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Mathematical Model for the Transmission Dynamics of Typhoid Fever Infection with Treatment

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ABSTRACT

In this paper, we develop a mathematical model for the transmission dynamics of typhoid fever infection and applied optimal control strategy for controlling the spread of the disease. We considered two control strategies such as sanitation and treatment of the infected individuals in order to minimize the transmission of the disease so as to maximize the number of susceptible and recovered individuals. We found the necessary conditions for the optimal solution for controlling the spread of the infection using Pontryagin's Maximum Principle (PMP). Runge-Kutta of order four was used for the numerical simulation to demonstrate the achievability of the control strategies in curtailing the transmission dynamics of the disease.

Keywords: Optimal Control, Transmission, Typhoid, Dynamics, Infection

1.0 INTRODUCTION

1.1 Introduction to Typhoid Fever

Typhoid fever is a life-threatening infection caused by the bacterium *Salmonella Typhi*. The species and strains of *Salmonella* that commonly cause typhoid fever in humans are *Salmonella paratyphi A*, *Salmonella paratyphi B*, *Salmonella paratyphi C* and *Salmonella paratyphi D*. It is usually spread through contaminated food or water. An estimated 11–20 million people get sick from typhoid and between 128 000 and 161 000 people die from it every year. Symptoms include prolonged fever, fatigue, headache, nausea, abdominal pain, and constipation or diarrhea. Some patients may have a rash. Severe cases may lead to serious complications or even death. Even when the symptoms go away, people may still be carrying typhoid bacteria, meaning they can spread it to others through their faeces. Typhoid fever is common in places with poor sanitation and a lack of safe drinking water, lack of access to safe water and adequate sanitation, poor health education, inappropriate hygiene among food handlers, and lack of typhoid vaccination, (WHO, 2019).

Mushayabasa, Bhunu and Mhlanga (2014) formulated an epidemiological model for direct and indirect transmission of typhoid is formulated and studied. The basic reproductive number is

calculated and the global stability is investigated. Sensitivity analysis of the reproductive number suggests that indirect typhoid transmission has more impact on determining typhoid prevalence compared to direct transmission.

Abboubakar and Racke (2019) derived and analyzed a model for the control of typhoid fever which takes into account an imperfect vaccine combined with some other control measures already studied in the literature. They began by analyzing the model without control. They also computed the basic reproduction number R_0 and prove the local and global stability of the disease free equilibrium whenever R_0 is less than one through Lyapunov's theory. When R_0 is greater than one, they prove the local asymptotic stability of the unique endemic equilibrium through the Centre Manifold Theory and they find that the model exhibits a forward bifurcation.

Mushayabasa (2012) formulated and analyzed a mathematical model for assessing the impact of educational campaigns on controlling the transmission dynamics of typhoid in the community. The reproductive number has been computed. Stability of the model steady-states has

been examined. The impact of educational campaigns on controlling the transmission dynamics of typhoid has been discussed through the basic reproductive number and numerical simulations. At its best the study suggests that targeted education campaigns, which are effective at stopping transmission of typhoid more than 40% of the time, will be highly effective at controlling the disease in the community.

Nthiiri (2017) developed a non-linear mathematical model of typhoid fever diseases incorporating protection is hereby considered to study the global stability of equilibrium points. To study the global stability of the disease free equilibrium point and endemic equilibrium point, the method by Castillo-Chavez and a suitable Lyapunov function are used respectively. The disease free equilibrium point was found not to be globally asymptotically stable while the endemic equilibrium point is globally asymptotically stable. This implies that the disease transmission can be kept quiet low or manageable with minimal deaths in the presence of protection. Several research works were carried-out on typhoid fever disease by some scholars; Tilahun, Makinde and Malonza (2017), Edward (2017), Peter, Ibrahim, Akinduko and Rabiu (2017), Karunditu, Kimathi and Osman (2019), Mushayabasa (2012) etc.

1.2 Model Formulation

We divide the human population into five (5) compartments as follows; Susceptible class (S), infected class (I), Treatment class (T), Carrier class (C) and Recovered class (R). While the other compartment is the Unhygienic Environment that causes the transmission of the disease to the Susceptible Individuals which is represented by (U).

The Susceptible (S) are those who are at risk of being infected by the *Salmonella typhi* bacteria. We assumed a constant recruitment rate (Λ) to the susceptible class. The susceptible population is increased by those who recovered from the infection after losing immunity, (ν). However, the class decreases by those who acquired the bacteria as a result of interaction with the unhygienic

environment or ingesting contaminated food or water, $\lambda = \frac{(e + \beta(I + \theta_1 T + \theta_2 C))S(t)}{N(t)}$. It is assumed

that whenever a susceptible individual interacts with the environment where an infected or carrier individual defecates. It is also assumed that the individuals in the treatment class could not transmit the disease since they are undergoing treatment. μ represents the natural died rate for the whole population.

While the Infected class increases by those who acquired the disease at a rate λ and it is decreases by those individuals who progresses to the treatment compartment after diagnosing at a rate ε , while d_1 represents the rate of interaction between the environment and infected individuals. δ_I represents the Infected class disease induced death rate. It is assumed that an individual who is receiving treatment can die at a rate δ_T . It is also assumed that an infected individual who could not recover fully due to partial treatment will move to the carrier class at a rate ω while γ_1 represents the rate of recovery of an infected individual due to complete treatment. The rate of interaction between the carriers and the environment is represented by d_2 , while δ_C represents the death rate as a result of typhoid disease. γ_2 represents the rate of full recovery from carrier class to recovery class. Lastly, ϕ represents the diminishing of the bacteria from the environment. Therefore, the model equations are given below;

1.3 Model Equations

$$\left. \begin{aligned} \frac{dS(t)}{dt} &= \Lambda + \nu R(t) - \frac{[e + \beta(I + \theta_1 T + \theta_2 C)]S(t)}{N(t)} - \mu S(t) \\ \frac{dI(t)}{dt} &= \frac{[e + \beta(I + \theta_1 T + \theta_2 C)]S(t)}{N(t)} - (\varepsilon + d_1 + \delta_I + \mu)I(t) \\ \frac{dT(t)}{dt} &= \varepsilon I(t) - (\omega + \gamma_1 + \delta_T + \mu)T(t) \\ \frac{dC(t)}{dt} &= \omega T(t) - (\gamma_2 + d_2 + \delta_C + \mu)C(t) \\ \frac{dR(t)}{dt} &= \gamma_1 T(t) + \gamma_2 C(t) - (\nu + \mu)R(t) \\ \frac{dU(t)}{dt} &= d_1 I(t) + d_2 C(t) - \phi U(t) \end{aligned} \right\} (1)$$

