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Research Article

Mathematical Model for Transmission Dynamics of Typhoid Fever With Optimal Control Strategies

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Abstract

In this study, we used a deterministic mathematical model to investigate the dynamics of typhoid disease in a community. Different control strategies were used to determine efficient control strategies that help to reduce the incidence of typhoid disease. Stability theory of differential equations is used to study the qualitative behavior of the system. The basic reproduction number that represents the epidemic indicator is obtained by using the condition of endemicity. A local stability and global stability conditions for disease free equilibrium is established. Uniqueness of endemic equilibrium point and global stability conditions are also proved. Finally we used Pontryagins maximum principle in order to determine optimal control strategies for the spread of the disease. The numerical simulation revealed that applying prevention has a significant impact in minimizing the incidence of the disease. If all the interventions strategies are implemented the disease will be eradicated in short period of time. However, this result agrees with global result in [22], in the present study drug resistant cases are not considered.

Keywords : Mathematical model; Typhoid fever; Carrier; Basic reproduction number.

1 Introduction

Typhoid disease is caused by the gram negative bacterium Salmonella enterica subspecies enterica serovar Typhi (S. Typhi). It is a disease that can be transmitted faeco-orally which is considered exclusive to humans and may present with prolonged fever, influenza-like-illness, headache, malaise, anorexia and abdominal symptoms. The most frequent species of Salmonella that cause typhoid disease are Salmonella paratyphi A, B, and C and Salmonella paratyphi D [22]. This disease is a life threatening illness with both ill persons and carriers shed salmonella Typhi in faeces or stool. Human being can get infected with typhoid fever by eating or drinking food or water contaminated with Salmonella Typhi.

Typhoid fever is a major problem in many developing countries where lack of clean water and contaminated food are common feeding practice. Flies can also move the bacteria onto food, especially when garbage and faeces are not disposed

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of properly. The bacterial pathogens can also be released from the infectious individuals or carriers and then contaminate food or drinking water as a consequence of unsafe hygiene practices. As a result, typhoid fever is a common disease in developing countries in Africa particularly in Ethiopia. The data taken from Ethiopia during the years 2008 – 2016 as shown in Figure 1, indicates that in each year the disease is increasing in alarming rate.

Typhoid incidence is considered high if greater

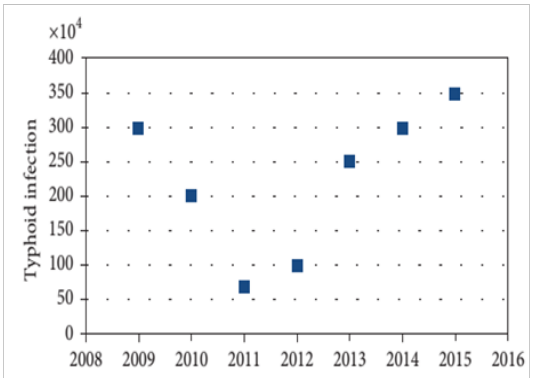


Figure 1: Reported cases of typhoid in Ethiopia.

than100 per 100,000 population, medium if 10 to 100 per 100,000 and low if less than10 [21]. From the Figure 1 we can observe that typhoid incidence is high in Ethiopia. In Western Europe and North America, typhoid fever has been largely eliminated or controlled, with water quality improvements and other public health reforms variously attributed to the disease decline [4]. Typhoid fever following ingestion of food or water that has been contaminated by the faeces of a case or carrier may dominate contemporary discourse [11]. Variation in factors that influence these modes of transmission, such as host behavior or environmental factors can result in fluctuating or complex seasonal dynamics [8].

Recently there have been a lot of case-control studies and other epidemiological investigations associated with the transmission of typhoid. Mathematical modeling of the spread of infectious diseases continues to become a vital tool in understanding the dynamics of diseases and it helps in decision making processes during the selection of intervention programs. For example, Getachew et al. developed a mathematical model to investigate dynamics of typhoid fever and used different control methods with cost-effective strategies [7]. In [15] a mathematical model is developed to study the effect of carriers on the transmission dynamics of typhoid fever. In his model he studied the dynamics of typhoid fever by incorporating vaccination rate as a control measure [17]. Some of the researcher who has contributed in this area is [1], [2], [3], [9], [16], [18] and [20]. In present study we considered the case of populations groups who are immune against the disease in the course of the disease transmission process.

2 Description and Formulation of Model

The compartments used in this model consist of six classes: E(t) is the compartment used for those who are immune against the disease over a period of time due to immunoprophylaxis. S(t) is used to represent the number of individuals who are prone to the disease at time t. C(t) denotes the number of individuals which are infected and are capable of transmitting the disease without showing the disease symptom, I(t) denotes the number of individuals who have been infected with the disease and are capable of spreading the disease to those in the susceptible categories. R (t) denote the number of individuals who are recovered from the disease. B (t) denotes the number of Salmonella bacteria at a time t. Immune populations are recruited into the population at per capita rate $p\pi$. These immune population groups are assumed to lose protection due to imperfect vaccine thus a fraction of them will join susceptible class. Susceptible individuals are recruited into the population at per capita rate $(1-p)\pi$. Susceptible individuals acquire typhoid fever at per capita rate λ. The susceptible class is increased by birth or emigration at a rate of $(1-p)\pi$ and also from Immune class by imperfect vaccine with φ rate. λ is the force of infection. γ is the rate at which carrier individuals become symptomatic infectious. μ is the natural mortality rate, d_1 is the disease induced mortality rate due to asymptomatic infection, d_2 is the disease induced mortality rate due to symptomatic infectious. β is the rate of recovery of symptomatic infectious

due to natural immunity and joining recovered class, θ is the rate of recovery of asymptomatic infectious due to natural immunity. The carriers (asymptomatic infectious) subclass is increased from susceptible subclass by $\rho\lambda$ screening rate. The infectious (symptomatic infectious) subclass is increased from susceptible subclass by $(1-\rho)\lambda$ screening rate. Those individuals in the carriers subclass can get recovered with a rate of θ and join recovered subclass. And those individuals in the infectious (symptomatic infectious) subclass can get recovered and join recovered subclass with a rate of β. The assumption of this model is that there is re-infection once an individual is recovered.

