that $n_0 > 0$ if and only if $\mathcal{R}_r > 1$ and $n_0 < 0$ if and only if $\mathcal{R}_r < 1$. Descartes' rule of signs can be applied to determine the number of possible positive real roots of the quadratic Eq (3.22).

Remark 3. The analysis for the resistant strain follows the same general approach as for the sensitive strain. In particular, the existence of equilibria and their stability can be obtained using the same approach. The results show that the resistant strain can also persist in the population and is governed by its own $\mathcal{R}_r$ (see Eq (3.6)).

3.6.3. Endemic equilibrium for the full model

Let us denote the endemic equilibrium point by $\mathcal{D}_1 = (S^*, I_s^*, I_r^*, T_{s1}^*, T_{r1}^*, T_{s2}^*, T_{r2}^*, R^*, B_c^*, H^*)$, a non-trivial equilibrium for the model (2.2). Setting the right-hand side of the model (2.2) to zero gives

$$\left\{ \begin{array}{l} T_{s1}^* = \Phi_1 I_s^*, \quad T_{r1}^* = \Phi_3 I_r^*, \quad T_{s2}^* = \Phi_2 I_s^*, \quad T_{r2}^* = \Phi_4 I_r^*, \quad H^* = \frac{\psi_1(I_s^* + I_r^*)}{\psi_1(I_s^* + I_r^*) + \psi_2}, \\ R^* = \Phi_5 I_s^* + \Phi_6 I_r^*, \quad B_c^* = (1 - \xi H^*)(\Phi_7 I_s^* + \Phi_8 I_r^*), \quad S^* = \frac{\Lambda + \epsilon(\Phi_5 I_s^* + \Phi_6 I_r^*)}{\lambda_s^* + \lambda_r^* + \mu}, \\ I_s^* = \omega \beta_b A(I_s^*, I_r^*) \frac{1}{\mathcal{Z}_1} \left(\frac{\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*)}{\psi_1(I_s^* + I_r^*) + \psi_2} \right) \frac{\Lambda + \epsilon(\Phi_5 I_s^* + \Phi_6 I_r^*)}{\lambda_s^* + \lambda_r^* + \mu}, \\ I_r^* = (1 - \omega)\beta_b A(I_s^*, I_r^*) \frac{1}{\mathcal{Z}_2} \left(\frac{\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*)}{\psi_1(I_s^* + I_r^*) + \psi_2} \right) \frac{\Lambda + \epsilon(\Phi_5 I_s^* + \Phi_6 I_r^*)}{\lambda_s^* + \lambda_r^* + \mu}, \end{array} \right. \quad (3.24)$$

with;

$$\begin{aligned} \lambda_s^* &= \omega \beta_b A(I_s^*, I_r^*) \left(\frac{\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*)}{\psi_1(I_s^* + I_r^*) + \psi_2} \right), \\ \lambda_r^* &= (1 - \omega)\beta_b A(I_s^*, I_r^*) \left(\frac{\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*)}{\psi_1(I_s^* + I_r^*) + \psi_2} \right), \\ A(I_s^*, I_r^*) &= \frac{(\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*))(\Phi_7 I_s^* + \Phi_8 I_r^*)}{(\psi_2 + (1 - \xi)\psi_1(I_s^* + I_r^*))(\Phi_7 I_s^* + \Phi_8 I_r^*) + \kappa(\psi_1(I_s^* + I_r^*) + \psi_2)}. \end{aligned}$$

The system of equations in (3.24) describes the equilibrium conditions for the infected classes (I_s^*) and (I_r^*). However, due to the strong nonlinearity, the resulting equations become highly complex, making it difficult to obtain an explicit expression for the endemic equilibrium. The trivial solution $I_s^* = 0$ and $I_r^* = 0$ corresponds to the DFE. The qualitative behaviour of the full model is further investigated using global stability analysis and numerical simulations.

4. Model calibration and sensitivity analysis

In this section, we first calibrate the proposed typhoid fever model using the available epidemiological data in order to estimate the key model parameters. We then examine the identifiability of these parameters to ensure that they can be reliably determined from the data. Finally, we perform a sensitivity analysis to determine how changes in model parameters influence the basic reproduction number and other important model outcomes. By combining calibration with identifiability analysis, this paper ensures that the estimated parameter values are not only capable of reproducing the observed dynamics but are also supported by sufficient information in the data. This

integrated approach strengthens the credibility of the model predictions and enhances the reliability of subsequent analyses, including sensitivity assessment and policy scenario simulations.

4.1. Model calibration

In this section, we calibrate the proposed typhoid fever model using epidemiological data obtained from Mbandjock, a town in the Centre Region of Cameroon [32]. The dataset consists of records of diagnostic tests conducted at Mbandjock District Hospital during the period from 01/08/2019 to 31/07/2020. A summary of the data collected is provided in Table 3.

Table 3. Tests carried out to detect the presence of *Salmonella typhi* in patients attending consultation at Mbandjock District Hospital from 01/08/2019 to 31/07/2020. These data were obtained from [32].

Month	Year	Number of tests performed	Number of positive tests	Number of negative tests
August	2019	83	60	23
September	2019	60	50	10
October	2019	56	38	18
November	2019	52	46	6
December	2019	69	62	7
January	2020	76	71	5
February	2020	80	74	6
March	2020	95	43	52
April	2020	68	29	39
May	2020	95	52	43
June	2020	65	38	27
July	2020	58	32	26

In our model (2.2), individuals infected with typhoid are represented by the compartments I_s and I_r , corresponding to infections with drug-sensitive and drug-resistant strains, respectively. We assume that infected individuals are reported to the hospital and begin first-line treatment at rates σ_s and σ_r .

The model (2.2) is calibrated using reported monthly confirmed typhoid cases.

$$\text{NTC}_{\text{data}}(t_i), \quad i = 0, 1, \dots, 11.$$

Let us denote the total number of cases reported at time t_i , where t_0 corresponds to August 2019 and t_{11} corresponds to July 2020. The available epidemiological data (see Table (3)) report only the total number of confirmed typhoid cases without distinguishing between drug-sensitive and drug-resistant strains. To capture the role of antimicrobial resistance, the model includes separate compartments for each strain. Because strain-specific incidence data are not available, several strain-dependent parameters are assigned values from the literature to ensure model identifiability. As a result, strain-specific components of the model cannot be directly calibrated using separate datasets. Instead, they are incorporated through the overall transmission framework of the model. This data limitation may influence the identifiability and precise estimation of the parameters associated with each strain.

This approach allows the model to represent the overall burden of the disease, while allowing exploration of resistance dynamics and intervention scenarios. The model calibration is performed using the total predicted incidence. The model predicted number of new reported cases is defined as

$$\text{NTC}_{\text{predict}}(t) = \sigma(I_s(t) + I_r(t)),$$

where $\sigma_s = \sigma_r = \sigma$ represents the rate of initiation of treatment, assumed to be the same for sensitive and resistant infections. Due to limited epidemiological data, only parameters that directly influence transmission dynamics and case reporting are estimated. The remaining parameters are fixed on the basis of available literature to ensure structural identifiability and numerical stability of the estimation procedure. In particular, recovery rates, disease-induced mortality rates, bacterial shedding rates, and the strain proportion parameter ω are not estimated but assigned fixed biologically plausible values.

