SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 6 | June 2025 - Peer Reviewed Journal

EXPLORING THE MATHEMATICAL MODEL OF DRUG-RESISTANT MALARIA AND TYPHOID FEVER CO-INFECTION

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Article DOI: https://doi.org/10.36713/epra22763
DOI No: 10.36713/epra22763

ABSTRACT

Antimalarial drug resistance has become one of the biggest threats to efforts employed in the tackle and control of malaria in endemic countries. This study presents and analyzes a new deterministic model for the co-infection of drug-resistant malaria and typhoid fever transmission dynamics. The model includes new compartments for individuals dually infected with drug resistant malaria and typhoid fever and allows for disease transmission by those individuals. The model is observed to exhibit the phenomenon of backward bifurcation when the associated reproduction number is less than unity, R0 < 1.

KEYWORDS: *Backward bifurcation, Drug resistant malaria, Typhoid fever, Co-infection*

2020 Mathematics Subject Classification: *34C60, 92C42, 92D30, 92D25, 93A30*

1.0 INTRODUCTION

Malaria, one of the leading fatal diseases globally, is caused by varied species of Plasmodium, including P. falciparum, P. vivax, P. ovale, P. malariae, and P. knowlesi. Nearly half of the global population is at risk for malaria, with the highest incidence and mortality rates occurring in Sub-Saharan Africa (Birhanle et al. (2014)). This life-threatening disease is transmitted through blood meals by infected female Anopheles mosquitoes (Ukaegbu et al. (2014); Mbah et al. (2015); Iwuafor et al. (2016); Odikamnoro et al. (2017)). World Malaria Report of 2019 stated that 228 million cases of malaria and 405,000 malaria-related deaths occurred worldwide in 2018 (WHO (2019b)). The emergence of drug resistance in malaria treatment has become one of the most significant obstacles in controlling the disease. It has contributed to malaria spreading to new areas and resurfacing in regions where it was once eradicated (Bloland (2001)).

Typhoid fever, another major public health concern in many developing nations, is a systemic infection caused by Salmonella typhi and characterized by high fever, weakness, headaches, abdominal pain, loss of appetite, and sometimes constipation or a rash. In rare instances, it can result in severe complications such as internal bleeding or even death (Pradhan (2011); Mushanyu et al. (2018)). The disease is typically contracted through the intake of contaminated food or water and is prevalent in regions with poor sanitation and inadequate clean water. Recent estimates suggest that between 11 and 21 million cases occur annually, leading to 128,000 to 161,000 deaths globally (WHO (2019a)).

Numerous studies have been conducted on the epidemiology of co-infection models (Mukandavire et al. (2009); Tilahun et al. (2018); Okongo et al. (2019); Ogunmiloro (2019); Egeonu et al. (2021); Omame et al. (2022); Agwu et al. (2023)). Mathematical modeling has been extensively used to explore the transmission dynamics of both malaria and typhoid fever. For example, Okosun (Okosun et al. (2011)) developed a deterministic model for malaria transmission, which exhibited backward bifurcation, meaning that the traditional threshold for malaria eradication, $R_0 < 1$, was insufficient on its own. The model also identified conditions under which eradication could be optimal. Simulations indicated that combining vaccination with effective treatment would substantially reduce malaria transmission.

Esteva (Esteva et al. (2009)) presented a model to study the impact of drug resistance on malaria transmission. The model considered both wild and resistant strains of the malaria parasite and the effect of high treatment rates on the dominance of resistant strains. Numerical results showed that when the two strains coexisted, the proportion of individuals with the resistant strain decreased as the resistance development rate increased. Okosun (Okosun and Makinde (2011)) further extended his work by considering drug resistance in the infective population, showing that optimal control of drug resistance could reduce disease spread.

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Fatmawati (Fatmawati and Tasman (2010)) examined malaria transmission, considering both sensitive and resistant strains of the parasite, and demonstrated the effectiveness of optimal control strategies in reducing infections. Mutua (Mutua et al. (2015)) developed models for the co-infection dynamics of malaria and typhoid, highlighting the need for simultaneous management of both diseases. The study showed that reducing the basic reproduction number, R_0, below one for both diseases would result in their eradication.

This research proposes a deterministic model to study the co-infection dynamics of drug-resistant malaria and typhoid fever. The model builds upon the work of Mutua (Mutua et al. (2015)) by:
i. Incorporating drug-resistant malaria dynamics into the co-infection model, where individuals who receive incomplete treatment may develop resistance.
ii. Including human-to-human transmission dynamics of typhoid, considering both direct and indirect transmission.
iii. Accounting for the transmission of both diseases in individuals co-infected with drug-resistant malaria and typhoid fever.

2.0 MODEL FORMULATION

The total human population at time t, denoted by $N_h(t)$, is divided into eleven mutually exclusive compartments as follows: Susceptible humans($S_h(t)$), individuals with untreated sensitive malaria strain ($I_{xs}(t)$), individuals with treated sensitive malaria strain (ie individuals whose treatment fail to completely clear the parasites in their system and so are still infectious) (I_{ts} (t)), individuals with resistant malaria strain ($I_r(t)$), individuals recovered from malaria ($R_m(t)$), individuals with typhoid Fever strain ($I_{tp}(t)$), individuals recovered from typhoid fever ($R_{tp}(t)$), individuals with untreated malaria strain and typhoid fever (I_{xstp} (t)), individuals with treated malaria strain and typhoid fever ($I_{tstp}(t)$), individuals with resistant malaria strain and typhoid fever ($I_{rtp}(t)$), individuals recovered from malaria and typhoid fever ($R_{mtp}(t)$). Similarly, the total vector population at time t, is divided into three compartments as follows: susceptible vectors ($S_v(t)$), vectors with infected strain ($I_{vs}(t)$) and vectors with resistant strain ($I_{vr}(t)$) while the compartment ($B_{tp}(t)$) stands for the bacteria concentration in the environment. Thus

$$N = S_h(t) + I_{xs}(t) + I_{ts}(t) + I_r(t) + R_m(t) + I_{tp}(t) + R_{tp}(t) + I_{xstp}(t) + I_{tstp}(t) + I_{rtp}(t) + R_{mtp}(t)$$
$$V = S_v(t) + I_{vs}(t) + I_{vr}(t)$$
$$B = B_{tp}(t) \quad (1)$$

