World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

A mathematical model for typhoid-helminth co-infection with typhoid-only treatment and persistent symptoms

Mutah Wadai [1,*], Ibekwe Jacob John [1], Agba Stephen Terungwa [1] and Adem Kilicman [2]

[1] *Department of Mathematics, Faculty of Physical Sciences, Federal University of Health Sciences, Otukpo, Benue State, Nigeria.*
[2] *Department of Computer and Mathematical Sciences, Faculty of Sciences, Universiti of Teknologi MARA, 40450 Shah Alam, Selangor, Malaysia.*

World Journal of Advanced Research and Reviews, 2026, 29(02), 1132-1153

Publication history: Received on 10 January 2026; revised on 18 February 2026; accepted on 20 February 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.29.2.0426

Abstract

Typhoid fever and helminthiasis are co-endemic in many low-resource settings and frequently present with overlapping clinical symptoms, complicating diagnosis and treatment. In practice, patients are often treated for typhoid fever alone, which may lead to temporary symptom relief while underlying helminth infection remains unrecognized. To investigate the epidemiological consequences of this scenario, we developed and analyzed a deterministic compartmental model that incorporates typhoid-helminth co-infection, a shared environmental transmission pathway, and a typhoid-only treatment strategy.

The model possesses a disease-free steady state and a threshold reproduction metric that determines whether infection dies out or persists. Analytical results establish conditions for stability of both disease-free and endemic equilibria, while sensitivity analysis identifies the most influential parameters governing transmission. Simulation results indicate that enhanced typhoid treatment lowers typhoid prevalence but does not eliminate the total symptomatic burden when helminth infection is untreated. In contrast, improvements in environmental sanitation substantially reduce both infections and co-infection by limiting shared transmission pathways.

These findings provide a mathematical explanation for persistent symptoms following typhoid treatment in co-endemic regions and highlight the importance of integrated diagnostic, therapeutic, and environmental interventions. The study underscores that sustainable reduction of disease burden requires coordinated strategies that address co-infection and environmental transmission rather than single-disease control alone.

Keywords: Co-infection; Environmental transmission; Helminthiasis; Mathematical modeling; Typhoid fever; Treatment strategies

1 Introduction

Typhoid fever and helminth infections continue to persist across many low- and middle-income regions, where limited diagnostic capacity and overlapping clinical manifestations complicate effective disease management. These infections frequently manifest with general symptoms including fever, abdominal discomfort, weakness, and gastrointestinal disturbances, which often lead to syndromic diagnosis and empirical treatment [1]. In such settings, patients are frequently treated for typhoid alone, while concurrent helminth infection may remain undetected and untreated, resulting in persistent or recurrent symptoms.

*Corresponding author: Mutah Wadai

Typhoid fever affects an estimated 9-14 million people annually worldwide, with the highest burden in South Asia and sub-Saharan Africa [8]. Mathematical modeling has become an essential tool for investigating transmission dynamics of typhoid fever and evaluating intervention strategies, including treatment, vaccination, and environmental control [2]. In addition, a rich body of literature exists on co-infection modeling involving typhoid and other diseases, particularly malaria, motivated by symptom overlap and diagnostic uncertainty [4]. Helminth infections have also been extensively modeled, both independently and in co-infection settings with other parasitic diseases. Soil-transmitted helminths infect more than 1.5 billion people globally and are strongly associated with poor sanitation and hygiene conditions [7].Despite clinical reports of concurrent infection, few mathematical studies have examined their combined transmission dynamics.

This gap is particularly important because helminthiasis may act as a hidden driver of prolonged illness when typhoid-only treatment is administered. Clinical reports indicate that co-infected patients may experience partial relief following typhoid therapy, yet continue to exhibit symptoms due to untreated helminth infection [1]. Such outcomes can be misinterpreted as treatment failure, relapse, or antimicrobial resistance, thereby increasing disease burden and healthcare costs.

Motivated by this clinical reality, this study develops a deterministic compartmental model for typhoid-helminth co-infection that explicitly incorporates typhoid-only treatment. The model tracks susceptible, exposed, infected, and co-infected populations, includes an environmental pathogen component, and allows co-infected individuals receiving typhoid therapy to transition to a helminth-only infection class. This structure captures the persistence of symptoms arising from untreated helminthiasis.

The objectives of this work are to establish the model's mathematical well-posedness through positivity and invariant region analysis, and to provide a theoretical explanation for persistent illness following typhoid treatment in co-infected populations. By focusing on the underexplored interaction between typhoid and helminthiasis under partial treatment, this study contributes to the co-infection modeling literature and highlights the epidemiological importance of integrated diagnosis and treatment strategies in endemic regions [2, 4].

2 Model Formulation and Assumptions

2.1 Model Formulation Description

Individuals enter the susceptible class S(t) at constant rate Λ and leave through infection or natural death at rate μ. Susceptible individuals acquire infection through environmental exposure P(t), moving to the exposed typhoid and helminth classes at rates $\beta_t PS$ and $\beta_h PS$, respectively. Exposed individuals (E_t and E_h) progress to the infectious classes(I_t and I_h)at constant rates ξ_t and ξ_h respectively.Typhoid-infected individuals receive treatment at rate γ_t or acquire helminth co-infection at rate $\beta_{th}P$, while helminth-infected individuals may acquire typhoid co-infection at rate $\beta_{ht}P$. Co-infected individuals receive treatment at rate γ_{th}. Disease-induced deaths occur at rates δ_t and δ_{th}, and all compartments experience natural mortality at rate μ.Treated individuals recover at rate ϕ, entering the recovered class R(t), and lose immunity at rate k, returning to S(t), thereby completing the transmission cycle.Environmental pathogens are generated by infectious individuals at rates α_t , α_h, and α_{th}, and decay at rate ω, linking both infections through a shared environmental pathway.

2.2 Model Assumptions

- The human population is homogeneously mixed and is replenished through recruitment at a constant rate. All individuals enter the population as susceptible, and natural death occurs in every compartment at a constant rate.
- Transmission of both typhoid and helminth infections occurs through a shared environmental pathogen reservoir representing environmental contamination pathways such as unsafe water, food, or soil exposure. Exposed individuals progress through latent stages before becoming infectious.
- Individuals may acquire typhoid infection alone, helminth infection alone, or both infections simultaneously. Co-infection arises when a singly infected individual acquires the second infection from the environment.
- Owing to overlapping clinical symptoms and limited diagnostic capacity, treatment is assumed to target typhoid infection only. Helminth-only infected individuals do not receive treatment within the model framework.

- Co-infected individuals receiving typhoid treatment clear the typhoid infection but remain infected with helminths, leading to persistent symptoms. Recovered individuals acquire temporary immunity and may return to the susceptible class.

