MATHEMATICAL MODELLING OF MALARIA AND TYPHOID CO-INFECTION INCORPORATING VECTOR AND LOSS OF IMMUNITY

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Abstract: This paper proposes a deterministic mathematical model for the transmission dynamics of malaria and typhoid co-infection incorporating loss of immunity. The sub models and the malaria and typhoid co-infection model were analyzed. The next generation matrix method was used to obtain the basic reproduction number; we also obtained the disease-free equilibrium for malaria sub model, typhoid sub model and co-infection model. Local and global stability of the system were obtained using Ruth Hurwitz criterion and Castillo-Chavez method, respectively. The results of our study shows that the disease-free point is locally asymptotically stable when $R < 1$. The model is also globally asymptotically stable, indicating that the disease eradication is independent on the initial population size. Numerical experiments were conducted using MATLAB R2015a, we observed that infected human population increases for both malaria and typhoid with an increased value of mosquito biting rate and the result also indicate that the best way of minimizing or eradicating malaria and typhoid in a population is to keep the transmission probabilities of malaria by mosquitoes and typhoid transmission rate to a barest minimal which will in turn keep reproduction number below one (a condition for disease eradication) $R < 1$.

Keywords: Co-infection, loss of immunity, local stability, global stability

Introduction

Malaria is an infectious disease caused by plasmodium parasite which is transmitted to humans through the bites of infectious female mosquitoes. In 2017, there were an estimated 219 million cases of malaria in 87 countries. The estimated number of malaria deaths stood at 435 000 in 2017. African Region carries a disproportionately high share of the global malaria burden (WHO, 2017). According to the estimations of World Health Organization (WHO) in 2015, 3.2 billion persons were at risk of infection and 2.4 million cases were detected with 438,000 cases of deaths. However Sub- Sahara Africa remain the most vulnerable region with rate due to malaria (Traoré *et al.,* 2017). Typhoid is a nickname given to *Salmonella typhi* named after "Typhoid Mary" popularly known as Mary Mallon, a healthy carrier who had become synonymous with the spread of the bacteria where many are infected due to her denial of being ill, Mary Mallon was born in 1869 thereafter emigrated to the US in 1884 (Marineli *et al.,* 2013).

Typhoid fever is a systemic infection caused by *Salmonella typhi*, usually through ingestion of contaminated food or water. There are other ways of getting infected by the bacteria. This acute illness is characterized by prolonged fever, headache, and nausea, loss of appetite, and constipation or sometimes diarrhea. Symptoms are often non-specific and clinically non-distinguishable from other febrile illnesses. However, clinical severity varies and severe cases may lead to serious complications or even death. It occurs predominantly in association with poor sanitation and lack of clean drinking water. Globally, according to the most recent estimates, between 11 and 21 million cases and 128 000 to 161 000 typhoid-related deaths occur annually worldwide. A similar but often less severe disease, paratyphoid fever, is caused by *Salmonella paratyphi* A and B (or uncommonly *Paratyphi* C) (WHO, 2018).

The *Salmonella typhi* enters through the mouth and spends 1 to 3 weeks in the intestine. After this, it makes its way through the intestinal wall and into the bloodstream. From the bloodstream, it spreads into other tissues and organs. The immune system of the host can do little to fight back because *S. typhi* can live within the host's cells, safe from the immune system. Typhoid is diagnosed by detecting the presence of *S. typhi* via blood, stool, urine, or bone marrow sample. According to the most recent estimates between 11 and 21 million cases and 128,000 to 161,000 typhoid-related deaths occur annually worldwide (WHO, 2017). A study on Incidence of Malaria/Typhoid Co-Infection among adult population in Unwana community, Afikpo North Local Government area, Ebonyi State, Nigeria was carried out.

The population of their study is 350 individuals, the 350 individuals were examined and their blood samples were subjected to microscopic examination and widal test for malaria and typhoid, also questionnaire was administered to obtained information on management practices of the infection. Their results show that out of the 350 blood samples analyzed, 190 positive for malaria, 173 positive for typhoid, while 127 were positive for both malaria and typhoid. However, their statistics in relation to sex, shows that, males had 50%, females 58% for malaria, for typhoid males had 57%, females 42%, while for co-infection of malaria/typhoid, males had 35%, while females had 37%. The management practices, 80% prefer environmental sanitation as best method for prevention/control, 48% preferred herbal remedies, while appreciable number adopted self-treatment method. They concluded that co-infection was higher in females than males, they advocate for improve living condition (Odikamnoro *et al.,* 2017).

Mathematical Formulation

For the purpose of describing the natural dynamics, we compartmentalized the population of this study into ten compartment namely: Protected human from malaria and typhoid P_T, Susceptible human S_H, Infected human with typhoid I_{HT}, Treated human from typhoid T_{HT}, Exposed human to malaria E_{HM}, Infected human with malaria I_{HM} Recovered human from malaria R_{HM}, infected human with both malaria and typhoid I_{HT}^{HM}, Susceptible mosquito S_M and Infected mosquito I_M. Now, human are recruited into the susceptible compartment by the following parameters; $(1-\alpha)\Lambda$, γP_T, $\rho_2 T_{HT}$, and $\rho_1 R_{HM}$, when susceptible

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humans come in contact with the parasite or bacteria or both, he may be infected. The infected human remain infected and infectious for some period before recovery (naturally or treatment) or die naturally or as result of the disease (Chin, 2000).