The population of susceptible humans (S_h) is increased by the recruitment of humans who are free from both malaria and typhoid fever at the rate Π_h. Susceptible humans acquire sensitive malaria infection following effective contacts with infected vectors (I_{vs}) with a force of infection λ_{ms} given by:

$$\lambda_{ms}(t) = \frac{\beta_m \zeta I_{vs}}{N_h} \quad (2)$$

In (2), $\beta_m\zeta$ is the transmission rate for the infected vectors that account for transmission of malaria infection, where β_m is the transmission probability for malaria (from vector to human) while ζ is the average biting rate of vectors. Susceptible humans develop resistance to malaria treatment following effective contacts with vectors with resistant strain (I_{vr}) with a force of infection λ_{mr} given by:

$$\lambda_{mr}(t) = \frac{\beta_m \zeta \epsilon_r I_{vr}}{N_h} \quad (3)$$

In (3), $\beta_m\zeta$ is the transmission rate for vectors with resistant strain that accounts for transmission of malaria infection with resistance to treatment while ϵ_r is the modification parameter for infectiousness of the resistant strain. Susceptible humans acquire typhoid fever infection following effective contacts with the bacteria (B_{tp}) with a force of infection λ_{tp} given by:

$$\lambda_{tp} = \frac{B_{tp}\varphi}{\kappa + B_{tp}} \quad (4)$$

In (4), φ stands for the rate of ingestion of the typhoid-causing bacteria while κ stands for the concentration of bacteria in foods and water. Susceptible humans can also acquire typhoid fever infection following effective contacts with already infected humans (i.e, those in $I_{tp}, I_{xstp}, I_{tstp}$ and I_{rtp} classes) with a force of infection λ_{tph} given by:

$$\lambda_{tph} = \frac{\beta_{tph}(I_{tp} + h_m(I_{xstp} + \epsilon_t I_{tstp} + I_{rtp}))}{N_h} \quad (5)$$

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In (5), β_{tph} is the infectious rate for humans already infected with typhoid fever which accounts for the transmission of typhoid fever infection, h_m is the additional modification parameter responsible for the increased infectiousness of dually infected individuals while the parameter ϵ_t with $0 < \epsilon_t < 0$, accounts for the reduction in infectiousness of treated humans when compared with untreated humans infected with malaria. Natural death rate for humans is represented by μ_h. ε stands for the modification parameter for reduced susceptibility to malaria by individuals who have recovered prior malaria infection. In our framework, individuals in the dually infected compartments $(I_{xstp}, I_{tstp}, I_{rtp})$ are allowed a natural progression (recovery) from either or both diseases. A fraction of the populations $(I_{xstp}, I_{tstp}, I_{rtp})$ progress to (I_{xs}, I_{ts}, I_r) at the rates $\chi_{tp\varrho 1}(1-\chi_m), \chi_{tp\varrho 2}(1-\chi_m)$ and $\chi_{tp\varrho 3}(1-\chi_m)$ respectively where ϱ_1, ϱ_2 *and* ϱ_3 account for rate of natural progression for dually infected, while $\chi_{tp}(1-\chi_m)$ accounts for fraction of the population progressing from $(I_{xstp}, I_{tstp}, I_{rtp})$ to (I_{xs}, I_{ts}, I_r), $(0 < \chi_m, \chi_{tp} \leq 1)$. The rates τ_{m1} *and* τ_{m2} account for administration of anti-malaria drugs for singly and dually infected (I_{xs}, I_{xstp}) respectively. α_{ms}, α_{mt} *and* α_{mr} stand for malaria natural recovery rates for singly infected (I_{xs}, I_{ts}, I_r) respectively. σ_{m1} *and* σ_{m2} represent the rates of development of resistance to malaria treatment for individuals in (I_{ts}, I_{tstp}) respectively, who receive incomplete treatment. δ_{ms} *and* δ_{mr} stand for malaria induced death rates for the sensitive and resistant malaria strains respectively, φ_1, φ_2 account for the modification parameters for δ_{ms} from (I_{ts}, I_{tstp}), while δ_{tp} represent typhoid fever induced death rate. γ_{tp} is the typhoid fever recovery rate due to treatment for infected. μ_b stands for bacteria death rate, ρ_{tp} accounts for rate of excretion of bacteria into the environment by the already infected humans while σ_t, σ_r are the modification parameters for bacteria recruitment for I_{tstp} *and* I_{rtp} respectively

The population of susceptible vectors (S_v) is increased by the recruitment (birth) of vectors free from infection at the rate Π_v, (All vectors are assumed to be born susceptible, hence no vertical transmission is allowed). Susceptible vectors acquire infection following effective contacts with the infected humans $(I_{xs}, I_{ts}, I_{xstp}$ *and* $I_{tstp})$ with the force of infection λ_{vs}, given by

$$\lambda_{vs}(t) = \frac{\beta_v \zeta (I_{xs} + I_{xstp} + \epsilon_t (I_{ts} + I_{tstp}))}{N_h} \tag{6}$$

In (6), $\beta_v \zeta$ is the effective contact rate of the infected humans with untreated sensitive malaria strain and treated sensitive malaria strain that accounts for the transmission of infection for vectors. Susceptible vectors also acquire infection following effective contacts with the infected humans $(I_r$ *and* $I_{rtp})$ with the force of infection λ_{vr}, given by

$$\lambda_{vr}(t) = \frac{\beta_v \zeta \epsilon_r (I_r + I_{rtp})}{N_h} \tag{7}$$

In (7), $\beta_v \zeta$ is the effective contact rate with the infected humans with resistant strain that accounts for the transmission of infection for vectors while ϵ_r is the modification parameter for infectiousness of the resistant strain. μ_v is the natural death of vectors.

Merging all the concepts and assumptions above, the model for the co-dynamics of drug-resistant malaria and typhoid fever is given by the following differential equations:

$$\frac{dS_h}{dt} = \Pi_h - (\lambda_{ms} + \lambda_{mr} + \lambda_{tp} + \lambda_{tph} + \mu_h) S_h$$

$$\frac{dI_{xs}}{dt} = \lambda_{ms}(S_h + \varepsilon R_m + \varepsilon R_{mtp} + R_{tp}) + \chi_{tp\varrho 1}(1-\chi_m) I_{xstp} - (\tau_{m1} + \mu_h + \alpha_{ms} + \delta_{ms1}) I_{xs} - (\lambda_{tp} + \lambda_{tph}) I_{xs}$$

$$\frac{dI_{ts}}{dt} = \tau_{m1} I_{xs} + \chi_{tp\varrho 2}(1-\chi_m) I_{tstp} - (\sigma_{m1} + \mu_h + \alpha_{mt} + \varphi_1 \delta_{ms1}) I_{ts} - (\lambda_{tp} + \lambda_{tph}) I_{ts}$$

$$\frac{dI_r}{dt} = \sigma_{m1} I_{ts} + \lambda_{mr}(S_h + \varepsilon R_m + \varepsilon R_{mtp} + R_{tp}) + \chi_{tp} \rho_3 (1-\chi_m) I_{rtp} - (\mu_h + \alpha_{mr} + \delta_{mr1}) I_r - (\lambda_{tp} + \lambda_{tph}) I_r$$

$$\frac{dR_m}{dt} = \alpha_{ms} I_{xs} + \alpha_{mt} I_{ts} + \alpha_{mr} I_r - \mu_h R_m - (\lambda_{tp} + \lambda_{tph}) R_m - \varepsilon(\lambda_{ms} + \lambda_{mr}) R_m$$

