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MATHEMATICAL MODEL OF DRUG-RESISTANT MALARIA AND TYPHOID FEVER CO-INFECTION WITH OPTIMAL CONTROL AND COST EFFECTIVENESS ANALYSIS

Amarachukwu I.O. Ofomata [1], C.O Agwu [2], S.O Adeyemo[3] , C.B Okereke [4],
[1,2] *Department of Mathematics, Federal University of Technology Owerri, Nigeria.*
[1,3,4] *Federal Polytechnic Nekede Owerri, Nigeria.*
Correspondence: C.O Agwu

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ABSTRACT
This study presents a deterministic model developed to examine how malaria drug resistance and various preventive strategies influence the transmission dynamics of malaria–typhoid fever co-infection. The model introduces additional compartments to account for individuals simultaneously infected with drug-resistant malaria and typhoid fever, and it considers their role in propagating the diseases. Numerical simulations suggest that preventing drug-resistant malaria significantly reduces the spread of malaria–typhoid co-infections within the population. Furthermore, cost-effectiveness analysis indicates that the most efficient intervention among the strategies examined is one that targets the prevention of typhoid fever from both bacterial sources and human carriers. This combined approach offers the greatest reduction in disease burden at the lowest cost.

KEYWORDS: *Cost Effectiveness, Drug resistant malaria, Typhoid fever, Co-infection,*
2020 Mathematics Subject Classification: 34C60, 92C42, 92D30, 92D25, 93A30

1.0 INTRODUCTION

Malaria remains one of the most deadly infectious diseases worldwide, primarily caused by several species of the *Plasmodium* parasite, namely *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae*, and *P. knowlesi*. According to Birhanle et al. (2014), nearly half of the world's population is at risk, with Sub-Saharan Africa bearing the highest burden in terms of both infection rates and fatalities. The disease is transmitted through the bite of an infected female *Anopheles* mosquito, typically during blood feeding (Ukaegbu et al., 2014; Mbah et al., 2015; Iwuafor et al., 2016; Odikamnoro et al., 2017). The World Health Organization's 2019 report recorded an estimated 228 million malaria cases and 405,000 deaths globally in 2018 (WHO, 2019b). A growing concern in malaria control efforts is the rise of drug-resistant strains of the parasite. As Bloland (2001) observed, drug resistance has significantly hindered treatment success and has led to the re-emergence of malaria in regions where it had previously been controlled.

Typhoid fever is another significant public health challenge, particularly in many developing countries. It is a systemic infection caused by *Salmonella typhi*, typically marked by high fever, fatigue, headaches, abdominal discomfort, loss of appetite, and, in some cases, constipation or a rash. Although uncommon, severe complications such as internal bleeding or even death can occur (Pradhan, 2011; Mushanyu et al., 2018). Transmission usually occurs through the consumption of food or water contaminated with fecal matter, making the disease especially prevalent in areas with poor sanitation and limited access to clean water. According to the World Health Organization (2019a), an estimated 11 to 21 million typhoid cases are reported annually, resulting in 128,000 to 161,000 deaths worldwide.

Several researchers have examined 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. Ofomata et al., 2025), with mathematical modeling serving as a key tool in understanding the transmission dynamics of both malaria and typhoid fever. For instance, Okosun et al. (2011) developed a deterministic model of malaria transmission that revealed the occurrence of backward bifurcation, where the basic reproduction number, $R_0 < 1$, is not sufficient for disease eradication. The study emphasized that combining vaccination with effective treatment could significantly lower malaria prevalence.

In another study, Esteva et al. (2009) modeled the effects of drug resistance on malaria transmission, considering both wild-type and resistant parasite strains. Their findings showed that while resistant strains may initially dominate under high treatment pressure, increasing the rate of resistance development could lead to a decline in the resistant population. Building on this, Okosun and Makinde (2011) incorporated drug resistance in the infective class and demonstrated that implementing optimal control strategies could effectively mitigate disease spread.

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Fatmawati and Tasman (2010) also modeled malaria transmission involving both sensitive and resistant strains, illustrating how optimal interventions could reduce infection levels. Additionally, Mutua et al. (2015) explored the co-infection dynamics of malaria and typhoid fever, stressing the importance of integrated control measures. Their results suggested that bringing the basic reproduction number below one for both diseases is crucial to achieving long-term 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 sensitive 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:

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

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

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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:

$$\begin{aligned}
\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 \\
\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} \\
\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}
\end{aligned} \tag{8}$$

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

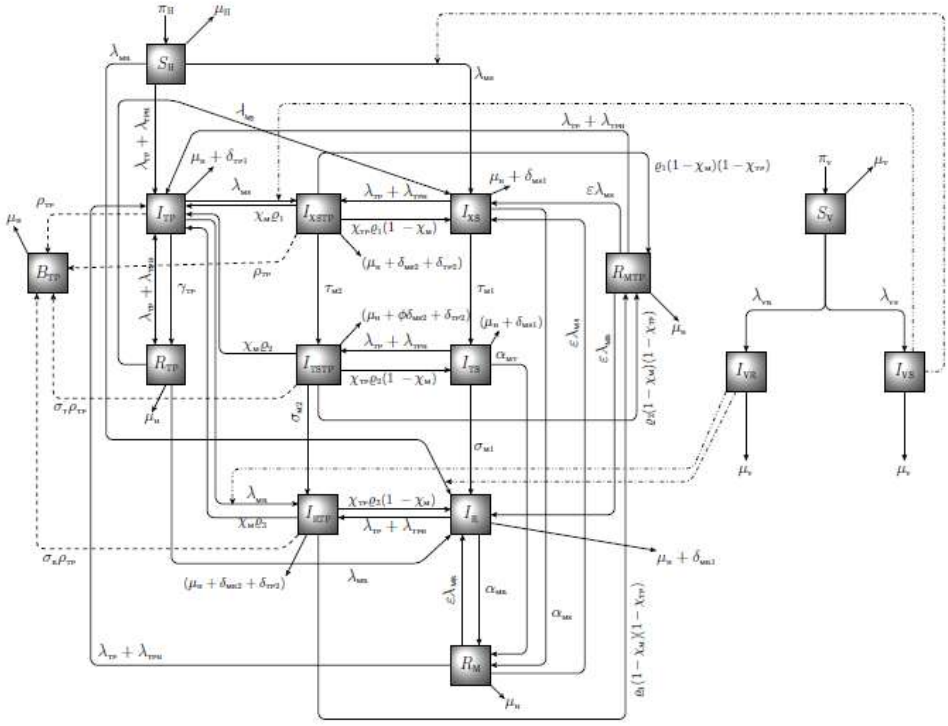
with the corresponding initial conditions,

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

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3.0 BASIC PROPE

The basic properties of the model were presented and analysed in the work of Ofomata et al.(2025).

