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Global Stability Analysis of a Malaria–Typhoid Fever Co-infection Model

Abah R.T., *Agbo, C.E., & Ibrahim, A.
Department of Mathematics University of Abuja, Abuja, Nigeria

***Corresponding author email**:agbo.ene@uniabuja.edu.ng

Abstract
Typhoid fever and malaria are two serious infectious diseases that are common in sub-Saharan Africa, and co-infection poses a serious threat to public health. Designing successful control measures requires an understanding of the dynamics of these illnesses. We created a deterministic compartmental model that divided the human population into seven groups: susceptible people, typhoid-only infected people, malaria-only infected people, co-infected people, recovered people, susceptible mosquitoes, and infected mosquitoes. We defined parameters for the global stability of the disease-free equilibria and determined the fundamental reproduction numbers for typhoid and malaria using the next-generation matrix technique. The global stability results were validated using Lyapunov functions and LaSalle's invariance principle. When both fundamental reproduction numbers are less than unity, the disease-free equilibrium is asymptotically stable worldwide.Numerical simulations highlight the threshold parameters that drive co-infection persistence and the combined impact of malaria–typhoid interventions. This study provides a theoretical basis for controlling malaria and typhoid co-infections through integrated interventions. The analytical thresholds derived can guide policymakers in optimizing combined control strategies in endemic regions such as Nigeria

Keywords: Global Stability, Analysis, Malaria Typhoid Fever, Co-infection, Model

Introduction

In sub-Saharan Africa and other tropical countries, typhoid fever and malaria continue to be two of the most serious infectious diseases that affect people (WHO, 2021; Crump & Mintz, 2010). A major source of morbidity and mortality, malaria is caused by Plasmodium parasites and is spread via the bite of infected Anopheles mosquitoes, especially in pregnant women and children under five (Snow et al., 2005). Salmonella enterica serovar Typhi is the causative agent of typhoid fever, a systemic bacterial infection that is mainly spread by contaminated food and water through the fecal-oral route (Buckle et al., 2010). According to Pruss-ustum et al. (2019), both diseases flourish in settings with low sanitation, restricted access to clean water, and inadequate healthcare infrastructure.The co-occurrence of malaria and typhoid fever within the same population and sometimes within the same individual is increasingly recognized as a significant public health challenge (Takem et al., 2014; Kang et al., 2020). Co-infection complicates clinical diagnosis due to overlapping symptoms such as fever, headache, and malaise, which can lead to misdiagnosis and inappropriate treatment (Nguyen et al., 2021). Furthermore, co-infection can exacerbate disease severity, prolong recovery times, and increase mortality risk (Adewuyi et al., 2018). Despite these challenges, most control programs and epidemiological studies have focused on the diseases individually, potentially underestimating the compounded burden of co-infection (Liu et al., 2017).

Mathematical modeling provides a powerful tool for studying infectious disease dynamics, including co-infection scenarios (Abu-Raddad et al., 2006). While numerous models have been developed for malaria and typhoid independently, relatively few have examined their joint transmission dynamics especially in a framework that integrates malaria's vector-host interactions with typhoid's human-to-human waterborne transmission (Mutua et al., 2015). Co-infection modeling can reveal how interactions between pathogens alter transmission thresholds, persistence conditions, and the effectiveness of combined control measures (Abah et al., 2023). In this study, we formulated a deterministic compartmental model dividing the human population into seven epidemiological states

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alongside susceptible and infected mosquito vectors. Using the next-generation matrix method, we derived the basic reproduction numbers for malaria and typhoid. Using Lyapunov functions and LaSalle's invariance principle, the global stability of the endemic and disease-free equilibria was thoroughly examined (Korobeinikov, 2006). Our results underscore the importance of integrated disease management strategies. By identifying the threshold conditions for eradication or persistence, the model provides a framework for guiding public health interventions in high-burden countries such as Nigeria, where malaria–typhoid co-infection is common (Okolo et al., 2023; Akinyemi et al., 2023).

Malaria and typhoid fever are endemic diseases that contribute substantially to the global burden of infectious diseases, particularly in sub-Saharan Africa and parts of Asia (WHO, 2021). Both share overlapping geographic distributions and risk factors, such as inadequate sanitation, unsafe water sources, and limited healthcare infrastructure factors that often facilitate co-infection (Crump & Mintz, 2010). Reported prevalence rates for malaria–typhoid co-infection in endemic regions range from 5% to over 20%, complicating diagnosis and management (Snow et al., 2005). Mathematical models have been pivotal in understanding the transmission dynamics of individual infectious diseases. For malaria, vector-host models have informed control strategies such as insecticide-treated nets and indoor residual spraying (Buckle et al., 2010). Typhoid fever models have focused on waterborne transmission and the impact of vaccination and sanitation improvements (Pruss-ustun et al., 2019). However, co-infection modeling remains comparatively underexplored. The benefits of co-infection modeling are evident in studies on other disease pairings, such as HIV–tuberculosis, which have highlighted synergistic effects on transmission and disease progression (Takem et al., 2019). Recently, Abah et al., (2023) developed models for malaria–typhoid and other infectious diseases, emphasizing integrated control strategies and the use of stability analysis for policymaking.The subsequent elements are esssential parts of the mathematical modeling about the co-infection dynamics of typhoid and malaria.