The above dynamics may results into the following; The susceptible S_H, is populated by natural birth $(1-\alpha)\Lambda$, and reduces by $\mu_1 S_H$, $\frac{\pi\theta I_{HT}S_H}{N}$, $\frac{\varepsilon\beta S_H I_M}{N}$ and while natural death is μ_1 disease induced death is δ_1, as the susceptible human S_H interact with infectious mosquito that usually attacks human they progresses into the exposed compartment E_{HM}. At this stage, the exposed human E_{HM}, becomes infectious and enters the infected compartment I_{HM} at a rate α_1 and ψ which is as a result of new birth, that is an infected nursing mother passing to her new born, and the infected I_{HM} individual remain infected and infectious for some time before they are recover either naturally or by way of treatment and the infected compartment reduces by $\mu_1 I_{HM}$, δI_{HM} due to natural death and death as a result of malaria infection. Those that recovered naturally or by way treatment enters the recovered compartment R_{HM} at the rate α_2 and the recovered individuals immediately loss there immunity and re-enters the susceptible compartment at a rate ρ_1 and the population of the recovered reduces by natural death. The recruitment into susceptible compartment of mosquito S_M is natural birth Λ_M , when susceptible mosquito interacts with infectious humans with malaria I_{HM}, at this stage, S_M become infected and enters mosquito infected compartment

I_M at a rate ε , the infected mosquito I_M remained infected and infectious, there after interacts with susceptible humans and get them infected, also I_{HM} is depopulated natural death μ_2 . The protection against typhoid is for a while, when the protected humans loss their immunity, they progress into susceptible compartment at the rate γ, at this stage, S_H is exposed and upon interaction with I_{HT} that is human with typhoid, they move to I_{HT} compartment at a rate π . The infected human with typhoid I_{HT} remains infected and infectious for some time before they recover either naturally or by way of treatment, the population of I_{HM} compartment is depleted by natural death or as a result of the infection μ_1 and δ_1 respectively. The infected I_{HT} after treatment, I_{HT} progress into the treated compartment T_{HT} at a rate β_1 and the population of T_{HT} reduces by natural death and the remaining proportion move back to susceptible compartment S_H at rate ρ_2. Chances are that an individual have both malaria and typhoid at the same time, one cannot stop the other and that is why we have a co-infection compartment I_{HT}^{HM} , the compartment is populated by $\frac{\varepsilon\beta I_{HM}I_{HT}}{N}$, $\frac{\pi\theta I_M I_{HT}}{N}$, and the co-infection compartment is depopulated by natural death μ_1 and death as a result of either one malaria or typhoid or both infections (Chin, 2000). Both infections are treatable and preventable.
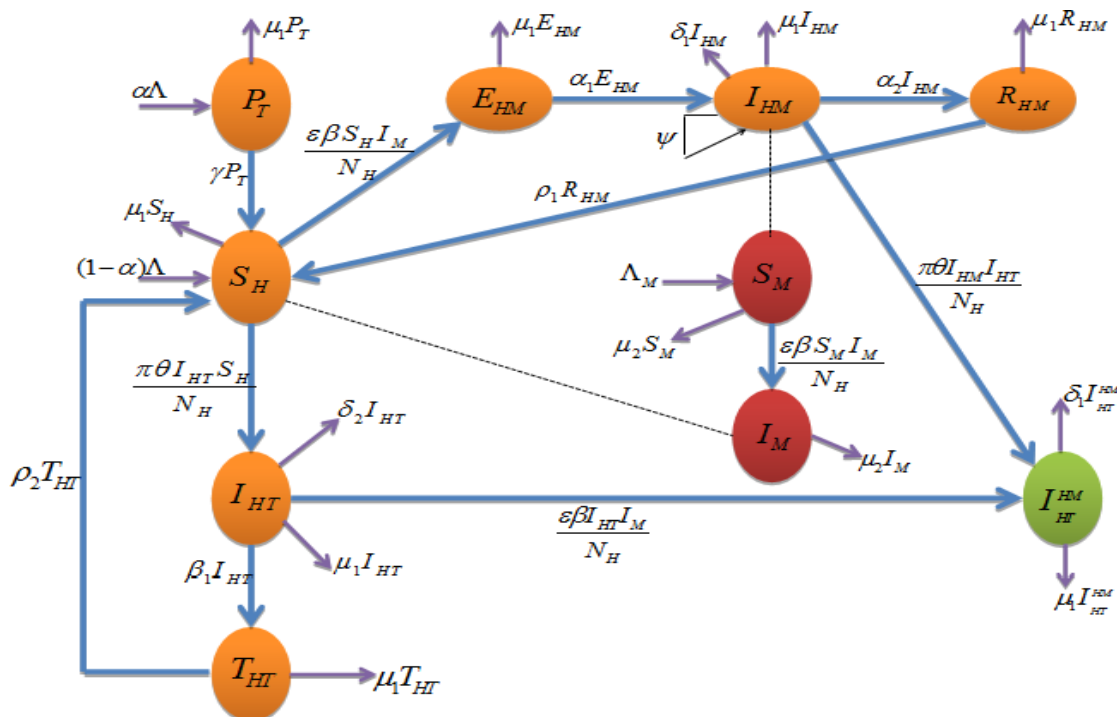

Fig. 1: Malaria and typhoid co-infection diagram

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Model equations

$$\frac{dP_T}{dt} = \alpha\Lambda - (\mu_1 + \gamma)P_T$$

$$\frac{dS_H}{dt} = (1-\alpha)\Lambda - \mu_1 S_H - \frac{\varepsilon\beta I_M S_H}{N_H} - \frac{\pi\theta I_{HT} S_H}{N_H} + \gamma P_T + \rho_2 T_{HT} + \rho_1 R_{HM}$$

$$\frac{dI_{HT}}{dt} = \frac{\pi\theta I_{HT} S_H}{N_H} - \frac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1 + \delta_2 + \beta_1)I_{HT}$$

$$\frac{dT_{HT}}{dt} = \beta_1 I_{HT} - (\mu_1 + \rho_2)T_{HT}$$

$$\frac{dE_{HM}}{dt} = \frac{\varepsilon\beta S_H I_M}{N_H} - (\mu_1 + \alpha_1)E_{HM}$$

$$\frac{dI_{HM}}{dt} = \alpha_1 E_{HM} - \frac{\pi\theta I_{HM} I_{HT}}{N_H} - (\mu_1 + \delta_1 + \alpha_2)I_{HM} + \psi I_{HM}$$

$$\frac{dR_{HM}}{dt} = \alpha_2 I_{HM} - (\mu_1 + \rho_1)R_{HM}$$

$$\frac{dI_{HT}^{HM}}{dt} = \frac{\pi\theta I_{HM} I_{HT}}{N_H} + \frac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1 + \delta_1)I_{HT}^{HM}$$

$$\frac{dS_M}{dt} = \Lambda_m - \frac{\varepsilon\beta S_M I_{HM}}{N_H} - \mu_2 S_M$$

$$\frac{dI_M}{dt} = \frac{\varepsilon\beta S_M I_{HM}}{N_H} - \mu_2 I_M$$

(1)