The vector of estimated parameters is given by:

$$\Pi = (\beta_b, \sigma, g, \xi).$$

Parameter estimation is performed by solving the nonlinear least squares problem

$$\min_{\Pi} \|\text{NTC}_{\text{predict}}(t; \Pi) - \text{NTC}_{\text{data}}(t)\|_2^2.$$

The hygiene variable $H(t)$ represents the level of hygiene behaviour in the population and scaled between 0 and 1. Since no data on hygiene awareness or behaviour are available, $H(t)$ is not directly measured or fitted to the data and is treated as an unobserved variable. Therefore, the parameters governing awareness dynamics are fixed at plausible values to ensure a stable and identifiable calibration process.

The system of differential equations of model (2.2) is fitted on the typhoid data of Table 3 using the nonlinear least squares fitting routine *lsqnonlin* in the optimization tool box of MATLAB (R2024a).

The initial conditions used in the simulations are the following:

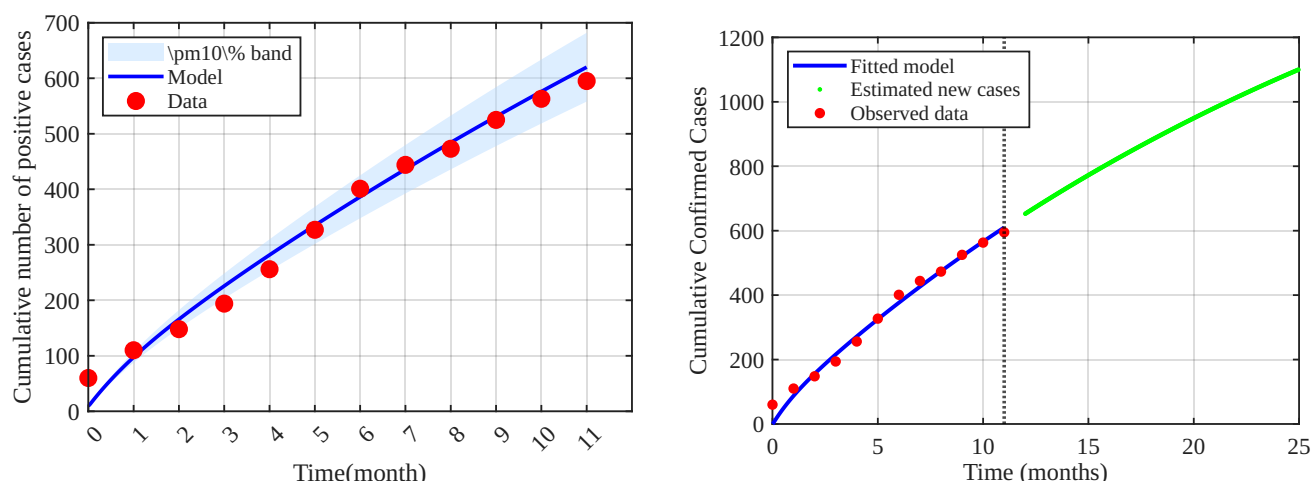
$$\begin{aligned} S(0) = 21076, \quad I_s(0) = 40, \quad I_r(0) = 20, \quad T_{s1}(0) = 2, \quad T_{r1}(0) = 1, \\ T_{s2}(0) = 0, \quad T_{r2}(0) = 0, \quad R(0) = 1, \quad B_c(0) = 500, \quad H(0) = 0.2. \end{aligned} \quad (4.1)$$

The initial susceptible population is set at $S(0) = 21076$, corresponding to the estimated population served by the Mbandjock District Hospital 2019–2020. The initial concentration of environmental bacteria, $B_c(0) = 500$, is obtained from [32], based on data from the same geographical setting.

Since direct measurements of the environmental bacterial load were unavailable, this value was chosen within a biologically plausible range to ensure realistic initial transmission dynamics and numerical stability during model calibration. The monthly cases reported vary considerably, ranging from 29 to 74 cases. Calibration is performed using the cumulative confirmed case data instead of the raw monthly counts. The cumulative number of cases predicted by the model is computed as $C(t) = \sigma \int_0^t (I_s(u) + I_r(u)) du$. This approach improves numerical accuracy and avoids errors that may arise from manually summing discrete model outputs.

This paper uses a limited dataset (monthly data from August 2019 to July 2020 at Mbandjock Hospital). The model provides reasonable projections up to September 2021, extending about 80%

beyond the observed period. The results are consistent with those of a related study conducted at Penka-Michel Hospital [13], which used a weekly dataset (from December 2019 to January 2023) collected at Penka-Michel Hospital and projected results up to November 2026. The results of this paper are in alignment with those reported in [13], although the data was collected from different hospitals, over different time periods, and in different geographical areas. This suggests that both studies are capturing similar patterns in the spread of typhoid fever, and that the findings are not limited to one specific location.



(a) Model fit on the cumulative number of positive cases per month (Aug 2019–Jul 2020)

(b) Model validation and prediction on the dynamics of typhoid disease for the period up to September 2021.

Figure 3. Model fit and calibration on the dynamics of typhoid fever in Mbandjock city.

Although monthly incidence data are available, the reported case counts are relatively small and subject to considerable fluctuations. To reduce the impact of this variability and obtain a stable fit, the model was fitted to cumulative reported cases (see Figure 3). Although incidence data are generally preferred for statistical inference, cumulative data provide a smoother representation of the outbreak trajectory. However, this approach may introduce dependencies between observations and potentially underestimate uncertainty, and results should be interpreted with this limitation.

Before presenting the simulation results, a parameter identifiability analysis is conducted to assess whether the estimated parameters can be reliably determined from the available data and to justify the selection of estimated and fixed parameters.

4.2. Parameter identifiability analysis

Given the limited number of observations (12 monthly data points) relative to the number of model parameters, we assess the practical identifiability of the estimated parameter vector $\Pi = (\beta_b, \sigma, g, \xi)$. With only $n = 12$ monthly observations and a model containing more than 20 parameters, direct estimation of the full parameter set is statistically ill-posed. Following the approach of [34, 35], we therefore partition the parameter space into a small set of estimated parameters Π that directly govern transmission intensity and case ascertainment, and a set of fixed parameters sourced from the literature

(Table 2). The estimated parameters enter the observation equation

$$\widehat{C}(t; \Pi) = \sigma \int_0^t (I_s + I_r) du,$$

directly or through the forces of infection, making them the primary drivers of the data-fit. Parameters governing compartmental transitions (recovery rates, disease-induced mortality, shedding rates) are obtained from the literature.

4.2.1. Calibration results

The calibrated parameter estimates together with their 95% confidence intervals (profile likelihood) are reported in Table 4. The model achieves a strong cumulative fit with $R^2_{\text{cumul}} = 0.9843$ close to one and a small root mean square error, confirming that the four-parameter subset $(\beta_b, \sigma, g, \xi)$ is sufficient to reproduce the observed trend in cumulative typhoid burden at Mbandjock District Hospital.

Table 4. Calibration of the model parameters (2.2) using nonlinear least squares fitting routine *lsqnonlin* of the optimization toolbox of MATLAB software.

Parameter	Lower bound	Upper bound	Estimated value	Source
β_b	0.0001	1	0.2	Fitted
σ	0.001	0.8	0.002	Fitted
g	0.001	0.03	0.014	Fitted
ξ	0.01	0.99	0.3	Fitted

4.3. Sensitivity analysis

After calibrating the model to observed typhoid data, a sensitivity analysis was carried out to examine how changes in the model parameters affect the effective reproduction number $\mathcal{R}_{0e}$. Although some parameters were estimated from data, there is still uncertainty in their values, as well as in the parameters obtained from the literature. Therefore, it is important to assess how variations in parameter values influence model outcomes.

