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Global Stability Analysis of Typhoid Fever Model

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Abstract: We analyze with four compartments a deterministic nonlinear mathematical model of typhoid fever transmission dynamics. Using the Lipchitz condition, we verified the existence and uniqueness of the model solutions to establish the validity of the model and derive the equilibria states of the model that is, disease-free equilibrium (DFE) and endemic equilibrium (EE). The computed basic reproductive number R_0 was used to establish that the disease-free equilibrium is globally asymptotically stable when its numerical value is less than one, the disease will be under control. In addition, the Lyapunov function was applied to investigate the stability property for the (DFE). The model was numerically simulated to validate the results of the analysis.

Keywords*:* typhoid fever, equilibria, stability, nonlinear mathematical model

1. INTRODUCTION

Typhoid fever is an infection caused by Salmonella typhi bacteria. Typhoid is usually triggered by the ingestion of food or water contaminated with feces or urine of infected individuals and is, therefore, a typical illness in regions with poor sanitation. (Brooks [1]; Roumagnac *et al.,* [2]). In developing economies, typhoid fever outbreaks occur from time to time in overwhelmed areas and refugee camps with high population density. The disease causes high morbidity in children below ten years of age, with at least seventeen million new cases globally and nearly 600 000 deaths annually. The disease is not rampant in North America: In the US, an estimated four hundred cases are reported every year; seventy percent of the cases are traced to those who return from endemic regions. The mortality of typhoid fever is ten percent, but with adequate treatment, it can be limited to one percent. (Lin *et al.,* [3]; Sinha *et al.,* [4]; Hyman, [5]).

Intestinal fever treatment is anchored on the blood culture condition of the patients. If the species is sensitive, the oral antibiotic is used. Typhoid fever is becoming an increasingly common illness worldwide, making antibiotic treatment more expensive and harder. Enteric fever signs vary and are similar to the signs of different microbial infections. Symptoms of typhoid fever are as follows: variable level of high fever in 75 cases, body aches and muscle pain, chills, shriveled loss of appetite, abdominal pain in 20 to 40 cases, nose bleeds, headache, dizziness, rashes on the skin, bowel constipation or looseness, weakness and fatigue, sore throat and cough (Lifshitz [6]). Some mathematical models have been formulated on the transmission of typhoid fever. (Adetunde [7]; Lauria *et al.,* [8];

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Kalajdzievska [9]; Mushayabasa [10]; Cvjetanovic *et al.,*[11]; Moffact [12]; Pitzer *et al.,* [13]; Date *et al.,*[14]; Muhammad, *et al.,*[15]; Watson & Edmunds [16]; Nthiiri [17]; Moatlhod & Gosaamang [18]; Mushayabasa [19]; Peter & Ibrahim [20]; Peter *et al.,*[21]; Peter and Ibrahim [22]). The aim of this study is to extend and complement previous works by formulating a model that captures the following controls; vaccination and education.

2. MATERIALS AND METHODS

The model comprises four compartments: Susceptible class; represents the proportion of those who are prone to typhoid. Infected class; represents the population of individuals who have been infected with typhoid fever and capable of transmitting the infection to susceptible populations through interaction. Carrier-class; represents the population of individuals who have been infected with typhoid fever but without signs of infectiousness in them. Recovered compartment; represent the number of individuals infected with typhoid fever but are now cured of typhoid as a result of treatment.

Recruitment into susceptible populations is by birth or immigration at the rate θ. It is assumed that a certain proportion in the susceptible class moved to the carrier infectious class at rate ρ while the complement $1-\rho$ moved to the infectious compartment. We also assume that the rate of disease transmission β of carrier individuals will be higher than the disease transmission rate γ of infected individuals this is because they are more likely to be unaware of their infection.

Carrier's disease symptom is noticeable at the rate α. Contagious individuals may receive treatment and recuperate at the rate δ. Susceptible individuals receive vaccination to protect themselves from contracting the disease at the rate Ψ. $1-\phi$ is an educational parameter that serves as the limitation for carriers and indicative persons from transmitting typhoid fever. This parameter lies within the range $0 \le \phi \le 1$. When $\phi = 0$ it implies that educational campaign not in position so that the vulnerable population are oblivious of typhoid fever and when $\phi = 1$ it denotes that all vulnerable persons are well informed of the causes of typhoid fever, that is, they explicitly understand what brings about the diseases, how the disease is being transmitted and how to safeguard themselves from being infected by the disease.