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:

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

4.2The Basic Reproduction Number (R_0)

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

$$R_{0ms} = \sqrt{\frac{\zeta^2 \beta_m \beta_v (K_2 + \epsilon_t \tau_{m1}) S_v^*}{K_1 K_2 \mu_v N_h^*}} \tag{10}$$

$$R_{0mr} = \sqrt{\frac{\zeta^2 \epsilon_r^2 \beta_m \beta_v S_v^*}{K_3 \mu_v N_h^*}} \tag{11}$$

and

$$R_{0tp} = \frac{\beta_{tph}}{K_4} + \frac{\rho_{tp} \varphi S_h^*}{\mu_b \kappa K_4} \tag{12}$$

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:

$$\lambda^2 + (K_3 + \mu_v)\lambda + K_3 \mu_v \left(1 - \frac{R_{0mr}^2}{\zeta}\right) = 0 \tag{13}$$

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$$\lambda^3 + (K_1 + K_2 + \mu_v)\lambda^2 + \left(K_1\mu_v + K_2\mu_v + K_1K_2 - \frac{\zeta^2\beta_m\beta_v S_v^*}{N_h^*}\right)\lambda + K_1K_2\mu_v(1 - R_{0ms}^2) \quad (14)$$

$$\lambda^2 + (\mu_b + K_4 - \beta_{tph})\lambda + K_4\mu_b(1 - R_{0tp}) \quad (15)$$

Implementing the Routh-Hurwitz criterion, the quadratic equations (13), (14) and (15) 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

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: (See result in Ofomata et al., (2025))

It is shown that 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.

5.0 ANALYSIS OF THE OPTIMAL CONTROL MODEL

Defining and incorporating time dependent controls into model (8), we have the following:

i. u_1 - Precautionary measures against development of malaria by the use of mosquito nets
ii. u_2 - Precautionary measures against development of typhoid fever from bacteria by periodic environmental sanitation (mass cleaning of surroundings and water bodies to protect humans from typhoid-causing bacteria).
iii. u_3 - Precautionary measures against development of typhoid from humans by proper personal sanitation and hygiene

Having incorporated u_1, u_2 and u_3 in the system (8), we derive the optimal control model as follows:

$$\frac{dS_h}{dt} = \Pi_h - \left[(1-u_1)(\lambda_{ms} + \lambda_{mr}) + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph} + \mu_h\right]S_h$$

$$\frac{dI_{xs}}{dt} = (1-u_1)\lambda_{ms}\left(S_h + \varepsilon R_m + \varepsilon R_{mtp} + R_{tp}\right) + D_1 I_{xstp} - K_1 I_{xs} - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_{xs}$$

$$\frac{dI_{ts}}{dt} = \tau_{m1} I_{xs} + D_2 I_{tstp} - K_2 I_{ts} - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_{ts}$$

$$\frac{dI_r}{dt} = \sigma_{m1} I_{ts} + (1-u_1)\lambda_{mr}\left(S_h + \varepsilon R_m + \varepsilon R_{mtp} + R_{tp}\right) + D_3 I_{rtp} - K_3 I_r - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_r$$

$$\frac{dR_m}{dt} = \alpha_{ms} I_{xs} + \alpha_{mt} I_{ts} + \alpha_{mr} I_r - \mu_h R_m - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]R_m - (1-u_1)\varepsilon(\lambda_{ms} + \lambda_{mr})R_m$$

$$\frac{dI_{tp}}{dt} = \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]\left(S_h + R_m + R_{tp} + R_{mtp}\right) - K_4 I_{tp} - (1-u_1)(\lambda_{ms} + \lambda_{mr})I_{tp} + \chi_{m\varrho 1} I_{xstp} + \chi_{m\varrho 2} I_{tstp} + \chi_{m\varrho 3} I_{rtp}$$

$$\frac{dR_{tp}}{dt} = \gamma_{tp} I_{tp} - \mu_h R_{tp} - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]R_{tp} - (1-u_1)(\lambda_{ms} + \lambda_{mr})R_{tp}$$

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

$$\frac{dI_{xstp}}{dt} = \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_{xs} + (1-u_1)\lambda_{ms} I_{tp} - K_5 I_{xstp}$$

$$\frac{dI_{tstp}}{dt} = \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_{ts} + \tau_{m2} I_{xstp} - K_6 I_{tstp}$$

$$\frac{dI_{rtp}}{dt} = \sigma_{m2} I_{tstp} + \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]I_r + (1-u_1)\lambda_{mr} I_{tp} - K_7 I_{rtp}$$

$$\frac{dR_{mtp}}{dt} = \mathrm{D}_4 I_{xstp} + \mathrm{D}_5 I_{tstp} + \mathrm{D}_6 I_{rtp} - \mu_h R_{mtp} - \left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]R_{mtp} - (1-u_1)\varepsilon(\lambda_{ms} + \lambda_{mr})R_{mtp}$$

$$\frac{dS_v}{dt} = \Pi_v - (\lambda_{vs} + \lambda_{vr})(1-u_1)S_v - \mu_v S_v$$

$$\frac{dI_{vs}}{dt} = \lambda_{vs}(1-u_1)S_v - \mu_v I_{vs} \quad (16)$$

$$\frac{dI_{vr}}{dt} = \lambda_{vr}(1-u_1)S_v - \mu_v I_{vr}$$

Hence, to analyse the optimal control levels of the system, a Lebesgue measurable control set is defined:

$$U = \{(u_1(t), u_2(t), u_3(t)) : 0 \le u_1 \le 1{,}0 \le u_2 \le 1{,}0 \le u_3 \le 1{,}0 \le t \le T\}$$

The focus here is to derive a control set u^* that minimizes the proposed objective function J:

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$$J(x;u_1,u_2,u_3)=\int_0^{t_f}\Big(A_1I_{xs}(t)+A_2I_{ts}(t)+A_3I_r(t)+A_4I_{tp}(t)+A_5B_{tp}(t)+A_6I_{xstp}(t)+A_7I_{tstp}(t)+A_8I_{rtp}(t)+A_9I_{vs}(t)+A_{10}I_{vr}(t)+\frac{A_{11}}{2}u_1^2(t)+\frac{A_{12}}{2}u_2^2(t)+\frac{A_{13}}{2}u_3^2(t)\Big)dt \quad (17)$$

Where t_f is the end time of control execution and the constants $A_i; i = 1,2,3,\dots,10$ are positive weights which help to stabilize each term in the integrand A_{11}, A_{12} and A_{13} are weight constants of the optimal controls u_1, u_2 and u_3 respectively. The quadratic terms $\frac{A_{11}}{2}u_1^2(t), \frac{A_{12}}{2}u_2^2(t), \frac{A_{13}}{2}u_3^2(t)$ stand for the cost of implementing the controls at any time t; the expressions are quadratic due to the fact that we assume cost to be non-linear in its nature. In the above optimal control problem, we suppose that the control parameters $u_1^*(t), u_2^*(t)$ and $u_3^*(t)$ are Lebesgue measurable with $a < u_1^*(t) \le b, \forall \in [0, t_f], a < u_2^*(t) \le b, \forall \in [0, t_f]$ and $a < u_3^*(t) \le b, \forall \in [0, t_f]$. Therefore, we aim to obtain optimal controls $u_1^*(t), u_2^*(t)$ and $u_3^*(t)$ such that $J(u_1^*, u_2^*, u_3^*) = min(J(u_i): u_i \in U, i = 1,2,3)$; where $U = u_i(t): 0 \le u_i(t) \le 1, 0 \le t \le t_f$ is a Lebesgue measurable non empty set for the controls.