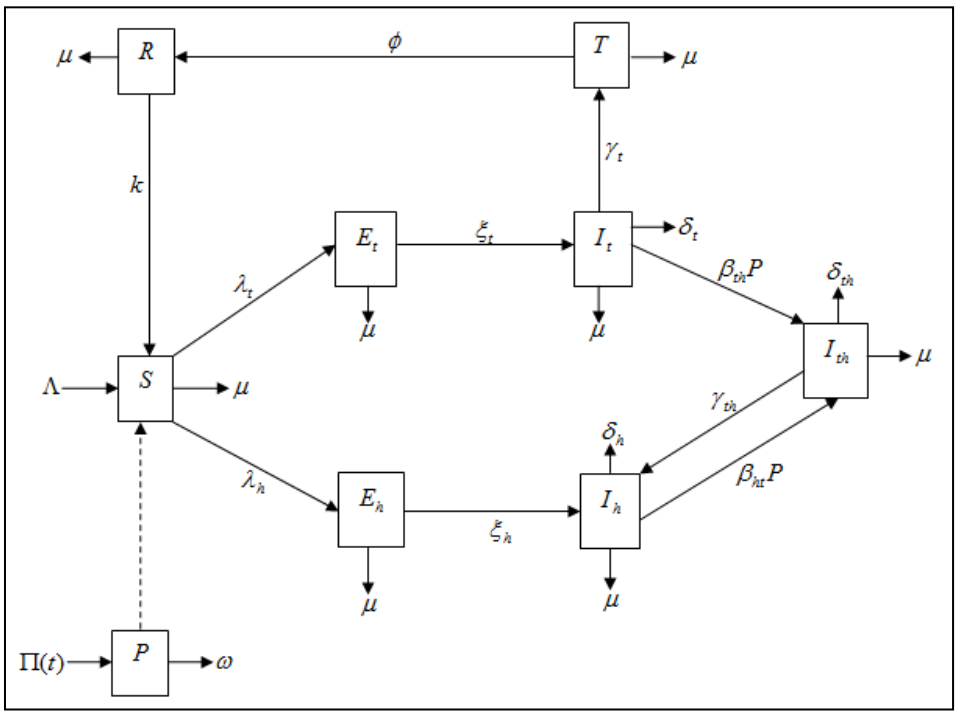

Figure 1 Schematic Diagram of Typhoid-Helminth Co-infection

3 Mathematical Analysis of the Model

Analytical techniques are used to examine the qualitative behaviour of the system, including solution feasibility, stability properties, and threshold conditions for disease persistence.

3.1 Model Equations of Typhoid-Helminth Co-infection

$$\frac{dS}{dt} = \Lambda + kR - (\lambda_t + \lambda_h + \mu)S, \tag{1}$$

$$\frac{dE_t}{dt} = \lambda_t S - (\xi_t + \mu)E_t, \tag{2}$$

$$\frac{dE_h}{dt} = \lambda_h S - (\xi_h + \mu)E_h, \tag{3}$$

$$\frac{dI_t}{dt} = \xi_t E_t - (\beta_{th}P + \gamma_t + \delta_t + \mu)I_t, \tag{4}$$

$$\frac{dI_h}{dt} = \xi_h E_h + \gamma_{th} I_{th} - (\beta_{ht}P + \delta_h + \mu)I_h, \tag{5}$$

$$\frac{dI_{th}}{dt} = \beta_{th}PI_t + \beta_{ht}PI_h - (\gamma_{th} + \delta_{th} + \mu)I_{th}, \tag{6}$$

$$\frac{dT}{dt} = \gamma_t I_t - (\phi + \mu)T, \tag{7}$$

$$\frac{dR}{dt} = \phi T - (k + \mu)R, \tag{8}$$

$$\frac{dP}{dt} = \Pi(t) - \omega P \tag{9}$$

With $\Pi(t) = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th}$, $\lambda_t = \beta_t P$, $\lambda_h = \beta_h P$, $\mu > 0, \omega > 0,$ and

$$N = S + E_t + E_h + I_t + I_h + I_{th} + T + R.$$

Table 1 Variable for Typhoid-Helminth Co-infection

Variable	Description
$S(t)$	Number of susceptible individuals at time t.
$E_t(t)$	Number of individuals exposed to typhoid fever but not yet infectious.
$E_t(t)$	Number of individuals exposed to helminth infection but not yet infectious.
$I_t(t)$	Number of individuals infectious with typhoid fever only.
$I_t(t)$	Number of individuals infectious with helminthiasis only.
$I_{th}(t)$	Number of individuals co-infected with typhoid fever and helminthiasis.
$T(t)$	Number of individuals under treatment for typhoid fever.
$R(t)$	Number of individuals who have recovered from typhoid infection.
$P(t)$	Concentration of pathogens in the environment (water/food contamination).
$\Pi(t)$	Total pathogen input from infected shedding.

Table 2 Parameter Description for Typhoid-Helminth Co-infection

Parameter	Description
Λ	Recruitment rate of individuals into the susceptible population.
μ	Natural death rate of individuals.
β_t	Transmission coefficient for typhoid infection from the environment to humans.
β_h	Transmission coefficient for helminth infection from the environment to humans.
β_{th}	Rate at which typhoid-infected individuals acquire helminth infection.
β_{ht}	Rate at which helminth-infected individuals acquire typhoid infection.
ξ_t	Progression rate from exposed to infectious stage for typhoid fever.

ξ_h	Progression rate from exposed to infectious stage for helminth infection.
γ_t	Treatment rate for individuals infected with typhoid fever.
γ_{th}	Typhoid treatment rate for co-infected individuals.
ϕ	Rate at which treated individuals complete treatment and move to recovery.
k	Rate of loss of immunity, returning recovered individuals to the susceptible class.
δ_t	Disease-induced death rate due to typhoid fever.
δ_h	Natural clearance/removal rate of helminth infection.
δ_{th}	Disease-induced death rate for co-infected individuals.
α_t	Rate at which typhoid-infected individuals shed pathogens into the environment.
α_h	Rate at which helminth-infected individuals shed infectious stages into the environment.
α_{th}	Rate at which co-infected individuals contaminate the environment.
ω	Environmental pathogen decay/removal rate representing sanitation and hygiene.

3.2 Positivity (non-negativity of solutions) of Typhoid-Helminth Co-infection

Let the nonnegative initial conditions be $S(0), E_t(0), E_h(0), I_t(0), I_h(0), I_{th}(0), T(0), R(0), P(0) \geq 0$

From equation (1),

At $S = 0$: $$\frac{dS}{dt} = \Lambda + kR \geq 0.$$

From equation (2),

At $E_t = 0$: $$\frac{dE_t}{dt} = \lambda_t S = \beta_t PS \geq 0.$$

From equation (3),

At $E_h = 0$: $$\frac{dE_h}{dt} = \lambda_h S = \beta_h PS \geq 0.$$

From equation (4),

At $I_t = 0$: $$\frac{dI_t}{dt} = \xi E_t \geq 0.$$

From equation (5),

At $I_h = 0$: $$\frac{dI_h}{dt} = \xi_h E_h + \gamma_{th} I_{th} \geq 0.$$

From equation (6),

At $I_{th} = 0$: $$\frac{dI_{th}}{dt} = \beta_{th} P I_t + \beta_{ht} P I_h \geq 0.$$

From equation (7),

At $T = 0$: $$\frac{dT}{dt} = \gamma_t I_t \geq 0.$$

From equation (8),

At $R = 0$: $$\frac{dR}{dt} = \phi T \geq 0.$$

From equation (9),

At $T = 0$: $$\frac{dP}{dt} = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th} \geq 0.$$

Hence, the nonnegative orthant is positively invariant, and all state variables remain greater than or equal to zero for t ≥ 0.

3.3 Invariant region of Typhoid-Helminth Co-infection

Let N be the total human population with respect to t. That is,

$$N = S + E_t + E_h + I_t + I_h + I_{th} + T + R. \tag{10}$$

Taking the derivative of (10), we have,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE_t}{dt} + \frac{dE_h}{dt} + \frac{dI_t}{dt} + \frac{dI_h}{dt} + \frac{dI_{th}}{dt} + \frac{dT}{dt} + \frac{dR}{dt}. \tag{11}$$

Substituting equation (1) – (8) into (11) and simplifying further gives,

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_t I_t - \delta_h I_h - \delta_{th} I_{th}. \tag{12}$$

In the absence of disease-induced death rate ($\delta_t - \delta_h - \delta_{th} = 0$). Therefore, equation (12) reduces to:

$$\frac{dN}{dt} = \Lambda - \mu N \tag{12a}$$

Applying comparison method on equation (12a) yields,

$$N(t) \leq \max\left\{ N(0), \frac{\Lambda}{\mu} \right\} \text{ for all } t \geq 0.$$

Therefore, the solutions eventually enter and remain in $N \leq \frac{\Lambda}{\mu}$.