In this paper, a global sensitivity analysis is conducted using latin hypercube sampling (LHS) combined with partial rank correlation coefficients (PRCC). LHS is an efficient statistical method that allows parameters to be sampled simultaneously across their entire ranges [36]. PRCC is then used to measure the strength and direction of the relationship between each parameter and the model output while accounting for the effects of other parameters [37]. This approach has been widely used in infectious disease modelling to identify key drivers of transmission and to guide control strategies.

A positive PRCC indicates that increasing the parameter increases $\mathcal{R}_{0e}$, while a negative PRCC suggests that increasing the parameter reduces $\mathcal{R}_{0e}$. Parameters with high absolute PRCC values are considered highly influential and may serve as key targets for intervention strategies. Therefore, this analysis provides information on which biological or treatment-related processes contribute the most to the spread of typhoid fever and antibiotic resistance dynamics in the model. The results identify

key parameters, such as the transmission rate, and hygiene effectiveness, that significantly influence disease dynamics and may inform targeted intervention strategies. The environmental transmission rate β_b has the highest positive sensitivity index (see Figure 4), confirming that reducing contact between susceptible individuals and contaminated water or food is the most impactful for reducing $\mathcal{R}_{0e}$.

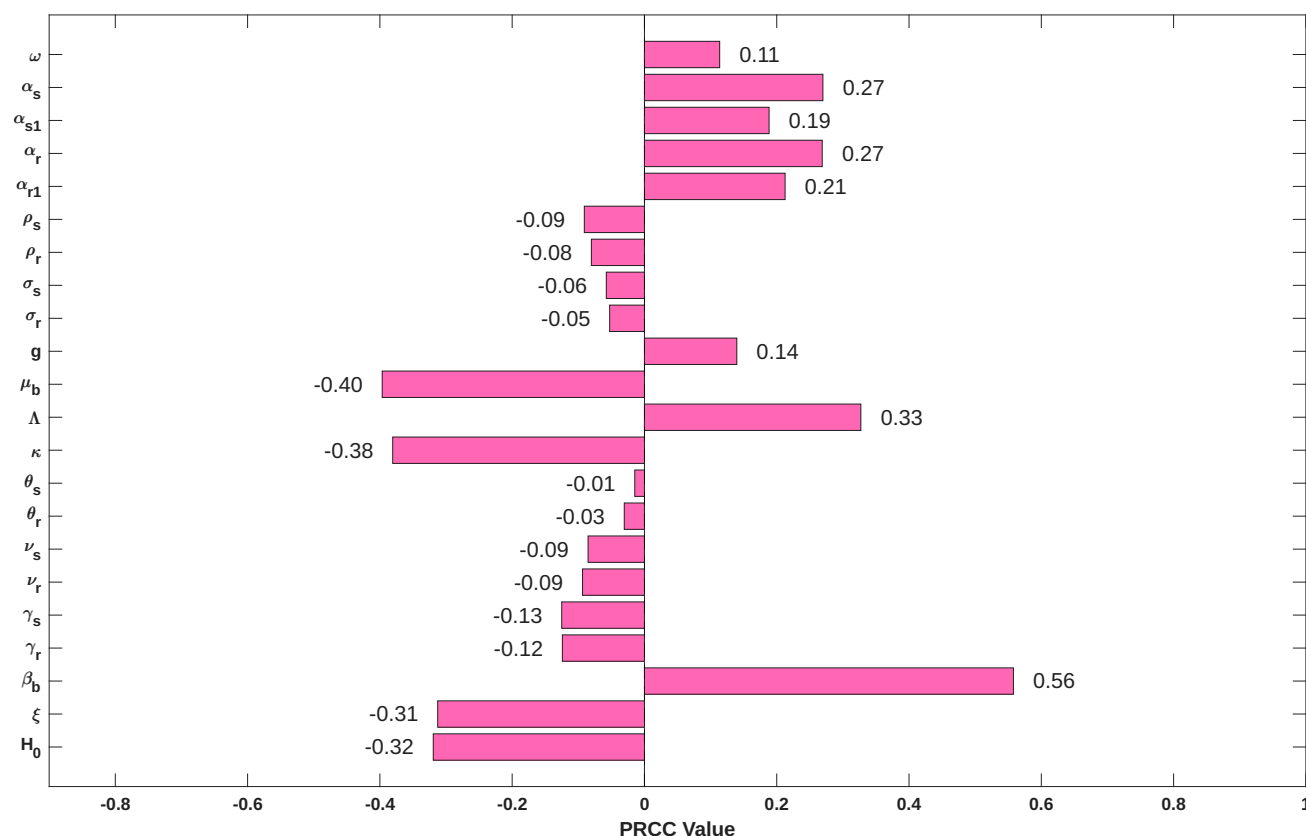


Figure 4. Partial rank correlation coefficient of $\mathcal{R}_{0e}$ with respect to model parameters.

5. Numerical simulations

Numerical simulations are performed to examine the effects of key parameters, including transmission, treatment, and hygiene-related factors, on the dynamics of the model. Using the values of the fitted parameters, together with the fixed parameters in Table 2, the model (2.2) is simulated over a period of 25 months. The initial conditions are given in (4.1).

5.1. Effect of hygiene effectiveness on transmission

Figure 5 presents a contour plot showing the combined effects of the environmental transmission rate, β_b , and hygiene effectiveness, ξ , on the effective reproduction number, $\mathcal{R}_{0e}$. The results show that $\mathcal{R}_{0e}$ increases as the transmission rate β_b increases, meaning that higher environmental contamination may lead to greater disease spread. In contrast, increasing hygiene effectiveness ξ reduces $\mathcal{R}_{0e}$. This indicates that improved hygiene practices, such as safe water use and sanitation, can significantly reduce transmission. Overall, the results emphasize the importance of environmental control and

hygiene interventions in reducing transmission and preventing sustained outbreaks.

Figure 6(a) shows the combined effects of the bacterial shedding rate, α_s , and hygiene effectiveness, ξ , on the effective reproduction number, $\mathcal{R}_{0e}$. The results indicate that $\mathcal{R}_{0e}$ increases as α_s increases. This means that when infected individuals shed more bacteria into the environment, the potential for disease transmission increases. In contrast, increasing hygiene effectiveness ξ reduces $\mathcal{R}_{0e}$, reflecting the important role of hygiene in limiting environmental contamination and transmission. Figures 6(a),(b) further demonstrate that higher values of α_s or α_r require stronger hygiene measures to reduce $\mathcal{R}_{0e}$ below one. When hygiene effectiveness is low, even moderate increases in the shedding rate can sustain disease transmission. In general, the findings emphasize the importance of reducing environmental contamination and improving hygiene practices to control the spread of infection.

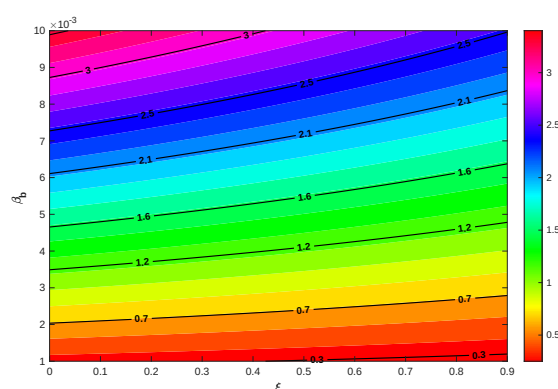


Figure 5. The effect of β_b and ξ on the basic reproduction number.