Table 1: Parameters of the Model

Symbols	Descriptions	Values and references
Λ	Recruitment rate into the susceptible class	1000 Assumed
ν	Recovery rate	0.3 Stephen ¹ (2017)
μ	Natural death rate	0.003 Assumed
δ_I	Typhoid induced death rate of infected class	0.012 Stephen ¹ (2017)
δ_T	Typhoid induced death rate of treatment class	0.001 Assumed
δ_C	Typhoid induced death rate of carrier individuals	0.01 Stephen ¹ (2017)
e	Ingestion rate contaminated water and food	0.9 Tilahum et al (2017)
β	Transmission rate coefficient	0.0001 Mushayabaya (2012)
θ_1	Modification parameter associated with reduced transmission rate by individuals in treatment class	0.001 Assumed
θ_2	Modification parameter associated with reduced transmission rate by carrier class	0.01 Stephen ¹ (2017)
d_1	Rate of interaction between the infected and the unhygienic Environment	0.9 Tilahum et al (2017)
d_2	Rate of interaction between the carrier and the unhygienic Environment	0.8 Tilahum et al (2017)
ε	Rate of movement of an infected individual to the treatment class	0.002 Tilahum et al (2017)
γ_1	Rate at which infected individual fully recovered after undergoing treatment	0.1 Assumed
γ_2	Rate at which carrier individual recovered from the disease	0.05 Stephen ² (2017)
ω	Rate of symptom disappearance after treatment	0.01 Assumed
ϕ	Diminishing rate for bacteria	0.4 Stephen ² (2017)

2.0 MODEL ANALYSIS

2.1 Disease Free Equilibrium

At DFE, we set the LHS of (1) to zero. i.e.

$$\dot{S}(t) = \dot{I}(t) = \dot{T}(t) = \dot{C}(t) = \dot{R}(t) = \dot{U} = 0 \quad (2)$$

In the absence of typhoid fever we have,

$$I(t) = T(t) = C(t) = R(t) = U(t) = 0 \quad (3)$$

Solving (4.8) and (4.9), we obtain the disease free equilibrium (DFE) denoted by Z_o

$$Z_o = (S^*, I^*, T^*, C^*, R^*, U^*) = \left(\frac{\Lambda N}{e + \mu N}, 0, 0, 0, 0, 0 \right) \quad (4)$$

2.2 Basic Reproduction Number R_0

The basic reproduction number can be defined as the average number of secondary infectious individual in a completely susceptible population. We used the next generation matrix method of and

computing R_0 described by (Van den Driessche & Watmough, 2002) on the model (1). Thus,

$$R_0 = \rho FV^{-1} \quad (5)$$

Theorem: the disease free equilibrium (DFE) point is said to be locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: to prove the theorem, we obtain and evaluate the non-negative matrix F of the new infection terms and non-singular matrix V of transition terms for (1) at DFE as follows;

$$F = \begin{bmatrix} \frac{\Lambda\beta}{e + \mu N} & \frac{\Lambda\beta\theta_1}{e + \mu N} & \frac{\Lambda\beta\theta_2}{e + \mu N} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (6)$$

$$V = \begin{bmatrix} (\varepsilon + d_1 + \delta_I + \mu) & 0 & 0 & 0 \\ 0 & (\omega + \gamma_1 + \delta_T + \mu) & 0 & 0 \\ 0 & 0 & (\gamma_2 + d_2 + \delta_C + \mu) & 0 \\ -d_1 & 0 & -d_2 & \phi \end{bmatrix} \tag{7}$$

$$FV^{-1} = \begin{bmatrix} \frac{\Lambda\beta}{(e + \mu N)(\varepsilon + d_1 + \delta_I + \mu)} & \frac{\Lambda\beta\theta_1}{(e + \mu N)(\omega + \gamma_1 + \delta_T + \mu)} & \frac{\Lambda\beta\theta_2}{(e + \mu N)(\gamma_2 + d_2 + \delta_C + \mu)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{8}$$

We obtained the characteristic equation by evaluating (8), gives

$$\lambda^4 - \frac{\Lambda\beta}{(e + \mu N)(\varepsilon + d_1 + \delta_I + \mu)} \lambda^3 = 0 \tag{9}$$

we solve (9) to obtain the eigenvalues

$$\lambda_1 = \lambda_2 = \lambda_3 = 0 \text{ and } \lambda_4 = \frac{\Lambda\beta}{(e + \mu N)(\varepsilon + d_1 + \delta_I + \mu)}$$

the dominant eigenvalue of the characteristics equation is λ_4 .

So the basic reproduction number R_0 is given as

$$R_0 = \frac{\Lambda\beta}{(e + \mu N)(\varepsilon + d_1 + \delta_I + \mu)} \tag{10}$$

2.3 Global Stability of the Disease Free Equilibrium

We adopt the method of Castillo-Chavez, *et al.*, (2002), using the following conditions;

$$\frac{dX}{dt} = F(X, Z) \tag{11}$$

$$\frac{dZ}{dt} = G(X, Z); G(X, 0) = 0 \tag{12}$$

where $X = (S, R)$ and $Z = (I, T, C)$. $X \in R^2$

denotes the uninfected individuals, while $Z \in R^3$ denotes the infected individuals.

$E_0 = \left(\frac{\Lambda N}{e + \mu N}, 0, 0, 0, 0, 0 \right)$ denotes the DFE of

model (1).

For global stability to be achieved, the following two conditions must be met;

- (i) $\frac{dX}{dt} = F(X, 0)$, X^* is globally asymptotically stable
- (ii) $G(X, Z) = AZ - \hat{G}(X, Z)$, where $\hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Pi$

$A = D_Z G(X, 0)$ is an M – matrix (off diagonal elements of A are non-negative) and Π is the biological feasible region.