From equation (9),

$$\frac{dP}{dt} = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th} - \omega P \text{ with } I_t + I_h + I_{th} \le N .$$

Let $\overline{\alpha} = \max\{\alpha_t, \alpha_h, \alpha_{th}\}$. Then, $\alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th} \le \overline{\alpha}(I_t + I_h + I_{th}) \le \overline{\alpha} N$. Thus,

$\frac{dP}{dt} \le \overline{\alpha} N - \omega P$. Since inside the set $N \le \frac{\Lambda}{\mu}$, we have, $\frac{dP}{dt} \le \overline{\alpha}\frac{\Lambda}{\mu} - \omega P$.

By comparison method, P(t) is bounded and eventually satisfies,

$$P(t) \le \max\left\{P(0), \frac{\overline{\alpha}}{\omega}, \frac{\Lambda}{\mu}\right\}.$$

Therefore, the positively invariant feasible region for analysis will be,

$$\Omega = \left\{(S, E_t, E_h, I_t, I_h, I_{th}, T, R) \in \Re_+^9 : N \le \frac{\Lambda}{\mu}, P \le \frac{\overline{\alpha}}{\omega}, \frac{\Lambda}{\mu}\right\}.$$

Finally, the positivity guarantees that the solutions stay in $\Re_+^9$ and the inequalities above show that the trajectories enter and remain bounded in Ω.

3.4 Disease-Free Equilibrium (DFE) of Typhoid-Helminth Co-infection

The disease-free equilibrium (DFE) is the steady state where no human infection is present (all exposed/infected/treated/recovered due to infection are zero). That is,

$$E_t = E_h = I_t = I_h = I_{th} = T = R = 0 . \quad (13)$$

Therefore, we will solve for equation (1) and (9).

From equation (1), $\frac{dS}{dt} = \Lambda + kR - (\lambda_t + \lambda_h + \mu)S$. Setting the derivative to zero and substituting the respective of (13), we have,

$$0 = \Lambda - \mu S_0 \quad \Rightarrow S_0 = \frac{\Lambda}{\mu}.$$

Therefore, disease-free equilibrium is given by $(S, E_t, E_h, I_t, I_h, I_{th}, T, R, P) = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0,0,0,0\right)$.

3.5 Basic Reproduction Number of Typhoid-Helminth Co-infection

The next-generation matrix framework is widely used to compute reproduction numbers in compartmental epidemic models [5].

From equation (1) – (9), the infected-state vector for the next-generation method is

$$x = (E_t, E_t, I_t, I_t, P)^T$$

Note: The I_{th} does notenter the linear R_0 calculation because near the disease free equilibrium, P, I_t, and I_h are small. The inflows to I_{th} are products like PI_t and PI_h, which are second order, so the linearization gives no first-ordergeneration of I_{th}. Therefore, I_{th} (and $\alpha_{th}I_{th}$) do not affect R_0 . We will split the system into F(x) and V(x) as shown below.

$$\frac{dx}{dt} = F(x) - V(x),$$

where F contains new infection termsonly.

New infection terms F(x) will be $F(x) = \begin{pmatrix} \beta_t PS \\ \beta_h PS \\ 0 \\ 0 \\ 0 \end{pmatrix}$

At the DFE $S=S_0$, the Jacobian matrix F will be $F = \begin{pmatrix} 0 & 0 & 0 & 0 & \beta_t S_0 \\ 0 & 0 & 0 & 0 & \beta_h S_0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$. (14)

Transition terms V(x) will be $V(x) = \begin{pmatrix} (\xi_t + \mu)E_t \\ (\xi_h + \mu)E_h \\ (\gamma_t + \delta_t + \mu)I_t - \xi_t E_t \\ (\delta_h + \mu)I_h - \xi_h E_h \\ (\omega P - \alpha_t I_t - \alpha_h I_h) \end{pmatrix}$

Taking the Jacobian matrix V at DFE will be $V = \begin{pmatrix} \xi_t + \mu & 0 & 0 & 0 & 0 \\ 0 & \xi_h + \mu & 0 & 0 & 0 \\ -\xi_t & 0 & \gamma_t + \delta_t + \mu & 0 & 0 \\ 0 & -\xi_h & 0 & \delta_h + \mu & 0 \\ 0 & 0 & -\alpha_t & -\alpha_h & \omega \end{pmatrix}$ (15)

Taking the inverse of (15) gives,

$$V^{-1} = \begin{pmatrix} \frac{1}{\xi_t + \mu} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\xi_h + \mu} & 0 & 0 & 0 \\ \frac{\xi_t}{(\xi_t + \mu)(\gamma_t + \delta_t + \mu)} & 0 & \frac{1}{\gamma_t + \delta_t + \mu} & 0 & 0 \\ 0 & & 0 & \frac{1}{\delta_h + \mu} & 0 \\ \frac{\alpha_t \xi_t}{(\xi_t + \mu)(\gamma_t + \delta_t + \mu)\omega} & \frac{\alpha_h \xi_h}{(\xi_h + \mu)(\delta_h + \mu)\omega} & \frac{\alpha_t}{(\gamma_t + \delta_t + \mu)\omega} & \frac{\alpha_h}{(\delta_h + \mu)\omega} & \frac{1}{\omega} \end{pmatrix}$$

Taking the next-generation matrix K = FV^{-1} and finding the spectral radius(ρ) gives $R_0 = \rho(K)$

Typhoid contribution; $R_t = \left(\frac{\alpha_t}{\omega}\right)(\beta_t S_0)\left(\frac{1}{\gamma_t + \delta_t + \mu}\right) = \frac{\beta_t S_0 \alpha_t \xi_t}{(\xi_t + \mu)(\gamma_t + \delta_t + \mu)\omega}$

Helminth contribution; $R_h = \frac{\beta_h S_0 \alpha_h \xi_h}{(\xi_h + \mu)(\delta_h + \mu)\omega}$

Because the two infection pathways contribute additively through the same environmental reservoir near the DFE, the dominant eigenvalue becomes the sum:

$$R_0 = R_t + R_h = \frac{\beta_t S_0 \alpha_t \xi_t}{(\xi_h + \mu)(\gamma_t + \delta_t + \mu)\omega} + \frac{\beta_h S_0 \alpha_h \xi_h}{(\xi_h + \mu)(\delta_h + \mu)\omega} \quad (16)$$

Substituting $S = \frac{\Lambda}{\mu}$ into (16) gives;

$$R_0 = \frac{\beta_t (\Lambda/\mu) \alpha_t \xi_t}{(\xi_h + \mu)(\gamma_t + \delta_t + \mu)\omega} + \frac{\beta_h (\Lambda/\mu) \alpha_h \xi_h}{(\xi_h + \mu)(\delta_h + \mu)\omega} \quad (17)$$

3.6 Local Stability of the Disease-Free Equilibrium of Typhoid-Helminth Co-infection

Theorem 1.

