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MATHEMATICAL ANALYSIS OF MALARIA-TYPHOID CO-INFECTION DYNAMICS WITH ENVIRONMENTAL DRIVERS

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ABSTRACT

Malaria and typhoid fever pose significant health challenges in endemic regions, particularly in sub-Saharan Africa, due to environmental factors such as poor sanitation, stagnant water, and fluctuating temperatures. This study presents a deterministic compartmental model for co-infection dynamics of malaria and typhoid fever, incorporating both direct (human-to-human) and indirect (environment-to-human) transmission. Sub-models for malaria, typhoid and co-infection are analyzed to establish positivity, boundedness, and disease-free equilibrium (DFE). The basic reproduction numbers are derived using the next-generation matrix method. Stability of the DFE is shown when R_0 is less than unity $(R_0^m < 1, R_0^t < 1, R_0^{mt} < 1)$, particularly under the condition, $g_t < e_t$ and $g_v < e_v$ and unstable when $R_0 > 1$ $(R_0^m > 1, R_0^t > 1, R_0^{mt} > 1)$. Numerical simulations assess the impact of environmental decay pathogen shedding and breeding vector growth on disease persistence. Sensitivity analysis reveals key drivers influencing disease persistence. The results underscore the importance of integrated interventions targeting both environmental and host-based transmission pathways to reduce the burden of malaria-typhoid co-infection.

1. INTRODUCTION

Infectious diseases continue to pose a serious global health burden, particularly in low and middle-income countries where deteriorating infrastructure and limited access to healthcare persist. In sub-Saharan Africa, malaria and typhoid fever remain endemic and are frequently implicated in co-infection scenarios that complicate diagnosis, treatment, and control efforts. Both diseases share overlapping symptoms, transmission conditions, and risk environments, leading to frequent misdiagnosis and improper treatment. often with fatal consequences. Malaria, a parasitic disease transmitted by the female *Anopheles* mosquito, is responsible for a considerable proportion of morbidity and mortality, especially among children under five and pregnant women.

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[1] in 2021 alone, over 240 million cases of malaria were reported globally, resulting in approximately 619,000 deaths, with the African region accounting for more than 90% of this burden. Despite the availability of effective preventive measures, such as insecticide-treated nets and antimalarial therapies, transmission persists due to environmental factors, such as standing water that facilitates mosquito breeding, and climatic conditions that favor vector survival. Typhoid fever, caused by Salmonella enteric serotype Typhi, is a water / fecal-oral and foodborne bacterial infection, and can be transmitted directly and indirectly. Despite the fact that chronic typhoid can be transmitted by individual, who do not have history of the illness [2]. Is common in areas where limited access to safe water, poor hygiene, and inadequate sanitation, typhoid outbreaks remain common

Co-infection of malaria and typhoid fever is a growing public health concern in regions where both diseases are endemic. The co-existence of these pathogens in a single host can significantly complicate clinical management due to symptom overlap and potential drug interactions. Environmental factors play a pivotal role in the transmission dynamics of both diseases. Stagnant water, fluctuating temperatures, and poor sanitation create reservoirs for both breeding vectors and typhoid-causing bacteria. While previous studies have modeled the co-infection dynamics of various diseases, few have explicitly captured the environmental dependency of malaria-typhoid co-infection within a unified mathematical framework [3].

In this study, we extend prior work by developing a novel compartmental model that incorporates both direct and indirect transmission pathways for malaria and typhoid fever [4]. Our model explicitly accounts for environmental reservoirs, on how vectors such as contaminated water sources and mosquito breeding sites can be reduced [5], and investigates how these reservoirs influence disease dynamics. the model is subjected to numerical simulations and sensitivity analysis to evaluate the impact of key parameters,

The remainder of this paper is structured as follows: Section 2 presents the model formulation, including all compartments and transmission pathways. Section 3 analyzes the sub-models for malaria and typhoid infection separately and co-infection. Investigated the Disease Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP), we derive the basic reproduction numbers and perform stability analysis. While, Section 4 presents numerical simulations and discussion.

2.0 MATERIAL AND METHODS

2.1 Model Formulation

We develop a deterministic compartmental model to study the transmission dynamics of malaria and typhoid fever co-infection within a human population, accounting explicitly for environmental reservoirs that influence the dynamics of both diseases. The human population is assumed to be homogeneously mixed, and the total population at time t is denoted as:

$$N_h = S_h(t) + I_t(t) + I_m(t) + I_{mt}(t) + R_t(t) + R_m(t) + R_{mt}(t) + S_v(t)\, I_v(t)$$

Incorporated into the model are the bacterial compartments, where $B_t(t)$ is the concentration of Salmonella Typhi bacteria in the environment and $B_v(t)$ is the concentration of aquatic-stage mosquito vectors. Also we have, $S_h(t)$ Susceptible humans, $I_t,(t)$ infected human with typhoid, $I_m(t)$ infected human with malaria, $R_t(t)$ recovered infected human with typhoid, $R_m(t)$ recovered infected human with malaria, and $R_{mt}(t)$ is the susceptible human with co-infection.

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2.2 Transmission Dynamics

a) Typhoid fever infection to susceptible Humans

Direct (person-to-person) and indirect (environment) transmission

$$\lambda_{t_1} = \frac{\beta_{t1}\ (\ I_t + \mathrm{n}_t I_{mt}\ )}{N_h}, \quad \lambda_{t_2} = \frac{\beta_{t2}\ \ B_t}{B_t + \ K_t}$$

b) **Malaria Infection to Susceptible Humans**

Direct (person-to-person) and indirect (environment) transmission

$$\lambda_{m_1} = \frac{\beta_{m1}\ (\ I_m + \mathrm{n}_m I_{mt}\ )}{N_h}, \ \lambda_{m_2} = \beta_{m_2}\left(\frac{B_v}{B_v + \ K_v}\right) I_m$$

Cross-Transmission:Typhoid and Malaria Co-Infection

c) Typhoid to Malaria-infected individual

$$\lambda_{t_3} = \frac{\beta_{t3}(\ I_t + \mathrm{n}_t I_{mt}\ )}{N_h}, \ \lambda_{t_4} = \frac{\beta_{t4}\ \ B_t}{B_t + \ K_t}$$

d) Malaria to Typhoid-infected individual

$$\lambda_{m_3} = \frac{\beta_{m3}\ (r\ I_m + \mathrm{n}_m I_{mt}\ )}{N_h}, \ \lambda_{m_4} = \beta_{m4}\ (\frac{B_v}{B_v + \ K_v}) I_m$$