With initial conditions

$$P_T(0) = P_{T_0}, S_H(0) = S_{H_0}, = I_{HT}(0) = I_{HT_0}, = T_{HT}(0) = T_{HT_0}, E_{HM}(0) = E_{HM_0},$$

$$I_{HM}(0) = I_{HM_0}, R_{HM}(0) = R_{HM_0}, I_{HT}^{HM}(0) = I_{HT_0}^{HM}(t), S_M(0) = S_{M_0}, I_M(0) = I_{M_0}$$

Table 1: Variables/Parameter of the co-infection model

Parameter	Description
$P_T(t)$	Protected human against malaria and typhoid at time t
$S_H(t)$	Susceptible human at time t
$I_{HT}(t)$	Infected human with typhoid at time t
$T_{HT}(t)$	Treated human with typhoid at time t
$E_{HM}(t)$	Expose human to mosquito bite at time t
$I_{HM}(t)$	Infected human with malaria at time t
$R_{HM}(t)$	Recovered human with malaria at time at time t
$I_{HT}^{HM}(t)$	Human infected with both malaria and typhoid at time t
$I_M(t)$	Infected mosquito at time t
$\alpha\Lambda$	Recruitment into protected compartment
$(1-\alpha)\Lambda$	Recruitment into susceptible compartment
Λ_m	Recruitment of mosquito into susceptible compartment
μ_1	Natural death of human
μ_2	Natural death of mosquito
δ_1	Disease induce death of human
δ_2	Disease induce death of mosquito
ρ_1	Recovery rate of humans from malaria in to susceptible
ρ_2	Recovery rate humans from typhoid into susceptible
α_1	Rate at which get infected with malaria
α_2	Recovery rate of human from malaria
γ	Protected humans rate into susceptible
β_1	Rate of human treated of typhoid
π	Probability of humans infected with typhoid
ε	Biting rate of mosquito
β	Transmission probability of malaria by mosquito
θ	Transmission probability of typhoid by humans

Analysis of malaria and typhoid co-infection model
Existence and positivity of solution
Here, we provide the following results which guarantee that the malaria and typhoid co-infection governed by the system (1) is epidemiologically and mathematically well-posed in a feasible region D given by

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$$D = D_H \times D_M \subset \mathbb{R}_+^5 \times \mathbb{R}_+^2,$$

Where

$$D_H = \left\{ (P_T, S_H, E_{HM}, I_{HM}, R_{HM}) \in \mathbb{R}_+^8 : N_H \leq \frac{\Lambda}{\mu_1} \right\}$$

$$D_M = \left\{ (S_M, I_M) \in \mathbb{R}_+^2 N_M \leq \frac{\Lambda_M}{\mu_2} \right\}$$

Malaria sub model
We first consider Malarial model only

$$\frac{dP_T}{dt} = \alpha\Lambda - (\mu_1 + \gamma)P_T$$

$$\frac{dS_H}{dt} = (1-\alpha)\Lambda - \mu_1 S_H - \frac{\varepsilon\beta I_M S_H}{N_H} + \rho_1 R_{HM}$$

$$\frac{dE_{HM}}{dt} = \frac{\varepsilon\beta I_M S_H}{N_H} - (\mu_1 + \alpha_1)E_{HM}$$

$$\frac{dI_{HM}}{dt} = \alpha_1 E_{HM} - (\alpha_2 + \mu_1 + \delta_1)I_{HM}$$

$$\frac{dR_{HM}}{dt} = \alpha_2 I_{HM} - (\mu_1 + \rho_1)R_{HM}$$

$$\frac{dS_M}{dt} = \Lambda_M - \mu_2 S_M - \frac{\varepsilon\beta I_{HM} S_M}{N_H}$$

$$\frac{dI_M}{dt} = \frac{\varepsilon\beta I_{HM} S_M}{N_H} - \mu_2 I_M$$

(2)

With initials conditions
$P_T(0) = P_{0T}, S_H(0) = S_{0H}, E_{HM}(0) = E_{0HM}, I_{HM}(0) = I_{0HM},$
$R_{HM}(0) = R_{0HM}, S_M(0) = S_{0M}, I_M(0) = I_{0M}$

Disease free equilibrium of malaria model
From the system (2) At malaria free,

$$I_{HM} = E_{HM} = R_{HM} = I_M = 0$$

Therefore, the disease free equilibrium for the malaria sub-model is;

$$E_0 = \left(\frac{\alpha\Lambda}{(\mu_1 + \gamma)}, \frac{(1-\alpha)\Lambda}{\mu_1}, 0, 0, 0, \frac{\Lambda_M}{\mu_2}, 0 \right)$$

Basic reproduction number of malaria sub model

The basic reproduction number (R_0) of malaria sub model

from system (2) is the number of secondary infections produced by an infectious individual introduced during the period of infectiousness into totally susceptible population.