Mushayabasa et al. (2014) introduces a deterministic mathematical framework for scrutinizing the dynamics associated with malaria and typhoid co-infection.This analysis first evaluates the transmission dynamics separately before combining the two diseases, showing that a typhoid epidemic in areas where malaria is endemic can result in a greater number of clinically symptomatic people with dual infections than those with single infections. The model illustrates the intricate relationships between the two diseases within the population by showing a backward bifurcation phenomena. Mutua et al. (2015) develop novel mathematical frameworks to clarify the co-infection dynamics of malaria and typhoid, accentuating distinctive features and interconnections between the two diseases. The research emphasizes the critical role of typhoid carriers in co-epidemics and demonstrates that effective simultaneous preventive measures can reduce the co-infection basic reproduction number to below unity, facilitating disease eradication. The models highlight the imperative for extensive research to effectively manage these infectious diseases in tropical developing countries. A mathematical model is proposed to analyze the dynamics of malaria and typhoid co-infection, focusing on the consequences of erroneously diagnosing typhoid as malaria and subsequently administering anti-malarial treatments. The findings reveal that such misdiagnosis significantly exacerbates the endemicity of typhoid and intensifies malaria infections, underscoring the critical need for accurate diagnostic methodologies as articulated by Akinyi et al. (2015). A mathematical framework addressing the dynamics of two infectious diseases within a population experiencing co-infection scenarios is posited. It suggests that infection with one disease increases susceptibility to another, while recovery provides partial immunity. The model utilizes a coupled system of differential equations to investigate these dynamics, yielding insights into the interactions among the diseases (Gutiérrez-Jara et al., 2019). A mathematical model specifically designed for the dynamics of malaria-dysentery co-infection is also presented. It examines the interplay between malaria and dysentery in relation to prevention strategies (Okosun, 2020). Notwithstanding, additional research is necessary to fully comprehend the dynamics of malaria and typhoid co-infection; hence, the main goal of this research is to develop and evaluate a mathematical model that clarifies the co-infection dynamics of malaria and typhoid fever in a human population. This investigation aims to explore the fundamental characteristics of the developed model, which includes an evaluation of local stability. A comprehensive analysis of the results is provided, clarifying the dynamics of malaria-typhoid transmission.

Global stability analysis using Lyapunov functions and LaSalle's invariance principle is a well-established approach in epidemiological modeling, offering rigorous guarantees for the global attractivity of equilibria (Korobeinikov, 2006). These techniques have been applied successfully to vector-borne and waterborne disease models, demonstrating their utility in deriving robust eradication conditions. In the Nigerian context, where malaria–typhoid co-infection prevalence is high, integrating both diseases into a unified model with global stability analysis provides

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a valuable theoretical framework for designing combined interventions and optimizing limited healthcare resources (Cheng et al., 2020).

Material and Methods

To elucidate the dynamics nature, we categorized the population of this study into seven compartments, namely: Susceptible humans against malaria and typhoid S_H, Infected human with typhoid fever only I_T, individual co-infected with Typhoid and malaria(I_C), infected human with malaria only(I_{HM}), recovered individual(R_H), susceptible mosquitoes(S_M), infected mosquitoes(I_M). Figure1 shows the schematic representation of the malaria and typhoid co-infection model

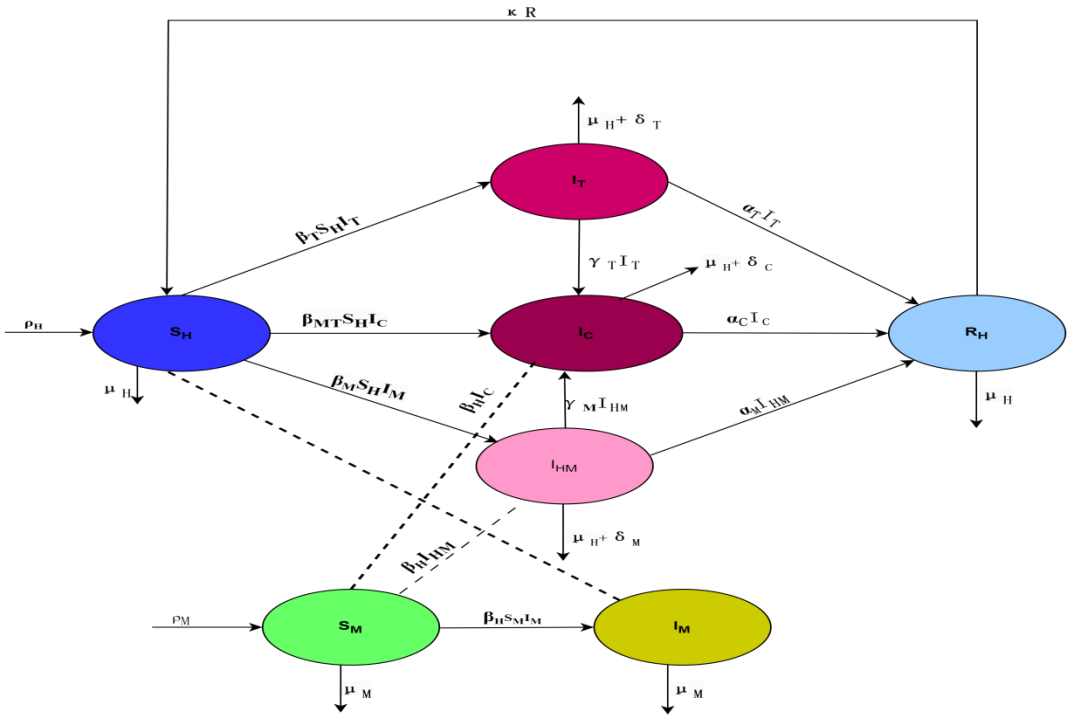

Figure 1: Malaria and Typoid co-infection model

Model Equation

$$\frac{dS_H}{dt} = \rho_H - \beta_T S_H I_T - \beta_{MT} S_H I_C - \beta_M S_H I_M - \mu_H S_H + \kappa R_H \quad (1)$$