As shown in Fleming and Rishel (1975), the existence of the optimal controls, $u_1^*(t), u_2^*(t), u_3^*(t)$ and the associated optimal solutions $(S_h^*, I_{xs}^*, I_{ts}^*, I_r^*, R_m^*, I_{tp}^*, R_{tp}^*, B_{tp}^*, I_{xstp}^*, I_{tstp}^*, I_{rtp}^*, R_{mtp}^*, S_v^*, I_{vs}^*, I_{vr}^*)$ are assured due to the fact that the state solutions are bounded as proved earlier, the state system satisfies the Lipschitz property with respect to the state variables, since the solutions exist and are unique. We then go ahead to prove the convexity of the integrand $L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1, u_2, u_3) = A_1I_{xs}(t)+A_2I_{ts}(t)+A_3I_r(t)+A_4I_{tp}(t)+A_5B_{tp}(t)+A_6I_{xstp}(t)+A_7I_{tstp}(t)+A_8I_{rtp}(t)+A_9I_{vs}(t)+A_{10}I_{vr}(t)+\frac{A_{11}}{2}u_1^2(t)+\frac{A_{12}}{2}u_2^2(t)+\frac{A_{13}}{2}u_3^2(t)$, with respect to the control parameters u_1, u_2, u_3 in the set Ω. Therefore, we need to show that $L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, \lambda u_1 + (1-\lambda)u_1', \lambda u_2 + (1-\lambda)u_2', \lambda u_3 + (1-\lambda)u_3') \le \lambda L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1, u_2, u_3) + (1-\lambda)L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1', u_2', u_3')$, for $\lambda \in (0,1)$.

Note that

$$L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, \lambda u_1 + (1-\lambda)u_1', \lambda u_2 + (1-\lambda)u_2', \lambda u_3 + (1-\lambda)u_3') = A_1I_{xs}(t)+A_2I_{ts}(t)+A_3I_r(t)+A_4I_{tp}(t)+A_5B_{tp}(t)+A_6I_{xstp}(t)+A_7I_{tstp}(t)+A_8I_{rtp}(t)+A_9I_{vs}(t)+A_{10}I_{vr}(t)+\frac{A_{11}}{2}(\lambda u_1+(1-\lambda)u_1')^2+\frac{A_{12}}{2}(\lambda u_2+(1-\lambda)u_2')^2+\frac{A_{13}}{2}(\lambda u_3+(1-\lambda)u_3')^2 \quad (18)$$

and

$$\lambda L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1, u_2, u_3) + (1-\lambda)L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1', u_2', u_3') = A_1I_{xs}(t)+A_2I_{ts}(t)+A_3I_r(t)+A_4I_{tp}(t)+A_5B_{tp}(t)+A_6I_{xstp}(t)+A_7I_{tstp}(t)+A_8I_{rtp}(t)+A_9I_{vs}(t)+A_{10}I_{vr}(t)+\frac{A_{11}}{2}\lambda u_1^2+\frac{A_{12}}{2}\lambda u_2^2+\frac{A_{13}}{2}\lambda u_3^2+\frac{A_{11}}{2}(1-\lambda)u_1'^2+\frac{A_{12}}{2}(1-\lambda)u_2'^2+\frac{A_{13}}{2}(1-\lambda)u_3'^2 \quad (19)$$

This means that from (18) and (19), we must prove that

$$(\lambda u_1+(1-\lambda)u_1')^2 \le \lambda u_1^2+(1-\lambda)u_1'^2, (\lambda u_2+(1-\lambda)u_2')^2 \le \lambda u_2^2+(1-\lambda)u_2'^2, (\lambda u_3+(1-\lambda)u_3')^2 \le \lambda u_3^2+(1-\lambda)u_3'^2 \quad (20)$$

Expanding $(\lambda u_1+(1-\lambda)u_1')^2$ gives

$$(\lambda u_1+(1-\lambda)u_1')^2 = \lambda^2u_1^2+2\lambda(1-\lambda)u_1u_1'+(1-\lambda)^2u_1'^2$$
$$= \lambda u_1^2+(1-\lambda)u_1'^2-\lambda(1-\lambda)[u_1-u_1']^2$$
$$\le \lambda u_1^2+(1-\lambda)u_1'^2 \quad (21)$$

Similarly, the same results can be obtained for expanding $(\lambda u_2+(1-\lambda)u_2')^2$ and $(\lambda u_3+(1-\lambda)u_3')^2$. This shows the convexity of the integral on the singleton Ω

By implementing the Pontryagin's Maximum principle (Pontryagin et al. (1963)) we obtained a Hamiltonian (H) defined as:

$$H(S_h, I_{xs}, I_{ts}, I_r, R_m, I_{tp}, R_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, R_{mtp}, S_v, I_{vs}, I_{vr}) = L(t, I_{xs}, I_{ts}, I_r, I_{tp}, B_{tp}, I_{xstp}, I_{tstp}, I_{rtp}, I_{vs}, I_{vr}, u_1, u_2, u_3) + \pi_1\frac{dS_h}{dt}+\pi_2\frac{dI_{xs}}{dt}+\pi_3\frac{dI_{ts}}{dt}+\pi_4\frac{dI_r}{dt}+\pi_5\frac{dR_m}{dt}+\pi_6\frac{dI_{tp}}{dt}+\pi_7\frac{dR_{tp}}{dt}+\pi_8\frac{dB_{tp}}{dt}+\pi_9\frac{dI_{xstp}}{dt}+\pi_{10}\frac{dI_{tstp}}{dt}+\pi_{11}\frac{dI_{rtp}}{dt}+\pi_{12}\frac{dR_{mtp}}{dt}+\pi_{13}\frac{dS_v}{dt}+\pi_{14}\frac{dI_{vs}}{dt}+\pi_{15}\frac{dI_{vr}}{dt} \quad (22)$$

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Where $\pi_i, i = 1,2,3, \dots, 15$, which satisfy the system of ordinary differential equations, are the adjoint variable functions to be resolved suitably by implementing pontryagin's maximum principle and also following the works in Fleming et al. (1982); Olaniyi et al (2020); Asamoah et al (2022), we determine the existence of the optimal control pairs.