We can find $R_0 = \rho(FV^{-1})$

$$R_0^M = \sqrt{\frac{\alpha_1\varepsilon^2\beta^2\Lambda_M\mu_1(1-\alpha)}{\mu_2^2\Lambda(\mu_1+\alpha_1)(\mu_1+\delta_1+\alpha_2-\psi)}}$$

Local stability of the disease free equilibrium for malaria model

Theorem 1: The disease free equilibrium E_0 point of

malaria model is locally asymptotically stable if $R_0 < 1$ and

unstable if $R_0 > 1$. Theorem1 is achieve by

$$\left| J(E_O) - \lambda I \right| = 0$$
(3)

Therefore, all the eigen values of the equation (3) have

negative real parts, implying that $\lambda_6 < 0, \lambda_7 < 0$ Since all

the values of $\lambda_i < 0$, for $i = 1, 2, 3, 4, 5, 6, 7$ when

$R_0 < 1$ we conclude that the disease-free equilibrium point is locally asymptotically stable.

Typhoid sub model
We consider Typhoid Sub model only.

$$\frac{dP_T}{dt} = \Lambda\alpha - (\mu_1 + \gamma)P_T$$

$$\frac{dS_H}{dt} = (1-\alpha)\Lambda - \frac{\pi\theta I_{HT} S_H}{N_H} - \mu_1 S_H + \gamma P_T + \rho_2 T_{HT}$$

$$\frac{dI_{HT}}{dt} = \frac{\pi\theta I_{HT} S_H}{N_H} - (\mu_1 + \delta_2 + \beta_1)I_{HT}$$

$$\frac{dT_{HT}}{dt} = \beta_1 I_{HT} - (\mu_1 + \rho_2)T_{HT}$$

(4)

With initial solution set
$\{ P_T(0) = P_{T0}, S_H(0) = S_{H0}, I_{HT}(0) = I_{HT0}, T_{HT}(0) = T_{HT0} \} \in \mathfrak{R}_+^4$

Disease free equilibrium of malaria model
From the system (4) at the disease free equilibrium

$I_{HT} = T_{HT} = 0$, the given system becomes

$$E_{0T} = \left(P_T^0, S_H^0, I_{HT}^0, T_{HT}^0 \right) = \left(\frac{\Lambda\alpha}{(\mu_1+\gamma)}, \frac{(1-\alpha)\Lambda}{\mu_1} + \frac{\gamma\Lambda\alpha}{\mu_1+\gamma}, 0, 0 \right)$$

Basic reproduction number for typhoid model

The basic reproduction number (R_0) of typhoid sub model

from system (4) is the number of secondary infections produced by an infectious individual introduced during the period of infectiousness into totally susceptible population.

We can find $R_0 = \rho(FV^{-1})$

$$R_0^T = \frac{\pi\theta(1-\alpha)}{(\mu_1 + \delta_2 + \beta_1)}$$

Local stability at disease free equilibrium of typhoid model
Theorem 2: The disease free equilibrium E_0 point of

typhoid model is locally asymptotically stable if $R_0^T < 1$ and

unstable if $R_0^T > 1$

$$|J(E_0) - \lambda I| = \begin{vmatrix} -b_1 - \lambda & 0 & 0 & 0 \\ b_2 & -b_3 - \lambda & -b_4 & b_5 \\ 0 & 0 & -b_6 - \lambda & 0 \\ 0 & 0 & b_7 & -b_8 - \lambda \end{vmatrix} = 0$$

(5)

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Where

$$b_1 = (\mu_1 + \gamma),$$
$$b_2 = \gamma, b_3 = \mu_1,$$
$$b_4 = \pi\theta(1-\alpha) + \frac{\gamma\alpha\mu_1\theta\pi}{\mu_1 + \gamma},$$
$$b_5 = \rho_2, b_7 = \beta_1, b_8 = (\mu_1 + \rho_2)$$
$$b_6 = (\mu_1 + \delta_2 + \beta_1) - \left(\pi\theta(1-\alpha) + \frac{\gamma\alpha\mu_1\theta\pi}{\mu_1 + \gamma}\right),$$

From system (5) all the values of $\lambda_i < 0$, for $i = 1, 2, 3, 4$ when $R_0^T < 1$ we conclude that the disease-free equilibrium point is locally asymptotically stable.

Co-infection model: The Malaria and typhoid model (1) has a DFE given by

$$E_0 = (\frac{\alpha\Lambda}{(\mu_1 + \gamma)}, \frac{(1-\alpha)\Lambda}{\mu_1} + \frac{\gamma\Lambda\alpha}{\mu_1 + \gamma}, 0, 0, 0, 0, 0, 0, \frac{\Lambda_M}{\mu_2}, 0)$$

The linear stability of E_0 can be established using next generation method (Diekmann *et al.,* 1990) on the system (1). It follows that the reproduction number of the malaria typhoid model (1) is denoted by R_0^{MT}. **where**

$$R_0^{MT} = \sqrt{\frac{\left(\varepsilon\beta(1-\alpha) + \frac{\gamma\alpha\varepsilon\beta}{(\mu_1 + \gamma)\mu_1}\right)\frac{\varepsilon\beta\alpha_1\Lambda_M\mu_1}{\mu_2\Lambda}}{(\mu_1 + \alpha_1)(\mu_1 + \delta_1 + \alpha_2 - \psi)\mu_2}}$$

Local stability of the disease equilibrium of co-infection model

Theorem 3: The disease free equilibrium point E_0 is locally asymptomatically stable if $R_0 < 1$ and unstable if $R_0 > 1$