$$\frac{dI_T}{dt} = \beta_T S_H I_T - \alpha_T I_T - \gamma_T I_T - (\mu_H + \delta_T) I_T \quad (2)$$

$$\frac{dI_C}{dt} = \beta_{MT} S_H I_C + \alpha_T I_T + \alpha_M I_{HM} - \alpha_C I_C - (\mu_H + \delta_c) I_C \quad (3)$$

$$\frac{dI_{HM}}{dt} = \beta_M S_H I_M - \alpha_M I_{HM} - \gamma_M I_{HM} - (\mu_H + \delta_M) I_{HM} \quad (4)$$

$$\frac{dR_H}{dt} = \alpha_T I_T + \alpha_C I_C + \alpha_M I_{HM} - (\mu_H + \kappa) R_H \quad (5)$$

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$$\frac{dS_M}{dt} = \rho_M - \beta_H S_M (I_{HM} + I_C) - \mu_M S_M \quad (6)$$

$$\frac{dI_M}{dt} = \beta_H S_M (I_{HM} + I_C) - \mu_M I_M \quad (7)$$

With initial condition

$$S_H(0) = S_{H0} > 0,\ I_T(0) = I_{T0} > 0,\ I_C(0) = I_{C0} > 0,\ I_{HM}(0) = I_{HM0} > 0,$$
$$R_H(0) = R_{H0} > 0,\ S_M(0) = S_{M0} > 0, I_M(0) = I_{M0} > 0 \quad (8)$$

The total population is human and mosquito is given as:

$$N_H = S_H(t) + I_T(t) + I_C(t) + I_{HM}(t) + R_H(t) \quad (9)$$

$$N_M = S_M(t) + I_M(t) \quad (10)$$

Model parameter and variable description

Table1: Variables

Variables	Description
S_H	Susceptible human
I_T	Infected individuals with Typhoid only
I_C	Individuals co-infected with Typhoid and Malaria
I_{HN}	Individuals infected with Malaria only
R_H	Recovered individuals
S_M	Susceptible mosquitoes
I_M	Infected mosquitoes (transmitting Malaria)

Table 2: Parameters

Parameter	Description
ρ_H	Recruitment rate of Human
ρ_M	Recruitment rate of Mosquito
μ_H	Natural death rate of humans
μ_M	Natural death rate of mosquitoes
β_T	Transmission rate of Typhoid
β_M	Transmission rate of Malaria
β_{TM}	Transmission rate of co-infection
β_H	Mosquito infection rate from humans
γ_T	Typhoid Interaction term for co-infection dynamics
γ_M	Malaria Interaction term for co-infection dynamics
α_T	Recovery rate from Typhoid
α_C	Recovery rate from co-infection
α_M	Recovery rate from Malaria

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δ_C	Death rate due to Co-infection
δ_T	Death rate due to Typhoid infection
δ_M	Death rate due to Malaria infection
κ	Loss of immunity and susceptibility to malaria
N_{H^H}	Total population of Human
N_M	Total population of Mosquitoes

Basic Properties of the Model

Invariant Region

The invariant region is used to determine where the model solution is bounded. The model equation (1) to (7) is divided into two groups: the mosquito population and the human population. The total human population is represented by $N_H = S_H(t) + I_T(t) + I_C(t) + I_{HM}(t) + R_H(t)$. The total mosquito population is $N_M = S_M + I_M$

Theorem:

Let $\omega_H = \left\{ S_H, I_T, I_C, I_{HM}, R_H \in \Re_+^5 : N_{Hh}(t) \le \frac{\rho_H}{\mu_H} \right\}$ and $\omega_M = \left\{ S_M, I_M \in \Re_+^2 : N_M(t) \le \frac{\rho_M}{\mu_M} \right\}$ so that $\omega = \omega_H * \omega_M \subset \Re^5 * \Re_+^2$. The biologically feasible region of the Ω of the model equation (1) to (7) is positively invariant.

Proof:

From the total human population represented by $N_H = S_H(t) + I_T(t) + I_C(t) + I_{HM}(t) + R_H(t)$,

It is clear that,

$$\frac{dN_H}{dt} \le \rho_H - \mu_H N_H \tag{11}$$

Applying separation of and integrating, that is,

$$\int \frac{1}{\rho_H - \mu_H N_H} dN_H \le \int dt \tag{12}$$

Integrating (12), we have

$$\rho_H - \mu_H N \le D_1 \ell^{-\mu_h t} \tag{13}$$

At $t = 0,\ N_H(t) = N_0$ equation (13) turns

$$\rho_H - \mu_H N = D_1$$

Therefore, equation (13) becomes

$$\rho_H - \mu_H N \le (\rho_H - \mu_H N)\ell^{-\mu_h t}$$

$$N_h \le \frac{\rho_H}{\mu_H} - \frac{(\rho_H - \mu_H N)\ell^{-\mu_h t}}{\mu_H} \tag{14}$$

If $t \to \infty$ equation (14) becomes

$$N_H(t) \le \frac{\rho_H}{\mu_H} \tag{15}$$

This implies that $0 \le N_H(t) \le \frac{\rho_H}{\mu_H}$.