The above model description can be written in

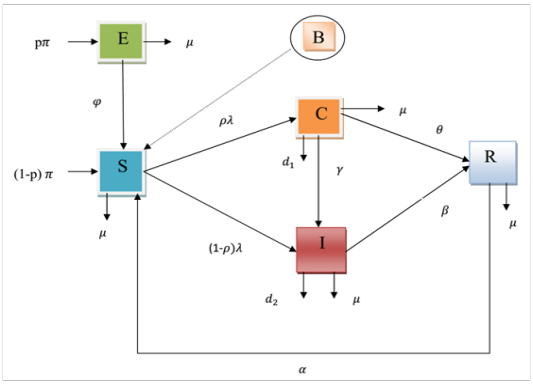


Figure 2: Flow diagram of the model.

six system of differential equation below.

$\frac{dE}{dt} = p\pi - (\varphi+\mu)E$ .....(1)

$\frac{dS}{dt} = (1-p)\pi + \varphi E + \alpha R - (\lambda+\mu)S$.....(2)

$\frac{dC}{dt} = \rho\lambda\ S - (\gamma+\theta+d_1+\mu)C$.....(3)

$\frac{dI}{dt} = (1-\rho)\lambda\ S + \gamma\ C - (\beta+d_2+\mu)I$.....(4)

$\frac{dR}{dt} = \theta\ C + \beta\ I - (\alpha+\mu)R$.....(5)

$\frac{dB}{dt} = r(1-\frac{B}{M})B$.....(6)

Where $\lambda = \frac{\nu B(t)}{K+B(t)}$ is force of infection, ν is injestion rate and K is concentration of bacteria in food or water.

Here $\frac{B(t)}{K+B(t)}$ is the probability of an individual getting infected by typhoid disease. M is the carrying capacity of the environment for the pathogens.

$N = E + S + C + I + R$ with initial conditions $E(0) = E_0, S(0) = S_0, C(0) = C_0, I(0) = I_0, R(0) = R_0$

3 Model Analysis

It is assumed that the initial condition of the model is non-negative, and now we will show that the solution of the model is positive.

3.1 Positivity of Solutions

Theorem 3.1 *Let* $\Omega = (S,E,C,I,R,B) \in \Re^6_+ : E_0 \geq 0, S_0 \geq 0$ $C_0 \geq 0, I_0 \geq 0, R_0 \geq 0, B_0 \geq 0\}$ *then the solutions* $\{E,S,C,I,R,B\}$ *are positive for* $t \geq 0$.

Proof. From the system (1-6), taking the first equation

$$\frac{dE}{dt} = p\pi - (\varphi+\mu)E$$

$$\Rightarrow \frac{dE}{dt} \geq -(\varphi+\mu)E$$

$$\Rightarrow \frac{dE}{E} \geq -(\varphi+\mu)dt$$

$$\Rightarrow \int \frac{dE}{E} \geq -\int (\varphi+\mu)dt$$

$$\Rightarrow lnE \geq -(\varphi+\mu)t + c_1 \text{ where}$$

c_1 is integration constant i.e. $E(0) = c_1$

Therefore, $E(t) \geq 0$ for all $t \geq 0$

Similarly we can show for the remaining state variables

This completes the theorem.

Therefore, the solution of the model is positive.

3.2 Invariant Region

Theorem 3.2 *The total population N of the system of model equation (1-6) is bounded in the invariant region* Ω. *That is the size of* $N(t)$ *is bounded for all t.*

Proof. In the given model total population (N) is

$N = E + S + C + I + R$.....(7)

Using the technique in [14] we differentiate N both sides with respect to t to get

$$\frac{dN}{dt} = \frac{dE}{dt} + \frac{dS}{dt} + \frac{dC}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$.....(8)

By substituting (1-6) into (8) we get

$\frac{dN}{dt} = \pi - \mu N - (d_1 C + d_2 I)$.....(9)

In the absence of mortality due to typhoid disease i.e. $d_1 = d_2 = 0$ equation (9) becomes

$\frac{dN}{dt} \leq \pi - \mu N$.....(10)

Rearrenging and integrating both sides of (10), we get

$\int \frac{dN}{\pi - \mu N} = \int dt$

$\Rightarrow \frac{-1}{\mu} ln(\pi - \mu N) \leq t + c_2$ where c_2 is integration constant.

$\Rightarrow ln(\pi - \mu N) \geq -\mu t + c_3$ where $c_3 = -\mu c_2$

$\Rightarrow \pi - \mu N \geq Ae^{-\mu t}$ where $A = e^{c_3}$

By applying the initial condition $N(0) = N_0$, we get $A = \pi - \mu N_0$

$\Rightarrow \pi - \mu N \geq (\pi - \mu N_0)e^{-\mu t}$

$\Rightarrow N \leq \frac{\pi}{\mu} - \frac{\pi - \mu N_0}{\mu} e^{-\mu t}$.....(11)

As $t \to \infty$ in (11), the population size $N \to \frac{\pi}{\mu}$.

Thus, the total population of the model remain in the region:

$\Omega = \{(E, S, C, I, R) \in \Re^5_+ : N \leq \frac{\pi}{\mu}\}$

Therefore, the basic model is well posed. Hence, it is sufficient to study the dynamics of the basic model in Ω.

Lemma 3.1 *Solution of the model equation (1-6) together with initial conditions* $E(0) \geq 0, S(0) \geq, C(0) \geq 0, I(0) \geq 0, R(0) \geq 0, B(0) \geq$ *exists in* $\Re^6_+$. *i.e. the solution of the model* $E(t), S(t), C(t), I(t), R(t)$ *and* $B(t)$ *exists for all* t *and will remain in* $\Re^6_+$.

