Mathematical Model For the Control of Typhoid Fever

[*]Peter, O.J[1], Ibrahim, M.O.[2], Akinduko, O.B[3] , Rabiu, M. [4]

Department of Mathematics, University of Ilorin, Ilorin, Kwara State, Nigeria, [1,2]
Department of Mathematical Sciences Adekunle Ajasin University Akungba,Ondo State Nigeria[3]
Department of Mathematics, Abdulraheem College of Advanced Studies,Igbaja.
(Affiliate of Alhikmah University, Ilorin, Kwara State) [4]
Corresponding Author: peterjames4real@gmail.com

Abstract: *In this paper, we studied the dynamics of typhoid fever model; we tested for the existence and uniqueness of solution for the model using the Lipchitz condition to ascertain the efficacy of the model and proceeded to determine both the disease free equilibrium (DFE) and the endemic equilibrium (EE) for the system of the equations. The local stability of the disease free equilibrium was obtained. The next generation matrix approach is used to determine the basic reproductive number R_o. We proved that the disease free equilibrium is globally asymptotically stable when $R_o < 1$ and the disease will always die out.*

Keywords: *basic reproduction number, equilibrium states, existence and uniqueness of solution, mathematical model, typhoid fever;*

I. Introduction

Typhoid fever is one of the most deadly disease in Africa especially where there is a poor sanitation, poor standard of personal hygiene and prevalence of contaminated water. Typhoid fever is endemic in many developing countries and remain a substancial public health concern despite recent progress in water sanitation coverage [1]. Many advances have been made towards the fight against typhoid fever such as treatment with drugs, vaccination and environmental sanitation. In recent years, data indicating that typhoid fever is a major cause of mortality among the urban and peri-urban population. In several community-base studies from South Asia, the incident rate seems to be high among young children, with rates exeeding 500-1000 cases per 100,000 populations [2]. Various studies including mathematical model of spread of typhoid fever, dynamic model for analyzing and predicting process of typhoid fever among others have been conducted by many researchers Globally. It is estimated that typhoid fever causes over 16 million cases of illness each year, resulting in over 600,000 deaths [3]. Several mathematical models have been developed on this disease [4, 5, 6, 7, 8, 9, 10, 11,12]. For example, in reference [9], the author proposed a mathematical model of the type P S, I, T. They divided the total human population into four subclasses, i.e., Susceptible, Protected, Infected and Treated. The existence of the steady states of the model were determined and the basic reproduction number was computed using the next generation matrix approach. Stability analysis of the model was carried out to determine the conditions that favors the spread of the disease. Complementing the work of [9], we constructed a mathematical model of the type PSITR. We added a recovered compartment in which all treated individuals recovered but after some time the recovered individuals lose immunity and return back to susceptible class.

II. Description and Formulation Of the Model

The compartments used in this model consists of five classes: $P(t)$ is the compartment used for those that have been vaccinated against the disease and loses protection over a period of time. S(t) is used to represent the number of individuals that are prone to the disease at time t or those susceptible to the disease. $I(t)$ 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. $T(t)$ denote the number of individuals who have been infected with the disease and are treated. $R(t)$ is the compartment used for those individuals who have been infected and then recovered from the disease. Those in this category are not able to be infected again or transfer to others. Susceptible individuals are recruited into the population at per capital rate $(1 - \sigma)\Lambda$. Susceptible individual aquired typhoid fever at a constant rate α. The disease is transmitted by direct transmssion or by ingestion of contaminated food or liquid by susceptible individuals. Hence we propose the above model with the following equations: The model *PSITR*.

$$\frac{dP}{dt} = \sigma\Lambda - (\gamma + \mu)P \tag{1}$$

$$\frac{dS}{dt} = (1-\sigma)\Lambda + \gamma P - \alpha SI - \mu S + kR \tag{2}$$

$$\frac{dI}{dt} = \alpha SI - (\delta + \beta + \mu)I \tag{3}$$

$$\frac{dT}{dt} = \beta I - (\mu + \varepsilon)T \tag{4}$$

$$\frac{dR}{dt} = \varepsilon T - \mu R - kR \tag{5}$$

Table 1: Description of parameters for the Model

Parameter	Description
Λ	Recruitment rate
σ	Adjustment parameter
μ	Natural death rate
δ	Disease induced death rate
γ	Loss of protection
β	Rate of treatment
α	Contact rate of infection
k	Relapse rate
ε	Progression rate from T to R

2.1 Existence and Uniqueness of Solution

The validity and authenticity of any mathematical model depends on whether the given system of equations has a solution, and if the solution is unique. We shall use the Lipchitz condition to verify the existence and uniqueness of solution for the system of equations (1)-(5)