As a result, the model's feasible solution set for the host population stays inside the region and is provided by

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$$\omega_H = \left\{ S_H, I_T, I_C, I_{HM}, R_H \in \Re_+^5 : N_{Hh}(t) \le \frac{\rho_H}{\mu_H} \right\}.$$

Similarly the equation given by the total mosquito population in the model equation (1) to (7), that is

$$\frac{dN_M}{dt} \le \rho_M - \mu_M N_M \tag{16}$$

By solving the equation (16), we have

$$N_M(t) \le \frac{\rho_M}{\mu_M} \tag{17}$$

From equation (17), if $t \to \infty$, $N_M(t) \to \frac{\omega_M}{\mu_M}$

Thus, for the total mosquitoes population the feasible solution set of the model remain within the region and is given by $\omega_M = \left\{ S_M, I_M \in \Re_+^2 : 0 \le N_M(t) \le \frac{\rho_M}{\mu_M} \right\}$.

Therefore, the feasible solution set for the model equations (1) to (7) together with initial conditions given by $\omega = \omega_H * \omega_M$, is a positive invariant and hence it is biologically meaningful and well posed in the domain ω.

Disease Free Equilibrium Point

A steady state solution in which there is no disease is known as the "disease free equilibrium point". A person's body is virus-free at this point. Setting the right sides of model differential equations (1) through (7) to zero will yield the disease-free equilibrium of these equations. That is,

$$\frac{dS_H}{dt} = \frac{dI_T}{dt} = \frac{dI_C}{dt} = \frac{dI_{HM}}{dt} = \frac{dR_H}{dt} = \frac{dS_M}{dt} = \frac{dI_M}{dt} = 0 \tag{18}$$

In the absence of the disease

$$N_H = S_H(t) = I_T(t) = I_C(t) = I_{HM}(t) = R_H(t) = S_M(t) = I_M(t) = 0.$$

Then, equation (18) reduces to

$$\rho_H - \mu_H S_H = 0 \tag{19}$$

$$\rho_M - \mu_M S_M = 0 \tag{20}$$

Then, from equation (19) and (20), we have

$$S_H = \frac{\rho_H}{\mu_H}, \quad S_M = \frac{\rho_M}{\mu_M}$$

Hence, the disease free equilibrium points E_0 are:

$$E_0 = (S_H, I_T, I_C, I_{HM}, R_H, S_M, I_M) = \left(\frac{\rho_H}{\mu_H}, 0, 0, 0, 0, \frac{\rho_M}{\mu_M}, 0 \right)$$

This suggests that the disease will die out in the population.

Sub Model of Malaria

$$\frac{dS_H}{dt} = \rho_H - \beta_M S_H I_M - \mu_H S_H + \kappa R_H \tag{21}$$

$$\frac{dI_{HM}}{dt} = \beta_M S_H I_M - \alpha_M I_{HM} - \gamma_M I_{HM} - (\mu_H + \delta_M) I_{HM} \tag{22}$$

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$$\frac{dR_H}{dt} = \alpha_M I_{HM} - (\mu_H + \kappa)R_H \quad (23)$$

$$\frac{dS_M}{dt} = \rho_M - \beta_H S_M I_{HM} - \mu_M S_M \quad (24)$$

$$\frac{dI_M}{dt} = \beta_H S_M I_{HM} - \mu_M I_M \quad (25)$$

With initial condition

$$S_H(0) = S_{H0} > 0,\ I_{HM}(0) = I_{HM0} > 0,\ R_H(0) = R_{H0} > 0,\ S_M(0) = S_{M0} > 0, I_M(0) = I_{M0} > 0$$

Results

Basic Reproduction Number R_0 of Sub Model of Malaria

An epidemic's course is determined by the basic reproduction number, a key idea in mathematical biology. According to Diekmann et al., (2020), the basic reproduction number is the number of secondary cases that a typical infected individual is expected to cause in a population that is completely susceptible.

It is a highly valuable threshold parameter that characterises mathematical problems related to infectious diseases. If it is less than one, this indicates that, on average, one infected individual generates fewer than one new infected individual over the infectious period, leading to the potential eradication of the illness. If it exceeds unity, then each infected individual generates, on average, more than one new infection, resulting in the disease proliferating within the community. Consequently, we calculate the fundamental reproduction number of the model equations (1) to (7) utilising the next generation method (Agbo et al., 2024). $R_0 = \rho(FV^{-1})$ Where, $\rho(A)$ is the spectral radius of matrix A(or the maximum modulus of the eigenvalues of A).

$F = \left[\frac{\partial F_i}{\partial x_j}(E_0)\right]$ and $V = \left[\frac{\partial V_i}{\partial x_j}(E_0)\right]$, with $1 \le i,\ j \le m$, where m represents the infected classes.