Proof. The RHS of the system (1-6) can be expressed as follows
$f_1(E, S, C, I, R, B) = p\pi - (\varphi + \mu)E$
$f_2(E, S, C, I, R, B) = (1-p)\pi + \varphi E + \alpha R - (\lambda + \mu)S$
$f_3(E, S, C, I, R, B) = \rho\lambda S - (\gamma + \theta + d_1 + \mu)C$
$f_4(E, S, C, I, R, B) = (1-\rho)\lambda\ S + \gamma C - (\beta + d_2 + \mu)I$
$f_5(E, S, C, I, R, B) = \theta C + \beta I - (\alpha + \mu)R$
$f_6(E, S, C, I, R, B) = r(1 - \frac{B}{M})B$
According to [5] theorem, let Ω denote the region
$\Omega = \{(E, S, C, I, R, B) \in \Re^6_+ : N \leq \frac{\pi}{\mu}\}$
Then the system (1-6) has a unique solution if f_i for $i = 1, 2, 3, 4, 5, 6$ w.r.t the state variables are continuous and bounded in Ω Using simple mathematical computation of partial derivatives we can show the partial derivative exists.
Hence, by the theorem in [5], the solution for the model (1-6) exists and is unique.

3.3 Disease Free Equilibrium (DFE)

To find the disease free equilibrium we consider the steady state of the system (1-6) which is
$p\pi - (\varphi + \mu)E = 0$

$(1 - p)\pi + \varphi E + \alpha R - (\lambda + \mu)S = 0$

$\rho\lambda\ S - (\gamma + \theta + d_1 + \mu)C = 0$.....(12)

$(1 - \rho)\lambda S + \gamma C - (\beta + d_2 + \mu)I = 0$

$\theta C + \beta I - (\alpha + \mu)R = 0$

$r(1 - \frac{B}{M})B = 0$

Equating (12) at $C = I = R = B = 0$ and solving the non-infected state variables we get the following

$E^0 = \frac{p\pi}{\varphi + \mu}$

$S^0 = \frac{p(\varphi + \mu - p\mu)}{\mu(\varphi + \mu)}$
Therefore, the disease free equilibrium point E_0 becomes
$E_0 = (\frac{p\pi}{\varphi + \mu}, \frac{p(\varphi + \mu - p\mu)}{\mu(\varphi + \mu)}, 0, 0, 0, 0)$

3.4 Endemic Equilibrium Point

To find the endemic equilibrium point F_0 we considered the steady state of the system (1-6) for all state variables. After applying some laborous and tidy calculation we get the following endemic equilibrium point

$F_0 = (E^*, S^*, C^*, I^*, R^*, B^*)$ where

$E^* = \frac{p\pi}{\varphi+\mu}$

$S^* = \frac{\pi(\gamma+\theta+d_1+\mu)[(1-p)(\varphi+\mu)+p\pi]}{(\lambda+\mu-a\rho\lambda\alpha)(\varphi+\mu)}$

$C^* = \frac{\rho\lambda\pi[(1-p)(\varphi+\mu)+p\pi]}{(\lambda+\mu-a\rho\lambda\alpha)(\varphi+\mu)}$

$I^* = \frac{\lambda[(1-\rho)(\gamma+\theta+d_1+\mu)+\rho\gamma][\pi(1-p)(\varphi+\mu)+p\varphi\pi]}{(\varphi+\mu)(\beta+d_2+\mu)(\lambda+\mu-a\rho\lambda\alpha)}$

$R^* = \frac{a\pi\rho\lambda[(1-p)(\varphi+\mu)+p\pi]}{(\lambda+\mu-a\rho\lambda\alpha)(\varphi+\mu)}$

$a = \frac{\theta\rho(\beta+d_2+\mu)+\beta[(1-\rho)(\gamma+\theta+d_1+\mu)+\rho\gamma]}{\rho(\beta+d_2+\mu)(\alpha+\mu)}$

$B^* = M$

3.5 Basic Reproduction Number ($\Re_0$)

The basic reproduction number is the average number of secondary cases a typical infectious individual will cause in a completely susceptible population. In this section we obtained the basic reproduction number which is the threshold parameter that governs the spread of the disease. For the given model the endemic equilibrium F_0 exists in the feasible region Ω the necessary and sufficient condition is that:

$0 < S^* \leq S_0$

Or $0 < S^* \leq \frac{p(\varphi+\mu-p\mu)}{\mu(\varphi+\mu)}$

Equivalently $\frac{p(\varphi+\mu-p\mu)}{\mu(\varphi+\mu)S^*} \geq 1$

Define $\Re_0 = \frac{p(\varphi+\mu-p\mu)}{\mu(\varphi+\mu)S^*}$ implies

$\Re_0 = \frac{p(\varphi+\mu-p\mu)(\lambda+\mu-a\rho\lambda\alpha)}{\mu\pi(\gamma+\theta+d_1+\mu)[(1-p)(\varphi+\mu)+p\pi]}$

3.6 Local Stability of Disease Free Equilibrium

Proposition 3.1 *The disease free equilibrium point is locally asymptotically stable if* $\Re_0 < 1$ *and unstable if* $\Re_0 > 1$.

Proof. To proof the proposition we first construct a Jacobean matrix for the system (1-6) at disease free equilibrium.

$$J = \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 & 0 \\ \varphi & -\mu & 0 & 0 & \alpha & a_2 \\ 0 & 0 & a_3 & 0 & 0 & a_4 \\ 0 & 0 & \gamma & a_5 & 0 & a_6 \\ 0 & 0 & \theta & \beta & a_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu \end{bmatrix} \text{.....(13)}$$

Where $a_1 = -(\varphi+\mu)$, $a_2 = \frac{-\nu S^0}{K}$

$a_3 = -(\gamma+\theta+d_1+\mu)$, $a_4 = \frac{\rho\nu S^0}{K}$

$a_5 = -(\beta+d_2+\mu)$, $a_6 = \frac{(1-\rho)\nu S^0}{K}$

$a_7 = -(\alpha+\mu)$, $S^0 = \frac{p(\varphi+\mu-p\mu)}{\mu(\varphi+\mu)}$

Now we compute the Jacobean matrix at disease free equilibrium and investigate its stability effect due to reproduction number $\Re_0$.