Consider the typhoid–helminth co-infection model with typhoid-only treatment and infection-generated environmental contamination, with disease-free equilibrium E_0. Let R_0 be the basic reproduction number. Then, the disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

Let $x = (E_t, E_t, I_t, I_t, P)^T$ denote the infected-state variables. The system can be written in the standard form;

$\frac{dx}{dt} = F(x) - V(x)$.

where F contains the rates of appearance of new infections and V represents all other transition terms. Evaluating the Jacobians of F and V at the disease-free equilibrium E_0 yields the next-generation matrix.

$K = FV^{-1}$

By definition, R_0 is the spectral radius of K.

From the result of van den Driessche and Watmough (2002), the disease-free equilibrium E_0 is locally asymptotically stable if $\rho(K) < 1$ and unstable if $\rho(K) > 1$. Since $\rho(K) = R_0$, it follows immediately that E_0 is locally asymptotically stable when $R_0<1$ and unstable when $R_0>1$.

3.7 Global Stability of the Disease-Free Equilibrium of Typhoid-Helminth Co-infection

Theorem 2.

Consider the typhoid-helminth co-infection model (1) – (8) with infection-generated environmental contamination and forces of infection. Let E_0 and R_0 be the disease-free equilibrium (DFE) and basic reproduction number respectively. Then, E_0 is globally asymptotically stable in the feasible region Ω when $R_0< 1$.

Proof:

From the positivity and invariant-region results (3.2) and (3.3), solutions with nonnegative initial data remain in Ω and the total human population satisfies,

$$N(t) \le \max\left\{N(0), \frac{\Lambda}{\mu}\right\}.$$

particularly, $0 \le S(t) \le N(t) \le \frac{\Lambda}{\mu}$ for all sufficiently large t. (18)

Let $x = (E_t, E_t, I_t, I_t, P)^T$. From the model equations, and using $S(t) \le \frac{\Lambda}{\mu}$ from (18), we have the bounds

$$\frac{dE_t}{dt} = \beta_t PS - (\xi_t + \mu)E_t \le \beta_t P\frac{\Lambda}{\mu} - (\xi_t + \mu)E_t,$$

$$\frac{dE_h}{dt} = \beta_h PS - (\xi_h + \mu)E_h \le \beta_h P\frac{\Lambda}{\mu} - (\xi_h + \mu)E_h.$$

For I_t and I_h, the co-infection acquisition terms are additional nonnegative removals (they appear with minus signs), so dropping them gives an upper bound (19) and (20).

$$\frac{dI_t}{dt} = \xi_t E_t - (\beta_{th}P + \gamma_t + \delta_t + \mu)I_t \le \xi_t E_t - (\gamma_t + \delta_t + \mu)I_t \quad (19)$$

$$\frac{dI_h}{dt} = \xi_h E_h + \gamma_{th}I_{th} - (\beta_{ht}P + \delta_h + \mu)I_h \le \xi_h E_h - (\delta_h + \mu)I_h + \gamma_{th}I_{th}. \quad (20)$$

Also, $\frac{dP}{dt} = \alpha_t I_t + \alpha_h I_h + \alpha_{th}I_{th} - \omega P$. Now observe that I_{th} satisfies

$$\frac{dI_{th}}{dt} = \beta_{th}PI_t + \beta_{ht}PI_h - (\gamma_{th} + \delta_{th} + \mu)I_{th},$$

so when $x(t) \to 0$, we automatically get $I_{th}(x) \to 0$ (it is driven by products and has linear decay). Similarly, T and R are driven by I_t and decay otherwise, so they also go to zero once $I_t \to 0$.

Thus, to prove global stability of the DFE, it is sufficient to show $x(t) \to 0$.

Considering the comparison linear system $\frac{dy}{dt} = (F - V)y$, where $y = (E_t, E_t, I_t, I_t, P)^T$ (21)

and F,V are exactly the next-generation matrices evaluated at the DFE with $S_0 \leq \frac{\Lambda}{\mu}$ (the same matrices used to compute R_0). By construction of the next-generation method, the condition $R_0 < 1$ is equivalent to the matrix (F−V) being Hurwitz (all eigenvalues have negative real parts), so the solution of (21) satisfies $y(t) \to 0$ as $t \to \infty$. (22)

Moreover, from the inequalities above and the fact that all omitted nonlinear terms are either nonnegative removals or higher-order infection terms, the infected vector x(t) satisfies a standard comparison inequality of the form $\frac{dx(t)}{dt} = (F - V)x(t)$, for large t, hence by the comparison theorem for cooperative linear systems and (22),

$$x(t) \to 0 \text{ as } t \to \infty. \quad (23)$$

From (23), $P(t) \to 0$, $I_t(t) \to 0$, and $I_h(t) \to 0$. Then the co-infected class satisfies $\frac{dI_{th}}{dt} = -(\gamma_{th} + \delta_{th} + \mu)I_{th} + \beta_{th}PI_t + \beta_{ht}PI_h$, and since the forcing terms $\beta_{th}PI_t$ and $\beta_{ht}PI_h$ tend to zero, it follows that $I_{th}(t) \to 0$. Next,

$$\frac{dT}{dt} = \gamma_t I_t - (\phi + \mu)T, \Rightarrow T(t) \to 0, \text{ and } \frac{dR}{dt} = \phi T - (k + \mu)R, \Rightarrow R(t) \to 0.$$

Finally, with $\lambda_t = \lambda_h = 0$ asymptotically and $R(t) \to 0$. The susceptible equation reduces to:

$\frac{dS}{dt} = \Lambda - \mu S$, so that $S(t) \to \frac{\Lambda}{\mu}$. Therefore, $(S, E_t, E_h, I_t, I_h, I_{th}, T, R, P) = \left(\frac{\Lambda}{\mu}, 0,0,0,0,0,0,0,0\right)$.

which proves that the DFE is globally asymptotically stable when $R_0 < 1$.

4 Endemic Equilibrium Analysis

When the basic reproduction number is greater than one, the disease may persist in the population, leading to the existence of an endemic equilibrium.

4.1 Endemic Equilibrium (EE) point of Typhoid-Helminth Co-infection

Let the derivative of equation (1)–(9) be zero.

Let the endemic equilibrium be $E_1 = (S^*, E_t^*, E_h^*, I_t^*, I_h^*, I_{th}^*, T^*, R^*, P^*)$ with at least one infected component positive.

Recall that $\Pi(t) = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th}$, $\lambda_t = \beta_t P$, and $\lambda_h = \beta_h P$.

