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On the Dynamical Analysis of a Deterministic Typhoid Fever Infection Model

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Abstract

In this paper, we develop a deterministic model of typhoid fever. The existence and uniqueness of solutions of the model were examined by actual solutions. Mathematical analysis is carried out to determine the transmission dynamics of typhoid in a community. We conduct local stability analysis for the model. The results show that the disease-free equilibrium which is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Keyword: Typhoid, treatment, transmission, reproduction number, disease-free

1. INTRODUTION

Typhoid is a major public health concern in tropical developing countries, especially in areas where access to clean water and other sanitation measures are limited [1-3]. Typhoid fever has complex pathogenesis and manifests as an acute febrile disease, with relatively long incubation period that involves the transmigration of the microorganism through the Peyer's patch, localized multiplication in the mesenteric lymph nodes, and subsequent spread to the liver and spleen prior to showing clinical symptoms [4]. It is a serious life-threatening infection characterised by false diagnosis due to similar signs and symptoms with malaria, which leads to improper controls and management of the disease. Despite extensive work on typhoid, not much is understood on the biology of the human-adapted bacterial pathogen and the complexity of the disease in endemic areas, especially in Africa [5]. Globally, the burden of the disease is estimated at 21 million cases and 222000 deaths annually with high rates reported among children and adolescents in South and Eastern Asia and uncertain in Africa [6-8]. The symptoms are alleviated with antibiotic medications, however, a proportion of people treated for typhoid fever usually experience relapse, after a week of antibiotic treatment with symptoms which are milder and Last for a shorter time compared with the original illness, requiring further treatment with antibiotics [9, 10]. Typhoid fever maybe prevented using vaccines, even though repeated mass vaccinations at intervals of 5 years interval may reduce the disease incidence, small gains re-observed at each subsequent vaccination [11]. The dynamics of typhoid fever involve multiple interactions between the human host, pathogen and environment, contributing to both direct human-to-human and indirect environment-to-human transmission pathways [12, 13]. Typhoid fever produces long-term asymptomatic carriers which play a pivotal role in the disease transmission.

In order to gain in-depth understanding of the complex dynamics of typhoid fever a number of studies have been conducted and published. Cvjetanovic et al. [11] constructed an epidemic model for typhoid fever in a stable population to study the transmission of infection at different levels of endemicity. Mushayabasa et al. [12] developed and analysed a deterministic mathematical model for assessment of the impact of treatment and educational campaigns on controlling typhoid out-break in Zimbabwe. Date et al. [6] reviewed various vaccination strategies using current typhoid vaccines to assess the rationale, acceptability, effectiveness, impact and implementation lessons in order to inform future public health typhoid control strategies. Watson and Edmunds [14] carried out an intensive review of typhoid fever transmission dynamics models and economic evaluation of vaccination. Clinicians, microbiologists, modellers, and epidemiologists worldwide need full understanding and knowledge of typhoid fever to effectively control and manage the disease [5].

This present study investigates the criteria under which the effectiveness of treatment could lead to the stability of the equilibrium point. We establish the conditions for existence and uniqueness of the solution of models, conducted local stability analysis of the models.

2.0 Model Formulation

Following [15], the equations describing typhoid fever epidemics are:

$$\frac{dS}{dt} = \Lambda - \frac{c\beta(I + k_1 I_c + k_2 T)}{N} S - \mu S \quad (1)$$

$$\frac{dI}{dt} = \frac{c\beta\rho(I + k_1 I_c + k_2 T)}{N} S + \alpha I_c - (\mu + \sigma + \delta_1) I \quad (2)$$

$$\frac{dI_c}{dt} = \frac{(1-\rho)c\beta(I + k_1 I_c + k_2 T)}{N} S + \tau T - (\mu + \alpha) I_c \quad (3)$$

$$\frac{dT}{dt} = \sigma I - (\mu + \gamma + \tau + \delta_2) T \quad (4)$$

$$\frac{dR}{dt} = \gamma T - \mu R \quad (5)$$

$$N(t) = S(t) + I(t) + I_c + T(t) + R(t) \quad (6)$$

As initial condition based on our assumptions, we choose

$$S(0) = S_0,\ I(0) = I_0,\ I_c(0) = I_{c(0)},\ T(0) = T_0,\ R(0) = R_0 \quad (7)$$