From the Jacobean matrix (13) we obtained a characteristic polynomial by evaluating $det(J-\lambda^* I) = 0$. Where λ^* is the eigen value of the characteristic polynomial.

$$\begin{vmatrix} a_1-\lambda^* & 0 & 0 & 0 & 0 & 0 \\ \varphi & -\mu-\lambda^* & 0 & 0 & \alpha & a_2 \\ 0 & 0 & a_3-\lambda^* & 0 & 0 & a_4 \\ 0 & 0 & \gamma & a_5-\lambda^* & 0 & a_6 \\ 0 & 0 & \theta & \beta & a_7-\lambda^* & 0 \\ 0 & 0 & 0 & 0 & 0-\mu-\lambda^* & \end{vmatrix} = 0.$$

Solving the determinant of the above matrix we obtained the following eigen values:

$\lambda_1^* = -(\varphi+\mu)$, $\lambda_2^* = -\mu$
$\lambda_3^* = -(\varphi+\theta+d_1+\mu)$, $\lambda_4^* = -(\beta+d_2+\mu)$
$\lambda_5^* = -(\alpha+\mu)$, $\lambda_6^* = -\mu$

Since all the eigen values are negative the disease free equilibrium point is locally asymptotically stable.

Therefore, the disease free equilibrium point E_0 is locally asymptotically stable if and only if $\Re_0 < 1$.

3.7 Global Stability of Disease Free Equilibrium

In this section we analyzed global stability of disease free equilibrium point by applying the tech-

niques used in [14]. We write the model equation (1-6) in the form:

$$\frac{dX_s}{dt} = A(X_s - X_{DFE,s}) + A_1 X_s$$

$$\frac{X_i}{dt} = A_2 X_i$$

Where X_s is the vector representing the non transmitting compartment and X_i is a vector representing the transmitting compartment. The disease free equilibrium is globally asymptomatically stable if a matrix A has negative eigen values and A_2 is Metzler matrix (i.e. the off-diagonal element of A_2 are non-negative).
For the model (1-6), we have

$$X_s = (E, S, R)^T$$

and

$$X_i = (C, I, B)^T$$

Where T refers to transpose of a matrix.
We need to check whether a matrix A for non-transmitting compartments has real negative eigen values and A_2 is Metzler matrix. From the equation for non-transmitting compartments in the model we obtained:

$$A = \begin{bmatrix} -(\varphi+\mu) & 0 & 0 \\ \varphi & -(\lambda+\mu) & \alpha \\ 0 & 0 & -(\alpha+\mu) \end{bmatrix}$$

From the matrix A we get the eigen values
$\lambda_1 = -(\varphi+\mu)$, $\lambda_2 = -(\lambda+\mu)$
and $\lambda_3 = -(\alpha+\mu)$ all the eigen values are real and negative. Now it remains to show that A_2 is Metzler.

Using suitable re-arrangement we can get

$$A_2 = \begin{bmatrix} -(\gamma+\theta+d_1+\mu) & 0 & 0 \\ \gamma & (\lambda+\mu) & \alpha \\ 0 & 0 & -(\alpha+\mu) \end{bmatrix}$$

and $A_1 = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \theta & \beta & 0 \end{bmatrix}$ Since the off-diagonal elements of A_2 are non-negative so it is a Metzler matrix.

Hence, the disease free equilibrium point is globally asymptotically stable.

3.8 Global Stability of Endemic Equilibrium Point

Theorem 3.3 *If $\Re_0 > 1$, then the endemic equilibrium point F_0 is globally asymptotically stable.*

Proof. To prove global asymptotic stability of the endemic equilibrium point we used the method of Lyapunov function below
Define $L(E^*, S^*, C^*, I^*, R^*, B^*) =$

$$(E - E^* - E^* ln\frac{E^*}{E}) + (S - S^* - S^* ln\frac{S^*}{S}) +$$

$$(C - C^* - C^* ln\frac{C^*}{C}) + (I - I^* - I^* ln\frac{I^*}{I}) + (R - R^* - R^* ln\frac{R^*}{R}) + (B - B^* - B^* ln\frac{B^*}{B}) \quad (14)$$

In (14) taking the derivative on both sides with respect to t and re-arrenging the terms we get
$\frac{dL}{dt} = (\frac{E-E^*}{E})\frac{dE}{dt} + (\frac{S-S^*}{S})\frac{dS}{dt} + (\frac{C-C^*}{C})\frac{dC}{dt} +$

$$(\frac{I-I^*}{I})\frac{dI}{dt} + (\frac{R-R^*}{R})\frac{dR}{dt} + (\frac{B-B^*}{B})\frac{dB}{dt} \quad (15)$$

Substituting the system (1-6) in (15) we get

$\frac{dL}{dt} = A - B$ Where

$A = (\varphi+\mu)E^* + (\lambda+\mu)S^* + (\gamma+\theta+d_1+\mu)C^* + (\beta+d_2+\mu)I^* + (\alpha+\mu)R^*$

and
$B = (\gamma+d_1)C - d_2 I - r(1-\frac{B}{M}) - p\pi\frac{E^*}{E} +$

$$[\alpha R + (1-p)\pi]\frac{S^*}{S} + \rho\lambda S\frac{C^*}{C} + (1-\rho)\lambda S\frac{I^*}{I} +$$

$$(\theta C + \beta I)\frac{R^*}{R} + r(1-\frac{B}{M})\frac{B^*}{B}$$

Thus, if $A < B$, the $\frac{dL}{dt} \leq 0$

Note that $\frac{dL}{dt} = 0$ if and only if
$E = E^*, S = S^*, C = C^*, I = I^*, R = R^*, B = B^*$
Therefore, the largest compact invariant set in $\Omega = (E, S, C, I, R, B) \in \Omega : \frac{dL}{dt} = 0$ is the singleton E^*.
Hence, by the principle in [10] we can infer that the endemic equilibrium point is globally asymptotically stable in Ω if $A < B$.

4 Sensitivity Analysis

The total mortality and morbidity attributed to typhoid disease can be best reduced by investigating the relative importance of the parameters featuring in the basic reproduction number $\Re_0$. In order to reduce mortality and morbidity due to typhoid disease, it is crucial to know the relative importance of different factors responsible for its transmission and prevalence. We calculate the sensitivity indices of the basic reproduction number $\Re_0$. This allows us to measure the relative change in the state variable when a parameter changes. When a state variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives. Thus, the values obtained for sensitivity indexes indicate which parameters should be targeted most for intervention purposes.