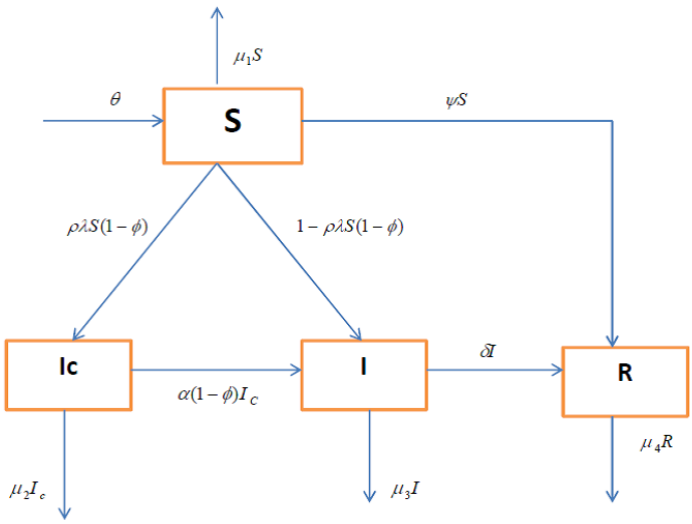

Figure 1. Pictorial Description of the Model

$$\left.\begin{aligned}
\frac{dS}{dt} &= \theta - \mu_1 S - \lambda S(1-\phi) - \psi S \\
\frac{dIc}{dt} &= \rho\lambda S(1-\phi) - \mu_2 I_c - \alpha(1-\phi) I_c \\
\frac{dI}{dt} &= (1-\rho)\lambda S(1-\phi) + \alpha(1-\phi) I_c - (\mu_3 + \delta) I \\
\frac{dR}{dt} &= \psi S + \delta I - \mu_4 R
\end{aligned}\right\} \quad (1)$$

Where $\lambda = \beta I_c + \gamma I$

$$\left.\begin{aligned}
\frac{dS}{dt} &= \theta - \mu_1 S - S(\beta I_c + \gamma I)(1-\phi) - \psi S \\
\frac{dIc}{dt} &= \rho S(\beta I_c + \gamma I)(1-\phi) - \mu_2 I_c - \alpha(1-\phi) I_c \\
\frac{dI}{dt} &= (1-\rho)(1-\phi) S(\beta I_c + \gamma I) + \alpha(1-\phi) I_c - (\mu_3 + \delta) I \\
\frac{dR}{dt} &= \psi S + \delta I - \mu_4 R
\end{aligned}\right\} \quad (2)$$

Table 1. Variables and parameters interpretation

Variables	Description
$S(t)$	vulnerable populations
$Ic(t)$	carrier contagious populations
$I(t)$	contagious populations
$R(t)$	recovered populations
Parameters	Interpretation
θ	recruitment rate in vulnerable class
μ_1	mortality rate of vulnerable class
μ_2	mortality rate for carrier infected class
μ_3	mortality rate for infected class
μ_4	mortality rate for recovered class
α	the rate at which individual carriers develops symptoms
ϕ	parameter for education
ψ	rate of vaccination
ρ	the probability of newly infected persons becoming asympt omatic or carrier
β	rate of transmission of infection for carrier individuals
γ	rate of transmission of infection for infectious individuals
λ	force of disease infection
δ	rate at which individual in the infectious class recovered

3. SOLUTION OF THE MODEL

3.1. Existence and Uniqueness of the Model

The validity of any mathematical model is a function of the solution of the model, provided the solution is unique. We verify the singularity of the solution of the model in equation (2) via the popular Lipchitz condition.

Given the system of equation (2) be as follows

$$\left.\begin{aligned} J_1 &= \theta - \mu_1 S - S(\beta I_c + \gamma I)(1-\phi) - \psi S \\ J_2 &= \rho S(\beta I_c + \gamma I)(1-\phi) - \mu_2 I_c - \alpha(1-\phi) I_c \\ J_3 &= (1-\rho)(1-\phi) S(\beta I_c + \gamma I) + \alpha(1-\phi) I_c - (\mu_3 + \delta) I \\ J_4 &= \psi S + \delta I - \mu_4 R \end{aligned}\right\} \quad (3)$$