Where β_{t_1} denote person to peron typhoid transmissioin, β_{t_2} is denotes the environment-to-humans per capital contact rates and the salmonella typhi in the contaminated environment, $\mathrm{n}_t \in (0,1)$. This assumption is motivated by the fewer numbers of co-infected individuals as compared to those infected with typhoid only

For malaria, the parameter r, is the total number of mosquitoes bite per day, while β_{m1} denotes effective person-to-person malaria transmission rate. The modification parameter n_m, account for the relative infectious individual in class I_m relative to individual in class $I_{mt.}$ we also assume that $\mathrm{n}_m \in (0,1)$, this assumption assumed that there are fewer numbers of co-infected individuals as compared to those infected with malaria alone. While β_{m2} denotes environment-to-human transmission rates of the vectors /pathogens interaction of infection with other compartments, once humans carries the diseases and move to the environments, also bitten by the parasite, which become infected too. You can see the interaction in the model figure 1, between compartment B_v and I_m. Maywhile, the term λ_{t_4} is the environmental transmission terms for typhoid, while λ_{m_4} is the environmental transmission terms for malaria as a result of infected human(I_m)-to-environment transmissions due to interaction.

2.3 Environmental Contamination

Contamination Dynamics in the environmental reservoirs are governed by logistic growth, shedding and decay:

Salmonella Typhi:

$$\frac{dB_t}{dt} = g_t B_t(1\ - \frac{B_t}{K_t}) + J_t I_t + \theta_t I_{mt} - e_t B_t$$

Aquatic-stage (breeding) vectors:

$$\frac{dB_v}{dt} = g_v B_v(1 - \frac{B_v}{K_v}) + J_v I_v + \theta_v I_{mt} - e_v B_v$$

where $J_t J_v$ are Salmonella Typhi bacteria and pathogens/vectors shedding rates from single infected classes, while $\theta_t \theta_v$are co-infection shedding rate of salmonella Typhi and pathogens/breeding vectors in the environment, and $e_t e_v$ are environmental decay rates.

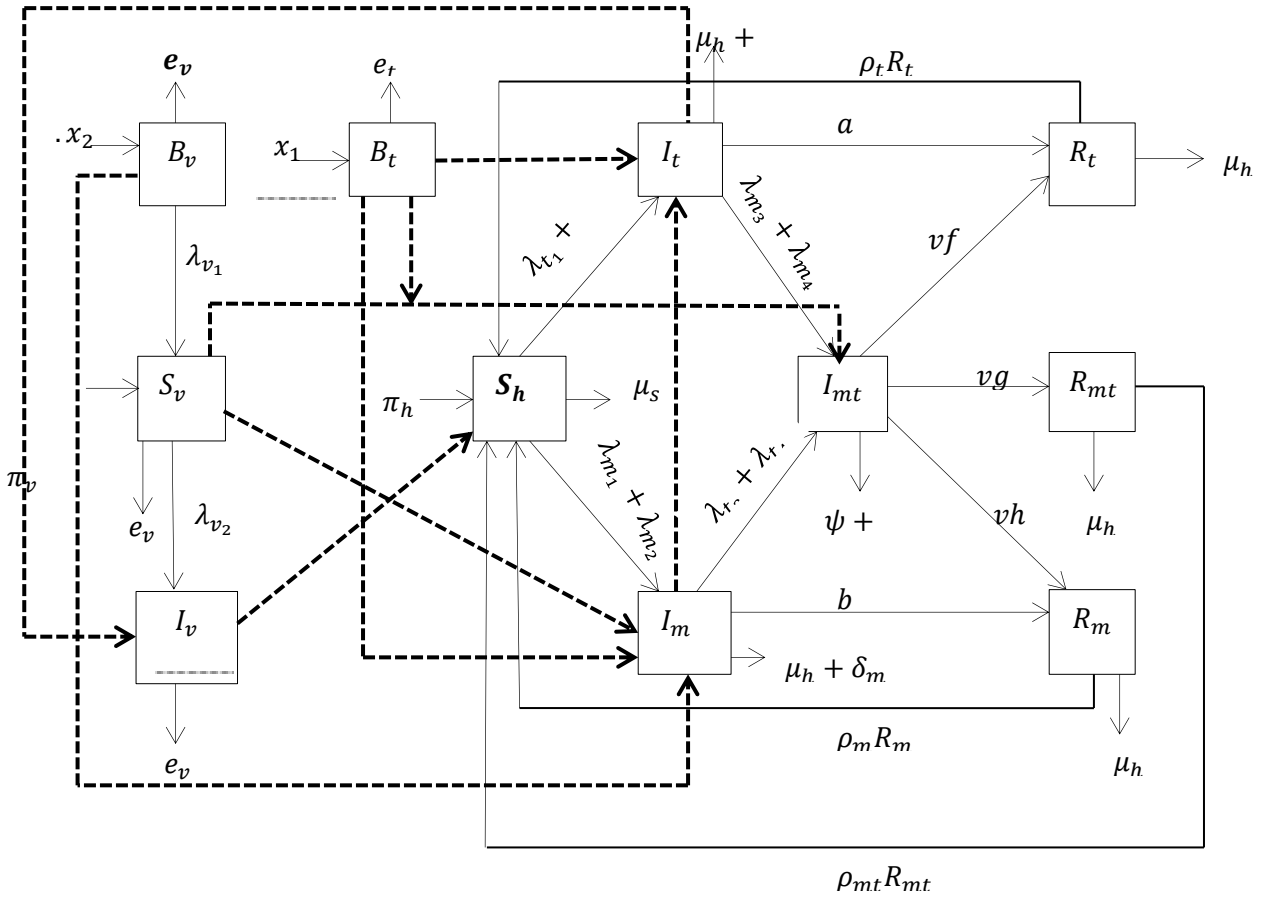

Figure 1: Schematic Flow Diagram for Malaria and Typhoid Fever Co-Infection

Substituted into the above flow diagram are:

$$x_1 = g_t\, B_t \left(1 - \frac{B_t}{k_t}\right) + \mathrm{J_t\, I_t} + \theta_t I_{mt} - \mathrm{e_t B_t}\,,\; x_2 = \mathrm{g_v\, B_v} \left(1 - \frac{B_v}{k_v}\right) + \mathrm{J_v\, I_v} + \theta_v I_{mt} - \mathrm{e_v B_v}$$

It is assumed that after treatment of individual with single infection and co-infection, immune system might likely wane, which make them move to the susceptible compartment to join new recruitments.