Numerical values of sensitivity indices of $\Re_0$ to parameter values for the typhoid model, evaluated using the parameter values listed in Table 2.

Table 1 shows the sensitivity indices of $\Re_0$ to the parameter for typhoid model, evaluated based on the values on table 2. The parameters are ordered from the most sensitive to the least sensitive. This result shows that, when the parameter values of π, μ, K, γ, φ and ρ increases while the other are kept constant they increase the value of $\Re_0$ which implies these parameters increases the endemicity of typhoid disease. Whereas the parameters μ, d_1, d_2, θ, β decreases the value of $\Re_0$ while the other are kept constant which implies these parameters decrease the endemicity of typhoid disease.

5 Characterization of an Optimal Control

In this section we apply optimal control method for the system (1-6) by using Pontryagin's maximum principles in [19]. The optimal control model is an extension of typhoid model in (1-6) by incorporating the following three controls mensioned below.

1. u_1 is the prevention effort for susceptible population.

2. u_2 is treatment used for infectious class.

3. u_3 is screaning used for carrier class.

After incorporating u_1, u_2 and u_3 in typhoid model (1-6), we get the following model.

$$\begin{cases} \frac{dE}{dt}=p\pi-\varphi(1-u_1)E-\mu E \\ \frac{dS}{dt}=(1-p)\pi+\varphi(1-u_1)E \\ +\alpha R-(1-u_1)\lambda S-\mu S \\ \frac{dC}{dt}=\rho(1-u_1)\lambda S-(\gamma+u_3)C \\ -(\theta+u_2)C-(d_1+\mu)S \\ \frac{dI}{dt}=(1-\rho)(1-u_1)\lambda S+(\gamma+u_3)C \\ -(\beta+u_2)I-(d_2+\mu)I \\ \frac{dR}{dt}=(\theta+u_2)C+(\beta+u_2)I-(\alpha+\mu)R \\ \frac{dB}{dt}=r(1-\frac{B}{M})B \end{cases} \quad \text{.....(16)}$$

The control functions u_1, u_2 and u_3 are bounded, Lebesgue integrable functions, which is defined as

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

Where g is the drug efficacy for typhoid infected individual. We need to obtain a control U, E, S, C, I, R and B that can minimize the proposed objective function J where the objective functional is taken from the literature on epidemic model [7], and it is given by

$$J=min_{u_1,u_2,u_3}\int_0^{t_f}(b_1C+b_2I+\tfrac{1}{2}\sum_{i=1}^{3}w_iu_i^2)dt$$

Where b_1, b_2 and w_i are positive. The expression $\frac{1}{2}w_iu_i^2$ represents costs which is associated with the controls u_i and t_f is the final time. The coefficients are balancing cost factors. Now we seek to find an optimal triple u_1^*, u_2^*, u_3^*, such that

$J(u_1^*,u_2^*,u_3^*)=min\{J(u_1,u_2,u_3):u_1,u_2,u_3\in U\}$ .....(17)

Where $U=\{J(u_1,u_2,u_3)\}$ is a measurable set and $t\in[0,t_f]$ for the control set.

Table 1: Parameter values for typhoid model.

Parameters	Descriptions	values	source
π	Recruitment rate	100	[7]
φ	Proportion of populations which loses immunity	0.8	[17]
μ	Natural mortality rate	0.004	Assumed
K	Concentration of Salmonella bacteria in food and water	10,000	Assumed
ν	Injestion rate of Salmonella	0.9	[7]
M	Carrying capacity of salmonnella	100,000	Assumed
$d_1 = d_2$	Typhoid induced death rate	0.052	[7]
γ	Rate at which carrier population enter into infectious class	0.36	Assumed
β	Recovery rate of infectious class by natural immunity	0.02	[7]
α	Removal rate from recovered class to susceptible class	0.000904	[2]
θ	Recovery rate of carriers by natural immunity	0.03	Assumed
ρ	Probability of susceptible population joining the carrier state	0.42	Assumed
p	Probability of immune population joining susceptible class	0.3	Assumed

Table 2: Sensitivity indeces.

Parameters	Sensitivity index
π	+ve
ν	+ve
K	+ve
γ	+ve
ρ	+ve
φ	+ve
μ	-ve
d_1	-ve
d_2	-ve
θ	-ve
β	-ve

5.1 Existence of an Optimal Control

The necessary condition that an optimal solution is guaranteed under [19]. The existence of an optimal control can be proved by the result in [6]. The system of equation (1-6) is bounded by a linear system for a finite time interval the detail of the prof is given in [6].
For the optimal control problems we need to check the following properties are satisfied.

1. The set of controls and corresponding state variables is non-empty.

2. The control set U is convex and closed.

3. The RHS of the system (1-6) is bounded by the linear function in the state and control.

4. The integrand of the objective functional is concave on U.

5. The function is bounded below by $a_2 - a_1(u_1^2 + u_2^2 + u_3^2)^{\frac{\alpha}{2}}$ where $a_1 > 0$, $a_2 > 0$ and $\alpha > 1$.

The existence result in [13] for the system (1-6) with bounded coefficients is used to satisfy the condition under (1).
The control set U is convex and is closed by definition.
The RHS of the state variables in (1-6) satisfies condition (3) as the state solutions are a priori bounded.
The integrand in the objective functional $b_1C + b_2I + \frac{1}{2}\sum_{i=1}^{3} w_i u_i^2$ is clearly concave on U. Finally, there are $a_1 > 0$, $a_2 > 0$ and $\alpha > 1$ satis-

fying
$b_1C + b_2I + \frac{1}{2}\sum_{i=1}^{3} w_i u_i^2 \leq a_2 - a_1(u_1^2 + u_2^2 + u_3^2)^{\frac{\alpha}{2}}$ because the state variables are bounded. Hence, there exist an optimal control (u_1, u_2, u_3) that minimize the objective functional, $J(u_1, u_2, u_3)$.