Theorem 5.1: For an optimal control set u_1, u_2, u_3 that minimizes J and U, there exists an adjoint variable $\pi_1 \dots \pi_{15}$, such that:

$$\frac{d\pi_1}{dt} = \pi_1\left[(1-u_1)(\lambda_{ms}+\lambda_{mr}) + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph} + \mu_h\right] - \pi_2[(1-u_1)\lambda_{mr}] - \pi_6\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]$$
$$\frac{d\pi_2}{dt} = \pi_2\left[K_1 + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - \pi_3\tau_{m1} - \pi_5\alpha_{ms} - \pi_9\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - A_1$$
$$\frac{d\pi_3}{dt} = \pi_3\left[K_2 + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - \pi_4\sigma_{m1} - \pi_5\alpha_{mt} - \pi_{10}\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - A_2$$
$$\frac{d\pi_4}{dt} = \pi_4\left[K_3 + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - \pi_5\alpha_{mr} - \pi_{11}\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right] - A_3$$
$$\frac{d\pi_5}{dt} = \pi_5\left[\mu_h + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph} + \varepsilon(1-u_1)(\lambda_{ms}+\lambda_{mr})\right] - \pi_2[(1-u_1)\varepsilon\lambda_{ms}] - \pi_4[(1-u_1)\varepsilon\lambda_{mr}] - \pi_6\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]$$
$$\frac{d\pi_6}{dt} = \pi_6[K_4 + (1-u_1)(\lambda_{ms}+\lambda_{mr})] - \pi_7\gamma_{tp} - \pi_8\rho_{tp} - \pi_9[(1-u_1)\lambda_{ms}] - \pi_{11}[(1-u_1)\lambda_{mr}] - A_4$$
$$\frac{d\pi_7}{dt} = \pi_7\left[\mu_h + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph} + (1-u_1)(\lambda_{ms}+\lambda_{mr})\right] - \pi_2[(1-u_1)\lambda_{ms}] - \pi_4[(1-u_1)\lambda_{mr}] - \pi_6\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]$$
$$\frac{d\pi_8}{dt} = \pi_8\mu_b - A_5$$
$$\frac{d\pi_9}{dt} = \pi_9 K_5 - \pi_2 D_1 - \pi_6\chi_{m\varrho 1} - \pi_8\rho_{tp} - \pi_{10}\tau_{m2} - \pi_{12}D_4 - A_6$$
$$\frac{d\pi_{10}}{dt} = \pi_{10}K_6 - \pi_3 D_2 - \pi_6\chi_{m\varrho 2} - \pi_8\rho_{tp}\sigma_t - \pi_{11}\sigma_{m2} - \pi_{12}D_5 - A_7$$
$$\frac{d\pi_{11}}{dt} = \pi_{11}K_7 - \pi_4 D_3 - \pi_6\chi_{m\varrho 3} - \pi_8\rho_{tp}\sigma_r - \pi_{12}D_6 - A_8$$
$$\frac{d\pi_{12}}{dt} = \pi_{12}\left[\mu_h + (1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph} + \varepsilon(1-u_1)(\lambda_{ms}+\lambda_{mr})\right] - \pi_2[(1-u_1)\varepsilon\lambda_{ms}] - \pi_4[(1-u_1)\varepsilon\lambda_{mr}] - \pi_6\left[(1-u_2)\lambda_{tp} + (1-u_3)\lambda_{tph}\right]$$
$$\frac{d\pi_{13}}{dt} = \pi_{13}[(1-u_1)(\lambda_{vs}+\lambda_{vr}) + \mu_v] - \pi_{14}(1-u_1)\lambda_{vs} - \pi_{15}(1-u_1)\lambda_{vr}$$
$$\frac{d\pi_{14}}{dt} = \pi_{14}\mu_v - A_9$$
$$\frac{d\pi_{15}}{dt} = \pi_{15}\mu_v - A_{10} \quad (23)$$

With transversality conditions, $\pi_i(t_f) = 0, i = 1, \dots, 15$. Also, we determine the control set (u_1^*, u_2^*, u_3^*) characterized by

$$u_1^*(t) = max\{0, min\{1, \eta_1\}\}$$
$$u_2^*(t) = max\{0, min\{1, \eta_2\}\} \quad (24)$$
$$u_3^*(t) = max\{0, min\{1, \eta_3\}\}$$

where

$$\eta_1 = \frac{\psi_1}{A_{11}}, \qquad \eta_2 = \frac{\psi_2}{A_{12}}, \qquad \eta_3 = \frac{\psi_3}{A_{13}}$$

Proof: By applying Pontryagin's maximum principle, we determine a system of differential equations involving the adjoint variable as follows:

$$\frac{d\pi_1}{dt} = \frac{dH}{dS_h}, \frac{d\pi_2}{dt} = \frac{dH}{dI_{xs}}, \frac{d\pi_3}{dt} = \frac{dH}{dI_{ts}}, \frac{d\pi_4}{dt} = \frac{dH}{dI_r}, \frac{d\pi_5}{dt} = \frac{dH}{dR_m}, \frac{d\pi_6}{dt} = \frac{dH}{dI_{tp}}, \frac{d\pi_7}{dt} = \frac{dH}{dR_{tp}}, \frac{d\pi_8}{dt} = \frac{dH}{dB_{tp}}, \frac{d\pi_9}{dt} = \frac{dH}{dI_{xstp}}, \frac{d\pi_{10}}{dt} = \frac{dH}{dI_{tstp}}, \frac{d\pi_{11}}{dt} = \frac{dH}{dI_{rtp}}, \frac{d\pi_{12}}{dt} = \frac{dH}{dR_{mtp}}, \frac{d\pi_{13}}{dt} = \frac{dH}{dS_v}, \frac{d\pi_{14}}{dt} = \frac{dH}{dI_{vs}}, \frac{d\pi_{15}}{dt} = \frac{dH}{dI_{vr}} \quad (25)$$

Similarly to obtain the controls, we solved the equation (58), $\frac{dH}{du_i} = 0$, at u_i^*, for $i = 1,2,3$ and derived the following:

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$$u_1^* = \frac{\psi_1}{A_{11}}, \quad u_2^* = \frac{\psi_2}{A_{12}}, \quad u_3^* = \frac{\psi_3}{A_{13}} \tag{26}$$

Where

$\psi_1 = [\lambda_{ms}(\pi_2 - \pi_1) + \lambda_{mr}(\pi_4 - \pi_1)]S_h + [\lambda_{ms}(\pi_2 - \pi_5) + \lambda_{mr}(\pi_4 - \pi_5)]\varepsilon R_m + [\lambda_{ms}(\pi_2 - \pi_{12}) + \lambda_{mr}(\pi_4 - \pi_{12})]\varepsilon R_{mtp} + [\lambda_{ms}(\pi_2 - \pi_7) + \lambda_{mr}(\pi_4 - \pi_7)]R_{tp} + [\lambda_{ms}(\pi_9 - \pi_6) + \lambda_{ms}(\pi_{11} - \pi_6)]I_{tp} + [\lambda_{vs}(\pi_{14} - \pi_{13}) + \lambda_{vr}(\pi_{15} - \pi_{13})]S_v$

$\psi_2 = \lambda_{tp}\big[(\pi_6 - \pi_1)S_h + (\pi_9 - \pi_2)I_{xs} + (\pi_{10} - \pi_3)I_{ts} + (\pi_{11} - \pi_4)I_r + (\pi_6 - \pi_5)R_m + (\pi_6 - \pi_7)R_{tp} + (\pi_6 - \pi_{12})R_{mtp}\big]$