The complete system of differential equations describing the dynamics is:

$$\frac{dS_h}{dt} = \pi_h + \rho_t R_t + \rho_m R_m + \rho_{mt} R_{mt} - [\,\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}]S - \mu_s \mathrm{S}$$

$$\frac{dI_t}{dt}. = (\,\lambda_{t_1} + \lambda_{t_2})\, S - \left(\,\lambda_{m_3} + \lambda_{m_4}\right) I_t - (\,\mu_h + \delta_T + a)\, I_t$$

$$\frac{dI_m}{dt} = (\,\lambda_{m_1} + \lambda_{m_2})\, S - \left(\,\lambda_{t_3} + \lambda_{t_4}\right) I_m - (\,\mu_h + \delta_m + b)\, I_m$$

$$\frac{dI_{mt}}{dt} = \left(\,\lambda_{t_3} + \lambda_{t_4}\right) I_m - \left(\,\lambda_{m_3} + \lambda_{m_4}\right) I_t - v\big((f + g + h)\big) I_{mt} - (\,\mu_h + \psi) I_{mt}$$

$$\frac{dR_t}{dt} = aI_T + v(f) I_{mt} - (\,\mu_h + \rho_t)\, R_t$$

$$\frac{dR_m}{dt} = bI_m + v(h) I_{mt} - (\,\mu_h + \rho_m)\, R_m \qquad (1.)$$

$$\frac{dRm_t}{dt} = vg\, I_{mt} - (\,\mu_h + \rho_{mT}) R_{mt}$$

$\frac{dB_t}{dt} = g_t B_t(1 - \frac{B_t}{K_t}) + J_t I_t + \theta_t I_{mt} - e_t B_t$

$\frac{dB_v}{dt} = g_v B_v(1 - \frac{B_v}{K_v}) + J_v I_v + \theta_v I_{mt} - e_v B_v$

$\frac{dS_v}{dt} = \pi_v + \lambda_{v_1} B_v - (e_v + \lambda_{v_2}) S_v$

$\frac{dI_v}{dt} = \lambda_{v_2} S_v - e_v I_v$

The Initial condition of the co-infection model
$S_h(0) > 0,\ I_t(0) \geq 0, I_m(0) \geq 0, I_{mt}(0) \geq 0, R_t(0) \geq 0, R_m(0) \geq 0, R_{mt}(0) \geq 0,$
$B_t(0) \geq 0, B_v(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$

2.4 Positivity and Boundedness of the Malaria-typhoid fever co-infection Model.

For the model to be epidemiologically meaningful, it is important to examine the following in the closed set n.

Theorem 1

The solution of the proposed model (1) is bounded

Proof

Let N_h denotes the number of total population for humans;

$X(t) = S_h + I_t + I_m + I_{mt} + R_t + R_m + R_{mt} + B_t + B_v + S_m + I_m$

$N_h(t) = S_h(t) + I_t(t) + I_m(t) + I_{mt}(t) + R_t(t) + R_m(t) + R_{mt}(t)$ (2)

$\frac{dN_h}{d_t} = \frac{dS_h}{d_t} + \frac{dI_T}{d_t} + \frac{dI_m}{d_t} + \frac{dI_t}{d_t} + \frac{dI_{mt}}{d_t} + \frac{dR_t}{d_t} + \frac{dRm}{d_t} + \frac{dR_{mt}}{d_t}$

$\frac{dN_h}{d_t} = \pi_h + P_t R_t + P_m R_m + P_{mt} R_{mt} - \mu_h N_h - [\sigma_t I_t + \sigma_m I_m + \psi \sigma_{mt} I_{mt}]$

Let $Z = P_t R_t + P_m R_m + P_{mt}$ and σ_t, σ_m, σ_{mt} represent infection induced death rate.

If. there is no mortality of malaria and typhoid fever infection, we get

$\frac{dN_h}{d_t} \leq \pi_h + z - \mu_h N_h$

Thus, $\frac{dN_h}{d_t} + \mu_h N_h \leq \pi_h + z$ (3)

Integrating and simplifying both sides of the inequality, we obtain

$N_h(t) \leq \frac{\pi_h + z}{\mu_h} + N_h(0) - \frac{\pi_h + z}{\mu_h}) e^{\mu h^t}$

$\lim_{n.\to\infty} sup N_h(t) \leq \lim_{n\to\infty} \sup (\frac{\pi_h + z}{\mu_h} + (N_h(0) - \frac{\pi_h + z}{\mu_h}) e^{\mu h^t}) = \frac{\pi_h + z}{\mu_h}$

$n_h = \left\{ (S_h, I_t, I_m, I_{mt}, R_t . R_m, R_{mt}) \in R_+^7 : 0 \leq N_h \leq \frac{\pi_h + z}{\mu_h} \right\}$ (4)

From equation (1), we consider the environmental bacteria B_t :

$\frac{dB_t}{dt} = g_t B_t(1 - \frac{B_t}{K_t}) + J_t I_t + \theta_t I_{mt} - e_t B_t$

$\frac{dB_t}{dt} \leq g_t B_t(1 - \frac{B_t}{K_t})$

This is a logistic-type of equation

Thus, B_t is bounded above by carrying capacity K_t: $0 \leq B_t(t) \leq K_t$ for all $t \geq 0$ (5)

Similarly, we consider the vectors population at a given time t, given by

$N_v = B_v + S_v + I_v$

Differentiating equation (1) with respect to t, we have

$\frac{dN_v}{d_t} = \frac{dS_v}{d_t} + \frac{dI_v}{d_t} + \frac{dB_v}{d_t}$ (6)

This means that

$\frac{d_{N_v}}{d_t} = \pi_v + \lambda_{v_1} B_v + \theta_v I_{mt} - \mu_v N_v$

$\frac{d_{N_v}}{d_t} \leq \pi_v + \lambda_{v_1} B_v + \theta_v I_{mt} - \mu_v N_v$

Since $B_v \leq K_v$

Integrating and simplifying both sides of the inequality, we obtain

$N_v(t) \leq \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v} + (N_v(o) - \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v}) e^{-\mu v^t}$

Taking the limit, we have

$\lim_{n \to \infty} sup N_v(t) \leq \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v} : 0 \leq N_v(t) \leq \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v}$

Hence the total vector population N_v is bounded in the region;

$\mathrm{n}_v = \left\{ (S_v, I_v, B_v) \in R_+^3 : 0 \leq N_v \leq \frac{\pi_v}{\mu_v} \right\}$ (7)

Conclusively;

$. \mathrm{n} = \left\{ X = R_+^{11} : 0 \leq N_h \leq \frac{\pi_h + Z}{\mu_h}, 0 \leq B_t \leq K_t, 0 \leq \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v} \right\}$ (8)

Theorem 2

For the system of differential equation (1) Co-infection model to be epidemiologically meaningful, permit the basic data of malaria-typhoid Co-infection model equation (1) given as :