Let the system of equation (1)-(5) be as follows

$$F_1 = \sigma\Lambda - (\gamma + \mu)P \tag{6}$$

$$F_2 = (1-\sigma)\Lambda + \gamma P - \alpha SI - \mu S + kR \tag{7}$$

$$F_3 = \alpha SI - (\delta + \beta + \mu)I \tag{8}$$

$$F_4 = \beta I - (\mu + \varepsilon)T \tag{9}$$

$$F_5 = \varepsilon T - \mu R - kR \tag{10}$$

Theorem 2.1 (Derrick and Groosman,1976)

Let D denote the region

$$|t - t_0| \le a, \|x - x_0\| \le 1, x = (x_1, x_2........, x_n), x_o = (x_{10}, x_{20},........x_{no})$$

And suppose that $f(t,x)$ satisfies the Lipchitz condition

$$\|f(t,x_1) - f(t,x_2)\| \le k\|x_1 - x_2\|$$

whenever the pairs (t,x_1) and (t,x_2) belong to D where k is a positive constant. Then, there is a constant $\delta \ge 0$ such that there exists a unique continuous vector solution of $x(t)$ of the system in the interval $t - t_o \le \delta$. It is important to note that the condition is satisfied by the requirement that $\dfrac{\partial f_i}{\partial x_j}$, $i, j = 1,2,\cdots,$ be continuous and bounded in D. Considering the model equation (1) to (5), we are interested in the region $0 \le \alpha \le R$. We look for the bounded solution in the region and whose partial derivatives satisfy $f \le \alpha \le 0$. where α and δ are positive constants

Theorem 2.2 Let D denote the region $0 \le \alpha \le R$, then equation (6) to (10) have a unique solution. We show that

$$\frac{\partial f_i}{\partial x_j}, \; i, j = 1,2,3,4,5$$

are continuous and bounded in D. For F_1

$$\left|\frac{\partial f_1}{\partial P}\right| = \left|\; -(\gamma + \mu)\;\right| < \infty$$

For F_2

$$\left|\frac{\partial f_2}{\partial P}\right| = |\gamma| < \infty, \; \left|\frac{\partial f_2}{\partial S}\right| = |-(\alpha I + \mu)| < \infty, \; \left|\frac{\partial f_2}{\partial R}\right| = |k| < \infty, \; \left|\frac{\partial f_2}{\partial I}\right| = |-\alpha S| < \infty, \; \left|\frac{\partial f_2}{\partial T}\right| = 0 < \infty,$$

These partial derivative exist, continuous and are bounded, similarly for F_3 through to F_5. Hence, by theorem 1, the model (6) to (10) has a unique solution

2.2 Equilibrum States of the Model
The disease free equilibrium of model system (1) to (5) is obtained by setting

$$\frac{dP}{dt} = \frac{dS}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0$$

and in the absence of disease

$$I = 0, R = 0, T = 0$$

Setting the RHS of (1-5) to zero we have

$$\sigma\Lambda - (\gamma + \mu)P = 0 \tag{11}$$

$$(1-\sigma)\Lambda + \gamma P - \alpha SI - \mu S + kR = 0 \tag{12}$$

$$\alpha SI - (\delta + \beta + \mu)I = 0 \tag{13}$$

$$\beta I - (\mu + \varepsilon)T = 0 \tag{14}$$

$$\varepsilon T - \mu R - kR = 0 \tag{15}$$

Hence model (1)-(5) has a disease free equilibrium

$$(P, S, I, T, R) = \left[\frac{\sigma\Lambda}{\gamma + \mu}, \frac{\Lambda(-\mu\sigma + \gamma + \mu)}{\mu(\gamma + \mu)}, 0, 0, 0\right] \tag{16}$$

For the endemic equilibrium, it means disease exist, solving the system of the equation (1) - (5). Hence the endemic equilibrium states are

$$P^* = \frac{\sigma\Lambda}{\gamma + \mu} \tag{17}$$

$$S^* = \frac{\delta + \beta + \mu}{\alpha} \tag{18}$$

$$I^* = \frac{(\mu + \varepsilon)(\mu + k)\left(-\Lambda\alpha\mu\sigma + \Lambda\alpha\gamma + \Lambda\alpha\mu - \beta\gamma\mu - \beta\mu^2 - \delta\gamma\mu - \delta\mu^2 - \gamma\mu^2 - \mu^3\right)}{\alpha[\beta\mu\Gamma_1 + (\delta\varepsilon + \delta\mu + \varepsilon\mu)\Gamma_2 + \Gamma_3]} \tag{19}$$

$$T^* = \frac{(\mu + k)\beta\left(-\Lambda\alpha\mu\sigma + \Lambda\alpha\gamma + \Lambda\alpha\mu - \beta\gamma\mu - \beta\mu^2 - \delta\gamma\mu - \delta\mu^2 - \gamma\mu^2 - \mu^3\right)}{\alpha[\beta\mu\Gamma_1 + (\delta\varepsilon + \delta\mu + \varepsilon\mu)\Gamma_2 + \Gamma_3]} \tag{20}$$

$$R^* = \frac{\beta\varepsilon\left(-\Lambda\alpha\mu\sigma + \Lambda\alpha\gamma + \Lambda\alpha\mu - \beta\gamma\mu - \beta\mu^2 - \delta\gamma\mu - \delta\mu^2 - \gamma\mu^2 - \mu^3\right)}{\alpha[\beta\mu\Gamma_1 + (\delta\varepsilon + \delta\mu + \varepsilon\mu)\Gamma_2 + \Gamma_3]} \tag{21}$$