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$$\frac{dI_{tp}}{dt} = (\lambda_{tp} + \lambda_{tph})(S_h + R_m + R_{tp} + R_{mtp}) - (\delta_{tp1} + \mu_h + \gamma_{tp})I_{tp} - (\lambda_{ms} + \lambda_{mr})I_{tp} + \chi_{m\varrho 1} I_{xstp} + \chi_{m\varrho 2} I_{tstp} + \chi_{m\varrho 3} I_{rtp}$$

$$\frac{dR_{tp}}{dt} = \gamma_{tp} I_{tp} - \mu_h R_{tp} - (\lambda_{tp} + \lambda_{tph})R_{tp} - (\lambda_{ms} + \lambda_{mr})R_{tp}$$

$$\frac{dB_{tp}}{dt} = \rho_{tp} I_{tp} + \rho_{tp}(I_{xstp} + \sigma_t I_{tstp} + \sigma_r I_{rtp}) - \mu_b B_{tp} \quad (8)$$

$$\frac{dI_{xstp}}{dt} = (\lambda_{tp} + \lambda_{tph})I_{xs} + \lambda_{ms} I_{tp} - (\mu_h + \delta_{ms2} + \delta_{tp2} + \tau_{m2} + \varrho_1)I_{xstp}$$

$$\frac{dI_{tstp}}{dt} = (\lambda_{tp} + \lambda_{tph})I_{ts} + \tau_{m2} I_{xstp} - (\mu_h + \varphi_2 \delta_{ms2} + \delta_{tp2} + \sigma_{m2} + \varrho_2)I_{tstp}$$

$$\frac{dI_{rtp}}{dt} = \sigma_{m2} I_{tstp} + (\lambda_{tp} + \lambda_{tph})I_r + \lambda_{mr} I_{tp} - (\mu_h + \delta_{mr2} + \delta_{tp2} + \varrho_3)I_{rtp}$$

$$\frac{dR_{mtp}}{dt} = \varrho_1(1 - \chi_m)(1 - \chi_{tp})I_{xstp} + \varrho_2(1 - \chi_m)(1 - \chi_{tp})I_{tstp} + \varrho_3(1 - \chi_m)(1 - \chi_{tp})I_{rtp} - \mu_h R_{mtp} - (\lambda_{tp} + \lambda_{tph})R_{mtp} - \varepsilon(\lambda_{ms} + \lambda_{mr})R_{mtp}$$

$$\frac{dS_v}{dt} = \Pi_v - (\lambda_{vs} + \lambda_{vr})S_v - \mu_v S_v$$

$$\frac{dI_{vs}}{dt} = \lambda_{vs} S_v - \mu_v I_{vs}$$

$$\frac{dI_{vr}}{dt} = \lambda_{vr} S_v - \mu_v I_{vr}$$

with the corresponding initial conditions,

$S_h(0) \geq 0, I_{xs}(0) \geq 0, I_{ts}(0) \geq 0, I_r(0) \geq 0, R_m(0) \geq 0, I_{tp}(0) \geq 0, R_{tp}(0) \geq 0, B_{tp}(0) \geq 0, I_{xstp}(0) \geq 0, I_{tstp}(0) \geq 0, I_{rtp}(0) \geq 0, R_{mtp}(0) \geq 0, S_v(0) \geq 0, I_{vs}(0) \geq 0, I_{vr}(0) \geq 0.$ (9)

Table 1: Table of parameters in the model (8).

Parameters	Interpretation
Π_h	Human Recruitment rate
β_m	Transmission Probability for malaria from vector to human
ζ	Average biting rate of vectors
ϵ_r	Modification parameter for infectiousness of the resistant strain
β_{tph}	Human-to-Human Typhoid Transmission Probability
ϵ_t	Modification parameter for malaria infectiousness of individual treating malaria
μ_h	Natural death rate
ε	Modification parameter for susceptibility to malaria by individuals who have recovered prior malaria infection
χ_{tp}	Fraction progressing from dually infected classes to singly infected or recovered class for typhoid
χ_m	Fraction progressing from dually infected classes to singly infected or recovered class for malaria
ϱ_1	Rate of progression out of I_{xstp}
ϱ_2	Rate of progression out of I_{tstp}
ϱ_3	Rate of progression out of I_{rtp}
τ_{m1}	Administration of Anti-Malaria Drugs for singly infected
δ_m	Malaria induced mortality rate
φ_1, φ_2	Modification parameter for δ_{ms} from treatment class
σ_m	Rate of development of resistance to malaria treatment
α_m	Malaria natural recovery rate for singly infected

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δ_{tp}	Typhoid induced mortality rate
γ_{tp}	Typhoid recovery rate due to treatment for infected
τ_{m2}	Administration of anti-malaria drugs for dually infected
μ_b	Mortality rate for bacteria
Π_v	Recruitment rate for vectors
β_v	Transmission Probability for malaria from human to vectors
μ_v	Natural death rate for vectors
h_m	Modification parameter for dually infected due to increased chance to infect
ρ_{tp}	Rate of excretion of bacteria into the environment
σ_t, σ_r	Modification parameter for bacteria recruitment
φ	Rate of ingestion of typhoid-causing bacteria

3.0 BASIC PROPERTIES OF THE MODEL

3.1 Positivity

For the model (8) to be epidemiologically meaningful, it is appropriate to show that all its state variables are non- negative over time.

Theorem 3.1: Assume the initial data $S_h > 0, I_{xs} > 0, I_{ts} > 0, I_r > 0, R_m > 0, I_{tp} > 0, R_{tp} > 0, B_{tp} > 0, I_{xstp} > 0, I_{tstp} > 0, I_{rtp} > 0, R_{mtp} > 0, S_v > 0, I_{vs} > 0, I_{vr} > 0$; Then the solutions $(S_h,\ I_{xs},\ I_{ts},\ I_r,\ R_m,\ I_{tp},\ R_{tp},\ B_{tp},\ I_{xstp},\ I_{tstp},\ I_{rtp},\ R_{mtp},\ S_v,\ I_{vs},\ I_{vr})$ of the basic model (8) are positive for all $t > 0$.

Proof: Let

$$t_1 = sup\{t > 0 :\ S_h > 0, I_{xs} > 0, I_{ts} > 0, I_r > 0, R_m > 0, I_{tp} > 0, R_{tp} > 0, B_{tp} > 0, I_{xstp} > 0, I_{tstp} > 0, I_{rtp} > 0, R_{mtp} > 0, S_v > 0, I_{vs} > 0, I_{vr} > 0\}.$$

Following from the first equation of the model (8), we have that

$$\begin{aligned}\frac{dS_h}{dt} &= \Pi_h - (\lambda_{ms} + \lambda_{mr} + \lambda_{tp} + \lambda_{tph} + \mu_h)S_h \\ &\geq \Pi_h - (\lambda_{ms} + \lambda_{mr} + \lambda_{tp} + \mu_h)S_h \\ &= \Pi_h - (\lambda_m + \lambda_{tp} + \mu_h)S_h\end{aligned}$$

$$where\ \lambda_m = \lambda_{ms} + \lambda_{mr} = \frac{\beta_m \zeta(I_{vs} + \epsilon_r I_{vr})}{N_h}\ and\ \lambda_{tp} = \frac{B_{tp}\varphi}{\kappa + B_{tp}}$$

which we can re-write as:

$$\frac{d}{dt}\left[S_h(t) exp\left[\int_0^t \left(\lambda_m(u) + \lambda_{tp}(u)\right) du + \mu_h(t)\right]\right] = \Pi_h exp\left[\int_0^t \left(\lambda_m(u) + \lambda_{tp}(u)\right) du + \mu_h(t)\right]$$