Condition (i);

$$\frac{dX}{dt} = F(X, 0) = \begin{bmatrix} \Lambda - eS - \mu S \\ -\nu R - \mu R \end{bmatrix} \tag{13}$$

from (13) we obtain the following differential equations;

$$\frac{dS}{dt} = \Lambda - (e + \mu)S \tag{14}$$

$$S(t) = \frac{\Lambda}{e + \mu} + Ce^{-(e + \mu)t} \tag{15}$$

at $t = 0$;

$$S(t) = \frac{\Lambda}{e + \mu} + \left(S^*(0) - \frac{\Lambda}{e + \mu} \right) e^{-(e + \mu)t} \tag{16}$$

$$\frac{dR}{dt} = -(v + \mu)R \tag{17}$$

$$\ln R = -(v + \mu)t + C \tag{18}$$

$$R(t) = R^*(0)e^{-(v + \mu)t} \tag{19}$$

$S^*(t) + R^*(t) \rightarrow N^*(t)$ as $t \rightarrow \infty$ irrespective of the values of $S^*(0)$ and $R^*(0)$ in (16) and (19).

therefore, $X^* = \left[\frac{\Lambda N}{e + \mu N}, 0 \right]$ is globally

asymptotically stable.

Condition (ii)

$$G(X, Z) = AZ - \hat{G}(X, Z) \tag{20}$$

$$A = \begin{bmatrix} -\varepsilon - d_1 - \delta_I - \mu & \frac{\beta\theta_1 S}{N} & \frac{\beta\theta_2 S}{N} & 0 \\ \varepsilon & -\omega - \gamma_1 - \delta_T - \mu & 0 & 0 \\ 0 & \omega & -\gamma_2 - d_2 - \delta_C - \mu & 0 \\ d_1 & 0 & d_2 & -\phi \end{bmatrix} \tag{21}$$

$$\hat{G}(X, Z) = \begin{bmatrix} \frac{[e + \beta(I + \theta_1 T + \theta_2 C)]}{N} S - (\varepsilon + d_1 + \delta_I + \mu) I \\ \varepsilon I - (\omega + \gamma_1 + \delta_T + \mu) T \\ \omega T - (\gamma_2 + d_2 + \delta_C + \mu) C \\ d_1 I + d_2 C - \phi U \end{bmatrix} \quad (22)$$

Thus, $G(X, Z) = AZ - \hat{G}(X, Z) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$, i.e.

$G(X, Z) = [0 \ 0 \ 0 \ 0]^T$. this shows that $G(X, Z) = 0$

2.4 Optimal Control Analysis

The optimal control problem is formulated for model (1) by considering the time-dependent control variables $u_1(t)$, $u_2(t)$ and $u_3(t)$ in order to determine the optimal typhoid control strategies (Sanitation and Treatments) with minimal implementation implication.

For the optimal control problem of the given system, we consider the control variables $u(t) = [u_1(t), u_2(t), u_3(t)] \in U$ relative to the state variables $S(t), I(t), D(t), C(t), R(t), U(t)$ where

$$\begin{aligned} H = & A_1 I + A_2 D + A_3 C + \frac{B_1 u_1^2}{2} + \frac{B_2 u_2^2}{2} + \frac{B_3 u_3^2}{2} \\ & + \lambda_S \left\{ \Lambda + \nu R(t) - (1 - u_1(t)) \left[\frac{e + \beta(I(t) + \theta_1 D(t) + \theta_2 C(t))}{N(t)} \right] S(t) - \mu S(t) \right\} \\ & + \lambda_I \left\{ (1 - u_1(t)) \left[\frac{e + \beta(I(t) + \theta_1 D(t) + \theta_2 C(t))}{N(t)} \right] S(t) - (\varepsilon + d_1 + \delta_I + \mu) I(t) \right\} \\ & + \lambda_D \left\{ \varepsilon I(t) - (\omega + u_2(t) \gamma_1 + \delta_D + \mu) D(t) \right\} \\ & + \lambda_C \left\{ \omega D(t) - (u_3(t) \gamma_2 + d_2 + \delta_C - \mu) C(t) \right\} \\ & + \lambda_R \left\{ u_2(t) \gamma_1 D(t) + u_3(t) \gamma_2 C(t) - (\nu + \mu) R(t) \right\} \\ & + \lambda_U \left\{ d_1 I(t) + d_2 C(t) - \phi U(t) \right\} \end{aligned} \quad (23)$$

where $\lambda_S, \lambda_I, \lambda_D, \lambda_C, \lambda_R$ and λ_U are the adjoint variables or co-state variables.

In order to find the necessary conditions for this optimal control, we apply the Pontryagin's Maximum Principle and the existence result for

control variables are bounded and measured with $\{U = (u_1, u_2, u_3)\}$ is Lebesgue measurable on $[0, 1]$, $0 \leq u_i(t) \leq 1, t \in [0, tf], i = 1, 2, 3$

The objective functional is given as

$$J = \min_{u_1, u_2, u_3} \int_0^{tf} \left(A_1 I + A_2 D + A_3 C + \frac{B_1 u_1^2}{2} + \frac{B_2 u_2^2}{2} + \frac{B_3 u_3^2}{2} \right) dt \quad (2)$$

where tf is the final time and the coefficients A_1, A_2, A_3, B_1, B_2 and B_3 are positive weights to balance the factors. The terms $A_1 I, A_2 D$ and $A_3 C$ are the cost of infection while $B_1 u_1^2, B_2 u_2^2$ and $B_3 u_3^2$ are the costs associated with u_1 (protection), u_2 (treatment) and u_3 (treatment of carrier individuals).

We seek optimal control u_1, u_2, u_3 such that

$$J(u_1^*, u_2^*, u_3^*) = \min \{ J(u_i, u_2, u_3), (u_i, u_2, u_3 \in U) \} \quad (3)$$

The necessary conditions that an optimal control must satisfy the Pontryagin's Maximum Principle (PMP), (Pontryagin, Boltyanskii, Gamkrelidze, & Mishchenko 1962) that converts (1) into a problem of minimizing a pointwise Hamiltonian H , with respect to u_1, u_2, u_3 .

the optimal control as described by Lenhart & Workman (2007).