From equation (7): $0 = \frac{dT}{dt} = \gamma_t I_t - (\phi + \mu)T, \Rightarrow T^* = \frac{\gamma_t}{\phi + \mu} I_t^*.$

From equation (8): $0 = \frac{dR}{dt} = \phi T - (k + \mu)R \Rightarrow R^* = \frac{\phi}{(k + \mu)} T^* = \frac{\phi \gamma_t}{(\phi + \mu)(k + \mu)} I_t^*.$

From equation (2): $0 = \frac{dE_t}{dt} = \lambda_t S - (\xi_t + \mu)E_t \Rightarrow E_t^* = \frac{\beta_t P^* S^*}{\xi_t + \mu}.$

From equation (3): $0 = \frac{dE_h}{dt} = \lambda_h S - (\xi_h + \mu)E_h \Rightarrow E_h^* = \frac{\beta_h P^* S^*}{\xi_t + \mu}.$

From equation (4): $0 = \frac{dI_t}{dt} = \xi_t E_t - (\beta_{th} P + \gamma_t + \delta_t + \mu)I_t,$

$$I_t^* = \frac{\xi_t E_t^*}{\beta_{th} P + \gamma_t + \delta_t + \mu} = \frac{\xi_t \beta_t P^* S^*}{(\xi_t + \mu)(\beta_{th} P + \gamma_t + \delta_t + \mu)}.$$

From equation (6): $0 = \frac{dI_{th}}{dt} = \beta_{th} P I_t + \beta_{ht} P I_h - (\gamma_{th} + \delta_{th} + \mu)I_{th},$

$$I_{th}^* = \frac{P^*(\beta_{th} I_t^* + \beta_{ht} I_h^*)}{\gamma_{th} + \delta_{th} + \mu}.$$

From equation (5): $0 = \frac{dI_h}{dt} = \xi_h E_h + \gamma_{th} I_{th} - (\beta_{ht} P + \delta_h + \mu)I_h,$

$$I_h^* = \frac{\xi_h E_h^* + \gamma_{th} I_{th}^*}{\beta_{ht} P^* + \delta_h + \mu} = \frac{\xi_h \frac{\beta_h P^* S^*}{\xi_h + \mu} + \gamma_{th} I_{th}^*}{\beta_{ht} P^* + \delta_h + \mu}.$$

From equation (9): $0 = \frac{dP}{dt} = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th} - \omega P,$

$$P^* = \frac{\alpha_t I_t^* + \alpha_h I_h^* + \alpha_{th} I_{th}^*}{\omega}.$$

From equation (1): $0 = \frac{dS}{dt} = \Lambda + kR - (\beta_t P + \beta_h P + \mu)S,$

$$S^* = \frac{\Lambda + kR^*}{\mu + (\beta_t + \beta_h)P^*} = \frac{\Lambda + k\left(\frac{\phi \gamma_t}{(\phi + \mu)(k + \mu)} I_t^*\right)}{\mu + (\beta_t + \beta_h)P^*}.$$

Therefore, $$S^* = \frac{\Lambda + \frac{k\phi\gamma_t}{(\phi+\mu)(k+\mu)} I_t^*}{\mu + (\beta_t + \beta_h)P^*}.$$

The endemic equilibrium point will be

$$E_1 = (S^*, E_t^*, E_h^*, I_t^*, I_h^*, I_{th}^*, T^*, R^*, P^*) =$$

$$\left(\begin{array}{l} \frac{\Lambda + \frac{k\phi\gamma_t}{(\phi+\mu)(k+\mu)} I_t^*}{\mu + (\beta_t + \beta_h)P^*}, \frac{\beta_t P^* S^*}{\xi_t + \mu}, \frac{\beta_h P^* S^*}{\xi_t + \mu}, \frac{\xi_t \beta_t P^* S^*}{(\xi_t + \mu)(\beta_{th} P + \gamma_t + \delta_t + \mu)}, \frac{\xi_h \frac{\beta_h P^* S^*}{\xi_h + \mu} + \gamma_{th} I_{th}^*}{\beta_{ht} P^* + \delta_h + \mu}, \\ \frac{P^*(\beta_{th} I_t^* + \beta_{ht} I_h^*)}{\gamma_{th} + \delta_{th} + \mu}, \frac{\gamma_t}{\phi + \mu} I_t^*, \frac{\phi\gamma_t}{(\phi+\mu)(k+\mu)} I_t^*, \frac{\alpha_t I_t^* + \alpha_h I_h^* + \alpha_{th} I_{th}^*}{\omega}. \end{array} \right)$$

The endemic equilibrium E_1 exists (with $P^* > 0$ and at least one infected class positive) whenever $R_0 > 1$.

4.2 Global stability of the endemic equilibrium of Typhoid-Helminth Co-infection

Theorem 3.

Consider the typhoid–helminth co-infection model with typhoid-only treatment and infection-generated environmental contamination $\Pi(t) = \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th}$, $\lambda_t = \beta_t P$, $\lambda_h = \beta_h P$, $\frac{dP}{dt} = \Pi(t) - \omega P$. Assume,

(C1) No disease-induced deaths: $\delta_t = \delta_h = \delta_{th} = 0$.
(C2)No loss of immunity: k=0 (so recovered do not return to S).
(C3) All parameters are nonnegative and $\mu, \omega > 0$.
(C4) The model admits a unique endemic equilibrium$E_1 \in \Omega$ (all components positive) whenever $R_0 > 1$. Then, if $R_0 > 1$, the endemic equilibrium E_1 is globally asymptotically stable in the interior of the feasible region Ω.

Proof:

Under (C1)–(C2), the human population satisfies $\frac{dN}{dt} = \Lambda - \mu N$, so $N(t) \to \frac{\Lambda}{\mu}$ as $t \to \infty$. Moreover, positivity and boundedness hold in Ω, so trajectories remain in a compact positively invariant set (as shown in section 3.2-3.3).

Let $E_1 = (S^*, E_t^*, E_h^*, I_t^*, I_h^*, I_{th}^*, T^*, R^*, P^*)$ be the unique endemic equilibrium for $R_0 > 1$. let the Volterra-type Lyapunov function be

$$V = \left(S - S^* - S^* In \frac{S}{S^*} \right) + \left(E_t - E_t^* - E_t^* In \frac{E_t}{E_t^*} \right) + \left(E_h - E_h^* - E_h^* In \frac{E_h}{E_h^*} \right) +$$
$$\left(I_t - I_t^* - I_t^* In \frac{I_t}{I_t^*} \right) + \left(I_h - I_h^* - I_h^* In \frac{I_h}{I_h^*} \right) + \left(I_{th} - I_{th}^* - I_{th}^* In \frac{I_{th}}{I_{th}^*} \right) + \left(T - T^* - T^* In \frac{T}{T^*} \right) \quad (24)$$
$$+ \left(R - R^* - R^* In \frac{R}{R^*} \right) + c \left(P - P^* - P^* In \frac{P}{P^*} \right),$$

where $c > 0$ is a constant to be chosen.

$x - x^* - x^* In(x/x^*) \geq 0$ for all $x > 0$ with equality only at $x = x^*$, it follows that $V \geq 0$ and $V = 0$ if and only if the state equals E_1.

Differentiate V along solutions. Using $\frac{d}{dt}\left(x - x^* - x^* In\frac{x}{x^*} \right) = \left(1 - \frac{x^*}{x} \right)\frac{dx}{dt}$, we obtain;

$$\frac{dV}{dt} = \sum_{X \in \{S, E_t, E_h, I_t, I_h, I_{th}, T, R\}} \left(1 - \frac{X^*}{X} \right)\frac{dX}{dt} + c\left(1 - \frac{P^*}{P} \right)\frac{dP}{dt}.$$

Now substitute the model equations and use the equilibrium identities satisfied at E_1. In particular, the equilibrium relations imply:

$$\beta_t P^* S^* = (\xi_t + \mu)E_t^*,\ \beta_h P^* S^* = (\xi_h + \mu)E_h^*,\ \xi_t E_t^* = (\beta_{th} P^* + \gamma_t + \mu)I_t^*,$$

$$\xi_h E_h^* + \gamma_{th} I_{th}^* = (\beta_{ht} P^* + \mu)I_h^*,\ \beta_{th} P^* I_t^* + \beta_{ht} P^* I_h^* = (\gamma_{th} + \mu)I_{th}^*,$$

$$\gamma_t I_t^* = (\phi + \mu)T^*,\ \phi T^* = \mu R^*,\ \alpha_t I_t + \alpha_h I_h + \alpha_{th} I_{th} = \omega P^*.$$

With these identities, V can be grouped into sums of terms of the form

$$-a\left(\frac{X}{X^*} + \frac{X^*}{X} - 2 \right) \text{ or } -a\left(\frac{X}{X^*} - 1 \right)^2 \ (a > 0),$$

which are all ≤0. The key step is applying the arithmetic-geometric mean inequality:

$$\left(\frac{\mu}{\mu^*} + \frac{\mu^*}{\mu} \right) \geq 2 \qquad (\mu > 0),$$

to each "paired flow" term created by transitions such as $(E_t \leftrightarrow I_t)$, $(I_t \leftrightarrow T)$ and the environmental coupling $(I_t, I_h, I_{th} \leftrightarrow P)$. Choosing c>0 so that the environmental terms cancel appropriately (a standard choice is to take c proportional to 1/ω so that the P-terms pair with the shedding terms), we obtain; $\frac{dV}{dt} \leq 0$ for all states in Ω.