Proof;
Let

$$F_1 = \alpha\Lambda - (\mu_1 + \gamma)P_T$$

$$F_2 = (1-\alpha)\Lambda - \mu_1 S_H - \frac{\varepsilon\beta S_H I_M}{N_H} - \frac{\pi\theta I_{HT} S_H}{N_H} + \gamma P_T + \rho_2 T_{HT} + \rho_1 R_{HM}$$

$$F_3 = \frac{\pi\theta I_{HT} S_H}{N_H} - \frac{\varepsilon\beta I_M I_{HT}}{N_H} - (\mu_1 + \delta_1 + \beta_1)T_{HT}$$

$$F_4 = \beta_1 I_{HT} - (\mu_1 + \rho_2)T_{HT}$$

$$F_5 = \frac{\varepsilon\beta S_H I_M}{N_H} - (\mu_1 + \alpha_1)E_{HM}$$

$$F_6 = \alpha_1 E_{HM} - \frac{\pi\theta I_{HT} I_{HM}}{N_H} - (\mu_1 + \delta_1 + \alpha_2)I_{HM} + \psi I_{HM}$$

$$F_7 = \alpha_2 I_{HM} - (\mu_1 + \rho_1)R_{HM}$$

$$F_8 = \frac{\pi\theta I_{HT} I_{HM}}{N_H} + \frac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1 + \delta_1)I_{HT}^{HM}$$

$$F_9 = \Lambda_M - \frac{\varepsilon\beta S_M I_{HM}}{N_H} - \mu_2 S_M$$

$$F_{10} = \frac{\varepsilon\beta S_M I_{HM}}{N_H} - \mu_2 I_M$$

(7)

Evaluating (7) at disease free we have

$$J(E_0) = \begin{pmatrix} -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_2 & -a_3 & a_4 & a_5 & 0 & 0 & a_6 & 0 & 0 & a_7 \\ 0 & 0 & -a_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & -a_{11} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_{12} & 0 & 0 & 0 & 0 & a_{13} \\ 0 & 0 & 0 & 0 & a_{14} & -a_{15} & 0 & a_{16} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & a_{17} & -a_{18} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_{19} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_{20} & 0 & 0 & -a_{21} & 0 \\ 0 & 0 & 0 & 0 & 0 & a_{22} & 0 & 0 & 0 & -a_{23} \end{pmatrix}$$

(8)

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Where

$$a_1 = (\mu_1 + \gamma), a_4 = \pi\theta - \pi\theta\alpha + \frac{\gamma\alpha\pi\theta}{(\mu_1 + \gamma)\mu_1}$$

$$a_2 = \gamma, a_3 = \mu_1, a_5 = \rho_2, a_6 = \rho_1, a_7 = \varepsilon\beta - \varepsilon\beta\alpha + \frac{\gamma\alpha\varepsilon\beta}{(\mu_1 + \gamma)\mu_1}$$

$$a_8 = \pi\theta\alpha - \pi\theta - \frac{\gamma\alpha\pi\theta}{(\mu_1 + \gamma)\mu_1}, a_9 = (\mu_1 + \delta_1 + \beta_1), a_{10} = \beta_1$$

$$a_{11} = (\mu_1 + \rho_2), a_{12} = (\mu_1 + \alpha_1), a_{13} = \left(\varepsilon\beta - \varepsilon\beta\alpha + \frac{\gamma\alpha\varepsilon\beta}{(\mu_1 + \gamma)\mu_1} \right),$$

$$a_{14} = \alpha_1, a_{15} = (\mu_1 + \delta_1 + \alpha_2 - \psi), a_{16} = \frac{\pi\theta\mu_1}{\Lambda}, a_{17} = \alpha_2, a_{18} = (\mu_1 + \rho_1)$$

$$a_{19} = (\mu_1 + \delta_1) - \frac{\pi\theta\mu_1}{\Lambda}, a_{20} = \frac{\varepsilon\beta\Lambda_M\mu_1}{\mu_2\Lambda}, a_{21} = \mu_2, a_{22} = \frac{\varepsilon\beta\Lambda_M\mu_1}{\mu_2\Lambda}, a_{23} = \mu_2$$

Given

$$\left| J(E_O) - \lambda I \right| = 0 \tag{9}$$

Evaluating equation (9) using equation (8) we obtain (10)

$$\begin{vmatrix} -a_1 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_2 & -a_3 - \lambda & a_4 & a_5 & 0 & 0 & a_6 & 0 & 0 & a_7 \\ 0 & 0 & -a_8 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & -a_{11} - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_{12} - \lambda & 0 & 0 & 0 & 0 & a_{13} \\ 0 & 0 & 0 & 0 & a_{14} & -a_{15} - \lambda & 0 & a_{16} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & a_{17} & -a_{18} - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_{19} - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_{20} & 0 & 0 & -a_{21} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & a_{22} & 0 & 0 & 0 & -a_{23} - \lambda \end{vmatrix} \tag{10}$$

From (10) the reduce system gives the following polynomial equation

$$A_0\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \tag{11}$$

Where

$$A_0 = 1, A_1 = (a_{12} + a_{15} + a_{23}),$$

$$A_2 = (a_{12}a_{15} + a_{12}a_{23} + a_{15}a_{23})$$

$$A_3 = (a_{12}a_{15}a_{23} - a_{13}a_{14}a_{22})$$

$$A_3 = \left((\mu_1 + \alpha_1)(\mu_1 + \delta_1 + \alpha_2 - \psi)\mu_2 - \left(\varepsilon\beta - \varepsilon\beta\alpha + \frac{\gamma\alpha\varepsilon\beta}{(\mu_1 + \gamma)\mu_1} \right)\left(\frac{\varepsilon\beta\alpha_1\Lambda_M\mu_1}{\mu_2\Lambda} \right) \right)$$

We apply Routh-Hurwitz criterion which states that all roots of the polynomial (11) have negative real part if and only if the coefficients A_i, are positive and the determinant of the matrices D_i for $i = 0, 1, 2, 3$. Therefore from (11) we have

$$D_1 = |A_1| > 0, D_2 = A_1A_2 > A_3,$$

$$D_3 = A_3A_1A_2 > A_3^2.$$ Therefore, all the eigen values of the polynomial (4.176) have negative real parts, implying that $\lambda_8 < 0, \lambda_9 < 0, \lambda_{10} < 0$. Since all the values of $\lambda_i < 0$, for $i = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10$ when $R_0 < 1$ we conclude that the disease-free equilibrium point is locally asymptotically stable.