Theorem: The optimal controls u_1^*, u_2^*, u_3^* and solutions S, I, D, C, R, U of the corresponding state system (1), then there exists adjoint variables $\lambda_S, \lambda_I, \lambda_D, \lambda_C, \lambda_R$ and λ_U satisfying

$$-\frac{d\lambda_S}{dt} = \frac{\partial H}{\partial S(t)}, \quad -\frac{d\lambda_I}{dt} = \frac{\partial H}{\partial I(t)}, \quad -\frac{d\lambda_D}{dt} = \frac{\partial H}{\partial D(t)}, \quad -\frac{d\lambda_C}{dt} = \frac{\partial H}{\partial C(t)}, \quad -\frac{d\lambda_R}{dt} = \frac{\partial H}{\partial R(t)}, \quad \text{and} \quad -\frac{d\lambda_U}{dt} = \frac{\partial H}{\partial U(t)} \quad (24)$$

then,

$$\left. \begin{aligned} \frac{\partial H}{\partial S} &= (1-u_1(t)) \frac{[e + \beta(I + \theta_1 T + \theta_2 C)]}{N(t)} (\lambda_S - \lambda_I) + \mu \lambda_S \\ \frac{\partial H}{\partial I} &= (1-u_1(t)) \frac{\beta S(t)}{N(t)} (\lambda_S - \lambda_I) + \varepsilon (\lambda_I - \lambda_D) + d_1 (\lambda_I - \lambda_U) + (\delta_I + \mu) \lambda_I - A_1 \\ \frac{\partial H}{\partial D} &= (1-u_1(t)) \frac{\beta \theta_1 S(t)}{N(t)} (\lambda_S - \lambda_I) + \gamma_1 u_2(t) (\lambda_D - \lambda_R) + \omega (\lambda_D - \lambda_C) + (\delta_D + \mu) \lambda_D - A_2 \\ \frac{\partial H}{\partial C} &= (1-u_1(t)) \frac{\beta \theta_2 S(t)}{N(t)} (\lambda_S - \lambda_I) + \gamma_2 u_3(t) (\lambda_C - \lambda_R) + d_2 (\lambda_C - \lambda_U) - (\delta_C + \mu) \lambda_C - A_3 \\ \frac{\partial H}{\partial R} &= v (\lambda_R - \lambda_S) + \mu \lambda_R \\ \frac{\partial H}{\partial U} &= \phi \lambda_U \end{aligned} \right\} \quad (25)$$

with transversality conditions

$$\lambda_S(tf) = \lambda_I(tf) = \lambda_D(tf) = \lambda_C(tf) = \lambda_R(tf) = \lambda_U(tf) = 0 \quad (26)$$

using the optimality conditions;

$$u_1^* = \max \{0, \min(1, \bar{u}_1)\},$$

$$u_2^* = \max \{0, \min(1, \bar{u}_2)\} \quad \text{and}$$

$$u_3^* = \max \{0, \min(1, \bar{u}_3)\}$$

we solve the given optimality conditions to find

$$\bar{u}_1 = u_1, \bar{u}_2 = u_2 \quad \text{and} \quad \bar{u}_3 = u_3, \quad \text{we let}$$

$$\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0 \quad \text{and} \quad \frac{\partial H}{\partial u_3} = 0, \quad (27)$$

we differentiate (4) with respect to u_i , $0 < u_i \leq 1$,

where $i = 1, 2, 3$ to obtain

$$\frac{\partial H}{\partial u_1} = -B_1 u_1 + \frac{(e + \beta(I + \theta_1 D + \theta_2 C))}{N(t)} S(t) \lambda_S \quad (28)$$

$$u_1 = \frac{(e + \beta(I + \theta_1 D + \theta_2 C))}{B_1 N(t)} S(t) (\lambda_I - \lambda_S)$$

$$\frac{\partial H}{\partial u_2} = -B_2 u_2 + \gamma_1 D(t) \lambda_D - \gamma_1 D(t) \lambda_R \quad (29)$$

$$u_2 = \frac{\gamma_1 D(t) (\lambda_D - \lambda_R)}{B_2}$$

$$\frac{\partial H}{\partial u_3} = -B_3 u_3 + \gamma_2 C(t) \lambda_C - \gamma_2 C(t) \lambda_R \quad (30)$$

$$u_3 = \frac{\gamma_2 C(t) (\lambda_C - \lambda_R)}{B_3}$$

therefore,

$$u_1^* = \max \left\{ 0, \min \left(1, \frac{(e + \beta(I + \theta_1 D + \theta_2 C))}{B_1 N(t)} S(t) (\lambda_I - \lambda_S) \right) \right\} \quad (31)$$

$$u_2^* = \max \left\{ 0, \min \left(1, \frac{\gamma_1 D(t) (\lambda_D - \lambda_R)}{B_2} \right) \right\} \quad (32)$$

$$u_3^* = \max \left\{ 0, \min \left(1, \frac{\gamma_2 C(t) (\lambda_C - \lambda_R)}{B_3} \right) \right\} \quad (33)$$

3.0 NUMERICAL SIMULATIONS

We investigate numerically, the effects of the control strategies on the spread of typhoid fever in a society. We solve the optimality system using forward backward iterative method which resulted into the optimal sanitation and two stages treatment strategies in controlling the typhoid fever transmission dynamics. The optimal control solution is obtained by solving the optimality system, which consists of the state system and the adjoint system. Because of the transversality conditions, we solve the state equations with a presumption for the controls over the simulated time using the Runge–Kutta scheme of fourth-order.

We use sanitation as the first control strategy in the susceptible class, the treatment on the infected individuals and the treatment on the carrier infected individuals to see the effect of each control in their respective compartments.

3.1 Implementing Sanitation (u_1) as Control

We implement sanitation u_1 as a control strategy against the transmission typhoid fever disease in a population. We apply u_1 to curtail the transmission

by optimizing the objective functional (J) while we put $u_2 = 0$ and $u_3 = 0$, which shows the effectiveness of the control in reducing the disease transmission. We detect from figure 1(a) a significant increase of the susceptible population, while, figures 1(b) - 1(e) shows a sizeable decrease in the infected, treatment and carrier individuals as against when none of the control is applied. Figure

(f) shows the sanitation as a control strategy reduces the disease transmission in the environment. Therefore, the sanitation is very effective in controlling the disease transmission in the population. While figure 1(g), the control profile shows a rapid decline in the typhoid fever transmission from 100% to the lower bound at $t = 99$ days.