Hence,

$$\begin{aligned}&\frac{d}{dt}\left[S_h(t) exp\left[\int_0^{t_1} \left(\lambda_m(u) + \lambda_{tp}(u)\right) du + \mu_h(t)_1\right]\right] - S_h(0) \\ &= \Pi_h \int_0^{t_1} exp\left[\int_0^x \left(\lambda_m(u) + \lambda_{tp}(u)\right) du + \mu_h(x)\right] dx\end{aligned}$$

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So that

$$S_h(t_1) = S_h(0)exp\left[-\int_0^{t_1}\left(\lambda_m(u)+\lambda_{tp}(u)\right)du - \mu_h t_1\right] + exp\left[-\int_0^{t_1}\left(\lambda_m(u)+\lambda_{tp}(u)\right)du - \mu_h t_1\right] \times \Pi_h\int_0^{t_1} exp\left[\int_0^{x}\left(\lambda_m(u)+\lambda_{tp}(u)\right)du + \mu_h(x)\right]dx \ > 0$$

In the same manner. It can be shown that:

$I_{xs} > 0, I_{ts} > 0, I_r > 0, R_m > 0, I_{tp} > 0, R_{tp} > 0, B_{tp} > 0, I_{xstp} > 0, I_{tstp} > 0, I_{rtp} > 0, R_{mtp} > 0, S_v > 0, I_{vs} > 0, I_{vr} > 0.$

3.2 Invariant Domain

To obtain the invariant region, in which the solution of the model is bounded, first examine the total human population (N_h), where

$$N_h = S_h + I_{xs} + I_{ts} + I_r + R_m + I_{tp} + R_{tp} + I_{xstp} + I_{tstp} + I_{rtp} + R_{mtp}$$

Differentiating both sides of N_h with respect to't' leads to

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_{xs}}{dt} + \frac{dI_{ts}}{dt} + \frac{dI_r}{dt} + \frac{dR_m}{dt} + \frac{dI_{tp}}{dt} + \frac{dR_{tp}}{dt} + \frac{dI_{xstp}}{dt} + \frac{dI_{tstp}}{dt} + \frac{dI_{rtp}}{dt} + \frac{dR_{mtp}}{dt} \quad (10)$$

This gives

$$\frac{dN_h}{dt} = \Pi_h - \mu_h N_h - (\delta_1 + \delta_2) \quad (11)$$

Where: δ_1= All deaths due to Malaria and δ_2 = All deaths due to Typhoid Fever.

In the absence of deaths resulting from malaria and typhoid fever diseases ($\delta_1 = \delta_2 = 0$), hence:

$$\frac{dN_h}{dt} \leq \Pi_h - \mu_h N_h$$

Integrating both sides, we have

$$\int \frac{dN_h}{\Pi_h - \mu_h N_h} \leq \int dt \xrightarrow{yields} \Pi_h - \mu_h N_h \geq Ae^{-\mu_h t}\ ; A \equiv Constant$$

Applying the initial condition $N(0) = N_0$, we have:

$$A = \Pi_h - \mu_h N_{h0} \xrightarrow{yields} \Pi_h - \mu_h N_h \ \geq (\Pi_h - \mu_h N_{h0})e^{-\mu_h t} \xrightarrow{yields} N_h \ \leq \frac{\Pi_h}{\mu_h} - \left[\frac{\Pi_h - \mu_h N_{h0}}{\mu_h}\right]e^{-\mu_h t}$$

As $t \to \infty$, the population size $N_h \to \frac{\Pi_h}{\mu_h}$ which implies $0 \leq N_h \leq \frac{\Pi_h}{\mu_h}$

Therefore, the set of feasible solutions of the model equation is in the region

$$\Omega = \left[\left(S_h, I_{xs}, I_{ts}, I_r, R_m, I_{tp}, R_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, R_{mtp}\right) \in \Re_+^{11}: N_h \leq \frac{\Pi_h}{\mu_h}\right]$$

4.0 MODEL ANALYSIS

4.1 Disease-Free Equilibrium (DFE)

To find the DFE, we equate the right hand side of the basic model (8) to zero, evaluating it at $I_{xs} = I_{ts} = I_r = I_{tp} = I_{xstp} = I_{tstp} = I_{rtp} = B_{tp} = I_{vs} = I_{vr} = 0$ and solving the non-infected state variables. This gives the disease-free equilibrium (DFE) ξ_0 as:

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$$\xi_0 = (S_h^*, I_{xs}^*, I_{ts}^*, I_r^*, R_m^*, I_{tp}^*, R_{tp}^*, B_{tp}^*, I_{xstp}^*, I_{tstp}^*, I_{rtp}^*, R_{mtp}^*, S_v^*, I_{vs}^*, I_{vr}^*)$$

4.2 The Basic Reproduction Number (R_0)

To determine the R_0, the next-generation matrix method is used.The next generation matrices are given by:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_m\zeta & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_m\zeta\epsilon_r \\ 0 & 0 & 0 & \beta_{tph} & \frac{\varphi S_h}{\kappa} & \beta_{tph}h_m & \beta_{tph}h_m\epsilon_t & \beta_{tph}h_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_v\zeta S_v}{N_h} & \frac{\beta_v\zeta\epsilon_t S_v}{N_h} & 0 & 0 & 0 & \frac{\beta_v\zeta S_v}{N_h} & \frac{\beta_v\zeta\epsilon_t S_v}{N_h} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_v\zeta\epsilon_r S_v}{N_h} & 0 & 0 & 0 & 0 & \frac{\beta_v\zeta\epsilon_r S_v}{N_h} & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 & -D_1 & 0 & 0 & 0 & 0 \\ -\tau_{m1} & K_2 & 0 & 0 & 0 & 0 & -D_2 & 0 & 0 & 0 \\ 0 & -\sigma_{m1} & K_3 & 0 & 0 & 0 & 0 & -D_3 & 0 & 0 \\ 0 & 0 & 0 & K_4 & 0 & -\chi_{m\varrho 1} & -\chi_{m\varrho 2} & -\chi_{m\varrho 3} & 0 & 0 \\ 0 & 0 & 0 & -\rho_t & \mu_b & -\rho_t & -\rho_t\sigma_t & -\rho_t\sigma_r & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & K_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\tau_{m2} & K_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\sigma_{m2} & K_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_v & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_v \end{bmatrix}$$

Where

$$K_1 = \tau_{m1} + \mu_h + \alpha_{ms} + \delta_{ms1}, K_2 = \sigma_{m1} + \mu_h + \alpha_{mt} + \varphi_1\delta_{ms1}, K_3 = \mu_h + \alpha_{mr} + \delta_{mr1}, K_4 = \delta_{tp1} + \mu_h + \gamma_{tp}, K_5 = \mu_h + \delta_{ms2} + \delta_{tp2} + \tau_{m2} + \varrho_1, K_6 = \mu_h + \varphi_2\delta_{ms2} + \delta_{tp2} + \sigma_{m2} + \varrho_2, K_7 = \mu_h + \delta_{mr2} + \delta_{tp2} + \varrho_3, D_1 = \chi_{tp\varrho 1}(1-\chi_m), D_2 = \chi_{tp\varrho 2}(1-\chi_m), D_3 = \chi_{tp\varrho 3}(1-\chi_m)$$