F_i and V_i are the number of new infections that arise in the compartment i and the number of people who enter and exit the compartment i by any means, respectively. The corresponding matrices at disease-fee equilibrium E_0 were obtained using the linearization method. Taking into account the main infection:

$$F_i = \frac{B_M I_M \mu_H S_H}{\rho_H} \quad (26)$$

Differentiating (26) partial regarding the infection class and replacing the values S_H at the disease free equilibrium, we obtained

$$F_i = \frac{B_M}{\rho_H} \quad (27)$$

Similarly, considering the secondary infected class:

$$V_i = (\mu_H + \gamma_M + \delta_M)I_{HM} \quad (28)$$

Differentiating (28) partial with respect to infection class, we have

$$V_i = (\mu_H + \gamma_M + \delta_M) \quad (29)$$

Taking the inverse of (29), we have

$$V_i^{-1} = \frac{1}{(\mu_H + \gamma_M + \delta_M)} \quad (30)$$

Then, we have

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$$F_i V_i^{-1} = \frac{\beta_M}{(\mu_H + \gamma_M + \delta_M)}$$

Hence, the basic reproduction number is

$$R_0^H = \frac{\beta_M}{(\mu_H + \gamma_M + \delta_M)} \tag{31}$$

Also, considering the primary and the secondary infection classes from the mosquito compartments, that is

$$F_i = \frac{B_H I_{HM} \mu_H S_H}{\rho_H} \tag{32}$$

$$V_i = (\mu_H + \gamma_M + \delta_M) I_{HM} \tag{33}$$

Differentiating (32) and (33) partial with respect to infection classes and substituting the value S_M for (32) at the disease free equilibrium point, and taking the inverse of (33), we obtained

$$F_i V^{-1} = \frac{B_H}{\mu_M} \tag{34}$$

Equation (34) is the basic reproduction number mosquito compartments that is,

$$R_0^Q = \frac{B_H}{\mu_M} \tag{35}$$

Therefore, the reproduction number of sub model of malaria is,

$$R_0^M = R_0^H \times R_0^Q = \frac{B_H B_M}{(\mu_H + \gamma_M + \delta_M)\mu_M} \tag{36}$$

Sub Model of Typhoid

$$\left\{\begin{array}{l} \dfrac{dS_H}{dt} = \rho_H - \beta_T S_H I_T - \mu_H S_H + \kappa R_H \\ \dfrac{dI_T}{dt} = \beta_T S_H I_T - \gamma_T I_T - (\mu_H + \delta_T) I_T \\ \dfrac{dR_H}{dt} = \alpha_T I_T + \alpha_C I_C + \alpha_M I_{HM} - (\mu_H + \kappa) R_H \\ \text{With initial condition} \\ S_H(0) = S_{H0} > 0,\ I_T(0) = I_{T0} > 0,\ R_H(0) = R_{H0} > 0 \end{array}\right\} \tag{37}$$

Basic Reproduction Number R_0 of Sub Model of Typhoid

Considering the primary and secondary infection in model system (37), we have

$$F_i = \frac{B_T I_T S_H}{N_H} \tag{38}$$

$$V_i = (\gamma_T + \mu_H + \delta_T) I_T \tag{39}$$

Differentiating (38) and (39) partial with respect to infection classes and substituting the value S_H for (38) at the disease free equilibrium point and take the inverse of (39), we obtained

$$F_i V^1 = \frac{B_T}{(\gamma_T + \mu_H + \delta_T)}$$

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(40)

Equation (40) is the basic reproduction number sub model of Typhoid, that is

$$R_0^T = \frac{B_T}{(\gamma_T + \mu_H + \delta_T)} \tag{41}$$

Therefore, the basic reproduction number R_0 for the co- infection is

$$R_0 = R_0^H \times R_0^T = \frac{B_M B_H B_T}{(\gamma_T + \mu_H + \delta_T)(\mu_H + \gamma_M + \delta_M)\mu_M} \tag{42}$$

Global Stability of Disease-free Equilibrium of the Malaria-typoid co-infection

Theorem 2

The disease-free equilibrium $E_0 = \left(\frac{\rho_H}{\mu_H}, 0, 0, 0, 0, \frac{\rho_M}{\mu_M}, 0\right)$ is globally asymptotically stable if $R_0 \leq 1$.

Proof

Consider the Lyapunov function variables $E_0\left(S_H, I_T, I_C, I_{HM}, R_H, S_M, I_M\right)$ according to the approach in (Agbo et al., 2025) be

$$E_0(S_H, I_T, I_C, I_{HM}, R_H, S_M, I_M) = (S_H - S_{H0} - S_{H0} In \frac{S_H}{S_{H0}}) + I_T + I_C + I_{HM} + R_H + (S_M - S_{M0} - S_{M0} In \frac{S_M}{S_{M0}}) + I_M \tag{43}$$

$$E_0 = (S_H - S_{H0} - S_{H0} In \frac{S_H}{S_{H0}}) + I'_T + I'_C + I'_{HM} + R'_H + (S_M - S_{M0} - S_{M0} In \frac{S_M}{S_{M0}}) + I'_M \tag{44}$$

Differentiating $E_0(S_H, I_T, I_C, I_{HM}, R_H, S_M, I_M)$ with respect to time gives

$$E_0 = (1 - \frac{S_H}{S_0})S' + I'_T + I'_C + I'_{HM} + R'_H + (1 - \frac{S_M}{S_{M0}})S'_M + I'_M \tag{45}$$

Substituting equation (1-7) into (44) and $S_{H0} = \frac{\rho_H}{\mu_H}$ and $S_{M0} = \frac{\rho_M}{\mu_M}$ to gives