Theorem 3.1

Assuming the region $\Re$ is denoted by

$$|c - c_0| \le a, \|y - y_0\| \le 1, y = (y_1, y_2 ....., y_n), y_o = (y_{10}, y_{20}, ...y_{no}) \quad (4)$$

And suppose that R' meets the Lipchitz condition

$$\|f(c, y_1) - f(c, y_2)\| \le k\|y_1 - y_2\|$$

On each occasion, the pairs (c, y_1) and $(c, y_2) \in J$, where k is a positive constant. Hence, there is a constant $\delta \ge 0$ such that there exists a unique continuous vector solution $y(c)$ of the system in the interval $c - c_0 \le \delta$. It is vital to mention that the requirement is fulfilled by the condition that $\frac{\partial f_i}{\partial x_j}, i, j = 1,2,..$ is continuous and bounded in $\Re$. Considering the model equation (2), we are of the interest in the region $0 \le \alpha \le \Re$. We look for the solution that is bounded in the region and whose partial derivatives satisfy $f \le \alpha \le 0$ where α and δ are positive constants.

Theorem 3.2

Let *J* represent the region α then equation (2) has a unique solution if it is established that

$$\frac{\partial j_i}{\partial x_j}, i, j = 1,2,3,4$$

are continuous and bounded in *R* for J_1

$$\left|\frac{\partial j_1}{\partial S}\right| = \left| -\mu_1 - (\beta I_c + \gamma I)(1-\phi) - \psi \right| < \infty \qquad \left|\frac{\partial j_1}{\partial I_c}\right| = \left| - S\beta(1-\phi)\right| < \infty \quad ,$$

$$\left|\frac{\partial j_1}{\partial I}\right| = \left| - S\gamma(1-\phi)\right| < \infty$$

$$\left|\frac{\partial j_1}{\partial R}\right| = 0 < \infty$$

For J_2

$$\left|\frac{\partial j_2}{\partial S}\right| = \left| \rho(\beta I_c + \gamma I)(1-\phi) \right| < \infty, \left|\frac{\partial j_2}{\partial I_c}\right| = \left| \rho S\beta(1-\phi) - \mu_2 - \alpha(1-\phi) \right| < \infty$$

$$\left|\frac{\partial j_2}{\partial I}\right| = \left| \ \rho S\gamma(1-\phi) \ \right| < \infty, \ \left|\frac{\partial j_2}{\partial R}\right| = 0 < \infty$$

For J_3

$$\left|\frac{\partial j_3}{\partial S}\right| = \left| \ (1-\rho)(1-\phi)(\beta I_c + \gamma I) \ \right| < \infty, \ \left|\frac{\partial j_3}{\partial I_c}\right| = \left| \ (1-\rho)(1-\phi)S\gamma + \alpha(1-\phi) \ \right| < \infty$$

$$\left|\frac{\partial j_3}{\partial I}\right| = \left| \ (1-\rho)(1-\phi)S\gamma - (\mu_3 + \delta) \ \right| < \infty, \ \left|\frac{\partial j_3}{\partial R}\right| = 0 < \infty$$

For J_4

$$\left|\frac{\partial j_4}{\partial S}\right| = \left| \ \psi \ \right| < \infty, \ \left|\frac{\partial j_4}{\partial I_c}\right| = 0 < \infty, \ \left|\frac{\partial j_4}{\partial I}\right| = \left| \ \delta \ \right| < \infty, \ \left|\frac{\partial j_4}{\partial R}\right| = \left| \ -\mu_4 \ \right| < \infty$$

The partial derivatives exist, continuous and bounded, so the model has a unique solution that completes the proof of theorem 3.1.

3.2. Feasible Region and Equilibrium

From equation (2) we have that

$\frac{dS}{dt} \le \theta - (\psi + \mu_1)S$ and thus, $\limsup_{t\to\infty} S(t) \le \frac{\theta}{\psi + \mu_1}$ along with each solution.

Also from (2), we see that $\frac{dN}{dt} \le \theta - c_1 S - c_2 I_c - c_3 I - c_4 R \le \theta - \bar{c}N$

Where $\bar{c} = \min\{c_1.c_2, c_3, c_4\}$. Therefore, $\limsup_{t\to\infty} N(t) \le \frac{\theta}{\bar{c}}$

We have omitted R because R does not appear in other equations. This reveals that the model can be studied in the feasible region; $\Gamma = \left\{(S, I_c, I) \in \Re_+^3 : S \le \frac{\theta}{\psi + \mu_1}, S + I_c + I \le \frac{\theta}{\bar{c}}\right\}$. Γ is positively invariant in relation to (2). Once the dynamics (S, I_c, I) are understood, those of R can then be determined from the equation $R' = \psi S - \delta I - \mu_4 R$.