The basic reproduction number of the Malaria-Typhoid co-infection model (8), applying the perspective illustrated in (van den Driessche et al. (2002)), is given by $R_0 = \rho(FV^{-1}) = max\{R_{0mp}, R_{0tp}\}$ where $R_{0mp}(ie\ R_{0ms}\ and\ R_{0mr})\ and\ R_{0tp}$ are, respectively, the Malaria and Typhoid associated reproduction numbers, given by

$$R_{0ms} = \sqrt{\frac{\zeta^2\beta_m\beta_v(K_2 + \epsilon_t\tau_{m1})S_v^*}{K_1K_2\mu_vN_h^*}}$$

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$$R_{0mr} = \sqrt{\frac{\zeta^2 \epsilon_r^2 \beta_m \beta_v S_v^*}{K_3 \mu_v N_h^*}}$$

and

$$R_{0tp} = \frac{\beta_{tph}}{K_4} + \frac{\rho_{tp} \varphi S_h^*}{\mu_b \kappa K_4}$$

4.3 Local Asymptotic Stability Of The DFE (LAS)

Theorem 4.1: The DFE ξ_0, of the Malaria-Typhoid co-infection model (8) is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof: The local stability of the co-infection model is studied using the Jacobian matrix of the system (8) at ξ_0. The eigenvalues of $J(\xi_0)$ are $\lambda_1 = -K_5, \lambda_2 = -K_6, \lambda_3 = -K_7, \lambda_4 = \lambda_5 = \lambda_6 = \lambda_7 = -\mu_h, \lambda_8 = -\mu_v$ and the solutions to the characteristic polynomials are as follows:

$$\lambda^2 + (K_3 + \mu_v)\lambda + K_3 \mu_v \left(1 - \frac{R_{0mr}^2}{\zeta}\right) = 0 \quad (12)$$

$$\lambda^3 + (K_1 + K_2 + \mu_v)\lambda^2 + \left(K_1 \mu_v + K_2 \mu_v + K_1 K_2 - \frac{\zeta^2 \beta_m \beta_v S_v^*}{N_h^*}\right)\lambda + K_1 K_2 \mu_v (1 - R_{0ms}^2) \quad (13)$$

$$\lambda^2 + \left(\mu_b + K_4 - \beta_{tph}\right)\lambda + K_4 \mu_b \left(1 - R_{0tp}\right) \quad (14)$$

Implementing the Routh-Hurwitz criterion, the quadratic equations (12), (13) and (14) will give roots with negative real parts if and only if $R_0 < 1$. Hence, the disease-free equilibrium, ξ_0 is locally asymptotically stable if $R_0 < 1$

4.4 Global Asymptotic Stability (GAS) of the DFE

The pattern developed in (Castillo-Chavez et al. (2002)) is applied to examine the global asymptotic stability of the DFE of the system (8). Two conditions that will guarantee the GAS of the DFE, if satisfied are pointed out.

First, the system (8) is to be written in the form:

$$\frac{dX}{dt} = P(X, Z)$$
$$\frac{dZ}{dt} = Q(X, Z), Q(X, 0) = 0$$

Where $X \in R^m$ signifies (its components) the number of uninfected individuals and $Z \in R^n$ signifies (its components) the number of infected individuals. $U_0 = (X^*, 0)$ signifies the disease-free equilibrium (DFE) of this system (8). The conditions (W1) and (W2) below must be satisfied in order to guarantee local asymptotic stability:

(W1): For $\frac{dX}{dt} = P(X, 0), X^*$ is globally asymptotically stable (GAS),

(W2): $Q(X, Z) = BZ - \hat{Q}(X, Z)X, Q(X, Z) \geq 0$ for $(X, Z) \in \Omega$,

where $B = D_z Q(X^*, 0)$ is an M-matrix (the off-diagonal elements of B are non-negative and Ω is the region where the model makes biological sense. If system (8) satisfies the above two conditions, then the following theorem holds:

Theorem 4.1: The fixed point $U_0 = (X^*, 0)$ is said to be a globally asymptotic stable (GAS) equilibrium point of the system (8) provided that $R_0 < 1$ (LAS) and the assumptions (W1) and (W2) are satisfied.

Proof: Having obtained that $R_0 < 1$, we go ahead to establish the conditions (W1) and (W2). For the system (8), we have the following:

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
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$$P(X,0) = \begin{bmatrix} \Pi_h - \mu_h S_h \\ 0 \\ 0 \\ 0 \\ \Pi_v - \mu_v S_v \end{bmatrix} \quad (16)$$

Equation (16) implies that the first condition (W1) is satisfied. Next,

$$Q(X,Z) = BZ - \hat{Q}(X,Z)X$$

Then B =

$$\begin{bmatrix} -K_1 & 0 & 0 & 0 & 0 & D_1 & 0 & 0 & \beta_m\zeta & 0 \\ \tau_{m1} & -K_2 & 0 & 0 & 0 & 0 & D_2 & 0 & 0 & 0 \\ 0 & \sigma_{m1} & -K_3 & 0 & 0 & 0 & 0 & D_3 & 0 & \beta_m\zeta\epsilon_r \\ 0 & 0 & 0 & -K_4 & \frac{\varphi S_h^*}{\kappa} & \chi_{m\varrho 1} & \chi_{m\varrho 2} & \chi_{m\varrho 3} & 0 & 0 \\ 0 & 0 & 0 & \rho_{tp} & -\mu_b & \rho_{tp} & \rho_{tp}\sigma_t & \rho_{tp}\sigma_r & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -K_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_{m2} & -K_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & & -K_7 & 0 & 0 \\ \frac{\beta_v\zeta S_v^*}{N_h^*} & \frac{\beta_v\zeta\epsilon_t S_v^*}{N_h^*} & 0 & 0 & 0 & \frac{\beta_v\zeta S_v^*}{N_h^*} & \frac{\beta_v\zeta\epsilon_t S_v^*}{N_h^*} & 0 & -\mu_v & 0 \\ 0 & 0 & \frac{\beta_v\zeta\epsilon_r S_v^*}{N_h^*} & 0 & 0 & 0 & 0 & \frac{\beta_v\zeta\epsilon_r S_v^*}{N_h^*} & 0 & -\mu_v \end{bmatrix} \quad (17)$$