5.2 Hamiltonian and Optimality Condition

The necessary condition for the optimal triple is obtained using the principle in [19]. Therefore, using this principle we get a Hamiltonian which is defined as:

$$H(E, S, C, I, R, B, t) = L(C, I, u_1, u_2, u_3) + \lambda_1 \frac{dE}{dt} + \lambda_2 \frac{dS}{dt} + \lambda_3 \frac{dC}{dt} + \lambda_4 \frac{dI}{dt} + \lambda_5 \frac{dR}{dt} + \lambda_6 \frac{dB}{dt}$$

Where

$$L(C, I, u_1, u_2, u_3) = b_1 C + b_2 I + \frac{1}{2}\sum_{i=1}^{3} w_i u_i^2,$$

λ_i is adjoint variable to be determined using Pontryagin's Maximum Principle.

Theorem 5.1 *For an optimal control set u_1, u_2 and u_3 that minimizes J over U there are adjoint variables $\lambda_1, \lambda_2, ...\lambda_6$ such that:*

$$\begin{cases} \frac{d\lambda_1}{dt} = \lambda_1[\varphi(1-u_1)+\mu] - \lambda_2\varphi(1-u_1) \\ \frac{d\lambda_2}{dt} = \lambda_2[\frac{(1-u_1)\nu B}{K+B} + \mu] - \lambda_3\rho(1-u_1)\frac{\nu B}{K+B} - \\ \lambda_4(1-\rho)(1-u_1)\frac{\nu B}{K+B} \\ \frac{d\lambda_3}{dt} = -b_1 + \lambda_3(\gamma + u_3 + \theta + u_2 + d_2 + \mu) - \\ \lambda_4(\gamma + u_3) - \lambda_5(\theta + u_2) \\ \frac{d\lambda_4}{dt} = -b_2 + \lambda_4(\beta + u_2 + d_2 + \mu) - \lambda_5(\theta + u_2) \\ \frac{d\lambda_5}{dt} = -\lambda_2\alpha + \lambda_5(\alpha + \mu) \\ \frac{d\lambda_6}{dt} = \lambda_6(-r + \frac{2rB}{M}) \end{cases}$$

With transversality conditions, $\lambda_i(t_f) = 0$ for $i = 1, 2, ...6$.

Furthermore, we obtained the control set (u_1^*, u_2^*, u_3^*) characterized by $\frac{\partial H}{\partial u_i^*} = 0$ for $i = 1, 2, ..., 6$. Hence, we obtained

$$u_1^*(t) = max\{0, min(1, \sigma_1)\}$$

$$u_2^*(t) = max\{0, min(0.85, \sigma_2)\}$$

$$u_3^*(t) = max\{0, min(1, \sigma_3)\}$$

Where $\sigma_1 = \frac{(-\lambda_1+\lambda_2)\varphi E + [-\lambda_2 + \lambda_2\rho + \lambda_4(1-\rho)]\lambda S}{w_1}$

$\sigma_2 = \frac{(\lambda_3-\lambda_5)C + (\lambda_4-\lambda 5)I}{w_2}$ and $\sigma_3 = \frac{(\lambda_3+\lambda_4)C}{w_3}$

Proof. The adjoint variables and transversality conditions are standard results of Potryagin's maximum principle in [19]. To obtain the adjoint equations we differentiate the Hamiltonian H w.r.t the state variables E, S, C, I, R and B respectively and the we obtain:

$$\begin{cases} \frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial E} = \lambda_1[\varphi(1-u_1)+\mu] - \lambda_2\varphi(1-u_1) \\ \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial S} = \lambda_2[(1-u_1)\frac{\nu B}{K+B} + \mu] \\ -\lambda_3\rho(1-u_1)\frac{\nu B}{K+B} - \lambda_4(1-\rho)(1-u_1)\frac{\nu B}{K+B} \\ \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial C} = -b_1 + \lambda_3(\gamma + u_3 + \theta + u_2 + d_2 + \mu) \\ -\lambda_4(\gamma + u_3) - \lambda_5(\theta + u_2) \\ \frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial I} = -b_2 + \lambda_4(\beta + u_2 \\ +d_2 + \mu) - \lambda_5(\theta + u_2) \\ \frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial R} = -\lambda_2\alpha + \lambda_5(\alpha + \mu) \\ \frac{d\lambda_6}{dt} = -\frac{\partial B}{\partial B} = \lambda_6(-r + \frac{2rB}{M}) \end{cases}$$

Again using the method in [19] we obtain the controls by solving $\frac{\partial H}{\partial u_i^*} = 0$ for $i = 1, 2, 3$ then

$$u_1^* = \frac{(-\lambda_1+\lambda_2)\varphi E + [-\lambda_2 + \lambda_3\rho + \lambda_4(1-\rho)]\lambda S}{w_1}$$

$$u_2^* = \frac{(\lambda_3-\lambda_5)C + (\lambda_4-\lambda_5)I}{w_2} \text{ and } u_3^* = \frac{(\lambda_3+\lambda_4)C}{w_3}$$

Thus, writing u_1^*, u_2^* and u_3^* using standard control arguments involving the bounds.

$$u_1^* = \begin{cases} \sigma_1, & \text{if } 0 < \sigma_1 < 1; \\ 0, & \text{if } \sigma_1 \leq 0; \\ 1, & \text{if } \sigma_1 \geq 1 \end{cases}$$

$$u_2^* = \begin{cases} \sigma_2, & \text{if } 0 < \sigma_2 < 0.85; \\ 0, & \text{if } \sigma_2 \leq 0; \\ 1, & \text{if } \sigma_2 \geq 1 \end{cases}$$

$$u_3^* = \begin{cases} \sigma_3, & \text{if } 0 < \sigma_3 < 1; \\ 0, & \text{if } \sigma_3 \leq 0; \\ 1, & \text{if } \sigma_3 \geq 1 \end{cases}$$

Hence, the following optimality system is formed.