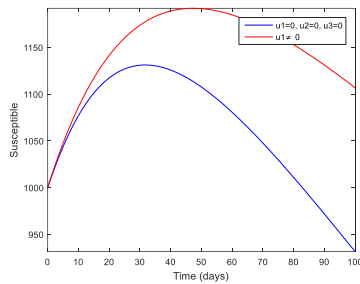


Figure 1(a)

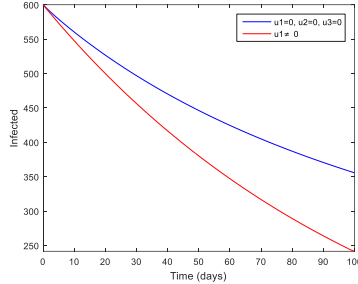


Figure 1(b)

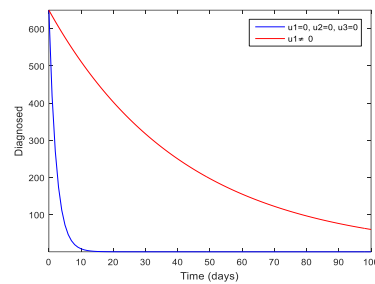


Figure 1(c)

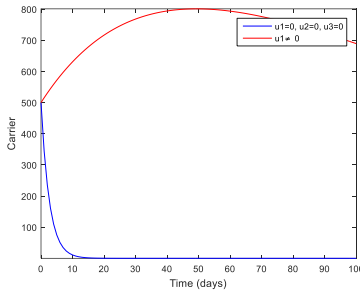


Figure 1(d)

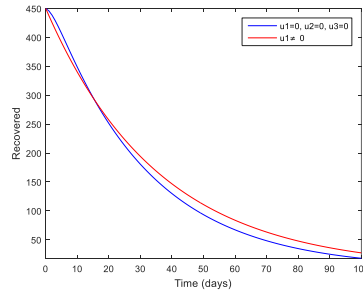


Figure 1(e)

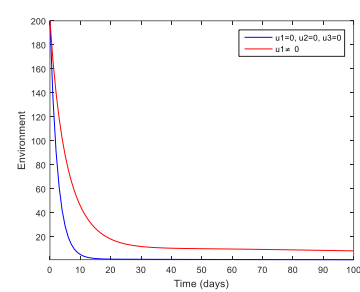


Figure 1(f)

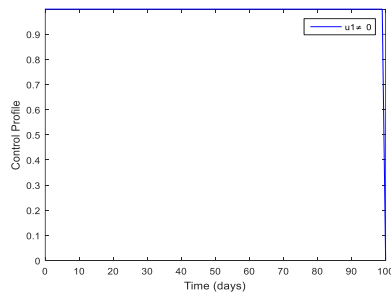


Figure 1(g)

Figure 1: Implementing Sanitation u_1 as Control Strategy for Protecting the Susceptible Individuals

3.2 Implementing (u_2) as control for treatment of infected individuals

The treatment u_2 of infected individuals was applied to optimize the objective functional (J) while setting $u_1 = 0$ and $u_3 = 0$. It has been observed from figure 2(a) – 2(e) that the number of infected individuals declines gradually due to the

drug efficacy of the infected treatment as compared to the situation where there is no control. Figure 2(f) shows how the environmental transmission of the diseases reduces due to the treatment of the infected individuals. While, the control profile, figure 2(g) shows how the control on treatment decreases the infection swiftly from the highest bound to the lower bound at $t = 98$ days.

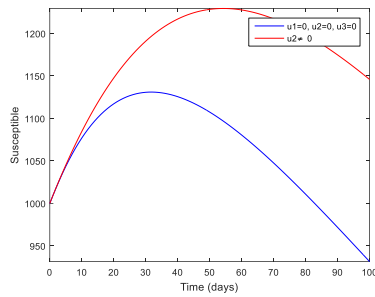


Figure 2(a)

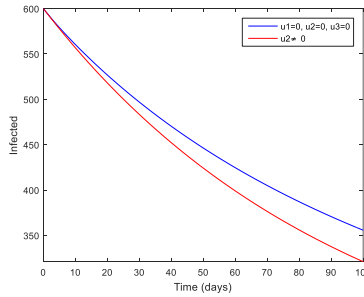


Figure 2(b)

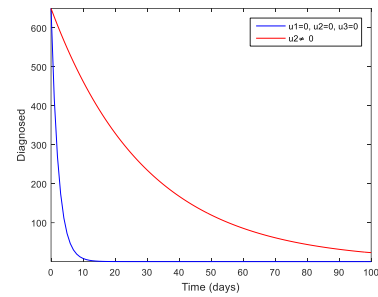


Figure 2(c)

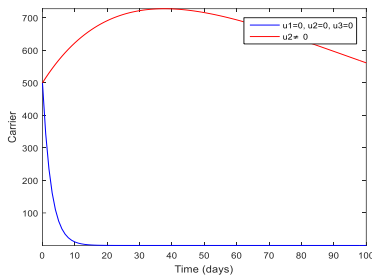


Figure 2(d)

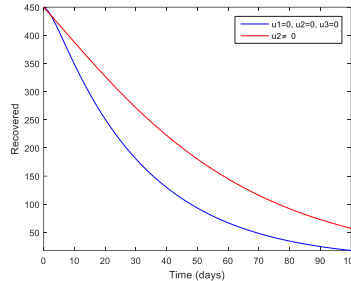


Figure 2(e)

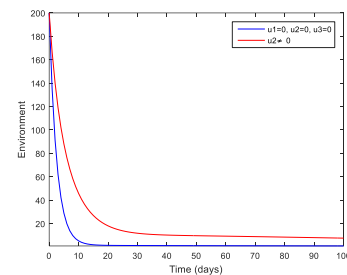


Figure 2(f)

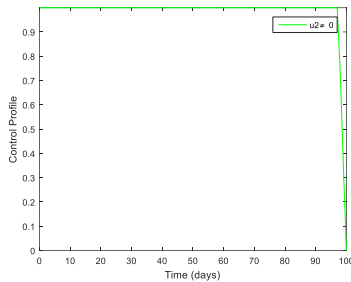


Figure 2(g)

Figure 2: Using (u_2) as Control Strategy for Treatment of Infected Individuals

3.3 Implementing Treatment on Carrier Individuals (u_3) as Control

The treatment u_3 as a control on carrier individuals, was implement to optimize the objective functional (J) by setting $u_1 = 0$ and $u_2 = 0$. It has been shown from figures 3(a) – 3(e) that the number of carrier individual's declines due to the drug

efficacy of control on carrier treatment as observed in the absence of any control. Figure 3(f) shows the reduction in environmental transmission of the diseases because of the treatment of the carrier individuals. While, the control profile, figure 3(g) shows how the control decrease the infection swiftly from the highest bound to the lower bound at $t = 99$ days.