and $\hat{Q} =$

$$\begin{bmatrix} \frac{\beta_m\zeta I_{vs}}{N_h}\left[1-\left(S_h+\varepsilon R_m+\varepsilon R_{mtp}+R_{tp}\right)\right]+\frac{B_{tp}\varphi}{\kappa+B_{tp}}I_{xs}+\frac{\beta_{tph}(D_8)}{N_h}I_{xs}^* \\ \frac{B_{tp}\varphi}{\kappa+B_{tp}}I_{ts}+\frac{\beta_{tph}(D_8)}{N_h}I_{ts}^* \\ \frac{\beta_m\zeta\epsilon_r I_{vr}}{N_h}\left[1-\left(S_h+\varepsilon R_m+\varepsilon R_{mtp}+R_{tp}\right)\right]+\frac{B_{tp}\varphi}{\kappa+B_{tp}}I_r+\frac{\beta_{tph}(D_8)}{N_h}I_r^* \\ B_{tp}\varphi\left[\frac{S_h^*}{\kappa}-\frac{\left(S_h+R_m+R_{tp}+R_{mtp}\right)}{\kappa+B_{tp}}\right]-\frac{\beta_{tph}(D_8)}{N_h}\left(S_h+R_m+R_{tp}+R_{mtp}\right)+\frac{\beta_m\zeta(I_{vs}+\epsilon_r I_{vr})}{N_h}I_{tp} \\ 0 \\ -\frac{B_{tp}\varphi}{\kappa+B_{tp}}I_{xs}-\frac{\beta_{tph}(D_8)}{N_h}I_{xs}^*-\frac{\beta_m\zeta I_{vs}}{N_h}I_{tp} \\ -\frac{B_{tp}\varphi}{\kappa+B_{tp}}I_{ts}-\frac{\beta_{tph}(D_8)}{N_h}I_{ts}^* \end{bmatrix}$$

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$$-\frac{B_{tp}\varphi}{\kappa+B_{tp}}I_r-\frac{\beta_{tph}(D_8)}{N_h}I_r^*-\frac{\beta_m\zeta\epsilon_r I_{vr}}{N_h}I_{tp}$$
$$\beta_v\zeta(I_{xs}+\epsilon_t I_{ts})\left[\frac{S_v^*}{N_h^*}-\frac{S_v}{N_h}\right]+\beta_v\zeta\left(I_{xstp}+\epsilon_t I_{tstp}\right)\left[\frac{S_v^*}{N_h^*}-\frac{S_v}{N_h}\right]$$
$$\beta_v\zeta\epsilon_r\left(I_r+I_{rtp}\right)\left[\frac{S_v^*}{N_h^*}-\frac{S_v}{N_h}\right]$$

(18)

where $D_8=I_{tp}+h_m\left(I_{xstp}+\epsilon_t I_{tstp}+I_{rtp}\right)$.

It is observed from equation (18) that $\hat{Q}(X,Z)$ is not positive semi-definite since entries 6, 7, 8 are negative, which implies that the second condition (W2) is not satisfied. Therefore the disease free equilibrium (DFE) U_0, may not be globally asymptotically stable (GAS) when $R_0<1$, implying that the condition $R_0<1$ is not sufficient in driving out the disease over time. This introduces the possible occurrence of a backward bifurcation.

However we see that by setting

$$\beta_m\zeta=B_{tp}\varphi=\beta_{tph}=0 \tag{19}$$

we have that $\hat{Q}(X,Z)\geq 0$, implying that the disease free equilibrium (DFE) U_0, may be globally asymptotically stable (GAS) when $R_0<1$.

4.5 Backward Bifurcation Analysis of The Model

This section discusses the occurrence of backward bifurcation in the model (8). A situation where a stable disease free equilibrium co-exists with a stable endemic equilibrium when the reproduction number associated with the model is less than unity, is known as backward bifurcation and has been observed in several disease models. The public health implication of the occurrence of backward bifurcation of model (8) is that, the standard epidemiological prerequisite of having $R_0<1$, although necessary, is no longer sufficient for the effective control of the diseases. Using the Centre Manifold Theory (Castillo-Chavez and Song (2004)), we establish the following result.

Theorem 4.2: Assume a backward bifurcation coefficient $a>0$, when $R_0<1$, then model (8) will display the attribute of a backward bifurcation at $R_0=1$. Also, when $a<0$, then the system (8) will display a forward bifurcation at $R_0=1$.

Proof: Suppose $\xi_e=\left(S_h^{**},I_{xs}^{**},I_{ts}^{**},I_r^{**},R_m^{**},I_{tp}^{**},R_{tp}^{**},B_{tp}^{**},I_{xstp}^{**},I_{tstp}^{**},I_{rtp}^{**},R_{mtp}^{**},S_v^{**},I_{vs}^{**},I_{vr}^{**}\right)$ is an arbitrary endemic equilibrium. In order to analyse the bifurcation of system (8), let,

$$S_h=x_1,I_{xs}=x_2,I_{ts}=x_3,I_r=x_4,R_m=x_5,I_{tp}=x_6,R_{tp}=x_7,B_{tp}=x_8,I_{xstp}=x_9,I_{tstp}=x_{10},I_r=x_{11},R_{mtp}=x_{12},S_v=x_{13},I_{vs}=x_{14},I_{vr}=x_{15} \tag{20}$$

with,

$$\frac{dx_1}{dt}=\Pi_h-\left(\lambda_{ms}+\lambda_{mr}+\lambda_{tp}+\lambda_{tph}+\mu_h\right)x_1$$
$$\frac{dx_2}{dt}=\lambda_{ms}(x_1+\varepsilon x_5+\varepsilon x_{12}+x_7)+\chi_{tp\varrho 1}(1-\chi_m)x_9-(\tau_{m1}+\mu_h+\alpha_{ms}+\delta_{ms1})x_2-\left(\lambda_{tp}+\lambda_{tph}\right)x_2$$
$$\frac{dx_3}{dt}=\tau_{m1}x_2+\chi_{tp\varrho 2}(1-\chi_m)x_{10}-(\sigma_{m1}+\mu_h+\alpha_{mt}+\varphi_1\delta_{ms1})x_3-\left(\lambda_{tp}+\lambda_{tph}\right)x_3$$
$$\frac{dx_4}{dt}=\sigma_{m1}x_3+\lambda_{mr}(x_1+\varepsilon x_5+\varepsilon x_{12}+x_7)+\chi_{tp}\rho_3(1-\chi_m)x_{11}-(\mu_h+\alpha_{mr}+\delta_{mr1})x_4-\left(\lambda_{tp}+\lambda_{tph}\right)x_4$$
$$\frac{dx_5}{dt}=\alpha_{ms}x_2+\alpha_{mt}x_3+\alpha_{mr}x_4-\mu_h x_5-\left(\lambda_{tp}+\lambda_{tph}\right)x_5-\varepsilon(\lambda_{ms}+\lambda_{mr})x_5$$