$$\begin{cases} \frac{dE}{dt} = p\pi - \varphi(1-u_1)E - \mu E \\ \frac{dS}{dt} = (1-p)\pi + \varphi(1-u_1)E + \alpha R - (1-u_1)\lambda S \\ -\mu S \\ \frac{dC}{dt} = \rho(1-u_1)\lambda S - (\gamma+u_3)C - (\theta+u_2)C - \\ (d_1+\mu)C \\ \frac{dI}{dt} = (1-\rho)(1-u_1)\lambda S + (\gamma+u_3)C - (\beta+u_2)I \\ -(d_2+\mu)I \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (18) \\ \frac{dR}{dt} = (\theta+u_2)C + (\beta+u_2)I - (\alpha+\mu)R \\ \frac{dB}{dt} = r(1-\frac{B}{M})B \\ \frac{d\lambda_1}{dt} = \lambda_1[\varphi(1-u_1)+\mu] - \lambda_2\varphi(1-u_1) \\ \frac{d\lambda_2}{dt} = \lambda_2[\mu + (1-u_1)\lambda] - \lambda_3\rho(1-u_1)\lambda \\ -\lambda_4(1-\rho)(1-u_1)\lambda \\ \frac{d\lambda_3}{dt} = -b_1 + \lambda_3(\gamma+\theta+u_3+u_2+d_2+\mu) \\ -\lambda_4(\gamma+u_3) - \lambda_5(\theta+u_2) \\ \frac{d\lambda_4}{dt} = -b_2 + \lambda_4(\beta+u_2+d_2+\mu) - \lambda_5(\theta+u_2) \\ \frac{d\lambda_5}{dt} = -\lambda_2\alpha + \lambda_5(\alpha+\mu) \\ \frac{d\lambda_6}{dt} = \lambda_6(-r + \frac{2rB}{M}) \end{cases}$$

$\lambda_i(t_f) = 0, for i = 1, 2, ...6, E(0) = E_0, S(0) = S_0,$

$C(0) = C_0, I(0) = I_0, R(0) = R_0, B(0) = B_0$

6 Numerical Simulations

In the present work, we have used an extension of SIR epidemic model with control measures. The simulations are carried out in order to determine the impact of control measures on the typhoid disease dynamics. Following parameter values are used in the model for simulation purpose.

$\pi = 100, \alpha = 0.000904, \mu = 0.004, d_1 = 0.052, \beta = 0.02, \theta = 0.0003, \rho = 0.42, \nu = 0.9, \gamma = 0.36, p = 0.3, \varphi = 0.8, K = 10,000, T = 4, b_1 = 2, b_2 = 1, w_1 = 2, w_2 = 3, w_3 = 5$ and initial values

$E(0) = 100, S(0) = 400, C(0) = 180, I(0) = 120, R(0) = 100, B(0) = 5000$

The optimal solution is obtained by solving the optimality system (18) consisting the state system, the adjoint system and transversality condition.

To solve the state system we use a forward fourth order Runge-Kutta method and solve the adjoint system using a backward fourth order Runge-Kutta method. The solution iterative scheme involves making a guess of the controls and solves the state system using forward fourth order Runge-Kutta scheme. Due to the transversality condition (18), the adjoint equations are then solved by the backward fourth order Runge-Kutta scheme using the current iteration solutions of the state equations. The controls are then updated using a convex combination of the previous controls and the values obtained using the characterizations. The updated controls are then used to repeat the solution of the state and adjoint systems. This process is repeated until the values in the current iteration are close enough to the previous iteration values in (17).

In this section we investigate numerically the effect of the following optimal control strategies on the spread of the disease in the population.

1. Using prevention effort (u_1), that protect susceptibles from contracting the disease ($u_2 = 0, u_3 = 0$)
2. Using treatment effort (u_2), for infectious individuals ($u_1 = 0, u_3 = 0$)
3. Using screaning effort (u_3), for carrier individuals ($u_1 = 0, u_2 = 0$)
4. Using prevention (u_1) for susceptible population and treatment (u_2) for infectious individuals ($u_3 = 0$)
5. Using prevension (u_1) and screening (u_3) effort ($u_1 = 0$)
6. Using treatment (u_2) and screening (u_3) effort. ($u_1 = 0$)
7. Using all the three controls.

6.1 Control with prevention only

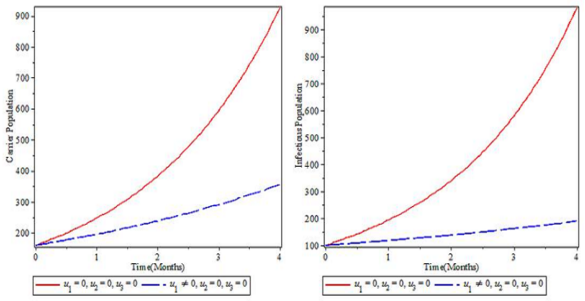
In figure 3 we observe that using prevention significantly affect the number of carriers and infectious population as the case compared without control. From this we can infer that applying prevention measure on infected population leads to a faster reduction of the proportion of both carrier and infected population as compared to the case without a control measure. This result agrees with result in [7].

6.2 Control with treatment only

u_2 is a treatment used to optimize the objective functional J; the other controls (u_1 and u_3) related to typhoid fever are set to zero. When a treatment is imposed on infectious population the simulation on figure 4 shows there will be a slight decline of the proportion of infectious population. As a result it is possible to deduce that applying a treatment for typhoid infected population has a slight effect in minimizing the spread of the disease. A similar result is obtained in [16].

6.3 Controls with screening only

The control measure with screening (u_3) is used to optimize the objective functional J; the other controls (u_1 and u_2) related to typhoid fever are set to zero. From figure 5 it is observed that the proportion of carrier population significantly decreased with time and there is a slight reduction of the proportion of infectious population. Therefore, using screening of the carrier population helps to reduce the rate of carrier populations as compared to the case without control.