The first stage of our analysis is to find the disease-free equilibrium states (S, I_c, I) from the equations by setting the right-hand side to zero i.e.,

$$\theta - \mu_1 S - S(\beta I_c + \gamma I)(1-\phi) - \psi S = 0$$

$$\rho S(\beta I_c + \gamma I)(1-\phi) - \mu_2 I_c - \alpha(1-\phi)I_c = 0$$

$$(1-\rho)(1-\phi)S(\beta I_c + \gamma I) + \alpha(1-\phi)I_c - (\mu_3 + \delta)I = 0$$

The model always has a disease-free- equilibrium $A_0 = \left[\frac{\theta}{\psi + \mu_1}, 0, 0\right]$.

And the endemic equilibrium $A^* = (S^*, I_c^*, I^*)$ satisfies $S^*, I_c^*, I^* > 0$. From the equilibrium equations we can show that a unique A^* exist with

$$S^* = \frac{(\mu_3 + \delta)(\mu_2 + \alpha k)}{k(\alpha\gamma k + \beta\delta\rho + \beta\mu_3\rho - \gamma\mu_2\rho + \gamma\mu_2)}$$

Where $k = (1-\phi)$

For A^* to exist in the feasible region Γ, the necessary and sufficient condition requires $0 < S^* \le \frac{\theta}{\psi + \mu_1}$, or equivalently, $\frac{\theta}{(\psi + \mu_1)S^*} \ge 1$. Define

$$R_0 = \frac{1}{S^*}\frac{\theta}{\psi + \mu_1} = \frac{k\theta\left[\alpha\gamma k + \beta\rho(\mu_3 + \delta) + \gamma\mu_2(1-\rho)\right]}{\mu_1 + \psi[(\mu_3 + \delta)(\mu_2 + \alpha k)]} \tag{4}$$

Then a threshold parameter determines the actual number of equilibria. In the next section, we shall explain the basic reproduction number.

Proposal 3.1

If $R_0 < 1$ then J_0 is the only equilibrium in Γ ; if $R_0 > 1$, so, two equilibria exist, A_0, and a unique endemic equilibrium, A^*

3.3. Basic Reproduction Number

The basic reproductive number measures the number of secondary cases that the bacterium is able to introduce into the whole population of fully susceptible individuals in a stable demographic state (Kalajdzievska [9]). The basic number of reproduction is a significant quantity in epidemiology since it sets the threshold for the analysis of an outbreak of disease and the evaluation of its control strategies. The value of the reproductive number, therefore, indicates whether a disease becomes endemic or dies out in a population.

If $R_0 < 1$, it indicates that each contagious person will not cause an infection and therefore, the disease will always disappear but when the basic reproduction number is greater than one i.e., $R_0 > 1$, each contagious person will cause at least one secondary infection which will subject the entire population to the attack of the disease. It is obtained by taking the largest eigenvalue(spectral radius) of the matrix

$$R_0 = \left[\frac{\partial F_i(x_o)}{\partial x_j}\right]\left[\frac{\partial V_i(x_o)}{\partial x_j}\right]^{-1}$$

Where F_i is the rate of new infections in infectious class, V_i is the transfer of persons out of the compartment by another means, x_o is the disease-free equilibrium. Applying the next generation matrix approach, the basic reproduction number for the model is given as

$$R_0 = \frac{k\theta}{\mu_1 + \psi}\left[\frac{\alpha\gamma k}{(\mu_3 + \delta)(\mu_2 + \alpha k)} + \frac{\beta\rho}{(\mu_2 + \alpha k)} + \frac{\gamma\mu_2(1-\rho)}{(\mu_3 + \delta)(\mu_2 + \alpha k)}\right] \tag{5}$$

In equation (5), the expression in the big square brackets is the per capita mean number of secondary infections. This expression is multiplied by $\frac{\theta}{\mu_1 + \psi}$, the number of susceptible individuals in the absence of infection to come about the basic reproduction number R_o