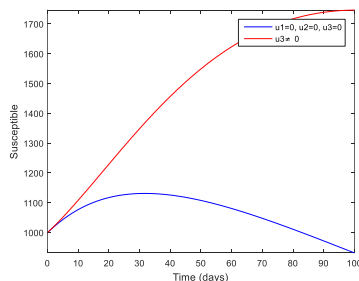


Figure 3(a)

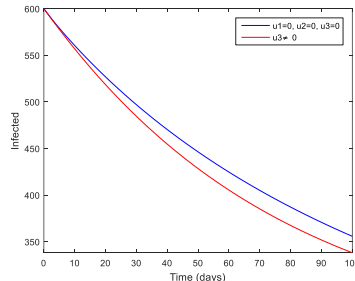


Figure 3(b)

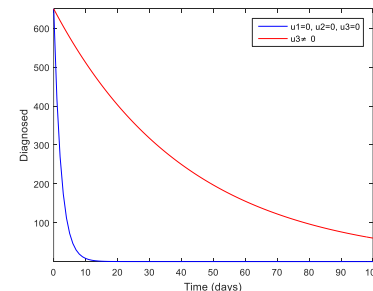


Figure 3(c)

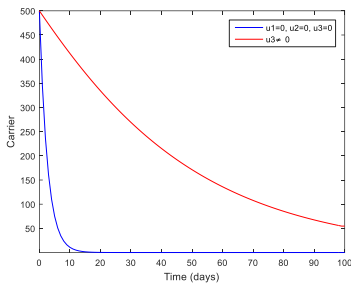


Figure 3(d)

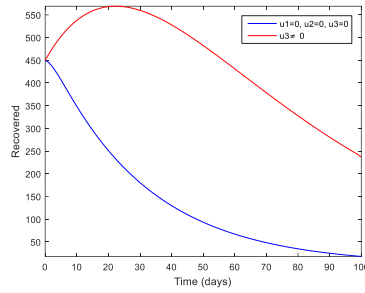


Figure 3(e)

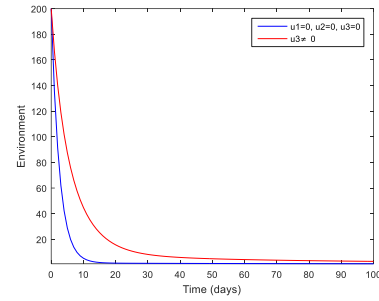


Figure 3(f)

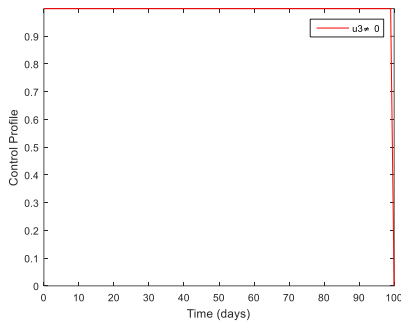


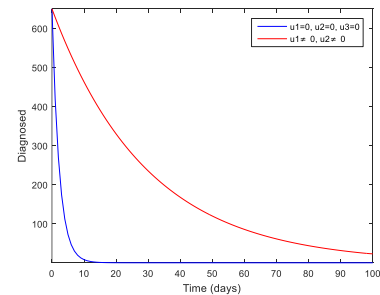
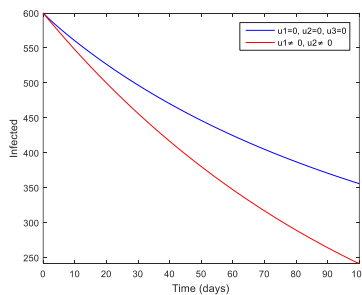
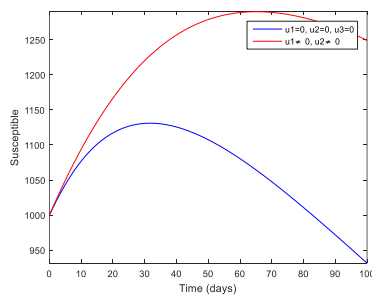
Figure 3(g)

Figure 3: Using Treatment on Carrier Individuals (u_3) as Control Strategy

3.4 Implementing sanitation u_1 and Treatment for Infected Individuals u_2 as Control Strategy

The control on sanitation u_1 and the control treatment of infected individuals u_2 to optimize the objective functional (J) while we set $u_3 = 0$, and it shows clearly the effectiveness of the two controls simultaneously. We observed from figures 4(a) –

4(e) the effect of applying the two (2) controls at the same time and it shows how the gradual declines in transmission as compared with when there is no control. The environmental transmission also decreases due to the implementation of the two controls simultaneously. While, the control profile, figure 4(g) shows a decrease in the infection transmission swiftly from the highest bound to the lower bound at approximately 100 days.