Where

$$\Gamma_1 = \gamma\varepsilon + \varepsilon\mu + \gamma k + \gamma\mu + k\mu + \mu^2$$

$$\Gamma_2 = \gamma k + \gamma\mu + k\mu + \mu^2$$

$$\Gamma_3 = \gamma k\mu^2 + \gamma\mu^3 + k\mu^3 + \mu^4$$

III. Estimation Of Basic Reproduction Number

The basic reproduction number denoted by R_o is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness (Diekmann et. al, 1990). The basic reproduction number is an important non-dimensional quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R_o . Furthermore, stability of equilibrium can be analyzed using R_o . If $R_o < 1$ it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_o > 1$ every infectious individual will cause more than one secondary infection and hence the disease will invade the population. It is Obtained by taking the largest (dominant) eigenvalue (spectral radius)

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

Where F_i be the rate of appearance of new criminal in compartments, V_i is the transfer of individuals out of the compartment by another means, x_o is the disease free equilibrium. We compute the basic reproduction number using the next generation matrix approch. The basic reproduction number for the model in system (1)-(5) is given as

$$R_0 = \frac{\alpha\Lambda(-\mu\sigma + \gamma + \mu)}{\mu(\gamma + \mu)(\delta + \mu + \beta)} \tag{22}$$

IV. Local Stability Of the Disease-Free Equilibrium

Theorem 3

The disease free equilibrium is locally asymptotically stable if

$$R_0 \leq 1$$

Proof

The variational (Jacobian matrix) to the system (1)-(5)

$$J = \begin{bmatrix} -(\gamma + \mu) & 0 & 0 & 0 & 0 \\ \gamma & -(\alpha I + \mu) & -\alpha S & 0 & k \\ 0 & \alpha I & \alpha S - (\delta + \beta + \mu) & 0 & 0 \\ 0 & 0 & \beta & -(\mu + \varepsilon) & 0 \\ 0 & 0 & 0 & \varepsilon & -(\mu + k) \end{bmatrix}$$

Where

$$S = \frac{\Lambda(-\mu\sigma + \gamma + \mu)}{\mu(\gamma + \mu)}, I = 0$$

At disease free

$$J = \begin{bmatrix} -(\gamma+\mu) & 0 & 0 & 0 & 0 \\ \gamma & -\mu & \dfrac{-\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)} & 0 & k \\ 0 & 0 & \dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu) & 0 & 0 \\ 0 & 0 & \beta & -(\mu+\varepsilon) & 0 \\ 0 & 0 & 0 & \varepsilon & -(\mu+k) \end{bmatrix}$$

The characteristics equation of the matrix above is obtained by

$$\det|J-\lambda I|=0$$

$$J = \begin{bmatrix} -(\gamma+\mu+\lambda) & 0 & 0 & 0 & 0 \\ \gamma & -\mu-\lambda & \dfrac{-\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)} & 0 & k \\ 0 & 0 & \dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)-\lambda & 0 & 0 \\ 0 & 0 & \beta & -(\mu+\varepsilon+\lambda) & 0 \\ 0 & 0 & 0 & \varepsilon & -(\mu+k+\lambda) \end{bmatrix}$$

$$-(\gamma+\mu+\lambda)\begin{vmatrix} -\mu-\lambda & \dfrac{-\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)} & 0 & k \\ 0 & \dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)-\lambda & 0 & 0 \\ 0 & \beta & -(\mu+\varepsilon+\lambda) & 0 \\ 0 & 0 & \varepsilon & -(\mu+k+\lambda) \end{vmatrix}$$

$$-(\gamma+\mu+\lambda)(-\mu-\lambda)\begin{vmatrix} \dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)-\lambda & 0 & 0 \\ \beta & -(\mu+\varepsilon+\lambda) & 0 \\ 0 & \varepsilon & -(\mu+k+\lambda) \end{vmatrix}$$

$$-(\gamma+\mu+\lambda)\left[(-\mu-\lambda)\left\{(\dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)-\lambda)\begin{vmatrix} -(\mu+\varepsilon+\lambda) & 0 \\ \varepsilon & -(\mu+k+\lambda) \end{vmatrix}\right\}\right]=0$$

$$-(\gamma+\mu+\lambda)\left[(-\mu-\lambda)\left\{(\dfrac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)-\lambda)(\mu+\varepsilon+\lambda)(\mu+k+\lambda)\right\}\right]=0$$