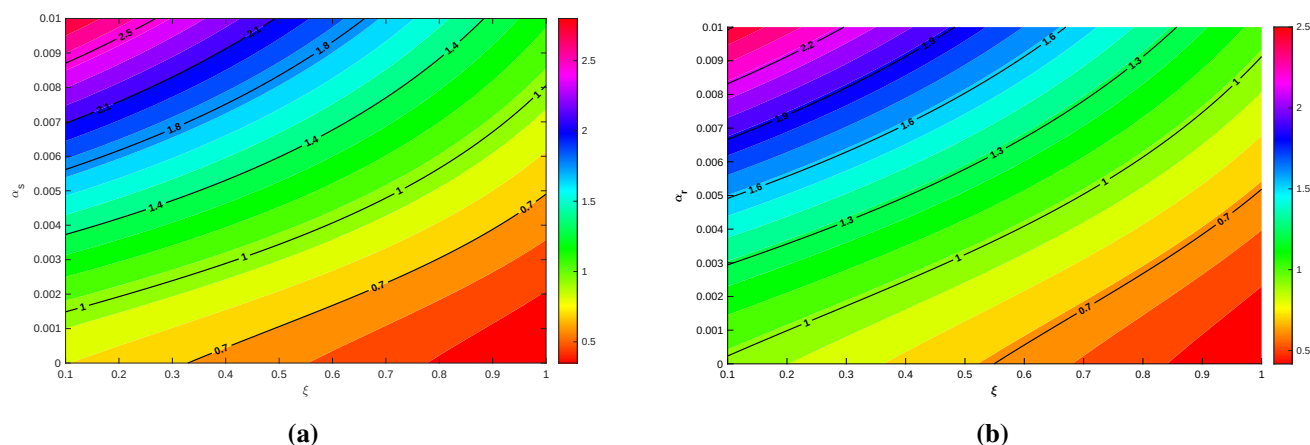


Figure 6. Comparison of the effects of ξ and α_s Figure 6(a) and ξ and α_r Figure 6(b) on the reproduction number.

Figure 7(c) shows how environmental bacteria $B_c(t)$ change over time under two hygiene scenarios, $\xi = 0$ and $\xi = 0.45$. When there is no hygiene, the bacterial concentration increases quickly because infected and treated individuals continuously shed bacteria into the environment. This leads to a steady

build-up of contamination, making the environment an important source of infection. In contrast, when hygiene is improved, the level of bacteria grows much more slowly. This is because hygiene reduces bacterial shedding by the factor $(1 - \xi H)$, which limits environmental contamination and reduces the risk of infection.

Figure 7(a),(b) show that in the absence of hygiene $\xi = 0$, both the sensitive and resistant infections increase rapidly, reaching a higher peaks, and persist for a longer period. When hygiene is introduced $\xi = 0.5$, transmission is reduced through the factor $(1 - \xi H)$, leading to lower infection peaks. Therefore, the results indicate that improved hygiene effectiveness reduces environmental contamination and the force of infection.

Figure Figure 8 shows how changes in behaviour affect the spread of infection. When there is no increase in awareness ($\psi_1 = 0$) (Figure 8(a)), people do not change their hygiene practices even as infections rise. As a result, both sensitive and resistant infections remain high. However, when awareness increases quickly ($\psi_1 = 0.5$), people improve their hygiene as infections grow. This reduces transmission and leads to a faster decline in both types of infections. This shows that responding to disease risk through better hygiene can significantly reduce the spread of infection.

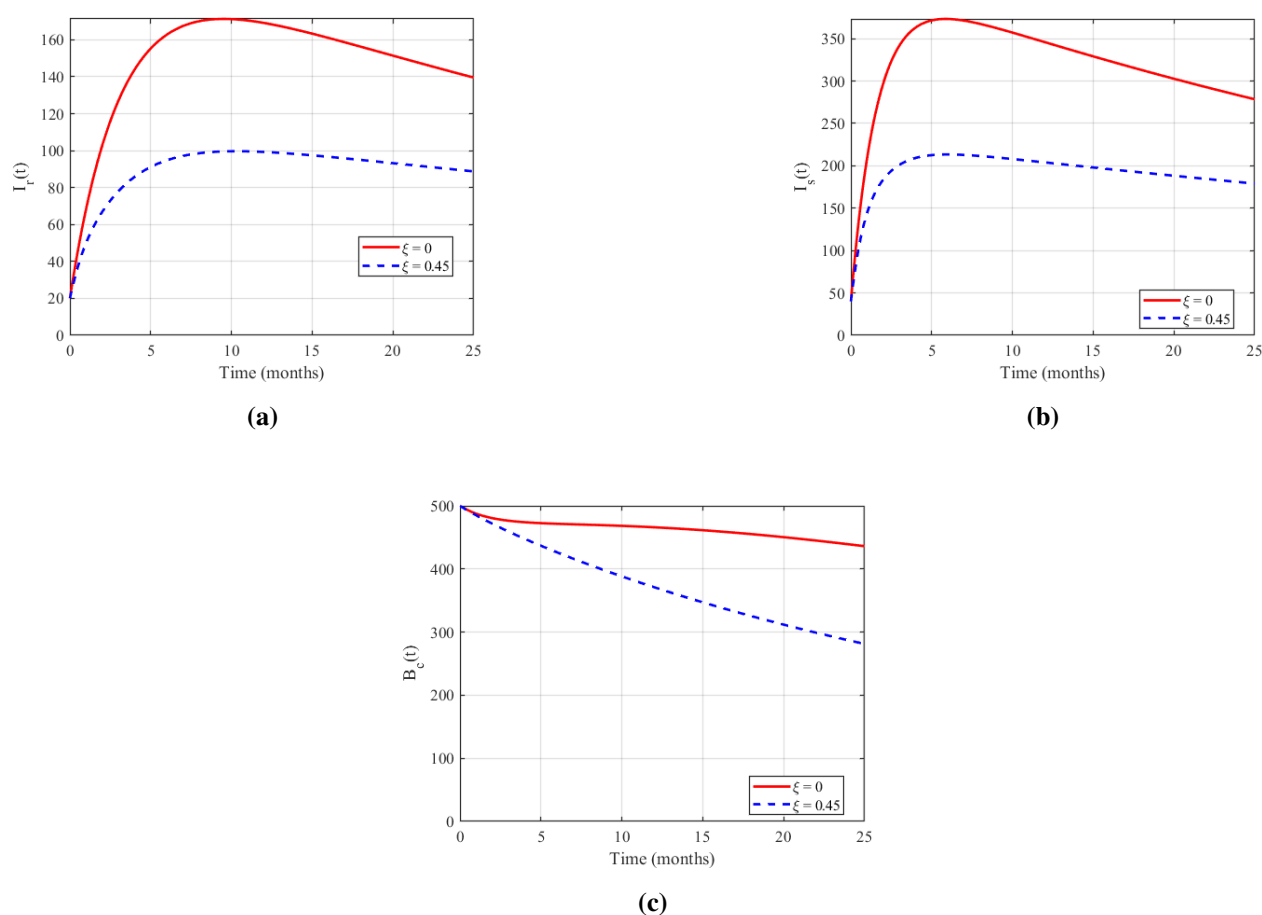


Figure 7. Time evolution of resistant infections $I_r(t)$ Figure 7(a), sensitive infections $I_s(t)$ Figure 7(b), and environmental bacterial concentration $B_c(t)$ Figure 7(c) under two hygiene scenarios, $\xi = 0$ and $\xi = 0.45$.