The uniqueness follows from monotonicity of the infection subsystem and linear environmental coupling.

Moreover, $\frac{dV}{dt} = 0$ holds only when every ratio $\frac{X}{X^*} = 1$ and $\frac{P}{P^*} = 1$ (only at the endemic equilibrium E_1). Therefore, the largest invariant set contained in $\left\{ \frac{dV}{dt} = 0 \right\}$ is the singleton $\{E_1\}$.

By LaSalle's invariance principle, every solution starting in Ω approaches E_1 as $t \to \infty$. Hence, E_1 is globally asymptotically stable in Ω whenever $R_0 > 1$.

5 Results

This section presents analytical and numerical results illustrating the transmission dynamics and evaluating the impact of control measures.

5.1 Sensitivity Analysis of the Basic Reproduction Number R_0

Sensitivity analysis evaluates how changes in parameter values affect model outcomes and helps identify effective control strategies [3].

Recall the expression (17) obtained via the next-generation matrix method,

$$R_0 = \frac{\beta_t(\Lambda/\mu)\alpha_t\xi_t}{(\xi_h+\mu)(\gamma_t+\delta_t+\mu)\omega} + \frac{\beta_h(\Lambda/\mu)\alpha_h\xi_h}{(\xi_h+\mu)(\delta_h+\mu)\omega}$$

For any parameter p, the normalized sensitivity index of R_0 is defined as

$$\gamma_p^{R_0} = \frac{\partial R_0}{\partial p}.\frac{p}{R_c}. \tag{25}$$

Using the parameter values in Table 3, we compute of R_t, R_h and weights

For Typhoid reproduction number: $R_t = \frac{(2.0\times10^{-8})(1.094\times10^{6})(1.0)(0.0833)}{0.25(0.0833+0.0000457)(0.1002)}$

$$\approx 0.873$$

For Helminth reproduction number: $R_h = \frac{(2.0\times10^{-9})(1.094\times10^{6})(0.4)(0.0333)}{0.25(0.0833+0.0000457)(0.1002)}$

$$\approx 0.348$$

Overall basic reproduction number: $R_0 = R_t + R_h \approx 1.221$

For Weights: $\omega_t = \frac{R_t}{R_0} = \frac{0.873}{1.221} \approx 0.715$ $\omega_t = \frac{R_h}{R_0} = \frac{0.348}{1.331} = 0.285$

About 71.5% of transmission potential comes from typhoid and 28.5% from helminth infection under the baseline in Table 3

Sensitivity analysis was carried out using the parameter values in Table 3 and the resultant analysis yields sensitivity index in table 4 as shown below.

Table 3 Model Parameters, baseline values and sources.

Parameter	Baseline value	Source
Λ	50 persons/day	Assumed
μ	4.57×10^{-5}	[6]
β_t	2.0×10^{-8}	Assumed
β_h	2.0×10^{-9}	Assumed
ξ_t	0.0833	[9]
ξ_h	0.0333	[7]

γ_t	0.10	[8]
δ_t	0.0002	[8]
δ_h	0.01	[11]
α_t	1.0	Assumed
α_h	0.4	Assumed
ω	0.25	[10]
β_{th}	1.0×10^{-9}	Assumed
β_{ht}	1.0×10^{-9}	Assumed
γ_{th}	0.10	[8]
ϕ	0.2	[9]
k	0.0005	Assumed

Parameter values were obtained from published demographic, clinical, epidemiological, and environmental studies where available. Parameters not reported in the literature were assumed within biologically reasonable ranges and their influence on model outcomes was assessed using sensitivity analysis.

Table 4 Normalized sensitivity indices of R_0

Parameter	**Sensitivity index**
ω	-1.000
β_t	$+0.715$
α_t	$+0.715$
γ_t	-0.713
β_h	$+0.285$
α_h	$+0.285$
δ_h	-0.284
δ_t	-0.001
ξ_t	$+0.0004$
ξ_h	$+0.0004$

Sensitivity analysis indicates that the most influential parameter on the basic reproduction number is the environmental pathogen decay rate ω, highlighting sanitation as the most effective control strategy. Typhoid-related transmission and shedding parameters (β_t , α_t) and the typhoid treatment rate (γ_t) have the next strongest influence, reflecting the dominant contribution of typhoid infection to overall transmission (w_t =0.715). Helminth-related parameters have a smaller but non-negligible impact (w_h = 0.285), indicating that untreated helminthiasis can sustain infection even when typhoid is partially controlled.

5.2 Numerical Simulation

Numerical experiments were performed to demonstrate the qualitative dynamics of the model under baseline and intervention scenarios. All parameter values used correspond exactly to those listed in Table 3, ensuring consistency between analytical results and numerical experiments. Initial conditions were chosen to represent a co-endemic setting with non-zero exposed and infectious classes, while maintaining positivity and biological feasibility.

The system of nonlinear ordinary differential equations was solved using a standard deterministic ODE solver (e.g. MATLAB ode45), which is appropriate for smooth, non-stiff epidemic systems. Time integration was performed over a sufficiently long horizon to capture transient and asymptotic dynamics. Tolerance settings were selected to ensure numerical stability and accuracy.

To assess intervention impact, one-parameter perturbations were applied to key control variables identified through sensitivity analysis. Specifically; the typhoid treatment rate was increased to evaluate clinical-only intervention effects, the environmental pathogen decay rate (ω) was increased to represent improved sanitation and the helminth transmission rate was amplified to represent high-exposure settings.

All other parameters were held constant during scenario comparisons to isolate the effect of each intervention. The qualitative trends observed in the simulations are consistent with analytical predictions derived from the reproduction number and sensitivity indices as shown in Figure 2 – 6.