$S_h(0) > 0, I_t(0) > 0, I_m(0) > 0, I_{mt}(0) > 0, R_t(0) > 0, R_m(0) > 0, R_{mt}(0) > 0, B_t(0) > 0, B_v(0) > 0, S_h(0) > 0$ and $I_t(0) > 0$. Then the orbits$(s_h(t), I_t(t), I_m(t), I_{mt}(t), R_t(t), R_m(t), R_{mt}(t), B_t(t), B_v(t), S_v(t), I_v(t))$ of the model with positive basic conditions, will continue to be positive for all time *t > 0.*

Proof.:

Let t_1= sup $\{t_1 > 0: S_h(0) > 0, I_t(0) > 0, I_m(0) > 0, I_{mt}(0) > 0, R_t(0) > 0, R_m(0) > 0, R_{mt}(0) > 0, B_t(0) > 0, B_v(0) > 0, S_h(0) > 0$, $I_t(0) > 0 \}$

Consider the first equation of model (1), given below as

$\frac{ds_h(t)}{dt} = \pi_h + z - [\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}]S - \mu_s \mathrm{S}$

Which can be re-expressed as:

$\frac{d}{dt} = \left[S_h(t) exp \left\{ \mu_s S^t + \int_0^t (\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}) dt \right\} \right] \geq$

$$\pi_h + \mathrm{z}\ exp\left\{\mu_s \mathrm{S}^t + \int_0^t \left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\right\}$$

$$S_h(t_1)exp\left\{\mu t_1 + \int_0^{t_1}\left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\right\} - S_h(0)$$
$$\geq \int_0^{t1} \pi_h + \mathrm{z}\left[exp\left\{\mu_s y + \int_0^y \left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\right\}\right]dy$$

So that

$S_h(t_1) \geq S_h(0)exp\left[-\mu_s t_1 - \int_0^{t_1}\left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\right] + \left[exp\left\{-\mu_s t_1 - \int_0^{t_1}\left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\right\}\right] \times \int_0^{t_1} \pi_h + \mathrm{z}\ \left[exp\{\mu_s y + \int_0^y \left(\lambda_{t_1} + \lambda_{t_2} + \lambda_{m_1} + \lambda_{m_2}\right)dt\}\right]dy > 0$ (9)

Hence, $S_h(t) > 0, \forall\ t > 0$.

Similarly, following same process prove for others in equations (1). We have established positivity for all the state variables in model (1) for all time t.

2.5 Disease-Free Equilibrium Point (EEP) for Co-infection Model

At the DFE, there are no infections in the system, hence:

$E_m^0 = (S_h^0,\ I_t^0, I_m^0, I_{mt}^0, R_t^0, R_m^0, R_{mt}^0, B_t^0, B_v^0, S_v^0, I_v^0) = (\frac{\pi_h}{\mu_h}, 0, 0, 0, 0, 0,\ \ 0, 0, 0, \frac{\pi_v}{\mu_v}, 0$ (10) .

Here, the human population is entirely susceptible, and both environmental reservoirs are free of pathogens.

2.6 Existence of Endemic Equilibrium Point (EEP) for Co-infection Model

We examine the existence of the endemic equilibrium point (EEP) of the model (1) and this describes that the disease is currently prevalent in the population [6]

$$E_{mT}^* = S_h^*(t), I_t^*(t), I_m^*(t), I_{mt}^*(t), R_m^*(t), R_t^*(t), R_{mt}^*(t), B_v^*(t), B_t^*(t), S_v^*(t), I_v^*(t) \quad (11)$$

Solving the right hand side of the equations in (1) for the states variables in terms of the forces of infection (λ_m^*) and (λ_t^*) at the endemic steady state, were all the derivatives are set to zero.

$\left(\frac{ds}{dt} = 0 \text{ and } \frac{dv}{dt} = 0\right)$, substituting the following equation below, to solve the RHS of the equation (1) produce our EEP.

$\lambda_m = \lambda_{m_1} + \lambda_{m_2},\ \lambda_t = \lambda_{t_1} + \lambda_{t_2},\ \lambda_1 = \lambda_{t_3} + \lambda_{t_4}\ ,\ \lambda_2 = \lambda_{m_3} + \lambda_{m_4}$

$\lambda_{t_1} = \frac{\beta_{t_1}(I_t + \mathrm{n}_t I_{mt})}{N_h}$, $\lambda_{t_2} = \frac{\beta_{t_2} B_t}{B_t + K_t}$, $\lambda_{m_1} = \frac{\beta_{m_1}(r I_m + \mathrm{n}_m I_{mt})}{N_h}$, $\lambda_{m_2} = \beta_{m_2}\left(\frac{B_v}{B_v + K_v}\right) I_m$

$\lambda_{t_3} = \frac{\beta_{t_3}(I_T + \mathrm{n}_t I_{mt})}{N_h}$, $\lambda_{t_4} = \frac{\beta_{t_4} B_t}{B_t + K_t}$, $\lambda_{m_3} = \frac{\beta_{m_3}(r I_m + \mathrm{n}_m I_{mt})}{N_h}$, $\lambda_{m_4} = \beta_{m_4}\left(\frac{B_v}{B_v + K_v}\right) I_m$

2.7 Malaria-Only Model

$\frac{dS_h}{dt} = \pi_h + P_m R_m - (\lambda_{m_1} + \lambda_{m_2})S - \mu_s$

$$\frac{dI_m}{dt} = (\lambda_{m_1} + \lambda_{m_2})\, S - (\mu_h + \delta_m + b)\, I_m$$

$$\frac{dR_m}{dt} = b\, I_m - (\mu_h + P_m) R_m \quad (12)$$

$$\frac{dB_v}{dt} = g_v B_v (1 - \frac{B_t}{K_t}) + J_v I_v - e_v B_v$$

$$\frac{dS_v}{dt} = \pi_v + \lambda_{v_1} B_v - (\mu_{Sv} + \lambda_{v_2})\, S_v$$

$$\frac{dI_v}{dt} = \lambda_{v2} S_v - e_v I_v$$

$$N_h(t) = S_h(t) + I_m(t) + R_m(t)\,,\ N_v(t) = S_v(t) + I_v(t) + B_v(t)$$

2.7.1 Positivity and Boundedness

Hence, the same process as proved above in theorem 1 and 2, malaria-only system is both positive invariant and bounded in the biologically feasible region:

$$\Omega = n_h \times n_v \subset R_+^3 \times R_+^3 = \{ (S_h, I_m, R_v, S_v, I_v, B_v) \in R_+^6 : 0 \le N_h \le \frac{\pi_h + z}{\mu_h}; 0 \le N_v \le \frac{\pi_v + \lambda_{v_1} B_v}{\mu_v} \} \quad (13)$$