3.4. Disease-Free Equilibrium's Stability

In order to check the local stability in the absence of infection A_0, the Jacobian matrix will be evaluated at $A_0 = \left[\frac{\theta}{\psi + \mu_1}, 0, 0\right]$

$$J(A_0)=\begin{bmatrix} -(\mu_1+\psi) & -\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right) & -\gamma(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right) \\ 0 & \rho\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right)-(\mu_2+\alpha(1-\phi)) & \rho\gamma(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right) \\ 0 & (1-\rho)(1-\phi)\beta\left(\frac{\theta}{\mu_1+\psi}\right)+\alpha(1-\phi) & (1-\rho)(1-\phi)\gamma\left(\frac{\theta}{\mu_1+\psi}\right)-(\mu_3+\delta) \end{bmatrix}$$

The following stability results indicate that R_0 is a sharp threshold.

Proposal 3.2

A_0 locally asymptotically stable if $R_0<1$ and is unstable if $R_0>1$.

Proof

One eigenvalue of $J(A_0)$ is $\lambda_1=-(\mu_1+\psi)<0$. The other two eigenvalues are 2×2 matrix.

$$J(D_0)=\begin{bmatrix} \rho\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right)-(\mu_2+\alpha(1-\phi)) & \rho\gamma(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right) \\ (1-\rho)(1-\phi)\beta\left(\frac{\theta}{\mu_1+\psi}\right)+\alpha(1-\phi) & (1-\rho)(1-\phi)\gamma\left(\frac{\theta}{\mu_1+\psi}\right)-(\mu_3+\delta) \end{bmatrix}$$

We want to show that whenever $R_0<1$ then the Routh-Hurwitz conditions hold, that is, $tra(A)<0$ and $\det(A)>0$.

$$tra(A)=(\mu_2+\alpha(1-\phi))\left[\frac{\rho\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right)}{\mu_2+\alpha(1-\phi)}-1\right]+(\mu_3+\delta)\left[\frac{(1-\rho(1-\phi)\gamma\left(\frac{\theta}{\mu_1+\psi}\right)}{(\mu_3+\delta)}-1\right]$$

Going by the assumption that

$$R_0=\frac{k\theta}{\mu_1+\psi}\left[\frac{\alpha\gamma k}{(\mu_3+\delta)(\mu_2+\alpha k)}+\frac{\beta\rho}{(\mu_2+\alpha k)}+\frac{\gamma\mu_2(1-\rho)}{(\mu_3+\delta)(\mu_2+\alpha k)}\right]<1$$

$$\frac{\rho\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right)}{\mu_2+\alpha(1-\phi)}<1 \text{ and } \frac{(1-\rho(1-\phi)\gamma\left(\frac{\theta}{\mu_1+\psi}\right)}{(\mu_3+\delta)}<1$$

Therefore,

$$\frac{\rho\beta(1-\phi)\left(\frac{\theta}{\mu_1+\psi}\right)}{\mu_2+\alpha(1-\phi)}<0 \text{ and } \frac{(1-\rho(1-\phi)\gamma\left(\frac{\theta}{\mu_1+\psi}\right)}{(\mu_3+\delta)}-1<0$$

This shows that $tra(A)<0$ and the $\det(A)>0$ whenever $R_0<1$. This establishes the proposition.

3.5. Global Stability of Disease-Free Equilibrium Point (DFE)

Theorem 3.3

A_0 is globally stable in the region Γ if $R_0 \le 1$.

Proof. To establish the global asymptotic stability of (DFE) we adopt the method of Lyapunov functions.

Define

$$L = XI_c + YI$$

Where $X = \dfrac{\rho\beta}{(\mu_2 + \alpha k)} + \dfrac{\gamma\mu_2(1-\rho)}{(\mu_3+\delta)(\mu_2+\alpha k)}$, $Y = \dfrac{\alpha\gamma k}{(\mu_3+\delta)(\mu_2+\alpha k)}$

$$\frac{dL}{dt} = X\frac{dI_c}{dt} + Y\frac{dI}{dt}$$

$$X\left[\rho S(\beta I_c + \gamma I)(1-\varphi) - \mu_2 I_c - \alpha(1-\varphi)I_c\right] + Y\left[(1-\rho)(1-\varphi)S(\beta I_c + \gamma I) + \alpha(1-\varphi)I_c - (\mu_3+\delta)I\right]$$

Substituting the values of both *X* and *Y* as in above and simplifying, we shall obtain

$$\frac{dL}{dt} = (R_0 S - 1)(\beta I_c + \gamma I)$$

Using the condition $0 < S^* < \dfrac{\theta}{\mu_1 + \psi}$ we have

$$\frac{dL}{dt} \le (R_0 S - 1)(\beta I_c + \gamma I) \le 0$$

So, $\dfrac{dL}{dt} \le 0$ if $R_0 \le 1$

Furthermore, $\dfrac{dL}{dt} = 0$ if $I_0 = I = 0$

Therefore, the largest invariant set in Γ where $\dfrac{dL}{dt} = 0$ is the singleton $\{A_0\}$. Hence, by LaSalle's Principle [23], J_0 is globally asymptotically stable in the region Γ.