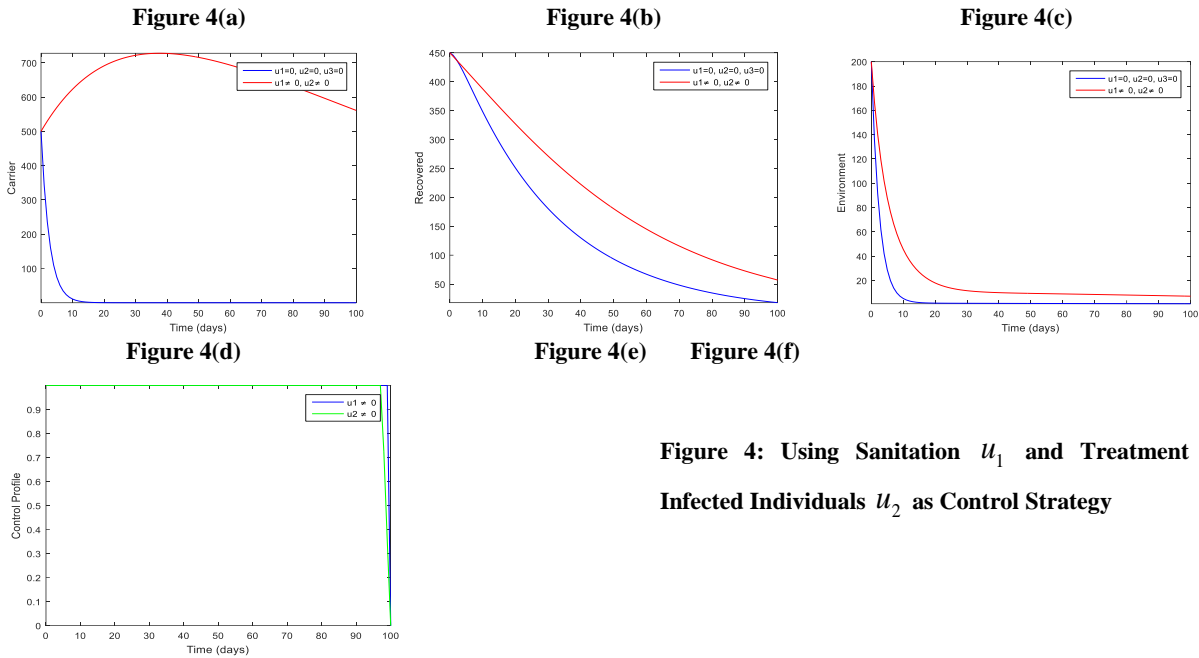


Figure 4: Using Sanitation u_1 and Treatment for Infected Individuals u_2 as Control Strategy

Figure 4(g)

3.5 Implementing Sanitation u_1 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

The control u_1 (sanitation) and the treatment of carrier individuals u_3 are used to optimize the objective functional (J) while we set $u_2 = 0$, and it

clearly shows the effectiveness of implementing the two controls concurrently. Figure 5(a) – 5(f) shows how the infection transmission declines gradually as compared with when there is no control. While the control profile, figure 5(g) show that the implementation of the two controls concurrently decrease the infection transmission swiftly from the highest bound to the lower bound at $t = 97$ days and $t = 99$ days respectively.

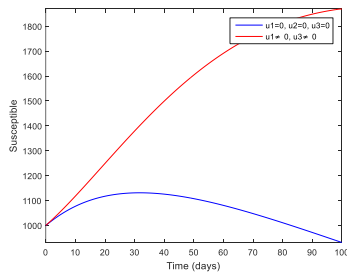


Figure 5(a)

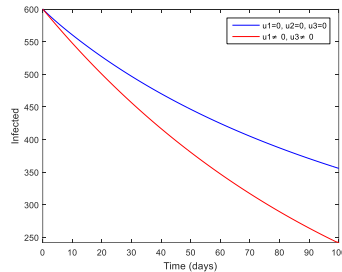


Figure 5(b)

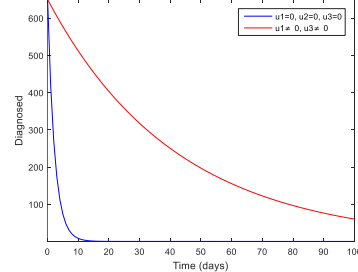


Figure 5(c)

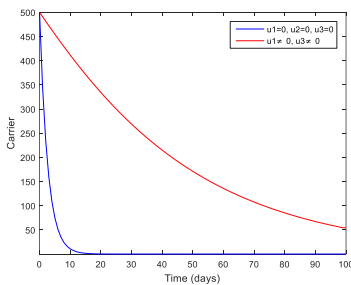


Figure 5(d)

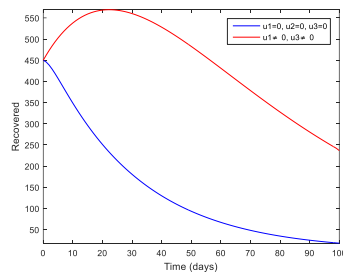


Figure 5(e)

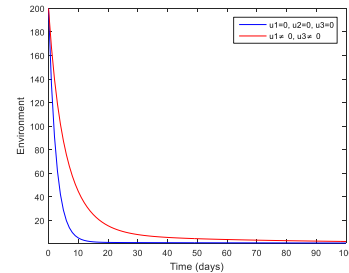


Figure 5(f)

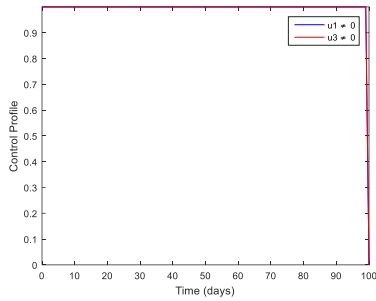


Figure 5(g)

Figure 5: Using Sanitation u_1 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

3.6 Implementing Sanitation u_2 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

The control u_2 (treatment on infected individuals) and the control u_3 (treatment of carrier individuals) are implemented concomitantly to optimize the objective functional (J) while setting $u_1 = 0$, and it

visibly shows the effectiveness of implementing the two controls concurrently. Figure 6(a) – 6(f) shows how the infection transmission declines gradually as compared with when there is no implementation of any control. While the control profile, figure 6(g) shows that the implementation of the two controls in parallel decreases the disease transmission rapidly from the highest bound to the lower bound at $t = 98$ days and $t = 99$ days respectively.