When awareness is maintained ($\psi_2 = 0$) (see Figure 8(b)), hygiene practices continue, and infection levels remain low. But when awareness fades quickly ($\psi_2 = 0.5$), people gradually stop following good hygiene practices, allowing infections to increase again. The shaded area in the figure shows the difference between these scenarios, showing how many infections are prevented when people maintain good hygiene. Overall, the results show that increasing awareness and maintaining it over time are both important for controlling typhoid transmission.

In Figure 9(b), when $\alpha_s = \alpha_r = 0.003$, the shedding rate of infected individuals is relatively low, meaning that infected people release only a small amount of bacteria into the environment. From the figure, the basic reproduction number is $\mathcal{R}_0 = 1.17$. This value is greater than one, which means that in the absence of hygiene measures, each infected individual produces more than one new infection on average. As a result, the disease can spread in the population and persist over time.

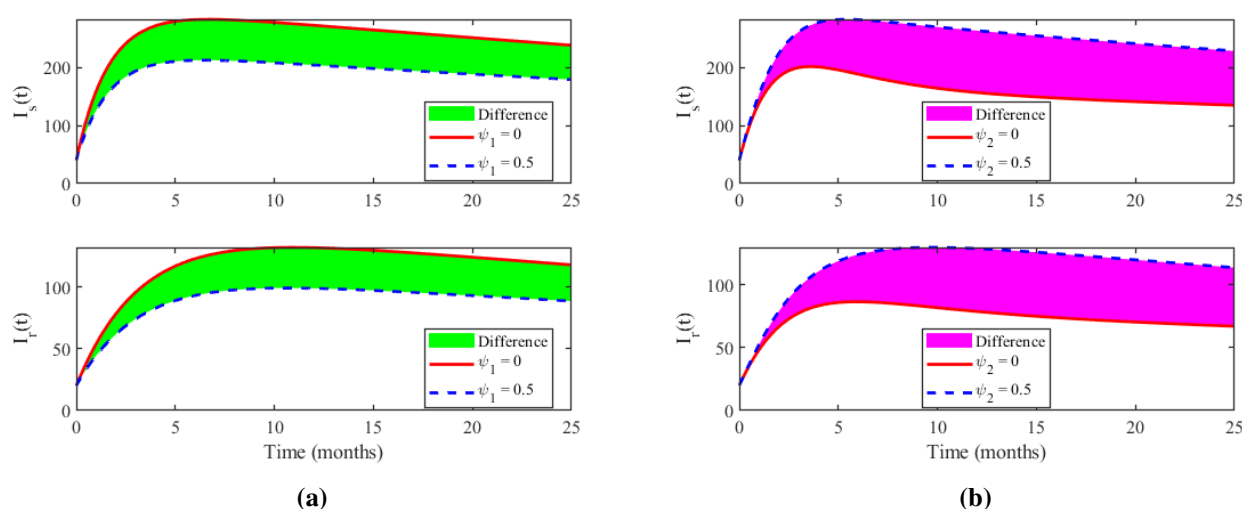


Figure 8. Comparison of the effects of $\psi_1 = 0$ and $\psi_1 = 0.5$ Figure 8(a) and $\psi_2 = 0$ and $\psi_2 = 0.5$ Figure 8(b) on the populations of infected individuals I_s and I_r , respectively.

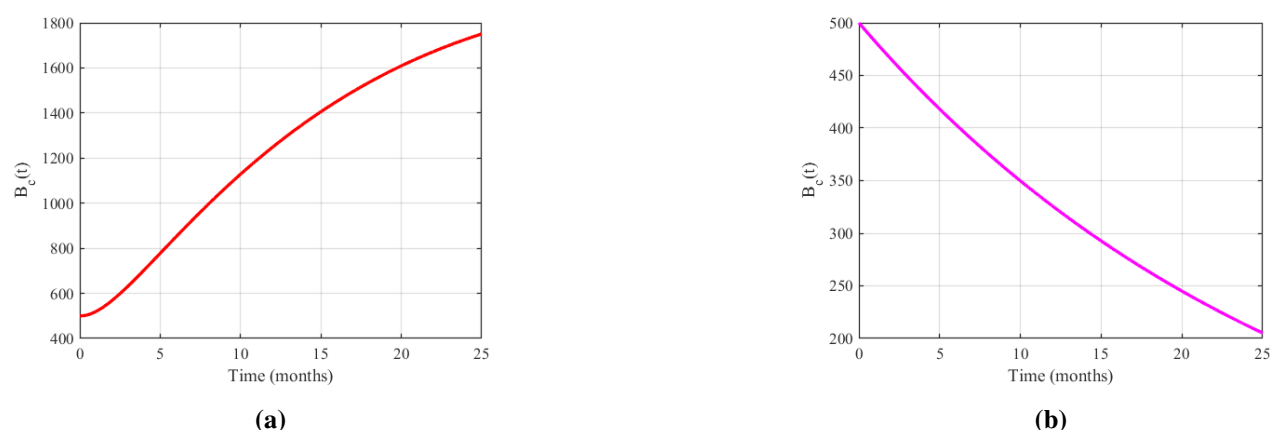


Figure 9. Comparison of the effects of $\alpha_s = \alpha_r = 0.3$ and $H = 0.0001$ Figure 9(a) and $\alpha_s = \alpha_r = 0.0003$ and $H = 0.6$ Figure 9(b) on the contaminated environment B_c , respectively.

However, when hygiene is included (see Figure 9), the effective reproduction number decreases to $\mathcal{R}_{0e} = 0.75$. Since $\mathcal{R}_{0e} < 1$, this means that each infected individual produces less than one new infection on average. In this case, the disease will gradually decline and eventually die out. The difference between $\mathcal{R}_0$ and $\mathcal{R}_{0e}$ clearly shows the impact of hygiene. While the shedding rates remain the same ($\alpha_s = \alpha_r = 0.003$), the introduction of hygiene reduces transmission sufficiently to move the system from a growing epidemic ($\mathcal{R}_0 > 1$) to a controlled situation ($\mathcal{R}_{0e} < 1$). This demonstrates that hygiene plays a crucial role in reducing the spread of infection even when bacterial shedding is present.