Table 2. Values of parameters for the model

Parameter	Value	Source
μ_2	0.20	Assumed
ψ	0.30	Assumed
μ_1	0.142(	Mushayabasa [10]
μ_3	0.2	Assumed
μ_4	0.142	Mushayabasa [10]
α	0.3	Assumed
ρ	0.5	Assumed
β	0.02	Assumed
γ	0.01	Mushayabasa [10]
δ	0.75	Assumed

ϕ	0.3	Estimated
θ	10^6	Lauria, *et al*. [8]

4. RESULTS AND DISCUSSION

We performed numerical simulations to describe graphically the long-term impact of early treatment on the dynamics of typhoid fever. Table 2 indicated the various set of parameter values adopted to validate the outcome of the analysis. Graphical illustrations showed time graphs of each state variable. Figure 2 described the time-sensitive graph. with high vaccination achievement, both the effective contact rate and the prevalence rate were low, thereby limiting the pool of susceptible populations over time. On the other hand, the population of susceptible individuals increased drastically when the rate of vaccination was low.

Figure 3 showed the diagram of infective carriers against time. The number of Infectious carriers $I_c(t)$ was rising because they are not aware of their conditions. However, as the symptoms of the disease began to manifest, the population began to fall as a result of early treatment. Figure 4 depicted the chart of infective against time. The population of the Infected $I(t)$ grows continuously which might be attributed to late treatment. In Figure 5, the graph for the Recovered $R(t)$ individuals continued to grow as a result of treatment and regular medical check-ups.