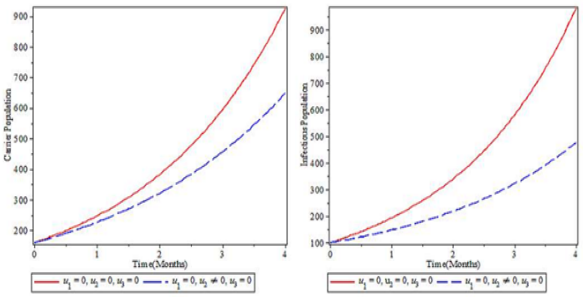


Simulation of optimal control with prevention only

Figure 3

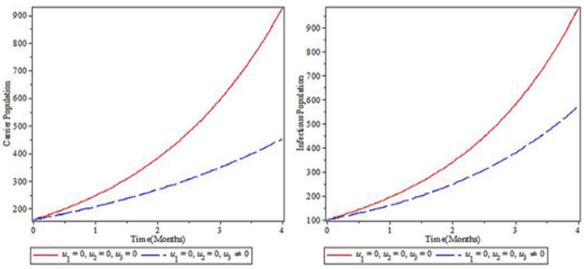
6.4 Controls with prevention and treatment

The controls u_1 and u_2 are used to optimize the objective functional J; while the control u_3 is set to zero. The simulation diagram in figure 6 shows that applying the controls prevention and treatment on typhoid infected population has a considerable impact in minimizing the incidence of typhoid cases in a community. From this we can



Simulation of optimal control with treatment infected population

Figure 4



Simulation of optimal control with screening only

Figure 5

infer that optimized prevention and treatment reduces the transmission of typhoid disease as compared to the case without control.

6.5 Controls with prevention and screening

The controls u_1 and u_3 are used to optimize the objective functional J; while the control u_2 is set to zero. From the simulation diagram on figure 7 we observe that this strategy shows there is higher reduction of the proportion of both carrier and infectious population.

6.6 Controls with treatment and screening

The controls u_2 and u_3 are used to optimize the objective functional J; while the control u_1 is set to zero. From the simulation diagram on figure 8 we observe that this strategy shows that there is a reduction of the proportion of both symp-

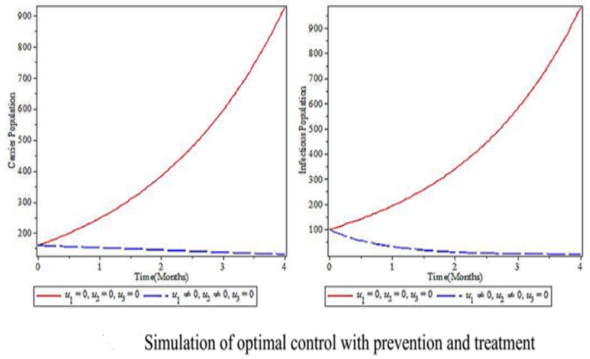


Figure 6

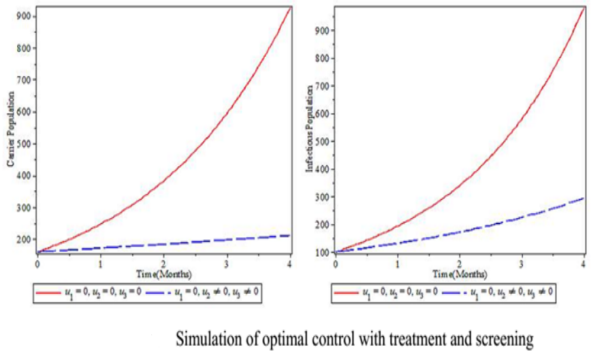


Figure 8

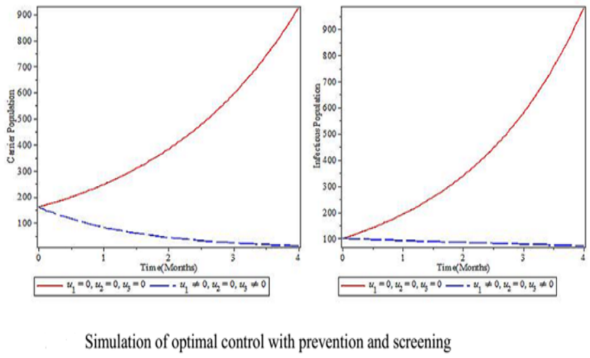


Figure 7

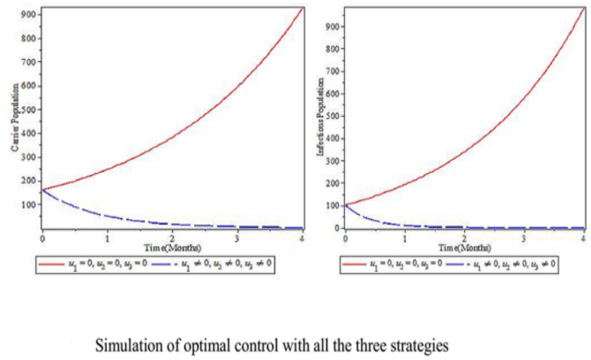


Figure 9

tomatic and asymptomatic populations. This result agrees with the results of some of the studies [2], [3], [8] and [17].

6.7 Controls with prevention, treatment and screening

Here we used all the intervention strategies which enable to minimize the objective functional J. From simulation diagram in figure 9 we observe that the proportion of both carriers and infectious population rapidly decline to zero before two months. Therefore, applying this strategy helps to eradicate typhoid disease from the community.

7 Discussions and Conclusions

In this study a deterministic mathematical model of typhoid fever consisting of immune, carrier and infectious stage has been established. The model consists of the assumption that certain population groups exists who are immune due to immuno prophylaxis against typhoid fever. Since vaccines are not 100% effecient a certain fraction of these population group will lose immunity and join susceptible class. A qualitative and numerical analysis of the model was done. We have shown that there exist a feasible region where the model is well posed biologically meaningful in which a unique disease free equilibrium point exists. The steady state point were obtained and the local and global stability conditions were investigated. The model has disease free equilibrium point if $\Re_0 < 1$ and has a unique endemic equilibrium point if $\Re_0 > 1$. Sensitivity analysis of the model parameter was done. The expression for the basic reproduction number $\Re_0$ shows ingestion rate of Salmonella bacteria has a direct effect either in increasing or decreasing the endemicity of the disease. If this is the case sanitation is expected to be a public health improvement priority.

For the given model an optimal control problem is formulated by incorporating different control strategies The optimality condition was estab-

lished by Pontryagin's Maximum Principle. A numerical simulation of the model was conducted and different combinations of control strategies were compared. It was observed that prevention has a significant impact in minimizing the incidence of the disease. It was also shown that a treatment given for infected group and screening of the carriers minimizes the transmission of the disease. Finally from the simulation diagram it was observed that a combination of the three control strategies rapidly eradicates typhoid disease before two months time. This result agrees with the global result in [22] only if drug resistant cases are not considered.

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