5.2. Effect of antibiotic switching

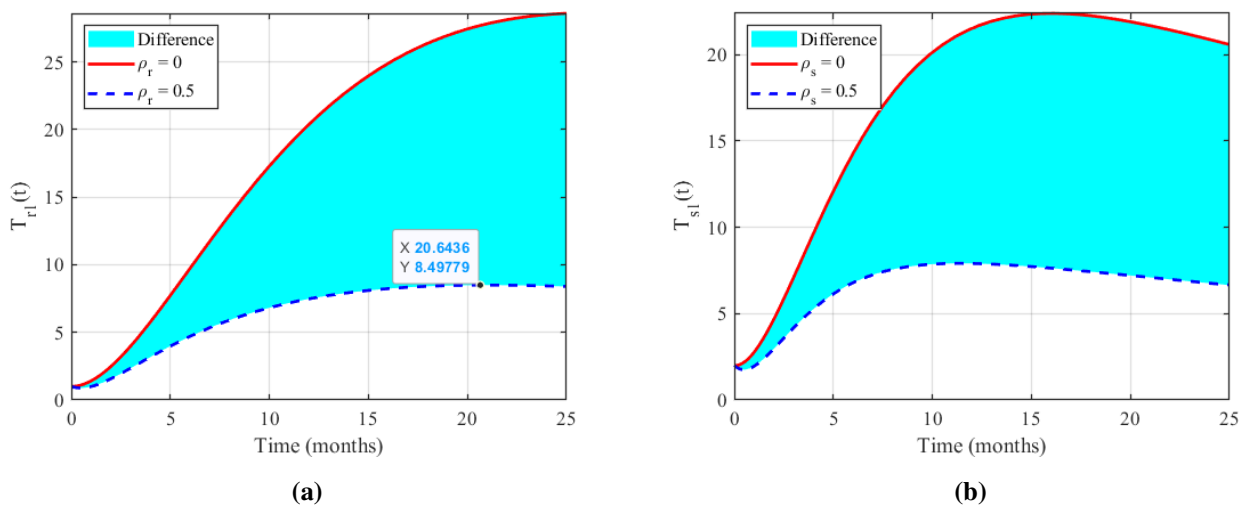


Figure 10. Comparison of the effects of second - line treatment ($\rho_s = 0$ and $\rho_s = 0.5$) and ($\rho_r = 0$ and $\rho_r = 0.5$) on sensitive and resistant strains, respectively.

Figure 10 shows the effect of second-line treatment. This is examined by comparing scenarios with and without switching to second-line treatment. When no switching is allowed ($\rho_s = 0$ and $\rho_r = 0$), the number of infected individuals remains relatively higher over time, indicating that reliance on first-line treatment alone is not sufficient to control the disease, especially in the presence of resistant strains. However, when switching is introduced at a moderate level ($\rho_s = 0.5$ and $\rho_r = 0.5$), there is a noticeable reduction in both sensitive and resistant infections, as shown by the shaded regions. The results further show that switching to second-line treatment leads to a substantial reduction in infection levels for both sensitive and resistant strains. Therefore, increasing the switching rate enhances disease control.

5.3. Effect of hygiene behaviour

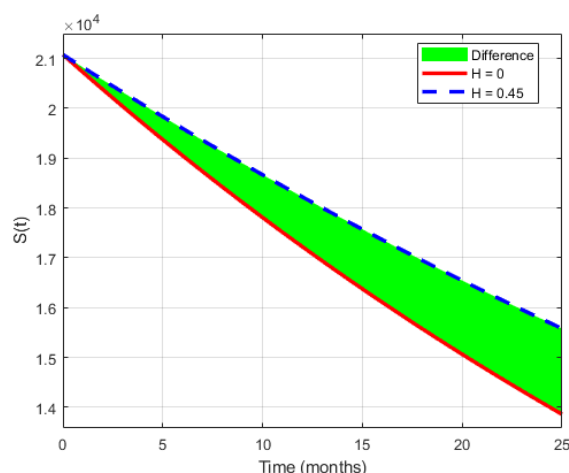


Figure 11. Trajectories of the susceptible classes are shown. The area between the two curves represents an improvement in hygiene.

In Figure 11, the trajectories of two susceptible classes are demonstrated in two hygiene scenarios, $H = 0$ and $H = 0.45$. The results show that higher hygiene levels lead to a slower decline in the susceptible population, indicating reduced transmission intensity. An increase in H reduces the force of infection, leading to fewer new infections and a higher proportion of individuals remaining in the susceptible class. The shaded area represents an improvement in hygiene. It can be seen that as time goes on, the difference increases, showing that hygiene plays an important role in reducing infection and protecting the population.

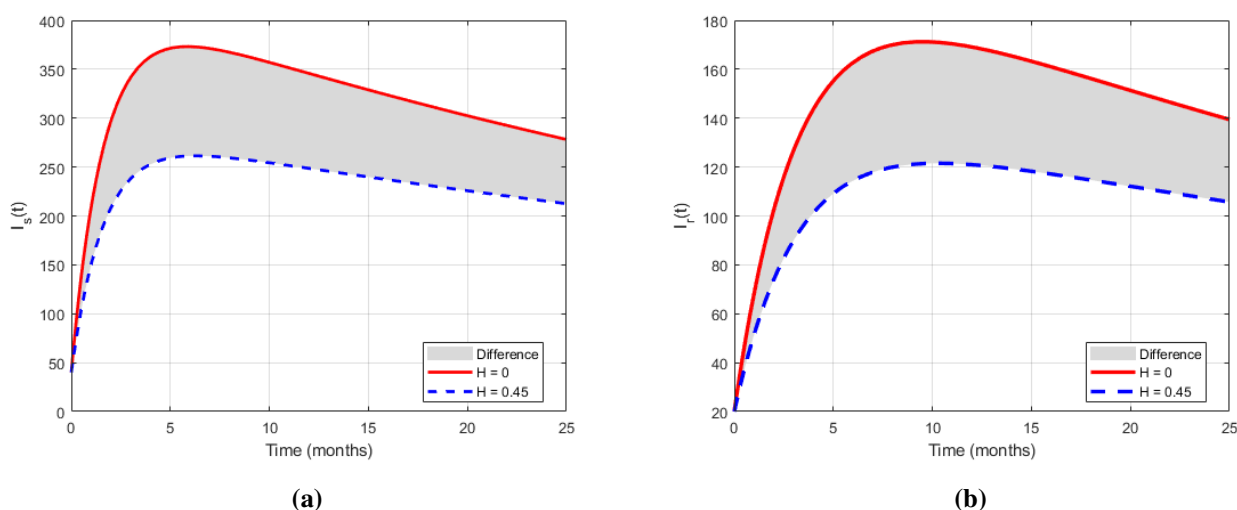


Figure 12. The behaviour of the infected population at different values of H , when $H = 0$ Figure 12(a) and $H = 0.45$ Figure 12(b).

Figure 12 shows that when there is no hygiene ($H = 0$), both the sensitive and resistant infected populations reach higher peak levels. However, when hygiene is improved to $H = 0.45$, the peak $I_s(t)$ decreases by about 45%, while the resistant infected population $I_r(t)$ is reduced by approximately 45%. This means that a substantial proportion of infections, including those that are harder to treat, can be prevented through improved hygiene practices.

6. Conclusions

Typhoid fever remains a major public health concern in settings with limited access to clean water, sanitation, and hygiene, and is further complicated by the emergence of antibiotic resistance. Understanding how behavioural and treatment-related factors interact is therefore essential for effective control.

In this paper, a mathematical model was developed to examine the combined effects of hygiene behaviour, antibiotic switching, and environmental transmission. The results show that these mechanisms interact in ways that can sustain infection. In particular, the presence of backward bifurcation indicates that reducing the reproduction number below one is not sufficient for elimination, as the disease may persist if infection levels remain above a critical threshold.

The model was calibrated using monthly reported cases from Mbandjock District Hospital in Cameroon, yielding a good fit to the data. Simulation results indicate that improved hygiene reduces both infection levels and environmental contamination, but its effectiveness depends on sustained behavioural adherence. A high level of awareness leads to a significant decline in cases, whereas a rapid loss of awareness weakens control efforts and allows transmission to continue.

The simulations suggest that improved hygiene reduces both infection levels and environmental contamination, but the effect depends on how consistently these practices are maintained. Strong and sustained awareness leads to a decrease in cases, while a rapid loss of awareness weakens the overall impact and allows transmission to continue. This highlights the role of behaviour not just as a protective factor, but as a dynamic process that can either support or undermine control efforts.