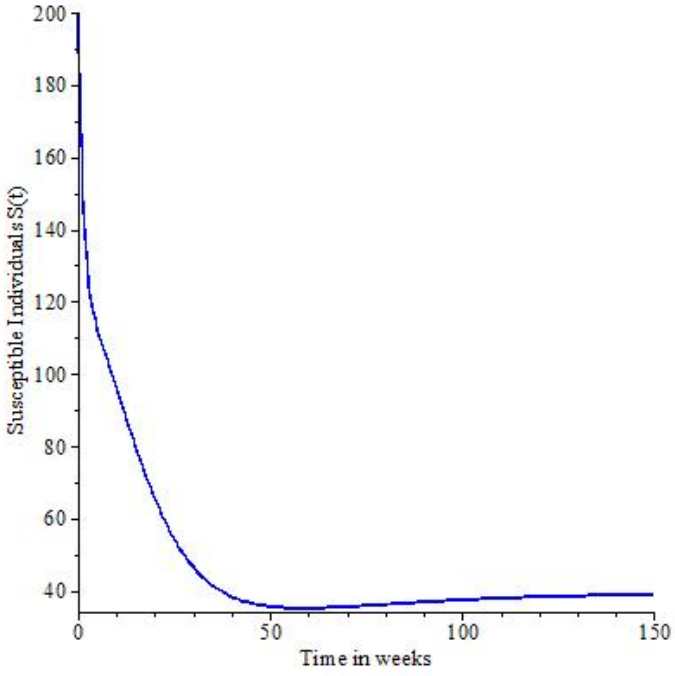

Figure 2. Susceptible Population

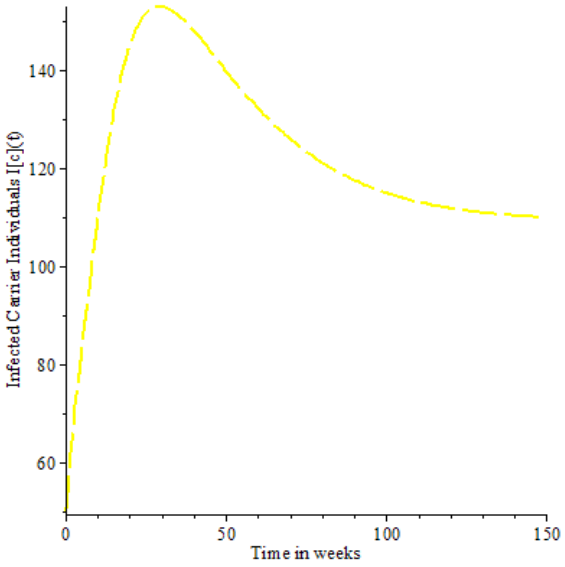

Figure 3. Infected Carrier Population

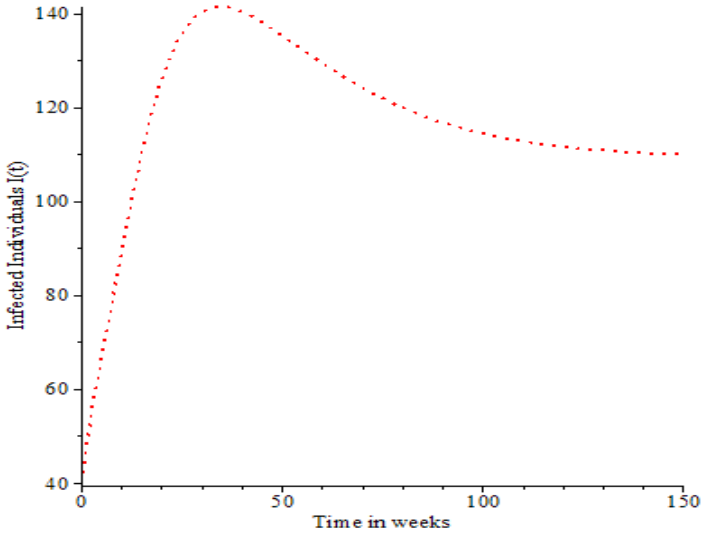

Figure 4. Infected Population

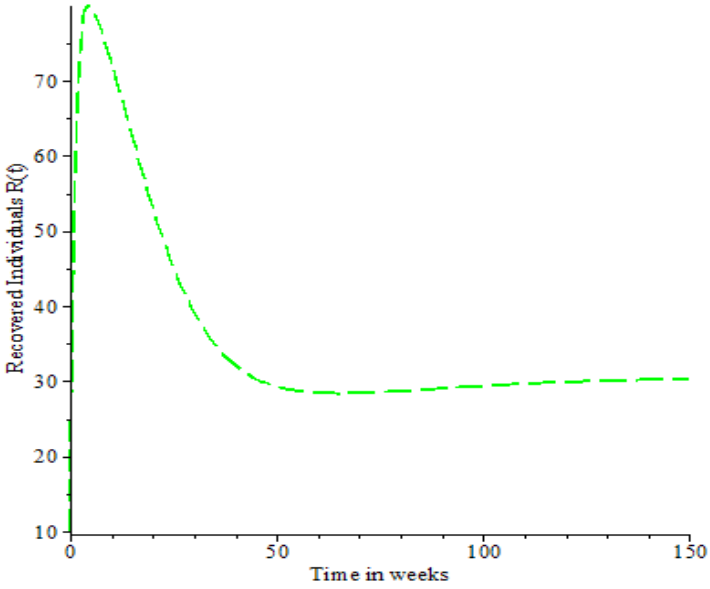

Figure 5. Recovered Population

5. CONCLUSION

We analyzed a deterministic model for the management of typhoid. The conditions for the existence and stability of the equilibria described by the basic reproduction number of the model were successfully derived. The analysis showed that there is a disease-free equilibrium that is locally and globally asymptotically stable as long as $R_0 < 1$ an unstable if $R_0 > 1$. The model revealed that the transmission of typhoid fever depended largely on the contact rate with the infected individuals in a population. Thus, timely detection and early treatment could reduce the infection rate.