Globally asymptotically stable

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The globally asymptotically stable (GAS) of the disease free equilibrium of the model will be investigated using the theorem by Castilo-Chavez and Feng (1997). The model is re-written as follows:

$$\left.\begin{array}{l} \dfrac{dx}{dt} = H(X,Z) \\[2mm] \dfrac{dz}{dt} = G(X,Z), G(X,0)=0 \end{array}\right] \quad (12)$$

Where the component of the column-vector $X \in R^M$ denoting the uninfected population, where $Z \in R^N$ denoting the infected population.

$$E_0 = (X^*,0) \text{ denoting the DFE of the system.}$$

Therefore, the fixed point $E_0 = (X^*,0)$ is globally asymptotically stable equilibrium for the system provided that $R_0 < 1$ that is, (locally asymptotically stable) and the following two conditions must be satisfy.

(H_1) For $\dfrac{dx}{dt} = H(X,0),$ X^* is globally asymptotically stable

(H_2) $G(X,Z) = PZ - \hat{G}(X,Z),$ $\hat{G}(X,Z) \geq 0$

For $(X,Z) \in \eta_H$.

Where $P = D_z G(X^*,0)$ is an M-matrix (the off diagonal elements of P are non-negative) and Ω_H is the region where the model makes biological sense. If the system (7) satisfy the above two conditions, then the below theorem holds.

Theorem 4: the disease free equilibrium point E_{0M} of malaria infection is globally asymptotically stable if $R_{0M} < 1$ and the above condition are satisfied.

Proof: We have from theorem 3 that E_{0M} is asymptotically stable if $R_{0M} < 1$. Now consider

$$\frac{dS}{dt} = F(S,0) = \begin{pmatrix} \alpha\Lambda - (\mu_1 + \gamma)P_T \\ (1-\alpha) - \mu_1 S_H + \gamma P_T \\ 0 \\ 0 \\ 0 \\ \Lambda_M - \mu_2 S_M \end{pmatrix} \quad (13)$$

$$\frac{dI}{dt} = G(S,I) = \begin{pmatrix} \dfrac{\pi\theta S_H I_{HT}}{N_H} - \dfrac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1 + \delta_2 + \beta_1)I_{HT} \\[3mm] \alpha_1 E_{HM} - \dfrac{\pi\theta I_{HT} I_{HM}}{N_H} - (\mu_1 + \delta_1 + \alpha_2)I_{HM} + \psi I_{HM} \\[3mm] \dfrac{\pi\theta I_{HT} I_{HM}}{N_H} + \dfrac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1 + \delta_1)I_{HT}^{HM} \\[3mm] \dfrac{\varepsilon\beta S_M I_{HM}}{N_M} - \mu_2 I_M \end{pmatrix} \quad (14)$$

Evaluating equation (14) at DFE, we have

$$\begin{bmatrix} \pi\theta(1-\alpha) + \dfrac{\gamma\alpha\pi\theta}{(\mu_1+\gamma)\mu_1} - (\mu_1+\delta_2+\beta_1) & 0 & 0 & 0 \\[3mm] 0 & -(\mu_1+\delta_1+\alpha_2-\Psi) & 0 & 0 \\[3mm] 0 & 0 & -\left(\dfrac{\pi\theta\mu_1}{\Lambda} + \mu_1 + \delta_1\right) & 0 \\[3mm] 0 & \dfrac{\varepsilon\beta\Lambda_M\mu_1}{\Lambda\mu_2} & 0 & 0 \end{bmatrix} \quad (15)$$

Evaluating (13) and (14) we obtain (16)

$$G(X,Z) = \begin{bmatrix} G(X,Z)_1 \\ G(X,Z)_2 \\ G(X,Z)_3 \\ G(X,Z)_4 \end{bmatrix} = \begin{bmatrix} \theta\pi I_{HT}(1 - \dfrac{S_H}{N_H}) - \dfrac{\varepsilon\beta I_{HT} I_M}{N_H} - (\mu_1+\delta_2+\beta_1) \\[3mm] \theta\pi I_{HT} I_{HM} - \alpha_1 E_{HM} + (\mu_1+\delta_1+\alpha_2)I_{HM} + \psi I_{HM} \\[3mm] (\mu_1+\delta_1)I_{HT}^{HM} - \dfrac{\theta\pi I_{HT} I_{HM}}{N_H} - \dfrac{\varepsilon\beta I_{HT} I_M}{N_H} \\[3mm] \varepsilon\beta I_{HM}(1 - \dfrac{S_M}{N_M}) - \mu_2 I_M \end{bmatrix} \quad (16)$$

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Since we have that, $G(X,Z)_1, G(X,Z)_2,$ ..., $G(X,Z)_4 \geq 0$, it shows that conditions (H_1), (H_2) are met. Thus, E^0 is globally asymptotically stable whenever $R_0^{MT} < 1$.

This means the fight against *malaria* and *typhoid* may be won if cases are kept at bear minimal.

Numerical Simulation
Table 2: Parameter/Variable values used for model simulations