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$$= (1-\frac{S_{H0}}{S_H})(\rho_H - \beta_T S_H I_T - \beta_{MT} S_H I_C - \beta_M S_H I_M - \mu_H S_H + \kappa R_H) + \beta_T S_H I_T -$$
$$\alpha_T I_T - \gamma_T I_T - (\mu_H + \delta_T) I_T + \beta_{MT} S_H I_C + \alpha_T I_T - \alpha_M I_{HM} - \alpha_C I_C -$$
$$(\mu_H + \delta_C) I_C + \beta_M S_H I_M - \alpha_M I_{HM} - \gamma_M I_{HM} - (\mu_H + \delta_M) I_{HM} +$$
$$\alpha_T I_T + \alpha_C I_C + \alpha_M I_{HM} - (\mu_H + \kappa) R_H + (1-\frac{S_{M0}}{S_M})(\rho_M - \beta_H S_M (I_{HM} + I_C)$$
$$- \mu_M S_M) + \beta_H S_M (I_{HM} + I_C) - \mu_M I_M \quad (46)$$

$$= \rho_H - \beta_T S_H I_T - \beta_{MT} S_H I_C - \beta_M S_H I_M - \mu_H S_H + \kappa R_H - \rho_H \frac{S_{H0}}{S_H} +$$
$$\beta_T S_H I_T \frac{S_{H0}}{S_H} + \beta_{MT} S_H I_C \frac{S_{H0}}{S_H} + \beta_M S_H I_M \frac{S_{H0}}{S_H} + \mu_H S_H \frac{S_{H0}}{S_H} - \kappa R_H \frac{S_{H0}}{S_H} +$$
$$\mu_H (S_H, I_T, I_{HM}, I_C, R_H) + \mu_M (S_M, I_M) + \rho_M - \rho_M \frac{S_{M0}}{S_M} +$$
$$\beta_H S_M \frac{S_{M0}}{S_M} (I_{HM} + I_C) - \mu_M I_M \quad (47)$$

simplifying

$$= \rho_H - \rho_H \frac{S_{H0}}{S_H} + \beta_T I_T S_{H0} + \beta_{MT} I_C S_{H0} + \beta_M I_M S_{H0} + \mu_H S_{H0} -$$
$$\kappa R_H \frac{S_{H0}}{S_H} + \rho_M - \rho_M \frac{S_{M0}}{S_M} + \beta_H S_M (I_{HM} + I_C) + \beta_H S_{M0} (I_{HM} + I_C) -$$
$$\mu_H (S_H, I_T, I_{HM}, I_C, R_H) - \mu_M (S_M, I_M) - \mu_M I_M \quad (48)$$
$$= \rho_H (1-\frac{S_{H0}}{S_H}) + (\beta_T I_T + \beta_{MT} I_C + \beta_M I_M + \mu_H) S_{H0} - \kappa R_H \frac{S_{H0}}{S_H} +$$
$$\rho_M (1-\frac{S_{M0}}{S_M}) - \mu_H (S_H, I_T, I_{HM}, I_C, R_H) - \mu_M (S_M, I_M) \quad (49)$$

From equation (1-7) $(\beta_T I_T + \beta_{MT} I_C + \beta_M I_M + \mu_H) S_{H0}$, $S_{H0} = \frac{\rho_H}{\mu_H}$ and $S_{M0} = \frac{\rho_M}{\mu_M}$ are non-negatives hence

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$$E_0 \le \rho_H(1-\frac{S_{H0}}{S_H}) + (\beta_T I_T + \beta_{MT} I_C + \beta_M I_M + \mu_H)S_{H0} - \kappa R_H \frac{S_{H0}}{S_H} + \rho_M(1-\frac{S_{M0}}{S_M}) - \mu_H(S_H, I_T, I_{HM}, I_C, R_H) - \mu_M(S_M, I_M) \quad (50)$$

By the inequality of arithmetic and geometric means

$$\rho_H(1-\frac{S_{H0}}{S_H}) + (\beta_T I_T + \beta_{MT} I_C + \beta_M I_M + \mu_H)S_{H0} - \kappa R_H \frac{S_{H0}}{S_H} + \rho_M(1-\frac{S_{M0}}{S_M}) - \mu_H(S_H, I_T, I_{HM}, I_C, R_H) - \mu_M(S_M, I_M) \le 0$$

This proved that E_0 is the lyapunov function of $E_0^{'}$=0 which implies that $I_T(t) = I_C(t) = I_{HM}(t) = R_H(t) = I_M(t) = 0$. Therefore, it follows that the larges invariant set in $(S_H + I_T + I_C + I_{HM} + R_H + S_M + I_M) : E'_0 = 0$ is $E_0 = \left(\frac{\rho_H}{\mu_H}, 0, 0, 0, 0, \frac{\rho_M}{\mu_M}, 0\right)$

Therefore, by Lasalle's invariance principle the disease free equilibrium is globally asymptotically stable.

Numerical Simulation Results

The global stability of the disease-free equilibrium will be quantitatively demonstrated in this section. The appropriate R programming instructions and packages were used to run the simulations. Table 3 displays the source and parameter values used, as well as the initial condition of $(S_H, I_T, I_C, I_{HM}, R_H, S_M, I_M) = (800, 345, 200, 550, 56, 1000, 500)$

Table 3: Parameters Values

Parameter	Values/day	Reference
ρ_H	100	(Okosun & Makinde , 2011)
ρ_M	1000	(Okosun & Makinde 2011)
μ_H	0.0236	estimated
μ_M	0.5	estimated
β_T	0.0035	estimated
β_M	0.0696	(Chitnis et al., 2006)
β_{TM}	0.15	(Mushayabasa et al., 2014)
β_H	0.059	(Okosun and Makinde 2011)
γ_T , γ_M	0.1	(Mushayabasa et al., 2014)
α_T	0.05	(Bhan et al., 2005)
α_C	0.2	(Mushayabasa et al., 2014)
α_M	0.03	(Bhan et al., 2005)
δ_C , δ_T , δ_M	0.01	(Mushayabasa et al., 2014)

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κ	0.0013	(Okosun & Makinde 2011)

The graph illustrates the simulation of human compartment over 100 days.