Where

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Variables	Parameters
$S(t)$ - Susceptible human	Λ - Recruitment rate
$I(t)$ -Infectives human	μ - per capital death rate
$I_c(t)$ - Carriers human	δ_1, δ_2 - Disease-induced deaths
$T(t)$ - Treated infectives	c - effective contacts
$R(t)$ - Recovered human	β - Rate of transmission
	α - Progression to symptomatic state
	γ - Rate of recovery from treatment
	ρ - New infections becoming carriers
	σ - Rate of treatment
	τ - Proportion of treated individuals
	k_1, k_2 - Modification parameters

3.0 Method of Solution

3.1 Positivity of Solutions

It is necessary to prove that all solutions of system (1) – (5) with positive initial data will remain positive for all times (t). This will be established by the following theorem.

Lemma 1: Let the closed set

$$\begin{bmatrix} S \\ I \\ I_c \\ T \\ R \end{bmatrix} \in \Re^5 \begin{bmatrix} S(0) \geq 0 \\ I(0) \geq 0 \\ I_c(0) \geq 0 \\ T(0) \geq 0 \\ R(0) \geq 0 \\ S + I + I_c + T + R \leq \frac{\Lambda}{\mu} \end{bmatrix}$$

Then, the solution of $(S(t), I(t), I(t), I_c(t), R(t))$ of the equations (1) to (5) are positive for all $t \geq 0$

Proof

from equation (1) we have that

$$\frac{ds}{dt} = \Lambda - BS - \mu S$$

$$\frac{ds}{dt} \geq -\mu S$$

$$\frac{ds}{S} \geq -\mu dt$$

$$\int_0^t \frac{1}{S} dS \geq -\mu \int_0^t dS$$

$$S(t) \geq e^{-\mu t} \geq 0$$

Similarly,

$$I(t) \geq e^{-(\mu - \sigma - \delta_1)t} \geq 0$$

$$I_c(t) \geq e^{-(\mu + \alpha)t} \geq 0$$

$$T(t) \geq e^{-(\mu + \gamma + \tau + \delta_2)t} \geq 0$$

$$R(t) \geq e^{-\mu t} \geq 0$$

Hence, the solution of $(S(t), I(t), I_c, T(t), R(t))$ of equation (1) to (5) are positive for all $t \geq 0$

3.2 Existence and Uniqueness of Solution

Lemma 2: Let $\delta_1 = \delta_2 = 0$, then the equation (1) to (6) with the initial condition has a unique solution for all $t \geq 0$

Proof: Let $\delta_1 = \delta_2 = 0$, $\Phi(t) = S(t) + I(t) + I_c(t) + T(t) + R(t)$. We obtain

$$\frac{d\Phi}{dt} = \Lambda - \Phi\mu, \ \Phi(0) = S(0) + I(0) + I_c(0) + T(0) + R(0) = \Phi_0 \qquad (8)$$

By direct integration, we obtain the solution of problem (8) as

$$\Phi(t) = \frac{\Lambda}{\mu}(1 - e^{-\mu t}) + \Phi_0 e^{-\mu t} \qquad (9)$$

Then, we obtain

$$S(t) = (\frac{\Lambda}{\mu} + (1 - e^{-\mu t})e^{-\mu t}) - (I(t) + I_c(t) + T(t) + R(t)) \qquad (10)$$

$$I(t) = (\frac{\Lambda}{\mu} + (1 - e^{-\mu t})e^{-\mu t}) - (S(t) + I_c(t) + T(t) + R(t)) \qquad (11)$$

$$I_c(t) = (\frac{\Lambda}{\mu} + (1 - e^{-\mu t})e^{-\mu t}) - (S(t) + I(t) + T(t) + R(t)) \qquad (12)$$

$$T(t) = (\frac{\Lambda}{\mu} + (1 - e^{-\mu t})e^{-\mu t}) - (S(t) + I(t) + I_c(t) + R(t)) \qquad (13)$$

$$R(t) = \frac{\Lambda}{\mu} + (1 - e^{-\mu t})e^{-\mu t}) - (S(t) + I(t) + I_c(t) + T(t)) \qquad (14)$$

Hence, there exists a unique solution of problem (1) – (6). This completes the proof.

3.3 Equilibrium State of the Model

At equilibrium,