2.8 Typhoid-Only Model

$$\frac{dS_h}{dt} = \pi_h + \rho_t R_t - [\lambda_{t_1} + \lambda_{t_2}] S - \mu_s S$$
$$\frac{dI_t}{dt} = (\lambda_{t_1} + \lambda_{t_2})\, S - (\mu_h + \delta_T + a)\, I_t$$
$$\frac{dR_t}{dt} = a I_t - (\mu_h + P_t)\, R_t \quad (14)$$
$$\frac{dB_t}{dt} = g_t B_t (1 - \frac{B_t}{K_t}) + J_t I_t - e_t B_t$$

With total human population:

$$N_h(t) = S_h(t) + t(t) + R_v(t) + B_t(\mathrm{t})$$

2.8.1 Positivity and Boundedness

Hence, the same process as proved above in theorem 1 and 2 the typhoid-only system is both positive invariant and bounded in the biologically feasible region:

$$\Omega_t = (S_h, I_t, R_t, B_t) \in R_+^4 : N_h \le \frac{\pi_h + z}{\mu_h}; 0 \le B_t(t) \le K_t \} \text{ for all } t \ge 0 \quad (15)$$

3.0 BASIC REPRODUCTION NUMBERS

The basic reproduction number(R_0) is a key threshold parameter in epidemiology, representing the expected number of secondary infections generated by one infectious individual in a whole susceptible population. If $R_0 < 1$, the disease tends to die out: if $R_0 > 1$, the disease may persist and become endemic.[6]

3.3.1 Malaria Only Reproduction Number(R_0^m)

The basic reproduction number(R_0^m) for malaria Sub- model (12), using Diekmann, et al (2010), and Sokhamoy, Susmita (2020). The computation of R_0 is done using next-generation matrix approach, and the Jacobian *(J_{E0})* matrix of F and V at the DFE evaluated is given as follows: from the equation (12) and differentiating F and V with respect to the state variables, we have the equations.

Mathematically, R_0 is defined as the spectral radius of the matrix product FV^{-1} express as

$R_0 = \rho(\mathrm{FV}^{-1})$ (16)

Where ρ is the spetra radius

$X = [I_m, B_v, I_v]\ ,\ V = V^- - V^+$

$$F = \begin{bmatrix} (\lambda_{m_1} + \lambda_{m_2})S_h \\ g_v B_v \left(1 - \frac{B_v}{K_v}\right) \\ 0 \end{bmatrix} \quad (17)\ ,\ V = \begin{bmatrix} (\mu_h + \delta_t + b)I_m \\ -J_v I_v + e_v I_v \\ \lambda_{v_2} S_v + e_v I_v \end{bmatrix} \tag{18}$$

Differentiating with respect to the state variables, Following the jacobian matrix process we have;

$$F.v^{-1} - \lambda I = \begin{bmatrix} \frac{r.\beta_{m_1}.\pi_h}{\mu_h N_h(\pi_h + \delta_m + b)} - \lambda & 0 & 0 \\ 0 & \frac{g_v}{e_v} - \lambda & \frac{g_v J_v}{e_v^2} \\ 0 & 0 & 0 - \lambda \end{bmatrix} \tag{19}$$

$$\lambda^3 - \left(\frac{\pi_h + \delta_m + bN_h g_v \mu_h}{e_v \mu_h N_h(\pi_h + \delta_m + b)} + \frac{g_v r \beta_{m_1} \pi_h}{e_v \mu_h N_h(\pi_h + \delta_m + b)}\right)\lambda$$

The eigenvalues of the jacobians(J_{E_0}) is given as

$$\lambda = 0\ ,\ \lambda = \frac{g_v}{e_v}\ ,\ \lambda = \frac{r\beta_{m_1}\pi_h}{\mu_h N_h(\pi_h + \delta_{m+} b)}$$

The effective basic reproduction number of the malaria R_0^m sub-model (12) is given by $\rho(\mathrm{FV}^{-1}) = \mathrm{Max}\,[R_0^m]$, with ρ the spectral radius of FV^{-1}

$$R_{.0}^m = \frac{r\beta_{m_1}\pi_h}{\mu_h N_h(\pi_h + \delta_m + b)} \tag{20}$$

3.3.2 Typhoid- Only Reproduction Number(R_0^t)

The basic reproduction number denoted by(R_0^t),using only the infected compartment I_t and B_t from system (14) , following same method in 3.3.1

$X = [I_t, B_t]\ ,\ V = V^- - V^+$

$$F = \begin{bmatrix} (\lambda_{t_1} + \lambda_{t_2})\mathrm{S} \\ g_t B_t(1 - \frac{B_t}{k_t}) + J_t I_t \end{bmatrix}, \tag{21}$$

$$v = \begin{bmatrix} (\mu_h + \delta_t + a)\ I_t \\ e_t B_t \end{bmatrix} \tag{22}$$

$$FV^{-1} - \lambda I = \begin{bmatrix} \frac{\beta_{t_1}\pi_h}{u.k} - \lambda & \frac{-\beta_{t_2}\pi_h}{G} \\ \frac{J_t}{k} & \frac{g_t}{e_t} - \lambda \end{bmatrix} = 0$$

$$\lambda^2 - \frac{(kug_t + \pi_h \beta_{t_1} e_t)\lambda}{e_t uk} + \frac{\pi_h(uJ_t\beta_{t_2}e_t + G\beta_{t_1}g_t)}{kGe_t u}$$

$$\left[\left[\lambda=\frac{1}{2}\frac{1}{kGe_tu}\left[Gkug_t+G\pi_h\beta_{t_1}e_t+\sqrt{4Gku^2J_t\pi_h\beta_{t_2}e_t^2+G^2k^2u^2g_t^2-2G^2ku\pi_h\beta_{t_1}e_tg_t+G^2\pi_h^2\beta_{t_1}^2e_t^2}\right]\right]\right],\left[\left[\lambda=\right.\right.$$

$$\left.\left.\frac{1}{2}\frac{1}{kGe_tu}\left[Gkug_t+G\pi_h\beta_{t_1}e_t-\sqrt{4Gku^2J_t\pi_h\beta_{t_2}e_t^2+G^2k^2u^2g_t^2-2G^2ku\pi_h\beta_{t_1}e_tg_t+G^2\pi_h^2\beta_{t_1}^2e_t^2}\right]\right]\right],$$