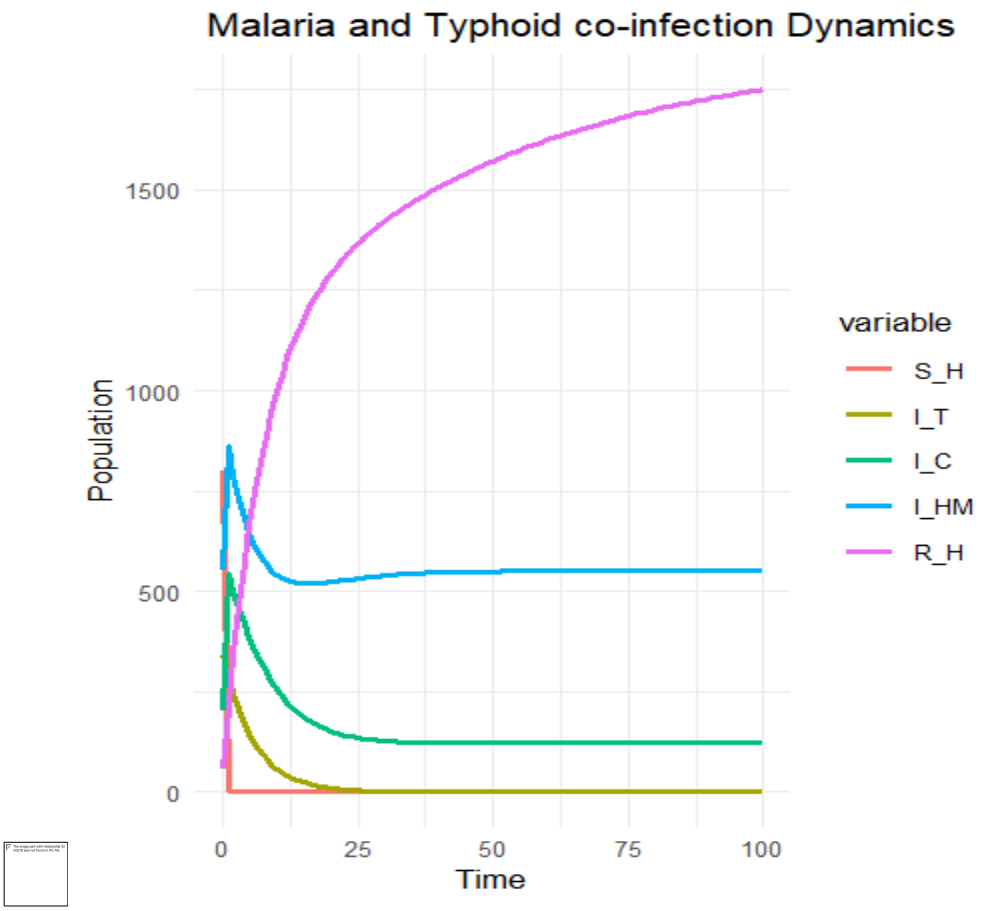

Figure2: Human Compartment of Malaria and Typhoid Co-infection Dynamics at the Disease-free Equilibrium

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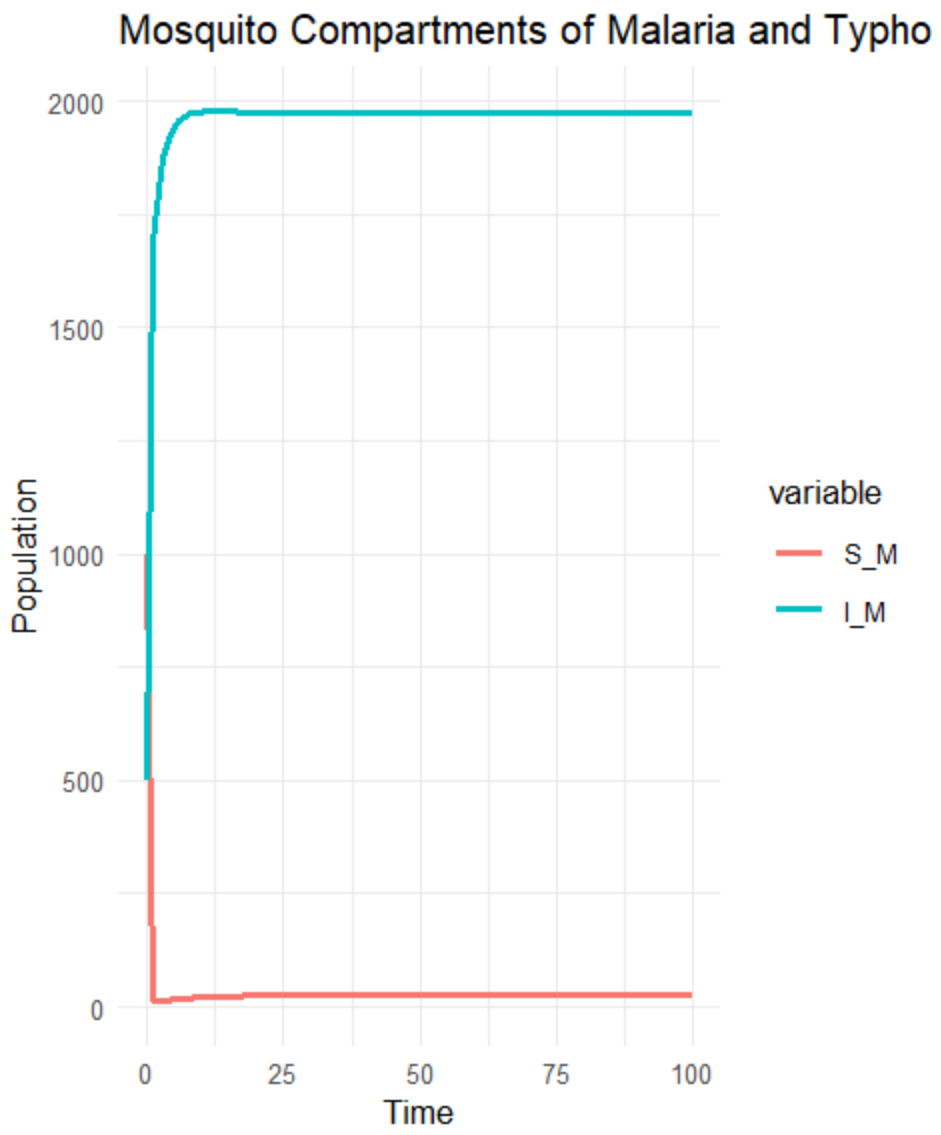

