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A Deterministic Mathematical Model for Direct and Indirect Transmission Dynamics of Typhoid Fever

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Abstract

Improvements in sanitation and the provision of clean drinking water led to the elimination of typhoid fever from developed countries in the beginning of the 20th century. However, *Salmonella typhi* and *paratyphi* remain a major source of morbidity and mortality in many developing countries today. The dynamics of typhoid transmission are poorly understood. In this study, we develop a novel mathematical model that captures the role of both human to human interaction and human to environment interaction in the transmission dynamics of typhoid fever. Our results have shown the feasible impact of different methods of typhoid control, including vaccination, improved treatment strategies, and investment in clean water and sanitation.

Subject Areas

Mathematical Modeling

Keywords

Modeling, Sanitation, Treatment, Vaccination, Epidemiology, Typhoid

1. Introduction

Typhoid fever is a communicable disease, found only in man and occurs due to systemic infection mainly by *Salmonella typhi* organism [1]. The disease is endemic in many developing countries and despite recent progress in water and sanitation coverage, it remains a substantial public health problem. Globally, it is estimated that typhoid causes over 16 million cases of illness each year, resulting in over 600,000 deaths [2]. Typhoid has a long storied history as a public health scourge. *Salmonella enterica serovar Typhi* (*S. Typhi*) is a human restricted bacterial pathogen transmitted via fecal contamination of food and water [3]. While

improvements in water and sanitation led to the elimination of typhoid from most developed countries during the twentieth century, the global burden of typhoid fever has recently been estimated to be between 13.5 and 26.9 million episodes and 190,000 to 216,000 deaths annually [4]. In many developing nations, the public health goals that can help prevent and control the spread of typhoid fever disease through safe drinking water, improved sanitation and adequate medical care may be difficult to achieve. Health education is paramount to raise public awareness and induce behavior change [5]. Several mathematical models have been developed to explain the dynamics of typhoid including [2] [6]-[12], but none has incorporated both direct and indirect transmission dynamics in typhoid fever model.

Our main objective in the present paper is to develop an *SIIcR-B* (susceptible, symptomatic infectious, asymptomatic infectious, recovered, bacteria concentration) model of typhoid fever with vaccination, treatment and water sanitation as control strategies that has not been investigated in prior studies. Our major assumption is existence of both direct transmission of typhoid from infected individuals to susceptible and indirect transmission of bacteria from the environment to the susceptible, the other assumptions; all susceptible individuals are equally likely to be infected by infectious individuals in case of contact, and we also assume direct transmission of typhoid from infected to susceptible individuals and that there is a constant recruitment rate to the susceptible population. Furthermore, we assume that the rate of transmission for carriers is greater than that of symptomatic infectious individuals.

2. Model Formulation

We first develop a more realistic model for typhoid. The model subdivides the human population of interest into four compartments: susceptible humans (S), infected humans (I), carrier humans (I_c), and recovered humans (R). Previous models of typhoid dynamics [2] [6]-[12], assume direct transmission of typhoid from infected individuals to susceptible individuals. However, typhoid is largely contracted from environmental bacteria through contaminated water and/or food and drinks [13] [14], and transmission of typhoid through direct person-to-person contact, if any, is negligible [15]. To incorporate this real biological phenomenon, we consider an additional compartment, B, which represents bacteria in the environment. We assume that susceptible individuals get infected with typhoid at a rate proportional to the susceptible population, S, and the environmental bacteria concentration, B, at a constant rate τ.

Individuals in the class, I, can recover from typhoid at the rate, η_1. The carrier individuals can also either progress to carrier class, I_c, at rate α or recover from typhoid, but with a significantly slow rate, η_2. Infected individuals in both infectious state and carrier state excrete bacteria into the environment. However, the rate of excretion by the infectious group, ϵ_1, is significantly higher than that by the carrier group, ϵ_2. Note that despite low excretion of bacteria by the carrier group, because of its extremely long duration without showing any sickness

the carrier group plays an important role on infection dynamics of typhoid. Growth curves of organisms are often described well with the 2 logistic models [16]-[21], so we assume that the bacteria in the environment grows according to a logistic growth rate and becomes non-infectious at a rate μ_b. r and K represent per capita growth rate and carrying capacity, respectively, and d_1, d_2 denotes the typhoid induced mortality in Infectious and carriers individuals respectively. The constant recruitment rate into the susceptible human is represented by Λ, while the natural death rate of human is represented by μ_h. The developed model can be expressed as the following differential equations detailed in section 2.1.