$\psi_3 = \lambda_{tph}\big[(\pi_6 - \pi_1)S_h + (\pi_9 - \pi_2)I_{xs} + (\pi_{10} - \pi_3)I_{ts} + (\pi_{11} - \pi_4)I_r + (\pi_6 - \pi_5)R_m + (\pi_6 - \pi_7)R_{tp} + (\pi_6 - \pi_{12})R_{mtp}\big]$ (27)

By using standard control arguments that involve the bounds on the control, we write and conclude:

$$u_1^*(t) = max\{0, min\{1, \eta_1\}\}$$
$$u_2^*(t) = max\{0, min\{1, \eta_2\}\} \tag{28}$$
$$u_3^*(t) = max\{0, min\{1, \eta_3\}\}$$

where $\eta_1 = \frac{\psi_1}{A_{11}}$, $\eta_2 = \frac{\psi_1}{A_{11}}$, $\eta_3 = \frac{\psi_1}{A_{11}}$

6.0 NUMERICAL SIMULATIONS

In this section we perform numerical simulations of the model (8) and the resulting optimal control model (16). We use the values of the parameters in Table 2 for the simulations, applying forward fourth-order Runge-Kutta method and backward fourth-order Runge-Kuta method for solving the state system (performed in MATLAB). The adjoint systems is solved by using the initial guess of the controls incorporating the obtained solution for the state system (Lenhart and Workman (2007)). We implement the following three control strategies for numerical simulation of the model (8) with the purpose of reducing the burden of the diseases (Drug-Resistant Malaria and Typhoid Fever) on the population

Table 2: Parameters in the model equation and their values

Parameter	Value	Reference
Π_h	750	Calculated, Agwu et al (2003)
Π_v	600	Assumed
β_m	2.8	Calculated
β_{tph}	1.0125	Calculated
β_v	2.72	Calculated
ζ	1.1	Assumed
ϵ_t	1.2	Assumed
ϵ_r	1.3	Assumed
μ_h	0.0189	Calculated, Agwu et al (2003)
μ_v	0.1429	Nakul et al. (2006)
μ_b	0.0345	Mutua et al. (2015)
ε	1.1	Assumed
χ_m	0.014	Assumed
χ_{tp}	0.015	Assumed
ϱ_1	0.009	Assumed
ϱ_2	0.008	Assumed
ϱ_3	0.007	Assumed
τ_{m1}	0.7	Assumed
τ_{m2}	0.6	Assumed
α_{ms}	0.038	Aguas et al. (2008), Akbari et al. (2012)
α_{mr}	0.2	Assumed
α_{mt}	0.05	Assumed
δ_{ms1}	0.0019	Akbari et al. (2012)

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δ_{ms2}	$1.2 * \delta_{ms1}$	Estimated
δ_{mr1}	0.0019	Akbari et al. (2012)
δ_{mr2}	$1.2 * \delta_{mr1}$	Estimated
δ_{tp1}	0.002	Mushayabasa (2011)
δ_{tp2}	$1.2 * \delta_{tp1}$	Estimated
σ_{m1}	1.1	Assumed
σ_{m2}	1.2	Assumed
σ_t	1.01	Assumed
σ_r	1.05	Assumed
γ_{tp}	0.0357	Adetunde (2008)
φ	$1.97 * 10^{-11}$	Mushayabasa (2011)
κ	10	Assumed
h_m	1.1	Assumed
ρ_{tp}	10	Mutua et al. (2015)
φ_1	1.01	Assumed
φ_2	1.02	Assumed

6.1 Strategy A: Precautionary Measures Against the Development Of Malaria ($u_1 \neq 0$)

Strategy A,when implemented gives simulation outcomes of the optimal control model. Figure 2 reveals that the number of individuals with untreated Malaria were decreased significantly as 1,053 new infected cases were prevented. The graph of this strategy reveals that by executing the control Strategy A ($u_1 \neq 0$), the Untreated Malaria population rose steadily to its apex (1,554) between the 2nd and 3rd months, then dropped to 599 individuals with the infection in 6months.

Figure 3 reveals that the population of individuals with Treated Malaria were significantly reduced, as 550 newly infected cases were prevented. This graph further shows that by executing the control Strategy A ($u_1 \neq 0$), the infected population rose to its apex (853) in the 3rd month and then dropped to 369 individuals with Treated Malaria in 6months

Figure 4 shows that the population of individuals infected with Resistant Malaria decreased significantly, as 8,406 newly infected cases were prevented. The graph also reveals that by executing the control Strategy A ($u_1 \neq 0$), the infected population rose consistently to its apex (6,574)in a little over 4months and then dropped to 4,244 individuals with Resistant Malaria in 6months.

For individuals co-infected with Untreated Malaria and Typhoid Fever, Figure 5 shows that Strategy A ($u_1 \neq 0$) had a significant impact having prevented 1,266 new cases of the co-infection. Figure 6 reveals that the population of individuals co-infected with Treated Malaria and Typhoid Fever were drastically decreased as 2,755 newly infected cases were prevented. Figure 7 as well reveals that the population of individuals co-infected with Resistant Malaria and Typhoid Fever were drastically reduced as 19,520 newly infected cases were prevented.

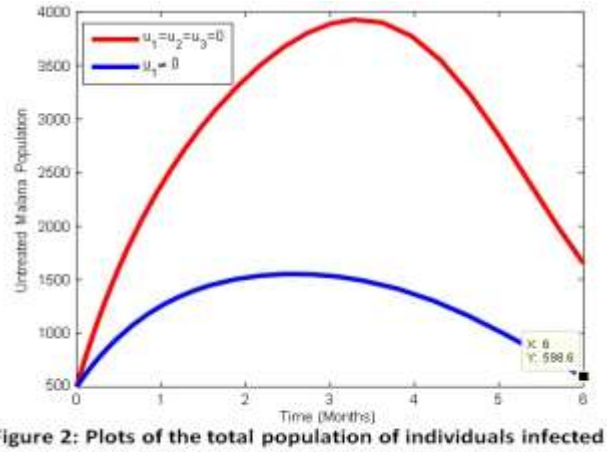


Figure 2: Plots of the total population of individuals infected wit Untreated malaria when strategy A is executed ($u_1 \neq 0$). All parameters are as in Table 2

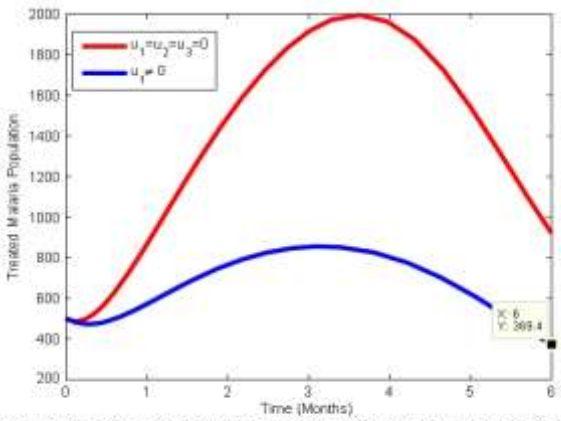


Figure 3: Plots of the total number of individuals infected with Treated malaria when strategy A is implemented ($u_1 \neq 0$). All parameters are as in Table 3