$R_0^T=\rho(\mathrm{FV}^{-1})=\mathrm{Max}\,[R_0^T]$, with ρ the spectral radius of FV^{-1}, is been given by

$$R_0^t=\left[\left[\lambda=\frac{1}{2}\frac{1}{kGe_tu}\left[Gkug_t+G\pi_h\beta_{t_1}e_t+\sqrt{4Gku^2J_t\pi_h\beta_{t_2}e_t^2+G^2k^2u^2g_t^2-2G^2ku\pi_h\beta_{t_1}e_tg_t+G^2\pi_h^2\beta_{t_1}^2e_t^2}\right]\right]\right] \quad (23)$$

Where:

$u=\mu_hN_h,\ k=\mu_h+\delta_t+a,\ G=k_te_t\mu_h$

3.3.3 Co-infection Reproduction Number (R_0^{mt})

Following the same method we generated in 3.1.1, the Jacobian matrix of F and V as follows: Infected compartments $I_m, I_t, I_{mt}, I_v, B_t, B_v$ from system (1) is differentiated partially with respect to the state variables follows;

$$\mathrm{X}=|I_m,I_t,I_{mt},I_v,B_t,B_v|$$

$$F.=\begin{bmatrix}(\lambda_{m_1}+\lambda_{m_2})S_h\\(\lambda_{t_1}+\lambda_{t_2})S_h\\(\lambda_{t_3}+\lambda_{t_4})I_m+(\lambda_{m_3}+\lambda_{m_4})I_t\\0\\g_tB_t\left(1-\frac{t}{k_t}\right)\\g_vB_v\left(1-\frac{B_v}{k_v}\right)\end{bmatrix}\ (24),\qquad V=\begin{bmatrix}(\lambda_{t_3}+\lambda_{t_4})I_m+(\mu_h+\delta_m+b)\\(\lambda_{m_3}+\lambda_{m_4})I_m+(\mu_h+\delta_m+a)\\V(f+g+h)I_{mt}+(\mu_h+\psi)I_{mt}\\-\lambda_{v_2}S_v+e_vI_v\\-J_tI_t-\theta_tI_{mt}+e_tB_t\\-J_vI_v-\theta_vI_{mt}+e_vB_v\end{bmatrix}\qquad (25)$$

From our computation, since co-infection contributes new infections, the basic reproduction number for the co-infection of malaria and typhoid fever, represented by R_0^{mt} is given as;

$$\rho(FV^{-1})=\mathrm{Max}\left(\frac{1}{2}\frac{D_1+\sqrt{D_2-2D_3+D_4+2D_5+D_6}}{\mu_hN_hZ_tk_te_t},\ \frac{r\beta_{m_1}\pi_h}{\mu_hN_hZ_m}\right) \quad (26)$$

$Z_t=\mu_h+\delta_t+a$, $Z_m=\mu_h+\delta_m+b$, $D_1=N_hZ_tg_tk_t\mu_h+\beta_{t_2}\pi_hJ_tN_h+\beta_{t_1}\pi_hk_te_t$,

$D_2=N_h^2Z_t^2g_t^2k_t^2\mu_h^2+2J_tN_h^2\pi_hZ_t\beta_{t_2}g_tk_t\mu_h$, $D_3=N_h\pi_h\beta_{t_1}e_tg_tk_t^2N_h$, $D_4=J_t^2N_h^2\pi_h^2Z_t\beta_{t2}^2$,

$D_5=2\left(J_tN_h\pi_h^2\beta_{t_1}\beta_{t_2}e_tk_t\right)$, $D_6=\pi_h^2\beta_{t1}^2e_t^2k_t^2$

3.4 Stability Analysis

3.4.1 Local Stability of the DFE

The disease-free equilibrium E_0 is locally asymptotically stable if:
$R_0^m<1, R_0^t<1$,

Proof

Linearizing the full system E_0, the Jacobian split into block matrices associated with new infection and transition terms. The eigenvalues are negative if and only if the spectral radius of the next-generation-matrix is less than one. This condition is equivalent to require

$R_0^m < 1,\ R_0^t < 1\ , R_0^{mt} < 1$ (27)

3.4.2 Stability Analysis of the Disease-Free Equilibrium for Typhoid fever infection Sub-model

Theorem 4

The Jacobian matrix of (14) at the DFE point $E_t^0 = \left(\frac{\pi_h}{\mu_h},\ 0, 0, 0\ ,0\right)$ is locally asymptotically stable (LAS) if $R_0^t < 1$ and unstable if $R_0^t > 1$

Proof: The Jacobians matrix of the model (15) at the DFE point E_t^0 is given by taking first-order partial derivatives of each equation with respect to each state variable, which give;

$$J_{E_0}^t = \begin{bmatrix} -\mu_h & -\beta_{t_1} & \rho_t & \frac{-\beta_{t_2}\pi_h}{\mu_h k_t} \\ 0 & \beta_{t_1} - (\mu_h + \delta_t + a) & 0 & \frac{\beta_{t_2}\pi_h}{\mu_h k_t} \\ 0 & a & -(\mu_h + \rho_t) & 0 \\ 0 & J_t & 0 & (g_t - e_t) \end{bmatrix} = 0 \quad (28)$$

All the infection will become zero, because we are interested in the linearized dynamics near DFE.

$$J_{E_0}^t = \begin{bmatrix} -\mu_h & 0 & \rho_t & 0 \\ 0 & \beta_{t_1} - (\mu_h + \delta_t + a) & 0 & 0 \\ 0 & a & -(\mu_h + \rho_t) & 0 \\ 0 & 0 & 0 & (g_t - e_t) \end{bmatrix} = 0$$

We observed that the Jacobian matrix is a block lower triangular matrix, meaning it has a special structure:

$$F = \begin{pmatrix} A & B \\ C & D \end{pmatrix}$$

Then the eigenvalues of the Jacobian $J_{E_0}^t$ matrix is given as:

$\lambda_1 = \mu_h < 0,\ \lambda_2 = \beta_{t_1} - (\mu_h + \delta_t + a) < 0$ If and only if $\beta_{t_1} < (\mu_h + \delta_t + a)$
$\lambda_3 = -(\mu_h + \rho_t) < 0\ ,\ \lambda_4 = (g_t - e_t) < 0$ If and only if $g_t < e_t$ (29)

Hence, the system DFE is locally asymptotically stable (LAS) if $\beta_{t_1} < (\mu_h + \delta_t + a)$ and $g_t < e_t$, and become unstable if $\beta_{t_1} > (\mu_h + \delta_t + a)$ and $g_t > e_t$, This complete the proof.

Similarly, stability for R_0^t and R_0^{mt}, is same process.

3.4.3 Global Stability of the Disease Free Equilibrium

Following the method of [7], we analyze the Global stability of the Disease free equilibrium point, is established under the conditions:

To investigate global stability of the uninfected system at the DFE of equation (1) using the next generation matrix (NGM),

Solving equation (1) at the DFE, we have;

$S_h = \frac{\pi_h}{\mu_h}\ ,\ S_v = \frac{\pi_v}{\mu_v}$

(30)

Since there is no infection, we say the system is globally asymptotically stable (GAS).