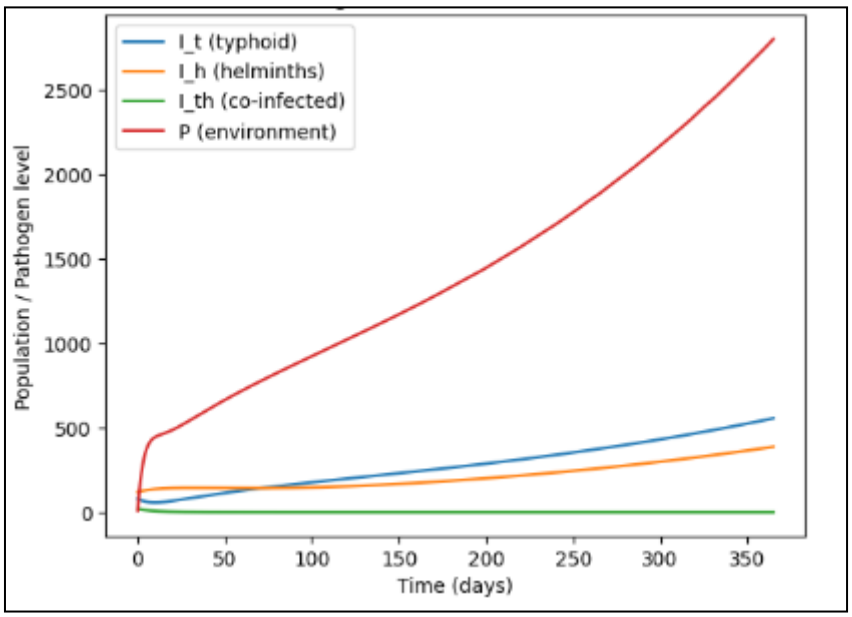

Figure 2 Baseline dynamics coexistence of typhoid I_t, helminths I_h, co-infection I_{th}, and the environmental pathogen P

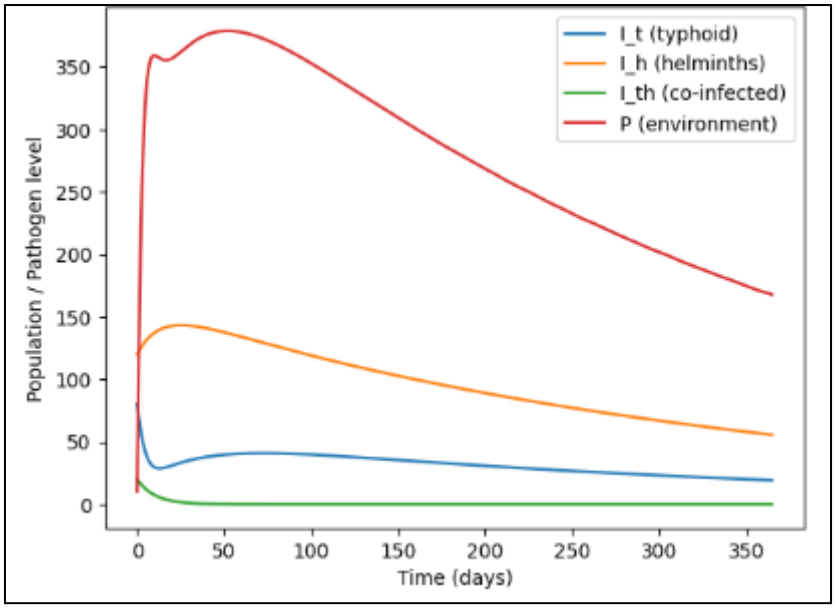

Figure 3 Higher typhoid treatment (γ_t ×2).

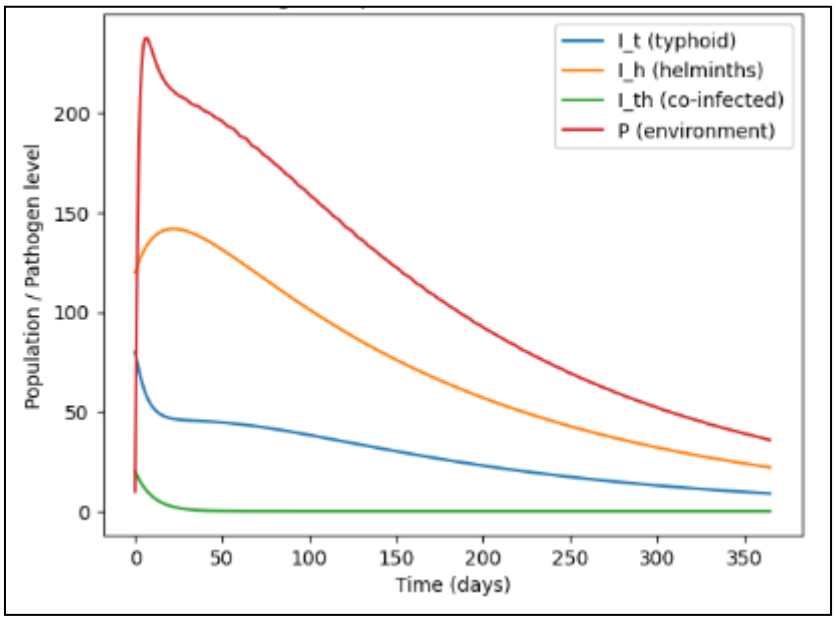

Figure 4 Improved sanitation (ω ×2).

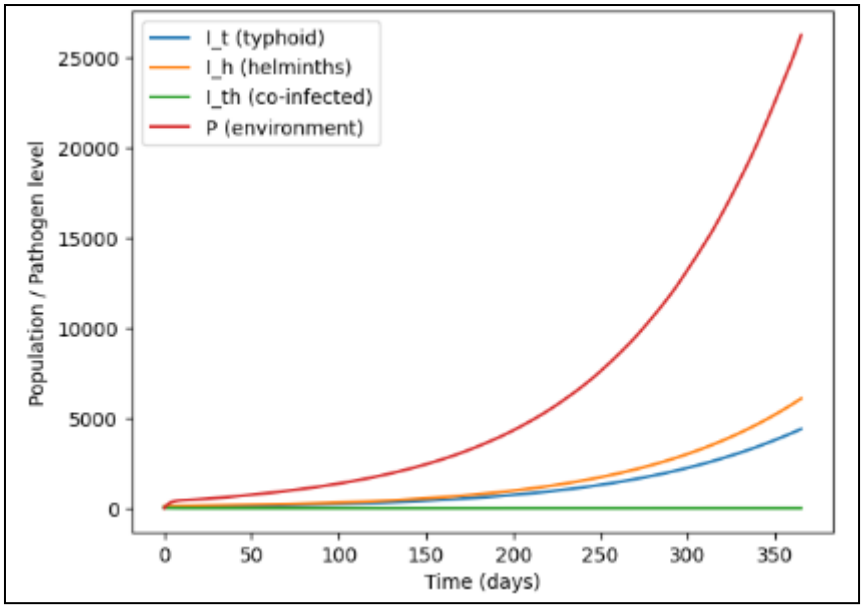

Figure 5 Higher helminth exposure (β_h ×3). Helminths dominate, co-infection rises, and typhoid-only treatment becomes less effective-exactly the "persistent symptoms" scenario.

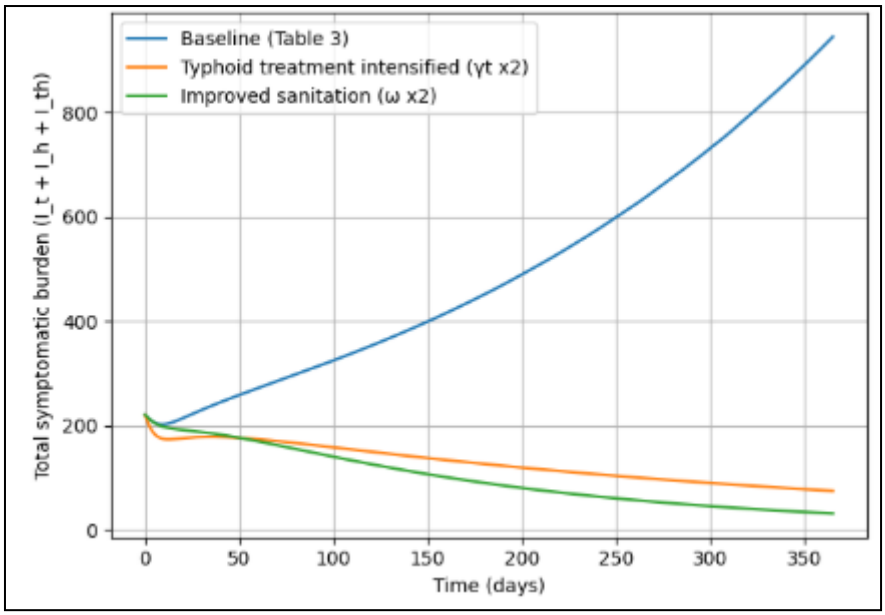

Figure 6 Total Symptomatic Burden under Key Scenarios (Table 3 Parameters)

5.2.1 Baseline dynamics

Under baseline conditions (Figure 2), typhoid infection, helminth infection, and co-infection coexist and are maintained through environmental pathogen persistence. Although typhoid infection dominates transmission, helminth infection persists and maintains a measurable co-infected population.