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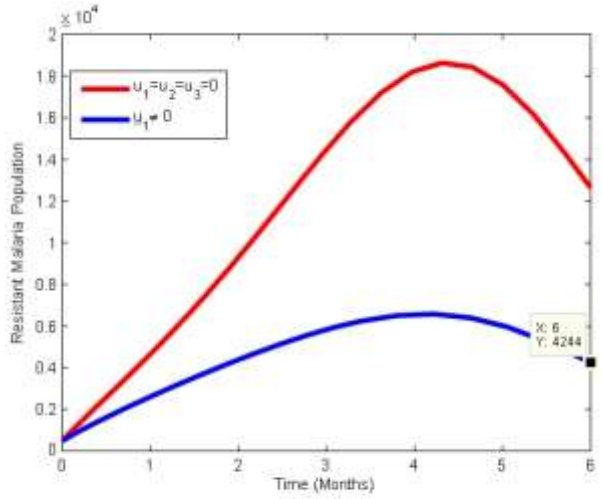


Figure 4: Plots of the total population of individuals infected with Resistant malaria when Strategy A is executed ($u_1 \neq 0$). All parameters are as in Table 2

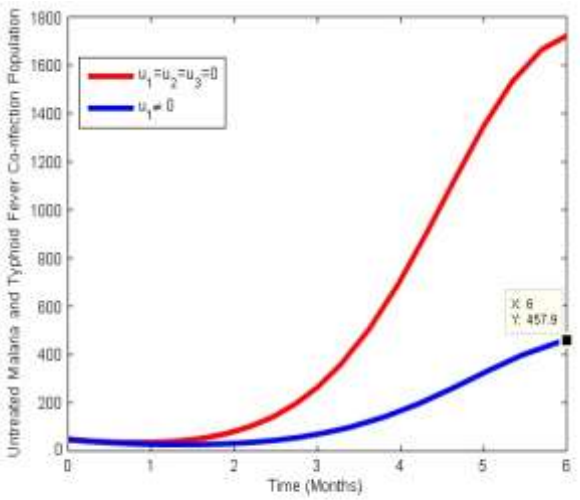


Figure 5: Plots of the total number of individuals co-infected with Untreated malaria and Typhoid fever when strategy A is implemented ($u_1 \neq 0$). All parameters are as in Table 2

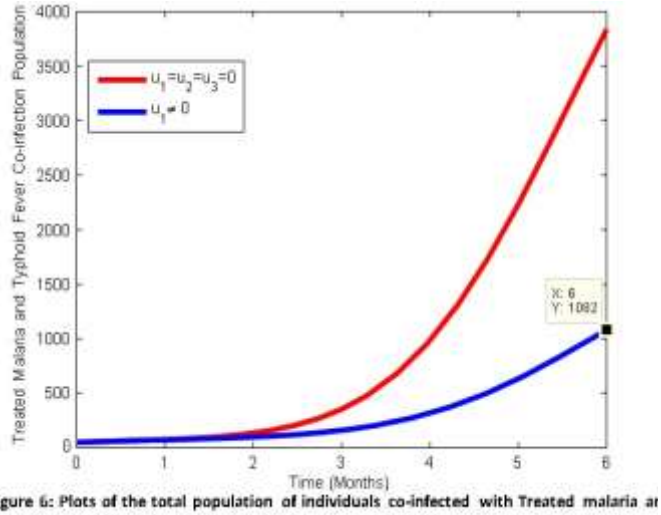


Figure 6: Plots of the total population of individuals co-infected with Treated malaria ar Typhoid fever when strategy A is executed ($u_1 \neq 0$). All parameters are as in Table 2

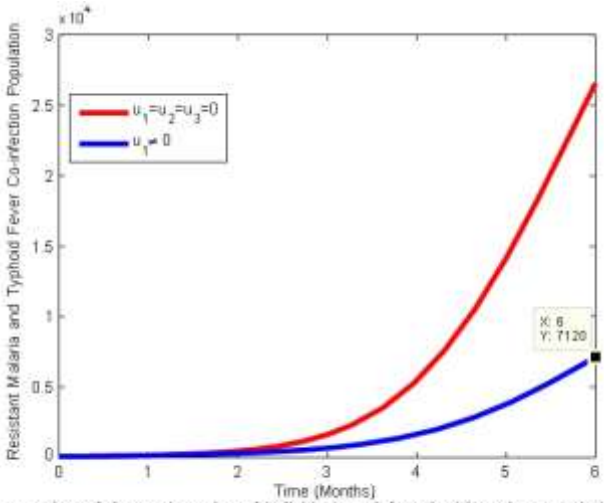


Figure 7: Plots of the total number of individuals co-infected with Resistant malaria an Typhoid fever when strategy A is implemented ($u_1 \neq 0$). All parameters are as in Table 2

6.2 Strategy B: Precautionary Measures Against the Development Of Typhoid Fever from bacteria ($u_2 \neq 0$) and Precautionary Measures Against the Development Of Typhoid Fever from humans ($u_3 \neq 0$)

Simulation results of the optimal control system when Strategy B (precautionary measures against the development of typhoid fever from bacteria ($u_2 \neq 0$) and precautionary measures against the development of typhoid fever from humans ($u_3 \neq 0$)) are obtained. Figure 8 reveals that the population of individuals infected with Typhoid Fever drastically reduced, as 783,000 new infections were prevented. Figure 9 shows that implementing Strategy B ($u_2 \neq 0$; $u_3 \neq 0$) on the co-infection population of Untreated Malaria and Typhoid Fever had a significant impact and prevented 1,230 new cases of the co-infection

Figure 10 reveals that the population of individuals co-infected with Treated Malaria and Typhoid Fever drastically decreased as 2,858 new cases of infection were prevented. Figure 11 reveals that the population of individuals co-infected with Resistant Malaria and Typhoid fever also decreased drastically as 19,438 new cases of infection were prevented.