Symbol	Values	References
Λ_M	0.000215	Olaniyi and Obabiyi (2013)
Λ_H	0.07	Olaniyi and Obabiyi (2013)
μ_1	0.0247	Estimated
μ_2	0.5	Estimated
δ_1	0.068	Estimated
δ_1	0.012	Mushayabasa (2012)
ρ_1	0.00017	Estimated
ρ_2	0.001	Estimated
α_1	0.0696	Chitnis *et al.* (2006)
α_2	0.0035	Estimated
γ	0.0044	Estimated
β_1	0.9	Estimated
θ	0.01	Mushayabasa (2012)
ε	0.46	Jia (2015)
β	0.1	Olaniyi and Obabiyi (2013)
π	0.354	Assumed
ψ	0.003	Assumed
P_T	100	Estimated
S_H	800	Silva. & Torres (2013)
I_{HT}	200	Estimated
T_{HT}	0	Estimated
E_{HM}	20	Okosun & Makinde (2013)
I_{HM}	10	Olaniyi and Obabiyi (2013)
R_{HM}	0	Olaniyi and Obabiyi (2013)
$I_{HM}I_{HT}$	5	Estimated
S_M	9500	Okosun & Makinde (2013)
I_M	30	Okosun & Makinde (2013)
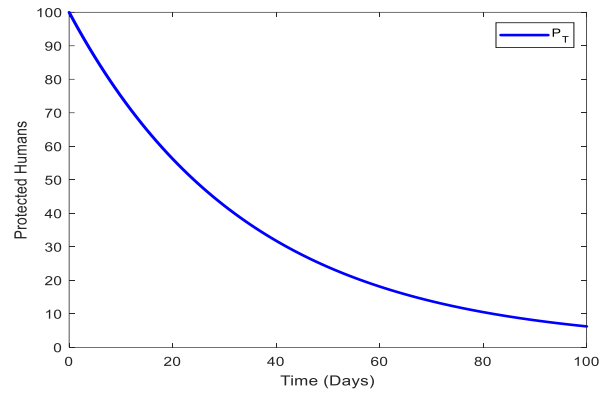
Fig. 2: The dynamics of protected humans against typhoid over a period of 100 days

The Fig. 2 shows that protected population reduced significantly because protection against the infection is not forever, therefore, as a result of lost immunity individuals progress into susceptible compartment.
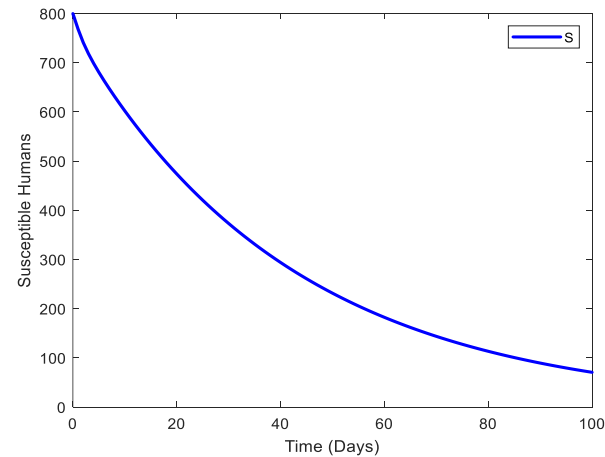

Fig. 3: The dynamics of susceptible humans against typhoid over a period of 100 days

As a result of the treatment and births the susceptible reaches a stable stage where it remains constant (Fig. 3). In Fig. 4, we observed that infected population with typhoid dropped drastically as result of treatment. Therefore, Fig. 5 increases significantly at a rapid rate until it reaches its peak and later gradually dropping as result of δ_2 and ρ_2 which is the decay function.
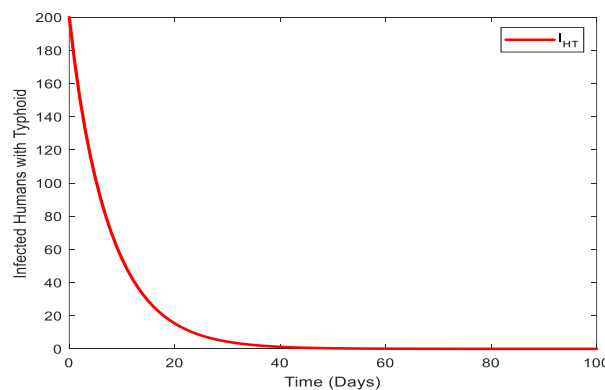

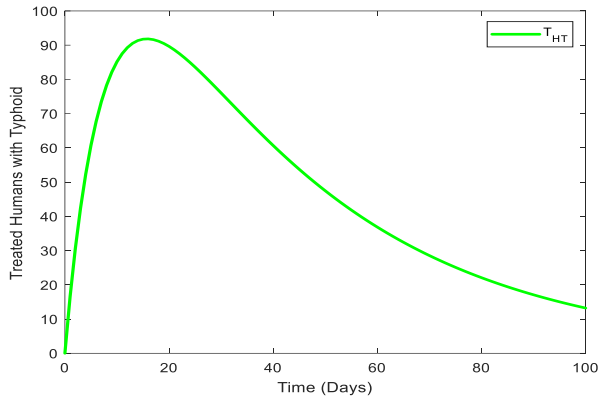**Fig. 4: The dynamics of infected humans with typhoid over a period of 100 days**

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Fig. 5: The dynamics of treated humans with typhoid over a period of 100 days

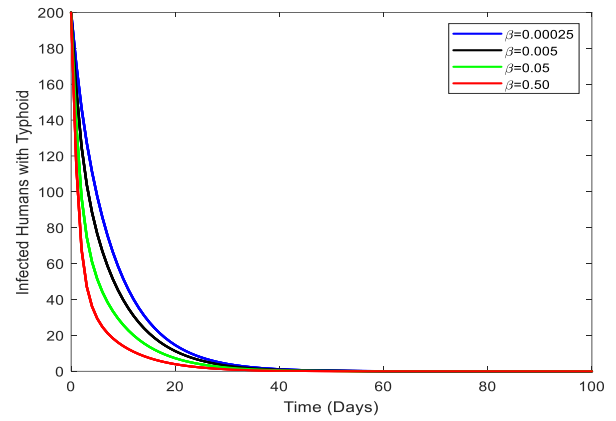**Fig. 6: The dynamics of exposed humans with malaria over a period of 100 days**

Fig. 7: Effect of transmission probability of humans with malaria on susceptible mosquito

Fig. 8: Effect of transmission probability of human with malaria on infected humans with typhoid

Figure 6 shows an increase just for a while but sharply decreases as a result of the presence of malaria infection. Figs. 7 and 8 as β decreases meaning reduction in biting rate of mosquito, shows the positive impact of the I_{HM} on both susceptible mosquito and human with typhoid, reverse will be negative.