The results suggest that improving hygiene behaviour can significantly reduce both sensitive and resistant infections, as well as the level of environmental bacterial contamination. The study also shows that behavioural parameters play an important role; a higher rate of awareness leads to a stronger reduction in infections, whereas a faster loss of awareness weakens the impact of hygiene interventions and allows the disease to persist. These findings confirm that sustained behavioural change is essential for effective disease control.

A key contribution of this study lies in demonstrating that the interplay between hygiene behaviour, antibiotic switching, and resistance dynamics can fundamentally change typhoid transmission patterns. While existing studies have established that improved hygiene reduces disease spread, the present model shows that this effect is not sufficient in isolation when treatment-related factors are considered. In particular, the results indicate that inappropriate or delayed switching between treatment lines, combined with declining behavioural adherence, can sustain transmission even under improved hygiene conditions. Moreover, the coexistence of sensitive and resistant strains introduces additional complexity, such that control efforts targeting only environmental or behavioural factors may fail to eliminate the disease.

These findings provide new insight into how coupled behavioural and clinical processes drive

persistent transmission dynamics, and show that control success depends on the interaction between behaviour, treatment strategies, and resistance evolution. This emphasizes the need for integrated intervention strategies that simultaneously promote sustained hygiene practices and ensure appropriate and timely antibiotic use.

A limitation of this study is the use of a relatively small dataset from a single location, without strain-specific information. As a result, some model parameters were obtained from the literature or assumed. In addition, although incidence data were available, the model was fitted to cumulative reported cases to reduce the effect of fluctuations in the data and obtain a more stable fit. However, this approach introduces dependence between observations and may underestimate uncertainty. Access to more detailed epidemiological data, particularly on resistant infections, would improve the accuracy of the model and enhance its predictive capability.

Overall, the findings suggest that sustained hygiene behaviour, together with effective treatment strategies, plays a critical role in reducing transmission. Their interaction is important, as it can either support or undermine disease control. The model could be extended to include vaccination strategies, which remain an important component of typhoid control in endemic regions. In addition, allowing hygiene behaviour to vary over time or across subpopulations may provide a more realistic representation of behavioural responses. Finally, integrating optimal control techniques could help identify cost-effective intervention strategies that combine treatment, sanitation, and behavioural measures.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare that there is no conflict of interest concerning the publication of this article.

References

1. World Health Organization, Typhoid, 2023. Available from: <https://www.who.int/news-room/fact-sheets/detail/typhoid>.
2. D. Piovani, G. Figlioli, G. K. Nikolopoulos, S. Bonovas, The global burden of enteric fever, 2017–2021: A systematic analysis from the global burden of disease study 2021, *EClinicalMedicine*, **77** (2024), 102883. <https://doi.org/10.1016/j.eclinm.2024.102883>
3. G. Liu, X. Zhang, Q. Cao, T. Chen, B. Hu, S. Shi, The global burden of typhoid and paratyphoid fever from 1990 to 2021 and the impact on prevention and control, *BMC Infect. Dis.*, **25** (2025), 919. <https://doi.org/10.1186/s12879-025-11223-8>

4. S. Murthy, N. N. Hagedoorn, S. Faigan, M. D. Rathan, K. J. Sharples, C. S. Marchello, et al., Global typhoid fever incidence: An updated systematic review with meta-analysis [version 2], preprint. <https://doi.org/10.12688/verixiv.403.2>
5. S. D. Bennett, S. A. Lowther, F. Chingoli, B. Chilima, S. Kabuluzi, T. L. Ayers, et al., Assessment of water, sanitation and hygiene interventions in response to an outbreak of typhoid fever in Neno District, Malawi, *PLoS One*, **13** (2018), e0193348. <https://doi.org/10.1371/journal.pone.0193348>
6. S. Brockett, M. K. Wolfe, A. Hamot, G. D. Appiah, E. D. Mintz, D. Lantagne, Associations among water, sanitation, and hygiene, and food exposures and typhoid fever in case–control studies: A systematic review and meta-analysis, *Am. J. Trop. Med. Hyg.*, **103** (2020), 1020. <https://doi.org/10.4269/ajtmh.19-0479>
7. C. Kim, G. R. Goucher, B. T. Tadesse, W. Lee, K. Abbas, J. H. Kim, Associations of water, sanitation, and hygiene with typhoid fever in case–control studies: A systematic review and meta-analysis, *BMC Infect. Dis.*, **23** (2023), 562. <https://doi.org/10.1186/s12879-023-08452-0>
8. O. J. Okesanya, G. Eshun, B. M. Ukoaka, E. Manirambona, O. N. Olabode, R. O. Adesola, et al., Water, sanitation, and hygiene (WASH) practices in Africa: Exploring the effects on public health and sustainable development plans, *Trop. Med. Health*, **52** (2024), 68. <https://doi.org/10.1186/s41182-024-00614-3>
9. E. J. Klemm, S. Shakoor, A. J. Page, F. N. Qamar, K. Judge, D. K. Saeed, et al., Emergence of an extensively drug-resistant *Salmonella enterica* serovar typhi clone harboring a promiscuous plasmid encoding resistance to fluoroquinolones and third-generation cephalosporins, *mBio*, **9** (2018). <https://doi.org/10.1128/mbio.00105-18>
10. S. Wu, J. P. Hulme, Recent advances in the detection of antibiotic and multi-drug resistant *Salmonella*: An update, *Int. J. Mol. Sci.*, **22** (2021), 3499. <https://doi.org/10.3390/ijms22073499>
11. V. E. Pitzer, N. A. Feasey, C. Msefula, J. Mallewa, N. Kennedy, Q. Dube, et al., Mathematical modeling to assess the drivers of the recent emergence of typhoid fever in Blantyre, Malawi, *Clin. Infect. Dis.*, **61** (2015), S251–S258. <https://doi.org/10.1093/cid/civ710>
12. S. T. Tresna, Subiyanto, S. Supian, Mathematical models for typhoid disease transmission: A systematic literature review, *Mathematics*, **10** (2022), 2506. <https://doi.org/10.3390/math10142506>
13. T. J. Tsafack, C. Kwa Kum, A. J. O. Tassé, B. Tsanou, Mathematical modelling of the dynamics of typhoid fever and two modes of treatment in a health district in Cameroon, *Math. Biosci. Eng.*, **22** (2025), 477–510. <https://doi.org/10.3934/mbe.2025018>
14. J. A. Crump, Progress in typhoid fever epidemiology, *Clin. Infect. Dis.*, **68** (2019), S4–S9. <https://doi.org/10.1093/cid/ciy846>
15. E. P. Fenichel, C. Castillo-Chavez, M. G. Ceddia, G. Chowell, P. A. G. Parra, G. J. Hickling, et al., Adaptive human behavior in epidemiological models, *Proc. Natl. Acad. Sci.*, **108** (2011), 6306–6311. <https://doi.org/10.1073/pnas.1011250108>
16. N. Perra, D. Balcan, B. Gonçalves, A. Vespignani, Towards a characterization of behavior-disease models, *PLoS One*, **6** (2011), e23084. <https://doi.org/10.1371/journal.pone.0023084>