Figure3: Mosquito Compartment of Malaria and Typhoid Co-infection Dynamics

Discussion

The findings of this study demonstrate the critical importance of fundamental reproduction numbers in determining the persistence or elimination of co-infection between malaria and typhoid in a particular population. The analytical analysis confirmed that the disease-free equilibrium shows global asymptotic stability when both reproduction numbers are less than unity, meaning that the co-infection will eventually disappear regardless of the original conditions. This finding reinforces the theoretical threshold condition as a formidable predictor for the efficacy of disease control measures.The global stability outcomes achieved through the application of Lyapunov functions and LaSalle's invariance principle accentuate the resilience of the model. Specifically, the methodology validated that the disease-free state is globally attractive under sub-threshold conditions. This is in concordance with previous epidemiological modeling investigations, which have consistently demonstrated that the reduction of the effective reproduction number below one remains fundamental to the eradication of infectious diseases.

Numerical simulations afforded additional insights into the temporal dynamics of both human and mosquito

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populations. The trajectories depicted that, in the absence of sustained transmission that is when $R_0 < 1$,the count of infected individuals diminishes progressively while the susceptible population reaches a state of stabilization. In contrast, parameter configurations characterized by reproduction numbers exceeding unity revealed the potential for the persistence of co-infection, thereby underscoring the synergistic interplay of malaria and typhoid transmission dynamics. These findings are particularly salient in endemic regions, where the intersecting epidemiology of the two diseases exacerbates the cumulative disease burden. The ramifications of these findings extend to the realm of public health policy. The model elucidates that interventions targeting single diseases may prove inadequate in regions with a high prevalence of co-infection. Instead, integrated strategies—such as eliminate co-infection in endemic regions. Specifically, the model shows that reducing both basic reproduction numbers below unity is essential for global disease eradication. Numerical simulations further illustrate that integrated interventions targeting vector control for malaria and improvements in sanitation, hygiene, and vaccination for typhoid can significantly reduce the burden of co-infection.

From a policy perspective, these findings emphasize the importance of adopting a holistic approach to infectious disease control. Tackling malaria and typhoid independently is insufficient in co-endemic areas, as the persistence of one disease may sustain the transmission of the other. Instead, combined intervention strategies offer the most effective pathway for reducing morbidity and mortality associated with the amalgamation of vector control initiatives for malaria with water, sanitation, and hygiene (WASH) programs for typhoid are imperative to effectively reduce both reproduction numbers beneath unity. Furthermore, the simulations suggest that neglecting one disease could inadvertently perpetuate or exacerbate the co-infection burden.The incorporation of interaction terms within the co-infection compartment illustrates how the presence of one infection may heighten susceptibility to the other. This outcome is congruent with clinical observations of heightened disease severity and diagnostic complexities in malaria and typhoid co-infections. It underscores the critical importance of precise and timely diagnoses, as misdiagnosis could perpetuate endemic and undermine control interventions.

Therefore, the results indicate that the malaria and typhoid co-infection system is exceedingly responsive to alterations in transmission and recovery parameters. Achieving and maintaining disease control necessitates the concurrent reduction of both diseases' reproduction numbers. This reinforces the advocacy for integrated intervention frameworks and provides a mathematical rationale for prioritizing combined malaria and typhoid control programs in resource-constrained, high-burden contexts such as sub-Saharan Africa.

Conclusion

This study has developed and analyzed a deterministic co-infection model for malaria and typhoid fever, incorporating both human and mosquito populations and the interactions that drive co-infection dynamics. By deriving the basic reproduction numbers for malaria, typhoid, and their co-infection, and establishing the global stability of the balance free of sickness The study offers solid theoretical support for the circumstances in which disease eradication is feasible by utilizing Lyapunov functions and LaSalle's invariance principle. The results demonstrate that simultaneous control of both infections is necessary to malaria–typhoid co-infection. This study contributes to the growing body of evidence supporting integrated disease management frameworks in sub-Saharan Africa and other high-burden regions. By highlighting the threshold conditions for disease elimination and the potential impact of joint interventions, the model provides a valuable tool for policymakers and public health practitioners seeking to optimize resource allocation and strengthen control programs against malaria and typhoid co-infection.

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