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
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$$\frac{dx_6}{dt} = (\lambda_{tp} + \lambda_{tph})(x_1 + x_5 + x_7 + x_{12}) - (\delta_{tp1} + \mu_h + \gamma_{tp})x_6 - (\lambda_{ms} + \lambda_{mr})x_6 + \chi_{m\varrho 1}\, x_9 + \chi_{m\varrho 2} x_{10} + \chi_{m\varrho 3}\, x_{11}$$

$$\frac{dx_7}{dt} = \gamma_{tp}x_6 - \mu_h x_7 - (\lambda_{tp} + \lambda_{tph})x_7 - (\lambda_{ms} + \lambda_{mr})x_7$$

$$\frac{dx_8}{dt} = \rho_{tp}x_6 + \rho_{tp}(x_9 + \sigma_t x_{10} + \sigma_r x_{11}) - \mu_b x_8 \qquad (21)$$

$$\frac{dx_9}{dt} = (\lambda_{tp} + \lambda_{tph})x_2 + \lambda_{ms}x_6 - (\mu_h + \delta_{ms2} + \delta_{tp2} + \tau_{m2} + \varrho_1)x_9$$

$$\frac{dx_{10}}{dt} = (\lambda_{tp} + \lambda_{tph})x_3 + \tau_{m2}x_9 - (\mu_h + \varphi_2\delta_{ms2} + \delta_{tp2} + \sigma_{m2} + \varrho_2)x_{10}$$

$$\frac{dx_{11}}{dt} = \sigma_{m2}x_{10} + (\lambda_{tp} + \lambda_{tph})x_6 + \lambda_{mr}I_{tp} - (\mu_h + \delta_{mr2} + \delta_{tp2} + \varrho_3)x_{11}$$

$$\frac{dx_{12}}{dt} = \varrho_1(1-\chi_m)(1-\chi_{tp})x_9 + \varrho_2(1-\chi_m)(1-\chi_{tp})x_{10} + \varrho_3(1-\chi_m)(1-\chi_{tp})x_{11} - \mu_h x_{12} - (\lambda_{tp} + \lambda_{tph})x_{12} - \varepsilon(\lambda_{ms} + \lambda_{mr})x_{12}$$

$$\frac{dx_{13}}{dt} = \Pi_v - (\lambda_{vs} + \lambda_{vr})x_{13} - \mu_v x_{13}$$

$$\frac{dx_{14}}{dt} = \lambda_{vs}x_{13} - \mu_v x_{14}$$

$$\frac{dx_{15}}{dt} = \lambda_{vr}x_{13} - \mu_v x_{15}$$

The Jacobian matrix of system (21) is as follows: $J(\xi_0) =$

$$\begin{bmatrix}
-\mu_h & 0 & 0 & 0 & 0 & -\beta_{tphh} & 0 & \frac{-\varphi x_{1h}^{**}}{\kappa} & -\beta_{tph}h_m & -\beta_{tph}h_m\epsilon_t & -\beta_{tph}h_m & 0 & 0 & -\beta_m\zeta & -\beta_m\zeta\epsilon_r \\
0 & -K_1 & 0 & 0 & 0 & 0 & 0 & 0 & D_1 & 0 & 0 & 0 & 0 & \beta_m\zeta & 0 \\
0 & \tau_{m1} & -K_2 & 0 & 0 & 0 & 0 & 0 & 0 & D_2 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \sigma_{m1} & -K_3 & 0 & 0 & 0 & 0 & 0 & 0 & D_3 & 0 & 0 & 0 & \beta_m\zeta\epsilon_r \\
0 & \alpha_{ms} & \alpha_{mt} & \alpha_{mr} & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -N_1 & 0 & \frac{\varphi x_1^*}{\kappa} & N_2 & N_3 & N_4 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \sigma_{tp} & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \rho_{tp} & 0 & -\mu_b & \rho_{tp} & \rho_{tp}\sigma_t & \rho_{tp}\sigma_r & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -K_5 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau_{m2} & -K_6 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\sigma_{m2} & -K_7 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -D_4 & D_5 & D_6 & -\mu_h & 0 & 0 & 0 \\
0 & -\theta & -\theta\epsilon_t & -\theta\epsilon_r & 0 & 0 & 0 & 0 & -\theta & -\theta\epsilon_t & -\theta\epsilon_r & 0 & -\mu_h & 0 & 0 \\
0 & \theta & \theta\epsilon_t & 0 & 0 & 0 & 0 & 0 & \theta & \theta\epsilon_t & 0 & 0 & 0 & -\mu_v & 0 \\
0 & 0 & 0 & \theta\epsilon_r & 0 & 0 & 0 & 0 & 0 & 0 & \theta\epsilon_r & 0 & 0 & 0 & -\mu_v
\end{bmatrix}$$

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Where

$$K_1 = \tau_{m1} + \mu_h + \alpha_{ms} + \delta_{ms1}, K_2 = \sigma_{m1} + \mu_h + \alpha_{mt} + \varphi_1\delta_{ms1}, K_3 = \mu_h + \alpha_{mr} + \delta_{mr1}, K_4 = \delta_{tp1} + \mu_h + \gamma_{tp}, K_5 = \mu_h + \delta_{ms2} + \delta_{tp2} + \tau_{m2} + \varrho_1, K_6 = \mu_h + \varphi_2\delta_{ms2} + \delta_{tp2} + \sigma_{m2} + \varrho_2, K_7 = \mu_h + \delta_{mr2} + \delta_{tp2} + \varrho_3, D_1 = \chi_{tp}\varrho_1(1-\chi_m), D_2 = \chi_{tp}\varrho_2(1-\chi_m), D_3 = \chi_{tp}\varrho_3(1-\chi_m), D_4 = \varrho_1(1-\chi_m)(1-\chi_{tp}), D_5 = \varrho_2(1-\chi_m)(1-\chi_{tp}), D_6 = \varrho_3(1-\chi_m)(1-\chi_{tp}), N_1 = (K_4 - \beta_{tph}), N_2 = \beta_{tph}h_m + \chi_{m}\varrho_1, N_3 = \beta_{tph}h_m\epsilon_t + \chi_{m}\varrho_2, N_4 = \beta_{tph}h_m + \chi_{m}\varrho_3, \theta = \frac{\beta_v\zeta x_{13}^*}{N_h^*}$$

Consider the situation when $R_{0mr} = 1$. Furthermore, suppose that the product $\beta_m\epsilon_r$ is selected as a bifurcation parameter. Solving for $\beta_m\epsilon_r = \beta^*$ from $R_{0mr} = 1$ gives

$$\beta_m^*\epsilon_r^* = \beta^* = \frac{\mu_v K_3 N_h^*}{\zeta^2\epsilon_r\beta_v S_v^*}$$