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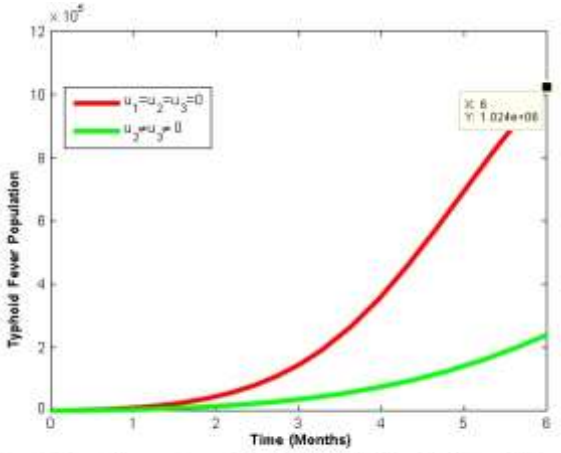


Figure 8: Plots of the total population of individuals infected with Typhoid fever when strategy B is executed ($u_2 \neq 0$; $u_3 \neq 0$). All parameters are as in Table 2

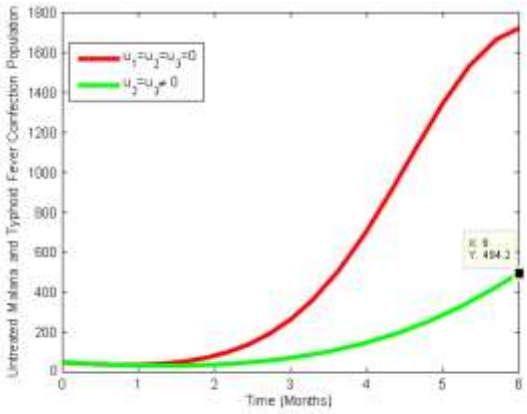


Figure 9: Plots of the total number of individuals co-infected with Untreated malaria and Typhoid fever when strategy B is implemented ($u_2 \neq 0$; $u_3 \neq 0$). All parameters are as in Table 2

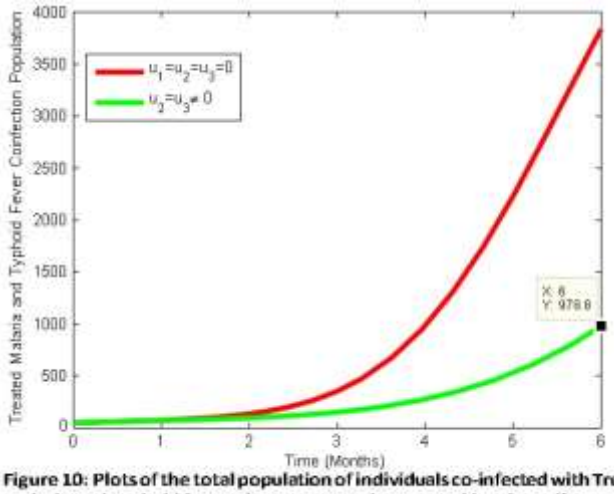


Figure 10: Plots of the total population of individuals co-infected with Treated malaria and Typhoid fever when strategy B is executed ($u_2 \neq 0$; $u_3 \neq 0$). All parameters are as in Table 2

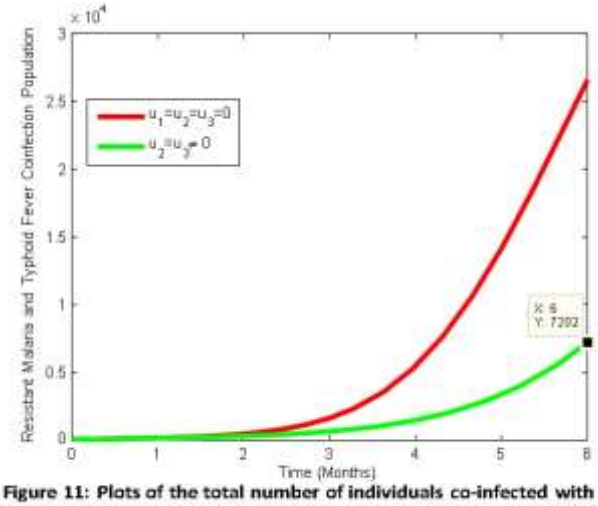


Figure 11: Plots of the total number of individuals co-infected with Resistant malaria and Typhoid fever when strategy B is implemented ($u_2 \neq 0$; $u_3 \neq 0$). All parameters are as in Table 2

he D from Against the Development Of Typhoid Fever from humans ($u_3 \neq 0$)

For Strategy C (precautionary measures against the development of malaria ($u_1 \neq 0$); precautionary measures against the development of typhoid fever from bacteria ($u_2 \neq 0$) and precautionary measures against the development of typhoid fever from humans ($u_3 \neq 0$)), Figure 12 shows that when this intervention strategy is implemented, the total number of individuals co-infected with Untreated Malaria and Typhoid Fever drastically dropped from 1,724 to 67, thereby preventing 1,657 new cases of the co-infection after a period of 6months.

Figure 13 reveals that the total population of individuals co-infected with Treated Malaria and Typhoid Fever drastically dropped from 3,837 to 227, thereby preventing 3,610 fresh cases of the co-infection after a period of 6months.
Figure 14 shows that the total population of individuals co-infected with Resistant Malaria and Typhoid fever drastically dropped from 26,640 to 1,518, thereby preventing 25,122 fresh cases of the co-infection after a period of 6months.

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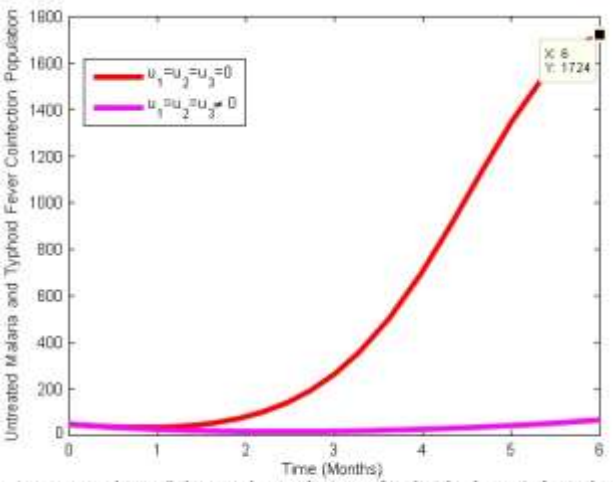


Figure 12: Plots of the total population of individuals co-infected with Untreated malaria and Typhoid fever when strategy C is executed ($u_1 \neq 0; u_2 \neq 0; u_3 \neq 0$). All parameters are as in Table 2

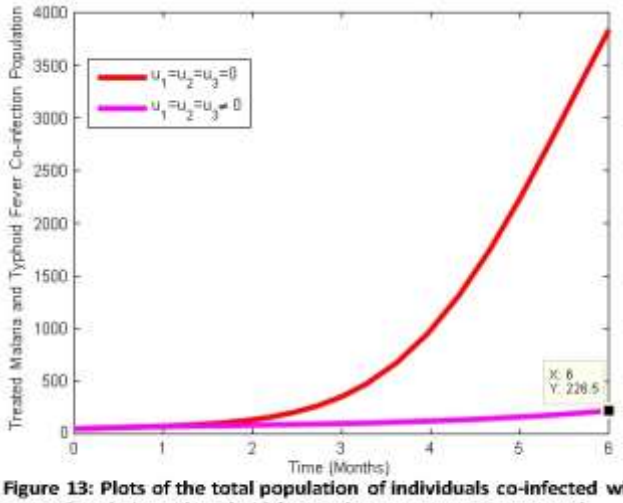


Figure 13: Plots of the total population of individuals co-infected with Treated malaria and Typhoid fever when strategy C is executed ($u_1 \neq 0; u_2 \neq 0; u_3 \neq 0$). All parameters are as in Table 2