Figure 9 as β gradually decreases the chances of human with typhoid getting infected with malaria are limited. Fig. 10 shows that as β increases there is a sharp rise of exposed population almost to the zenith.
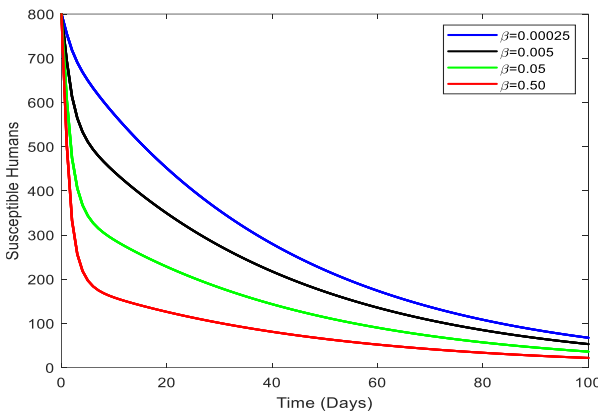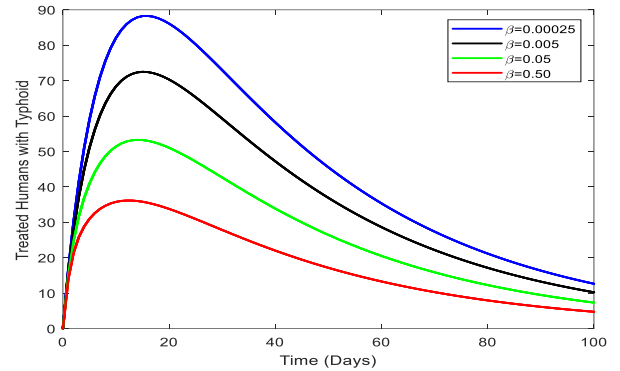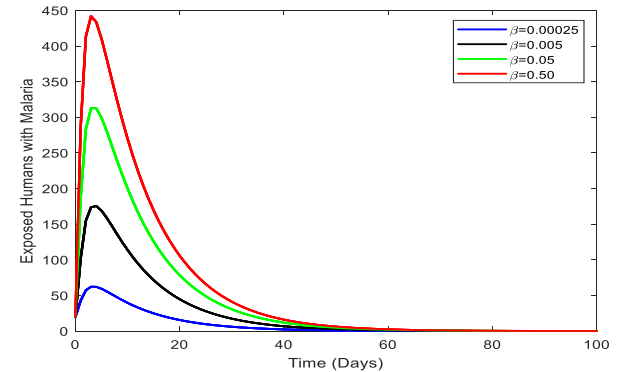

Fig. 9: Effect of transmission probability of malaria on treated humans with typhoid

Fig. 10: Effect of transmission probability of malaria on exposed humans with malaria

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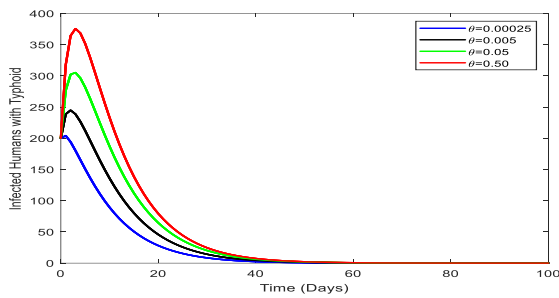
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Fig. 11: Effect of transmission probability of typhoid on infected humans with typhoid
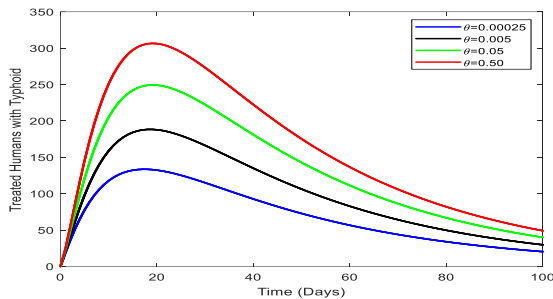

Fig. 12: Effect of transmission probability of typhoid on treated humans with typhoid
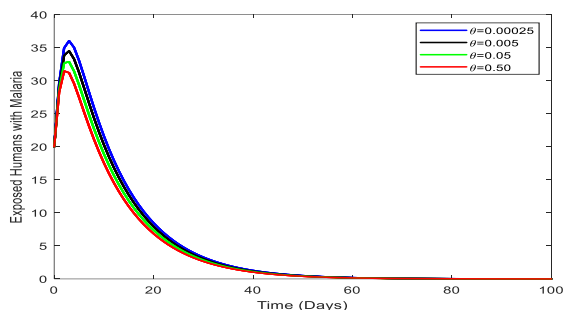

Fig. 13: Effect of transmission probability of typhoid on exposed humans with malaria

As θ increases implies an increase in the susceptibility population. Fig. 11 shows that as θ increases imply an increase in the population of human with typhoid. Fig. 12 shows that presence of malaria increases the susceptibility of typhoid, there is an increase in the population of human with typhoid, as a result of the treated becoming susceptible by losing their immunity. Fig. 13 shows that as θ increases or decreases there is a little change, because the presence of malaria increases the susceptibility of typhoid, meaning the exposed humans with malaria are more likely to have both infections.

Conclusion

We combined models due to Osman *et al.* (2017) and (Nthiiri *et al.* (2016), and come up with a co-infection model incorporating vector and loss immunity. Analytical studies were carried out using Routh-Hurwitz criteria. The disease free equilibrium points were obtained and our results showed that the equilibrium point of the system is locally asymptotically stable if $\left(R_0^{MT} < 1\right)$ and also globally asymptotically stable using (Castilo-Chavez and Feng, 1997). The result of the numerical experiment carried out indicates that the best way of minimizing or eradicating the

transmission of malaria and typhoid by increasing the death rate of the vector, taking proper medication to kill the parasite, destroying the vector habitats, and also to ensure that the reproduction number should less than unitary always $\left(R_0^{MT} < 1\right)$.

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