REFERENCES

1. J. Brooks (1996). The sad and tragic life of Typhoid Mary, *The Canadian Medic Assoc J.*, 154(6), 915-916.
2. P. Roumagnac, F.X Weill, C. Dolecek, S. Baker, S. Brises, N.T. Chinh et. al. (2006). Evolutionary history of Salmonella Typhi, *Science*, 314, 1301-1304, DOI: 10.1126/science.1134933.
3. F.Y. Lin, A.H. Vo, V.B. Phan, T.T. Nguyen, D. Bryla & C.T. Tran (2000). The epidemiology of typhoid fever in the Dong Thap Province, Mekong Delta region of Vietnam, *Amer. J. Trop. Med. Hygiene*, 62, 644-648.
4. A. Sinha, S. Sazawal, R. Kumar, S. Sood, V.P. Reddaiah, and B. Singh (1999). Typhoid fever in children aged less than 5 years, *Lancet*, 354, 734-737.
5. J.M. Hyman (2006). Differential Susceptibility and Infectivity Epidemic Models, *Math. Biosci. Eng.*, 3, 88-100.
6. E.L Lifshitz (1996). Travel trouble: typhoid fever-A case presentation and review, *J. Amer. College Health*, 45(3), 99-105.
7. I.A. Adetunde (2008). Mathematical models for the dynamics of typhoid fever in Kassenanankana district of the upper east region of Ghana, *J. Modern Math Stat*, 2, 45-49.
8. D.T. Lauria, B. Maskery, C. Poulos, and D. Whittington (2009). An optimization model for reducing typhoid cases in developing countries without increasing public spending, *Vaccine*, 27(10), 1609-1621.
9. D. Kalajdzievska (2011). Modeling the effects of carriers on the transmission dynamics of infectious diseases, *Math. Biosci. & Eng.*, 8(3), 711-722.
10. S. Mushayabasa (2011). Impact of vaccines on controlling typhoid, *J. Modern Math Stat*, 5(2), 54-59.
11. B. Cvjetanovic, B. Grab, K. Uemura (2014). Epidemiological model of typhoid fever and its use in the planning and evaluation of antityphoid immunization and sanitation programs, *Bulletin of the WHO*, 45(1), 53-75.
12. N. Moffact (2014). Mathematical Model and Simulation of the Effects of Carriers on the Transmission Dynamics of Typhoid Fever, *Trans. on Comp. Sci. Eng. and its Applic*, 2(3), 14-20.
13. V.E. Pitzer, C.B. Cayley, B. Stephen, K. Gagandeep, B. Veeraraghavan et. al. (2014). Predicting the impact of vaccination on the transmission dynamics of Typhoid in South Asia: A Mathematical modeling study, *PlosNegleted Trop. Dis.*, 8(1), e2642, DOI:10.1371/journal.pntd.0002642.
14. K.A. Date, A. Bentsi-Enchill, F. Marks & K. Fox (2015). Typhoid fever vaccination strategies, *Vaccine*, 33, 55-61.
15. A.K. Muhammad, S.I. Muhammad, K. Ilyas, S. Sharidan & G. Taza (2015). Mathematical analysis of Typhoid model with saturated incidence rate, *Adv. Stud. in Biology*, 7(2), 65-78.

16. C.H. Watson & W.J. Edmunds (2015). Review of typhoid fever transmission dynamic models and economic evaluations of vaccination, *Vaccine*, 33(3), 42-54.
17. J.K. Nthiiri (2016). Mathematical modeling of typhoid fever disease incorporating protection against infection, *British J. Math Comp. Sci*, 14(1), 1-10.
18. K. Moatlhodl & R. Gosaamang (2017). Mathematical analysis of Typhoid infection with treatment, *J. Math Sci: Adv. Applic.*, 40(1), 75-91.
19. S. Mushayabasa (2017). A simple epidemiological model for typhoid with saturated incidence rate and treatment effect, *Int. J. Sci., Basic Appl. Res.* (IJSBAR), 32(1), 151-168.
20. O.J. Peter. & M.O. Ibrahim (2017). Application of differential transform method in solving a Typhoid fever model, *Int. J. of Math. Anal. and Optim.: Theo. and Appli.*, 1(1), 250-260.
21. O.J. Peter, M.O. Ibrahim, O. B. Akinduko & M. Rabiu (2017). Mathematical model for the control of typhoid fever. *IOSR J. of Math. (IOSR-JM)* 13(4), 60-66.
22. P. O. James and M. O. Ibrahim (2019). Application of Variational Iteration Method in Solving Typhoid Fever Model, *Big Data, Knowledge and Control Systems Engineering (BdKCSE)*, Sofia, Bulgaria, 2019, 1-5, doi: 10.1109/BdKCSE48644.2019.9010598.
23. J.P. LaSalle (1976). The Stability of Dynamical Systems, *Regional Conference Series in Appl. Math., SIAM*, Philadelphia.