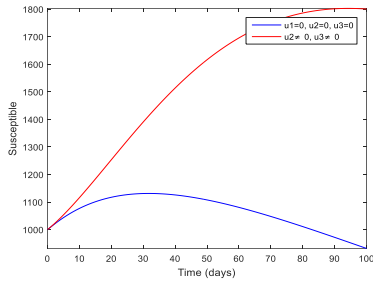


Figure 6(a)

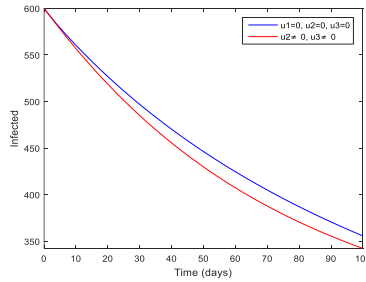


Figure 6(b)

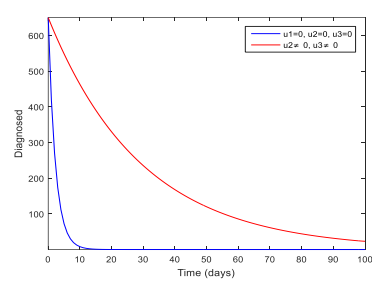


Figure 6(c)

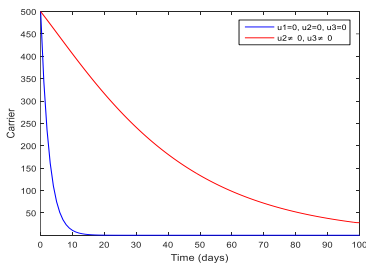


Figure 6(d)

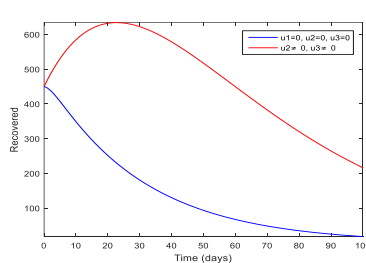


Figure 6(e)

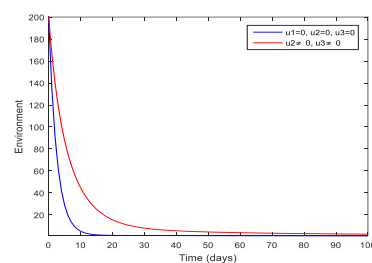


Figure 6(f)

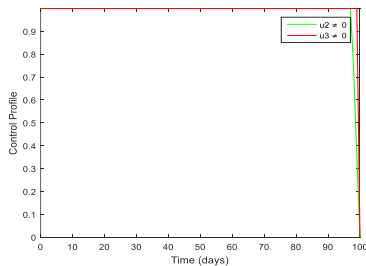


Figure 6(g)

Figure 6: Using Sanitation u_2 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

3.7 Implementing Sanitation u_1 , Treatment of Infected Individuals u_2 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

The controls u_1 (sanitation), u_2 (treatment of infected individuals) and u_3 (treatment of carrier individuals) are used to optimize the objective

functional (J) in order to effectively implement all the three controls concurrently. Figure 5(a) – 5(f) shows how the infection transmission declines gradually as compared with when there is no control. While the control profile, figure 5(g) shows that the implementation of all the three controls concurrently decreases the transmission of the disease from the highest bounds to the lower bounds at $t= 97$ days, $t = 99$ days and 100 days respectively.

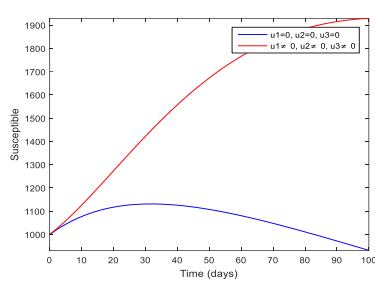


Figure 7(a)

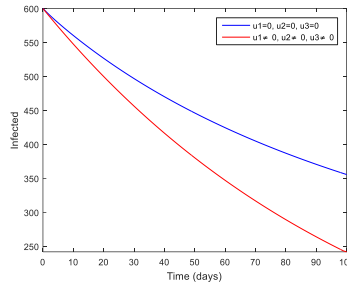


Figure 7(b)

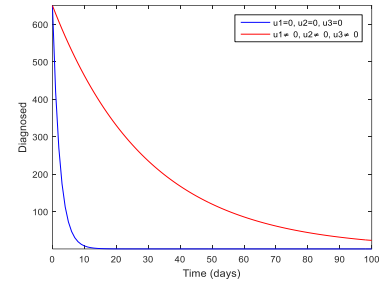


Figure 7(c)

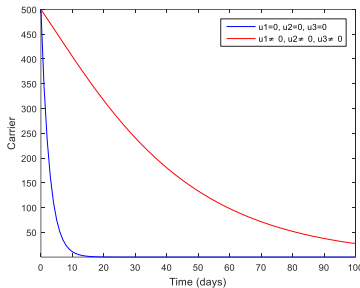


Figure 7(d)

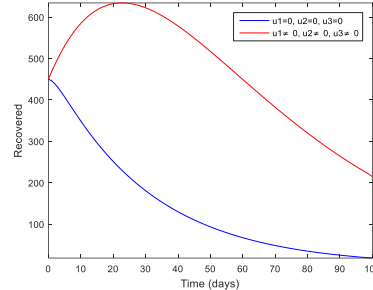


Figure 7(e)

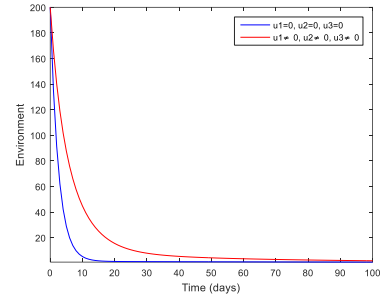


Figure 7(f)

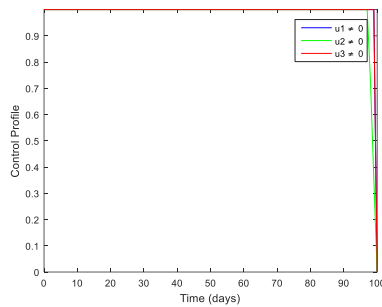


Figure 7(g)

Figure 7: Using Sanitation u_1 , Treatment of Infected Individuals u_2 and Treatment of Carrier Individuals u_3 at Stage as Control Strategy

4.0 CONCLUSION

In this work, a mathematical model of typhoid fever transmission dynamics was formulated, by incorporating three control strategies which will help in curtailing the transmission dynamics of the disease in the population. We considered optimal control strategy using Pontryagin’s maximum principle in analyzing the effectiveness of the three (3) controls: sanitation u_1 , treatment of infected individuals u_2 and treatment of carrier individuals u_3 . We observed that the implementation of the control(s) either individually or concurrently will have a very significant impact in decreasing the transmission dynamics of the disease in the population after sometimes.

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