17. L. Matsebula, F. Nyabadza, A mathematical model of cholera–typhoid coinfection dynamics with a hygiene-driven contact rate, *Sci. Afr.*, **26** (2024), e02348. <https://doi.org/10.1016/j.sciaf.2024.e02348>
18. L. Matsebula, F. Nyabadza, J. Mushanyu, Mathematical analysis of typhoid fever transmission dynamics with seasonality and fear, *Commun. Math. Biol. Neurosci.*, **2021** (2021). <https://doi.org/10.28919/cmbn/5590>
19. J. Wain, C. Kidgell, The emergence of multidrug resistance to antimicrobial agents for the treatment of typhoid fever, *Trans. R. Soc. Trop. Med. Hyg.*, **98** (2004), 423–430. <https://doi.org/10.1016/j.trstmh.2003.10.015>
20. M. M. Shaale, J. Mushanyu, F. Nyabadza, S. M. Nuugulu, Fighting typhoid fever: Modeling antibiotic resistance and antibiotic switching, *Franklin Open*, **11** (2025), 100274. <https://doi.org/10.1016/j.fraope.2025.100274>
21. M. Haber, B. R. Levin, P. Kramarz, Antibiotic control of antibiotic resistance in hospitals: A simulation study, *BMC Infect. Dis.*, **10** (2010), 1–10.
22. M. Lipsitch, C. T. Bergstrom, B. R. Levin, The epidemiology of antibiotic resistance in hospitals: paradoxes and prescriptions, *Proc. Natl. Acad. Sci.*, **97** (2000), 1938–1943. <https://doi.org/10.1073/pnas.97.4.1938>
23. P. Van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002), 29–48. [https://doi.org/10.1016/S0025-5564\(02\)00108-6](https://doi.org/10.1016/S0025-5564(02)00108-6)
24. P. Van den Driessche, J. Watmough, Further notes on the basic reproduction number, in *Mathematical Epidemiology*, Springer, (2008), 159–178. https://doi.org/10.1007/978-3-540-78911-6_6
25. J. Mushanyu, F. Nyabadza, G. Muchatibaya, P. Mafuta, G. Nhawu, Assessing the potential impact of limited public health resources on the spread and control of typhoid, *J. Math. Biol.*, **77** (2018), 647–670. <https://doi.org/10.1007/s00285-018-1219-9>
26. C. Castillo-Chavez, Z. Feng, W. Huang, On the computation of $\mathcal{R}_0$ and its role on global stability, in *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction*, Springer, **1** (2002), 229–250.
27. C. P. Bhunu, W. Garira, Z. Mukandavire, Modeling HIV/AIDS and tuberculosis coinfection, *Bull. Math. Biol.*, **71** (2009), 1745–1780. <https://doi.org/10.1007/s11538-009-9423-9>
28. C. Castillo-Chavez, B. Song, Dynamical models of tuberculosis and their applications, *Math. Biosci. Eng.*, **1** (2004), 361–404.
29. F. Nyabadza, S. D. Hove-Musekwa, From heroin epidemics to methamphetamine epidemics: Modelling substance abuse in a South African province, *Math. Biosci.*, **225** (2010), 132–140. <https://doi.org/10.1016/j.mbs.2010.03.002>
30. S. Mushayabasa, Modeling the impact of optimal screening on typhoid dynamics, *Int. J. Dyn. Control*, **4** (2016), 330–338. <https://doi.org/10.1007/s40435-014-0123-4>

31. S. Mushayabasa, C. P. Bhunu, E. T. Ngarakana-Gwasira, Assessing the impact of drug resistance on the transmission dynamics of typhoid fever, *Comput. Biol. J.*, **2013** (2013), 303645. <https://doi.org/10.1155/2013/303645>
32. H. Abboubakar, R. Racke, Mathematical modeling, forecasting, and optimal control of typhoid fever transmission dynamics, *Chaos Solitons Fractals*, **149** (2021), 111074. <https://doi.org/10.1016/j.chaos.2021.111074>
33. I. H. Spicknall, B. Foxman, C. F. Marrs, J. N. S. Eisenberg, A modeling framework for the evolution and spread of antibiotic resistance: Literature review and model categorization, *Am. J. Epidemiol.*, **178** (2013), 508–520. <https://doi.org/10.1093/aje/kwt017>
34. A. Raue, C. Kreutz, T. Maiwald, J. Bachmann, M. Schilling, U. Klingmüller, et al., Structural and practical identifiability analysis of partially observed dynamical models by exploiting the profile likelihood, *Bioinformatics*, **25** (2009), 1923–1929. <https://doi.org/10.1093/bioinformatics/btp358>
35. H. Miao, X. Xia, A. S. Perelson, H. Wu, On identifiability of nonlinear ODE models and applications in viral dynamics, *SIAM Rev.*, **53** (2011), 3–39. <https://doi.org/10.1137/090757009>
36. M. D. McKay, R. J. Beckman, W. J. Conover, A comparison of three methods for selecting values of input variables in the analysis of output from a computer code, *Technometrics*, **42** (2000), 55–61.
37. S. Marino, I. B. Hogue, C. J. Ray, D. E. Kirschner, A methodology for performing global uncertainty and sensitivity analysis in systems biology, *J. Theor. Biol.*, **254** (2008), 178–196. <https://doi.org/10.1016/j.jtbi.2008.04.011>

Appendix

Appendix A. Endemic equilibrium point coefficients for Section 3.6.3

where,

$$\Phi_1 = \frac{\sigma_s}{\mathcal{Z}_3}, \quad \Phi_2 = \frac{\rho_s}{\mathcal{Z}_5} \Phi_1, \quad \Phi_3 = \frac{\sigma_r}{\mathcal{Z}_4}, \quad \Phi_4 = \frac{\rho_r}{\mathcal{Z}_6} \Phi_3; \quad \Phi_5 = \frac{\theta_s + \nu_s \Phi_1 + \gamma_s \Phi_2}{\mathcal{Z}_7};$$

$$\Phi_6 = \frac{\theta_r + \nu_r \Phi_3 + \gamma_r \Phi_4}{\mathcal{Z}_7}, \quad \Phi_7 = \frac{\alpha_s + \alpha_{s1} \Phi_1}{\mathcal{Z}_8}, \quad \Phi_8 = \frac{\alpha_r + \alpha_{r1} \Phi_3}{\mathcal{Z}_8}$$

$$\lambda_s^* = \omega \beta_b A(I_s^*, I_r^*) \left(\frac{\psi_2 + (1 - \xi) \psi_1 (I_s^* + I_r^*)}{\psi_1 (I_s^* + I_r^*) + \psi_2} \right), \text{ and}$$

$$\lambda_r^* = (1 - \omega) \beta_b A(I_s^*, I_r^*) \left(\frac{\psi_2 + (1 - \xi) \psi_1 (I_s^* + I_r^*)}{\psi_1 (I_s^* + I_r^*) + \psi_2} \right),$$

$$A(I_s^*, I_r^*) = \frac{(\psi_2 + (1 - \xi) \psi_1 (I_s^* + I_r^*)) (\Phi_7 I_s^* + \Phi_8 I_r^*)}{(\psi_2 + (1 - \xi) \psi_1 (I_s^* + I_r^*)) (\Phi_7 I_s^* + \Phi_8 I_r^*) + \kappa (\psi_1 (I_s^* + I_r^*) + \psi_2)}.$$

Appendix B. Endemic equilibrium point for Section 3.6.2

$$\phi_3 = \frac{\sigma_r}{\mathcal{Z}_4}, \quad \phi_4 = \frac{\rho_r}{\mathcal{Z}_6} \phi_3, \quad \phi_6 = \frac{\theta_r + \nu_r \phi_3 + \gamma_r \phi_4}{\mathcal{Z}_7}, \quad \phi_8 = \frac{\alpha_r + \alpha_{r1} \phi_3}{\mathcal{Z}_8}.$$



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