Therefore

$$\lambda_1=-(\gamma+\mu)<0 \;,\; \lambda_2=-\mu<0 \;,\; \lambda_3=-(\mu+\varepsilon)<0 \;,\; \lambda_4=-(\mu+k)<0$$

$$\left[\frac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}-(\delta+\beta+\mu)\right]<0 \quad\Rightarrow\quad \frac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)}<(\delta+\beta+\mu) \tag{23}$$

Dividing both side of (23) by $(\delta+\beta+\mu)$

$$\Rightarrow \frac{\alpha\Lambda(-\mu\sigma+\gamma+\mu)}{\mu(\gamma+\mu)(\delta+\beta+\mu)}<1 \tag{24}$$

Substituting (22) into (23)

$$\therefore \quad R_o<1$$

Hence the proof.

V. Discussion and Conclusion

In this paper, we discussed a mathematical model for the control of typhoid fever. We proved the existence and uniqueness of solution in order to ascertain the existence of the model. We can control the disease burden by controlling the effective contact rate of the infected population. The model strongly indicated that the spread of a disease largely depend on the contact rates with infected individuals within a population. The next generation approach is used to determine the basic reproductive number R_o. We proved that the disease free equilibrium is globally asymptotically stable when $R_o<1$ and the disease will always die out.

References

[1].   Lauria, D. T., Maskery ,B., Poulos, C., and Whittington, D. An optimization model for reducing typhoid cases in developing countries without increasing public spending, Vaccine, JVAC-8805 (2009). http://dx.doi.org/10.1016/j.vaccine.2008.12.032
[2].   World Health Organisation(WHO/V and B/03.07),2003. Background document: The diagnosis, treatment and prevention of typhoid fever.www.who.int/vaccines-document/
[3].   Adetunde, I. A,. Mathematical models for the dynamics of typhoid fever in kassena-nankana district of upper east region of Ghana. J. Modern Math Stat., 2: 45-49, (2008).
[4].   Joshua, E. and Etukudo . Mathematical model of the spread of Typhoid fever, World Journal of Applied Science and Technology 3 (2) 10-12 (2011).
[5].   Kalajdzievska, D. and LI, M. Y, "Modeling the effects of carriers on transmission dynamics of infectious disease", Math. Biosci. Eng., 8(3):711-722, (2011), http://dx.doi.org/10.39 34/mbe.2011.8.711
[6].   Khan, M.A., Parvez, M., Islam, S., Khan, I., Shafie, S., and Gul, T, "Mathematical Analysis of Typhoid Model with Saturated Incidence Rate", Advanced Studies in Biology, 7(2):65 – 78, (2015)
[7].   Mushayabasa, S,"A simple epidemiological model for typhoid with saturated incidence rate and treatment effect", World Academy of Science, Engineering and Technology, International Journal of Sciences: Basic and Applied Research (IJSBAR) 32(1) 151-168.(2017).
[8].   Mushayabasa, S. (2014) Modeling the impact of optimal screening on typhoid dynamics, Int. J. Dynam. Control, Springer. DOI 10.1007/40435-014-0123-4
[9].   Nthiiri, J. K. Lawi,G. O. Akinyi,C. O. Oganga,D. O. Muriuki,W. C. Musyoka,M. J. Otieno,P. O. andKoech,L. Mathematical modelling of typhoid fever disease incorporating protection against the disease. British Journal of Mathematics & Computer Science14(1): 1-10, (2016), Article no.BJMCS.23325
[10].   Pitzer, V. E., Cayley, C., Bowles, C. C., Baker, S., Kang,G., Balaji, V., Jeremy J. Farrar, J.J,Bryan, T., Grenfell, B.T, "Predicting the Impact of Vaccination on the Transmission Dynamics of Typhoid in South Asia: A Mathematical Modeling Study", PLoS Neglected Tropical Diseases, 8(1): e2642, (2014).
[11].   Derrick, N.R and Grossman, S. L (1976) Differential Equation with application. Addison Wesley Publishing Company, Inc. Philippines.

[12]. Peter, O. J, Ibrahim, M. O. Application of Differential Transform Method in Solving a Typhoid Fever Model. International Journal of Mathematical analysis and Optimization.1(1), 250-260. (2017), Retrieved from http://ijmao.unilag.edu.ng/article/view/12

[13]. Diekmann, O., Heesterbeek, J. A., Metz, J. A. On the definition and computation of basic reproduction ratio R_o in models for infectious disease in heterogeneous populations. J. Math. Biology. 28(4) 365-382 (1990).