5.2.2 Effect of typhoid-only treatment

Increasing the typhoid treatment rate (γ_t ×2; Figure 3) substantially reduces typhoid prevalence and lowers environmental pathogen levels. However, helminth infection remains persistent, leading to a slower decline in the co-infected class. This result highlights that typhoid-only treatment may alleviate acute typhoid symptoms while leaving underlying helminth-driven morbidity unresolved.

5.2.3 Effect of sanitation and helminth exposure

Improved sanitation (ω ×2; Figure 4) results in a pronounced decline across all infected compartments, including helminth infection and co-infection, confirming the dominant role of environmental control identified by sensitivity analysis.

In contrast, increased helminth exposure (β_h ×3; Figure 5) leads to a marked rise in helminth prevalence and co-infection, even when typhoid transmission remains partially controlled. Figure 5 demonstrates that high helminth exposure can sustain infection and symptom persistence despite effective typhoid treatment, emphasizing the epidemiological importance of helminthiasis in co-endemic settings.

5.2.4 Total symptomatic burden

The total symptomatic burden, defined as $I_t + I_h + I_{th}$, is shown in figure 6. Intensifying typhoid treatment reduces the burden relative to baseline but does not eliminate symptoms due to the continued presence of helminth infection. In contrast, improved sanitation produces the greatest reduction in total burden. These results provide a mechanistic explanation for persistent symptoms following typhoid treatment alone.

6 Discussion and Policy Implications

This study examined the transmission dynamics of typhoid fever and helminthiasis under a co-endemic setting, with particular emphasis on scenarios where clinical management targets typhoid infection alone. The results provide both mechanistic and policy-relevant insights into why persistent symptoms may occur following standard typhoid treatment and how integrated control strategies can substantially improve health outcomes.

6.1 Why typhoid-only treatment can mislead clinicians

The numerical simulations demonstrate that increasing the typhoid treatment rate markedly reduces typhoid prevalence and environmental pathogen load, yet fails to eliminate the overall symptomatic burden when helminth infection remains untreated. In particular, helminth infection persists and continues to sustain a non-negligible co-infected population, even as typhoid incidence declines. Clinically, this may produce temporary clinical improvement followed by continued symptom persistence, which can be misinterpreted as treatment failure, drug resistance, or relapse of typhoid fever. The model therefore provides a plausible explanation for persistent symptoms observed in co-endemic regions despite adequate typhoid therapy.

6.2 Importance of combined diagnosis and integrated treatment

The relative contribution of helminth infection to overall transmission ($\omega_h \approx 0.285$) indicates that helminthiasis plays a meaningful supporting role in sustaining infection dynamics. Simulation results under increased helminth exposure further show that high helminth prevalence amplifies co-infection and symptom persistence, undermining the effectiveness of typhoid-only interventions. These results highlight the need for integrated diagnostic strategies that screen for both typhoid and helminth infections in symptomatic patients. From a public health perspective, integrating de-worming programs with typhoid management, either through routine presumptive treatment or targeted parasite screening, could significantly reduce long-term morbidity and prevent misclassification of persistent symptoms.

6.3 Sanitation as the dominant control lever

Sensitivity analysis identified the environmental pathogen decay rate ω, which captures the effects of sanitation and environmental hygiene, as the most influential parameter governing the basic reproduction number. Unlike treatment-based interventions that act on a single infection pathway, improvements in sanitation reduce both typhoid and helminth transmission simultaneously by limiting environmental contamination. This dual impact was confirmed in simulations, where increased ω produced the largest reduction in all infected classes and in the total symptomatic burden. These findings reinforce the role of water, sanitation, and hygiene (WASH) improvements as fundamental components of disease control in co-endemic settings and suggest that investments in sanitation may yield broader and more sustainable benefits than disease-specific interventions alone [7].

6.4 Public health implications

Taken together, the results suggest that reliance on typhoid-only treatment strategies may underestimate the true drivers of persistent morbidity in co-endemic regions. Integrated control strategies such as combining accurate diagnosis, appropriate treatment of both infections, and sustained improvements in sanitation are likely to be far more effective in reducing disease burden. The model highlights that addressing environmental transmission pathways and

neglected co-infections is essential for achieving long-term symptom resolution and improving clinical decision-making in resource-limited settings.

7 Conclusion

This study provides a mechanistic interpretation for persistent clinical symptoms observed in regions where typhoid fever and helminthiasis co-exist but are not jointly managed. By explicitly modeling typhoid-only treatment in the presence of untreated helminth infection and a shared environmental transmission pathway, the results show that successful reduction of typhoid prevalence does not necessarily translate into symptom resolution. Helminth infection can remain epidemiologically active and continue to sustain both co-infection and overall morbidity. The analysis further demonstrates that interventions acting solely at the clinical level have limited reach when environmental transmission persists. In contrast, improvements in sanitation exert a system-wide effect by simultaneously suppressing multiple infection pathways, making environmental control the most effective lever for reducing transmission and symptomatic burden. These findings emphasize that persistent symptoms should not automatically be interpreted as treatment failure or resistance, but may instead reflect overlooked co-infections maintained by environmental exposure. Overall, the findings emphasize the necessity of integrated diagnostic and control strategies that align clinical practice with the ecological realities of co-endemic diseases. Addressing co-infection and environmental transmission together is essential for achieving durable reductions in disease burden and improving patient outcomes in resource-limited settings.

Recommendations

Health systems operating in co-endemic regions should avoid managing persistent febrile illness through single-disease protocols. Routine clinical evaluation for suspected typhoid fever should incorporate parallel screening or presumptive management for helminth infection where laboratory capacity is limited. Integrating short-course de-worming into typhoid case management pathways may reduce recurrent symptoms and unnecessary repeat antibiotic use.

At the population level, disease-specific treatment campaigns should be aligned with environmental interventions. Investments in sanitation infrastructure, safe water access, and hygiene promotion should be prioritized as foundational control measures, since they simultaneously disrupt multiple transmission pathways.

Policy frameworks should therefore shift from isolated disease control models toward integrated infection management strategies that combine diagnosis, targeted therapy, and sustained environmental improvement to achieve durable reductions in symptomatic burden.

Compliance with ethical standards

Acknowledgments

The authors acknowledge the support of the Department of Mathematics for providing an enabling academic environment and computational facilities required for this study. Gratitude is also extended to mentors, colleagues, and reviewers whose constructive insights and encouragement contributed to the successful completion of this work.

Disclosure of conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Statement of ethical approval

This study is based entirely on mathematical modeling and numerical simulations and does not involve human participants, animal subjects, or the use of identifiable personal data. All analyses were conducted using theoretical frameworks and hypothetical parameter values obtained from publicly available literature sources.

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