7.0 COST-EFFECTIVENESS ANALYSIS

In this section, we aim to evaluate the most cost-effective intervention strategy in confronting Drug-Resistant Malaria and Typhoid co-infection. Two methods are utilized to accomplish this: the average cost-effectiveness ratio (ACER) and the incremental cost-effectiveness ratio (ICER). The cost-effectiveness analysis is used to assess the health-related benefits of the intervention strategies with the sole purpose of justifying the costs of the strategies (Tilahun et al. (2017)). This evaluation is achieved by comparing the differences among the health outcomes and costs of those interventions. ACER which deals with a single intervention strategy and weighing the intervention against its baseline option, is the ratio of the total cost generated by the intervention to the total number of infection prevented by the intervention. The formula is given below:

$$ACER = \frac{Total\ cost\ produced\ by\ the\ intervention\ strategy}{Total\ number\ of\ infections\ prevented\ by\ the\ strategy}$$

Similarly, ICER which is concerned with the comparison of the differences between the costs and health outcomes of two alternative intervention strategies competing for the same resources, is the ratio of the change in costs of two alternative strategies to the change in the total number of infections prevented by the two strategies. The ICER formula is given below:

$$ICER = \frac{Difference\ in\ costs\ between\ strategies}{Difference\ in\ health\ effects\ between\ strategies}$$

We calculate the total number of cases prevented and the total cost of the strategies implemented and present it in Table 4. The total number of cases prevented is derived by calculating the difference between the total number of individuals when controls are executed and the total number when control is not implemented. In like manner, we employ the cost functions $\frac{A_{11}}{2}u_1^2, \frac{A_{12}}{2}u_2^2, \frac{A_{13}}{2}u_3^2$, over time, to compute the total cost for the various strategies that were implemented. We suppose the weight constants to be: $A_{11} = 700, A_{12} = 600, A_{13} = 500$. We also assume here that:

- The cost of executing the malaria prevention control is higher when compared with the cost of executing the preventive control for bacteria transmitted typhoid fever which mainly involves mass cleaning of environments.
- The cost of implementing the bacteria transmitted typhoid fever prevention control is higher when compared with the cost of executing the preventive control for human-to-human transmitted typhoid fever which mainly involves personal hygiene.

Employing the formula given above, we compute the ACER for the control strategies as shown in Table 4.

Table 4: Increasing Order of the Total Infections Prevented Due to the Control Strategies (ACER)

Strategy	Total Infection Prevented	Total Cost	ACER
Strategy C	30,389	1,800	0.05923195893
Strategy A	33,550	700	0.02086438152
Strategy B	803,668	1,100	0.001368724399

Also, employing ICER method above, we then compare the cost effectiveness of Strategy C (precautionary measures against the development of malaria ($u_1 \neq 0$); precautionary measures against the development of typhoid fever from bacteria ($u_2 \neq 0$) and precautionary measures against the development of typhoid fever from humans ($u_3 \neq 0$)) and Strategy A

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(precautionary measures against the development of malaria $(u_1 \neq 0)$).

$$ICER(C) = \frac{1800}{30{,}389} = 0.05923195893$$

$$ICER(A) = \frac{1800 - 700}{30{,}389 - 33{,}550} = -0.347991142$$

Looking at the above computation of ICER(C) and ICER(A), we observe that ICER(C) is greater than ICER(A), revealing that Strategy A is less expensive and more cost effective than Strategy C, therefore Strategy C is removed from the subsequent ICER computation. The result can be seen in Table 5.

Table 5: Increasing Order of the Total Infection Prevented Due to the Control Strategies C and A

Strategy	Total Infection Prevented	Total Cost	ACER	ICER
C	30,389	1,800	0.059232	0.059232
A	33,550	700	0.0208644	-0.347991

Having eliminated Strategy C, we then compare the cost effectiveness of Strategy A (precautionary measures against the development of malaria $(u_1 \neq 0)$) and Strategy B (precautionary measures against the development of typhoid fever from bacteria $(u_2 \neq 0)$ and precautionary measures against the development of typhoid fever from humans $(u_3 \neq 0)$).

$$ICER(A) = \frac{700}{33{,}550} = 0.02086438152$$

$$ICER(B) = \frac{700 - 1{,}100}{33{,}550 - 803.668} = 0.000519400923$$

Observing the above computation of ICER(A) and ICER(B), we notice that ICER(A) is greater than ICER(B), revealing that Strategy A is more expensive and less cost effective than Strategy B. Table 6 shows the comparison between the cost effectiveness of Strategy C, Strategy A and Strategy B.

Table 6: Increasing Order of the Total Infection Prevented Due to the Control strategies C, A and B

Strategy	Total Infection Prevented	Total Cost	ACER	ICER
C	30,389	1,800	0.059232	0.059232
A	33,550	700	0.0208644	-0.347991
B	803,668	1,100	0.0013687	0.000519

From the above computations and comparisons, we observe that Strategy B has the least ICER and is therefore the most cost effective of the three control strategies for the prevention of the co-infection of Drug-Resistant Malaria and Typhoid Fever.

8.0 CONCLUSION

We 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. We extended the full co-infection model by incorporating optimal controls and enforcing optimal control intervention strategies. In order to accomplish this, we obtained the Hamiltonian, the adjoint variables , the characterization of the controls and the optimality system. We simulated the optimality system by considering and implementing three different control strategies as follows:

i. Precautionary Measures Against the Development Of Malaria $(u_1 \neq 0)$
ii. Precautionary Measures Against the Development Of Typhoid Fever from bacteria $(u_2 \neq 0)$ and Precautionary Measures Against the Development Of Typhoid Fever from humans $(u_3 \neq 0)$
iii. Precautionary Measures Against the Development of Malaria $(u_1 \neq 0)$; Precautionary Measures Against the Development Of Typhoid Fever from bacteria $(u_2 \neq 0)$ and Precautionary Measures Against the Development Of Typhoid Fever from humans $(u_3 \neq 0)$

For the purpose of simulation, data relevant to the dynamics of the diseases in Lagos, Nigeria was used. We numerically analysed for cost effectiveness, determining the least and most expensive strategies by using ACER and ICER technique. From the comparison result we deduced that, Strategy B, which is implementing precautionary measures against development of Typhoid fever from bacteria by periodic environmental sanitation (mass cleaning of environments and ponds to protect humans from typhoid causing bacteria) and precautionary measures against development of Typhoid fever from humans by proper personal sanitation and hygiene, is the most cost effective strategy with health benefits as it had the ability to significantly prevent 783,000 fresh cases of Typhoid fever infection. Furthermore, for the Untreated Malaria and Typhoid fever co-infection population , the strategy prevented 1,230 new cases of infection while the strategy prevented 2,858 new cases of infection when implemented on the Treated Malaria and Typhoid fever co-infection population. This strategy also prevented 19,438 new cases of infection when implemented on the Resistant Malaria and Typhoid fever co-infection population.

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9.0 CONFLICT OF INTEREST

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

10.0 FUNDING NOT APPLICABLE

11.0 DATA AVAILABILTY

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

12.0 ACKNOWLEDGEMENTS

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