Analysing the Jacobian of the system (20) at the DFE, $J(\xi_0)$ and applying the approach in (Castillo-Chavez and Song (2004)), we obtain that $J(\xi_0)$ has a right eigenvector as follows:

$$w = [\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7, \omega_8, \omega_9, \omega_{10}, \omega_{11}, \omega_{12}, \omega_{13}, \omega_{14}, \omega_{15}]^T$$

where,

$$\omega_1 = -\frac{1}{\mu_h}\left[\left(\beta_{tph} + \frac{\varphi\rho_{tp}x_1^*}{\mu_b\kappa}\right)\omega_6 + \beta_v\beta_m\zeta^2 x_{13}^*\left[\left(1 + \frac{\tau_{m1}}{\kappa}\right)\omega_2 + \epsilon_r^2\omega_4\right]\right]$$

$$\omega_2 = \omega_2 > 0, \omega_3 = \frac{\tau_{m1}}{K_2}\omega_2, \omega_4 = \omega_4 > 0, \omega_5 = \frac{1}{\mu_h}\left[\left(\alpha_{ms} + \frac{\alpha_{mt}\tau_{m1}}{K_2}\right)\omega_2 + \alpha_{mr}\omega_4\right]$$

$$\omega_6 = \omega_6 > 0, \omega_7 = \frac{\gamma_{tp}}{\mu_h}\omega_6, \omega_8 = \frac{\rho_{tp}}{\mu_b}\omega_6, \omega_9 = \omega_{10} = \omega_{11} = \omega_{12} = 0$$

$$\omega_{13} = -\frac{\beta_v\zeta x_{13}^*}{\mu_v N_h^*}\left[\left(1 - \frac{\epsilon_t\tau_{m1}}{K_2}\right)\omega_2 + \epsilon_r\omega_4\right], \omega_{14} = \frac{\beta_v\zeta x_{13}^*}{\mu_v N_h^*}\left(1 + \frac{\epsilon_t\tau_{m1}}{K_2}\right)\omega_2$$

$$\omega_{15} = \frac{\beta_v\zeta\epsilon_r x_{13}^*}{\mu_v N_h^*}\omega_4$$

In like manner, the elements of the left eigenvector of $J(\xi_0)|_{\beta=\beta_m^*\epsilon_r^*}, v = (v_1, v_2, \dots, v_{15})$, satisfying $v.w = 1$, are as follows:

$$v_1 = v_5 = v_7 = v_{12} = v_{13} = 0, v_2 = v_2 = 0$$

$$v_3 = \frac{1}{K_2}\left[\frac{\sigma_{m1}\theta\epsilon_r}{\mu_v K_3} + \frac{\theta\epsilon_t\beta_m\zeta v_2}{\mu_v}\right], v_4 = \frac{\theta\epsilon_r}{\mu_v K_3}, v_6 = \frac{\rho_{tp}}{\mu_b N_1}, v_8 = \frac{\kappa N_1}{\rho_{tp}\varphi x_1^*}$$

$$v_9 = \frac{1}{K_5}\left[\left(\frac{\mu_v D_1 + \theta\beta_m\zeta}{\mu_v}\right)v_2 + \frac{N_2\rho_{tp}}{\mu_b N_1} + \frac{N_1\kappa}{\varphi x_1^*} + \tau_{m2}v_{10}\right]$$

$$v_{10} = \frac{\mu_v D_2 v_3 + \mu_v N_3 v_6 + \mu_v\rho_{tp}\sigma_t v_8 + \mu_v\sigma_{m2}v_{11} + \theta\epsilon_t\beta_m\zeta v_2}{\mu_v K_6}$$

$$v_{11} = \frac{D_3 v_4 + N_4 v_6 + \rho_{tp}\sigma_r v_8 + \theta\epsilon_r v_{15}}{K_7}, v_{14} = \frac{\beta_m\zeta v_2}{\mu_v}, v_{15} = \frac{K_3}{\theta\beta_m\zeta\epsilon_r^2}$$

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
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The bifurcation coefficients given by:

$a = \sum_{k,i,j=1}^{n} v_k \omega_i \omega_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$ and $b = \sum_{k,i=1}^{n} v_k \omega_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_m^*}(0,0)$

are computed to be:

$$a = -\frac{\omega_8 \varphi}{\kappa}\left[\omega_2(v_2 - v_9) + \omega_3(v_3 - v_{10}) + \omega_4(v_4 - v_{11}) - v_6\left(\omega_1 + \omega_5 + \omega_7 + \omega_8 \frac{2x_1^*}{\kappa}\right)\right]$$
$$- \frac{\omega_6 \beta_{tph}}{N_h^*}\left[\omega_2(v_2 - v_9) + \omega_3(v_3 - v_{10}) + \omega_4(v_4 - v_{11})\right.$$
$$\left. - v_6\left(\omega_1 + \omega_5 + \omega_7 - \frac{x_1^*}{N_h^*}(\omega_1 + \omega_2 + \omega_3\omega_4 + \omega_5 + \omega_7)\right)\right]$$
$$+ \frac{\beta_m \zeta}{N_h^*}\left[v_4 \omega_{15} \epsilon_r \left[\omega_1 + \omega_7 - (\omega_1 + \omega_2 + \omega_3 + \omega_4 + \omega_5 + \omega_6 + \omega_7)\frac{x_1^*}{N_h^*}\right]\right.$$
$$\left. - \omega_6[v_6(\omega_{14} + \omega_{15}\epsilon_r) - v_9\omega_{14} - v_{11}\omega_{15}\epsilon_r]\right]$$
$$- \frac{2\beta_v \zeta v_{14}(\omega_2 + \omega_3 \epsilon_t)}{N_h^*}\left[(\omega_1 + \omega_2 + \omega_3 + \omega_4 + \omega_5 + \omega_6 + \omega_7)\frac{x_{13}^*}{N_h^*} - \omega_{13}\right]$$
$$+ \frac{v_{15}\omega_4 \beta_v \zeta \epsilon_r}{N_h^*}\left[\omega_{13} - (\omega_1 + \omega_2 + \omega_3 + \omega_5 + \omega_6 + \omega_7)\frac{x_{13}^*}{N_h^*}\right]$$

and $b = (v_2\omega_{14} + v_4\omega_{15}\epsilon_r)\frac{\zeta x_1^*}{N_h^*} > 0$

Hence, according to (Castillo-Chavez and Song (2004)) we deduce that the model (8) will display a backward bifurcation if the coefficient, a, above is positive.

5.0 CONCLUSION

We developed and analyzed a 15-compartmentalized mathematical model for the dynamics of the co-infection of drug-resistant malaria and typhoid fever transmission. Our results show that model (8) possesses a disease free equilibrium point that is locally asymptotically stable when $R_0 < 1$ and globally asymptotically stable when there is no external reinfection. By applying theorem (4.2), we investigated and explored the possibility of backward bifurcation of the model. The model was shown to exhibit the phenomenon of backward bifurcation when the bifurcation coefficient a is less than unity, so the basic threshold $R_0 < 1$ is no longer sufficient for controlling the spread of the diseases in the population.

6.0 CONFLICT OF INTEREST

On behalf of all authors, the corresponding author states that there is no conflict of interest.

7.0 FUNDING NOT APPLICABLE

8.0 DATA AVAILABILTY

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

9.0 AUTHOR CONTRIBUTIONS

A.I.O. Ofomata and A. Omame concieved the idea and designed the model; A.I.O. Ofomata drafted the manuscript; A.I.O. Ofomata, C.O. Agwu and A. Omame performed numerical simulation; A.I.O. Ofomata, C.O. Agwu and A. Omame contributed to the overall analysis and interpretation of the findings. M.C. Obi, N.N. Iheonu and N.N. Araka supervised the overall work. All authors read and approved the original manuscript.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 6 | June 2025 - Peer Reviewed Journal

10.0 ACKNOWLEDGEMENT

We would like to thank all the lecturers in the department of mathematics, FUTO, for their support in this study.

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