To investigate global stability of the infected system at the DFE of equation (1) using the next generation matrix (NGM),

The Jacobian matrix (J_{E0}) of the infected compartments [I_t , I_m , I_{mt} , I_v] is given as: $J =$

$$\begin{bmatrix} -(\mu_h + \delta_t + a) & 0 & 0 & 0 \\ 0 & -(\mu_h + \delta_m + b) & 0 & 0 \\ \dot{\lambda_{t_1}} & \dot{\lambda_{m_1}} & -(v(f+g+h) + (\mu_h + \psi)) & 0 \\ 0 & 0 & 0 & -e_v \end{bmatrix} \quad .(31)$$

Theorem 5

The infected subsystem $\frac{dx}{dy} = F(X, 0)$ has a globally asymptotically stable equilibrium; if the infected system's Jacobian matrix (J_{E_0}) has all the off diagonal elements non-negatives and the diagonal elements are all negative. This further implies that the determinant of the Jacobian matrix (J_{E_0}) is positive and the trace negative (Conditions for stability)

$$\left.\begin{array}{l} \det(J_{Eo}) = -(\mu_h + \delta_t + a) - (\mu_h + \delta_t + b) - (\mu_h + \psi + v(f+g+h)) - e_v > 0 \\ \text{(4 negative signs = positive)} \\ \text{Trace } (J_{E0}) = -3\mu_h - \delta_t - a - \delta_m - \psi - v(f+g+h) - e_v < 0 \\ \text{Therefore, the DFE is stable under these thresholds.} \end{array}\right\} \quad (32)$$

Table 1 Baseline Parameters ans variables Values used in the Numerical Experiments

Variables	Values	Soumrce		Parameters	Values	Soumrce
$S_t(t)$	118341511	Assumed		N_h	206553865	WHO (2023)
$S_m(t)$	1800	Assumed		N_v	16800	WHO(2023)
$I_t(t)$	18123413	Assumed		θ_t	0.02	Assumed
$I_m(t)$	19234785	Assumed		θ_v	0.05	Assumed
$I_{mt}(t)$	21045767	Assumed		π_h	20544	World Bank (2022)
$I_v(t)$	500	Assumed		π_v	41000	WHO (2010-2023)
$R_t(t)$	9248875	Assumed		k_t	0.006	Okuonghae & Inyama (2018)
$R_m(t)$	8711883	Assumed		k_v	0.0065	Okuonghae & Inyama (2011)
$R_{mt}(t)$	11847571	Assumed		A	0.008	LHT (2025)
e_t	0.2	Assumed		f	0.5	Assumed
$B_t(t)$	10000	Assumed		g	0.01	Junehyul, *et al* (2017)
$B_v(t)$	10000	Assumed		h	0.8	NACA (2011)
λ_{t_1}	0.0006	Assumed		ψ	2.5	Operah et al., (2022)
λ_{t_2}	0.02	Assumed		J_1	30	Assumed
λ_{m_1}	0.021	WHO, 2024		J_v	25	Assumed
λ_{m_2}	0.002	WHO, 2024		β_t	0.001	Akinyi et al., 2020
λ_{t_3}	0.021	Assumed		β_m	0.003	Okosun et al., 2013
λ_{t_4}	0.1	Assumed		β_{m_1}	0.0025	Okosun et al., 2013
λ_{m_3}	0.01	Assumed		β_{t_1}	0.005	Akinyi et al., 2020
λ_{m_4}	0.02	Assumed		β_{t_2}	0.001	Assumed
λ_{v_1}	0.1	Assumed		β_{m_2}	0.004	Assumed
r	0.3	Okosun et al., (2013)		e_v	0,05	Assumed
μ_h	0.0004	Assumed		μ_m	0.1429	Assumed
δ_t	0.007	Assumed		g_t	0.3	Assumed
δ_m	0.03	Assumed		g_v	0.25	Assumed

4.0 NUMERICAL SIMULATIONS

The system (1) of non-linear ODEs was solved numerically with MATLAB 7.10.0 (R2010a) Using the ode45 (Runge-Kutta 4,5 method). Simulations time is expressed over time (days) with relative tolerance 10^{-6}, the initial conditions for all compartments in equation (1) are given in0.

table1. The λ_{t_2} and λ_{m_2} were used in the simulations because they are already defined as force of infection terms in the ODEs

Experiment 1: To determine the effect of varying shedding rate (J_t) on the Salmonella Typhi reservoir in the environment (B_t) due to infected humans with typhoid (I_t) and co-infected humans
(I_{mt}), with $(J_t = 20, 25, 35, 50)$ over time (days).

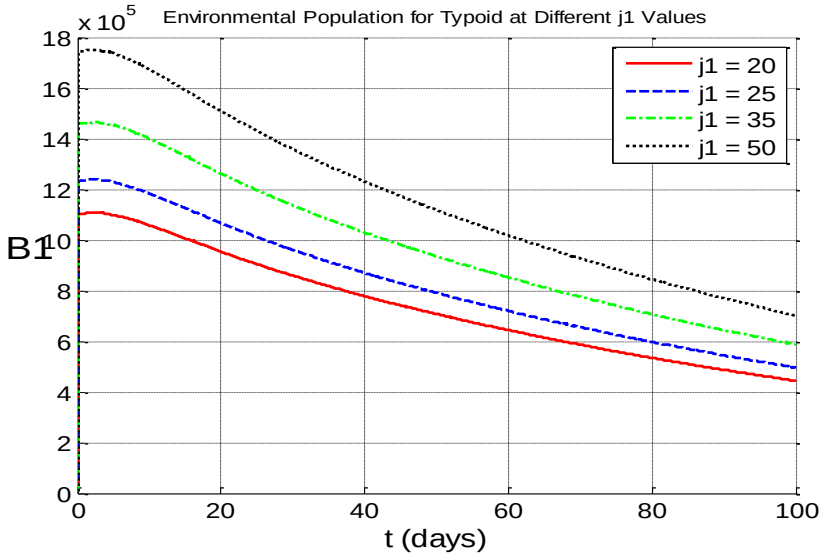

Figure 4.1 The shedding rate (J_t) with typhoid-infected (I_t) and co-infected (I_{mt}) humans over time (days). This graph initially rises from shedding (I_t, I_{mt}) later decline due to recovery and decay, for varying (J_t).

Experiment 2: To determine the effect of varying rate of indirect transmission (λ_{t_2}) on prevalence of only typhoid fever infection on the Infectious class for (I_t)due to interaction of infected individuals with the environment $(\lambda_{t_2} = 0.02, 0.05, 0.08, 0.11)$.

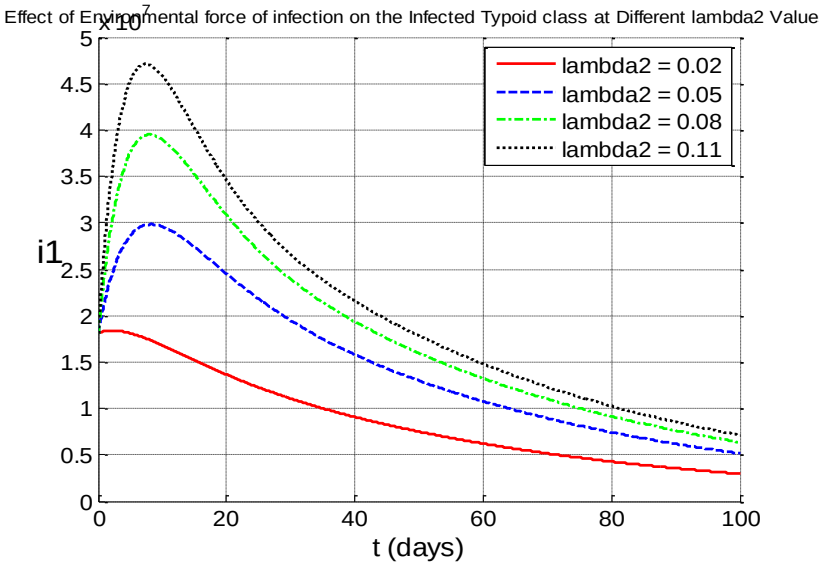

Figure 4.2 Prevalence of Typhoid fever infection (I_t) with varying rate of indirect transmission (λ_{t_2}) from interaction with infected individuals with the environment . This graph effect of varying indirect transmission rate λ_{t_2} on typhoid only infected class (I_t); initial rise followed by decline due to recovery, treatment and decay .

Experiment 3: To determine the effect of varying rate of indirect transmission (λ_{m_2})on prevalence of malaria only infection on the Infectious class of human (I_m)due to interaction of infected individuals with the environment$(\lambda_{m_2} = 0.03, 0.07, 0.11, 0.15)$.

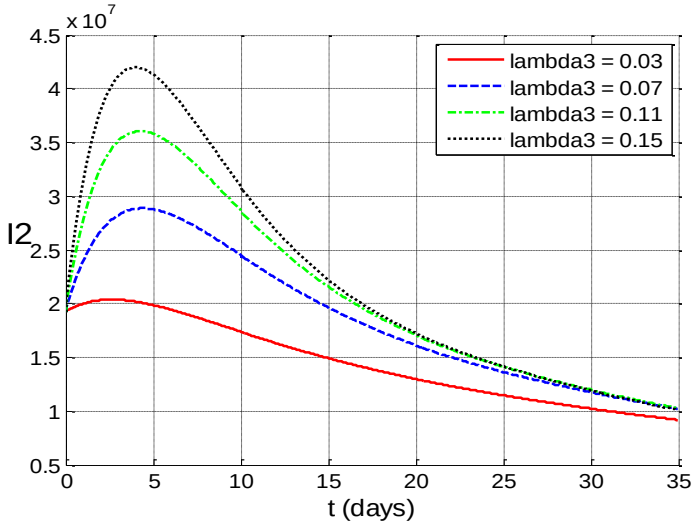

Figure 4.3 Prevalence of Malaria infection (I_m) with varying rate of indirect transmission (λ_{m_2}). This graph varying λ_{m_2} show an initial rise in prevalence due to strong environmental interaction, but later decline as recovery and control effects dorminate.

Experiment 4: To determine the effect of varying shedding rate (J_v) of the pathogens and breeding vectors reservoir in the environment (B_v) due to infected humans with malaria (I_m) and co-infected humans as a result of interaction $(J_v = 30,\ 45,\ 55,\ 65)$

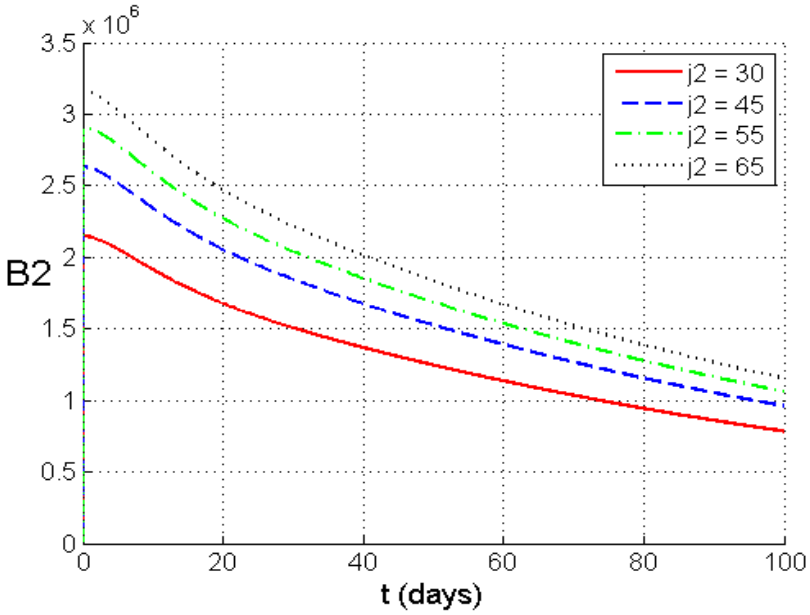

Figure 4.4 Concentration of the pathogens and breeding vectors reservoir in the environment (B_v) with malaria (I_m) and co-infected (I_{mt}) humans . This graph varying J_v increases B_v as vectors bite infected humans $(I_m,\ I_{mt})$, with transmission later decline through decay and control.

DISCUSSION

The findings from our model highlight how both direct and indirect transmission pathways, together with environmental reservoirs, influence disease spread. We develop a deterministic model to study this research and investigated the disease-free and endemic equilibria, derived basic reproduction number, and performed stability analysis to identify the conditions for disease persistence. Numerical solution shows that environmental factors, such as salmonella typhi bacteria and breeding vectors/pathogen shedding, significantly affect infection prevalence, emphasizing the critical role of environmental drivers in shaping disease dynamics.
However, the model demonstrates that strengthening community health interventions by improving access to clean water, improve sanitation, public health campaigns, along with vector control could help reduce disease burden. This reflects real-world trends where eradication remains elusive, but continued efforts produce measurable declines in prevalence and mortality.

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