2.1. Model Equations

From the assumptions, descriptions and the compartmental diagram in Figure 1, we formulate the following system of differential equations.

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda + \phi R - (\lambda + \mu_h + \theta) S \tag{1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = p\lambda S + \alpha I_c - (\mu_h + d_1 + \eta_1 + \epsilon_1) I \tag{2}$$

$$\frac{\mathrm{d}I_c}{\mathrm{d}t} = (1-p)\lambda S - (\alpha + \mu_h + d_2 + \eta_2 + \epsilon_2) I_c \tag{3}$$

$$\frac{\mathrm{d}B}{\mathrm{d}t} = r\left(1 - \frac{B}{K}\right) B + \epsilon_1 I + \epsilon_2 I_c - \mu_b B \tag{4}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \eta_1 I + \eta_2 I_c + \theta S - (\mu_h + \phi) R \tag{5}$$

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu_h N - d_1 I - d_2 I_c \tag{6}$$

where

$$\lambda = \beta I + \gamma I_c + \frac{\tau B}{K + B}$$

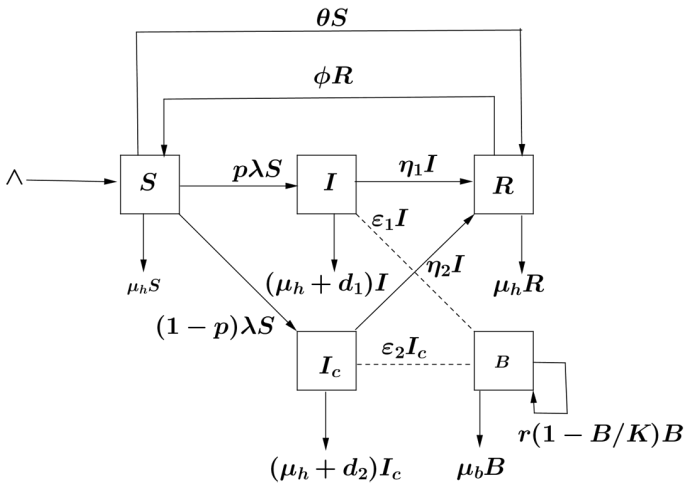

Figure 1. A compartmental diagram for a typhoid model with control strategies.

Equation (1) describes the dynamics of susceptible in the community of size *N*. The death rate of the susceptible individuals is represented by, is the rate of recruitment into susceptible class, is the rate of exposure to contaminated food and water, is the probability of susceptible catching typhoid fever, is the susceptible and is the concentration of Salmonella typhi bacteria in food and water. Equations (2) and (3) describes the dynamics of infected people in the community, their number increases as susceptible become infected and decreases as the infected recovers or die from the disease or natural death. Measures to limit the spread of the disease, such as hygiene and total sanitation reduce the amount of Salmonella typhi bacteria in the environment. Equation (4) describes the dynamics of pathogenic Salmonella typhi bacteria in environment, comprising the contaminated food or water consumed by people and unhygienic handling of typhoid fever patients and their waste products. Equation (5) describes the dynamics of effect of treatment or lack of treatment to the population of infected people.

2.2. Basic Properties of the Model

2.2.1. Positivity of Solutions

We show that if the system starts with non-negative initial conditions $\left(S^0, I^0, I_c^0, R^0, B^0\right)$, the solutions/trajectories of (1)-(5) will remain non-negative for all $t \in (0,\infty)$. This is an ideal condition to check since the model monitors human population and the pathogen concentration in the aquatic environment. We thus have the following theorem: ***Theorem* 1.** *Given that the initial conditions of the system* (1)-(5) $\left(S^0 > 0, I^0 > 0, I_c^0 > 0, R^0 > 0, B^0 > 0\right)$, *the resulting solutions* $\left(S(t), I(t), I_c(t), R(t), B(t)\right)$ *are all non-negative for all* $t \in (0,\infty)$.

Proof. To show positivity of solution, it is enough to show that each of the tra-jectories of system (1)-(5) is non negative for all $t > 0$. From Equation (1), the differential inequality describing the evolution of the susceptible population over time is given by

$$\frac{\mathrm{d}S}{\mathrm{d}t} \geq \Lambda - (\lambda + \mu_h + \theta) S \tag{7}$$

The resulting differential inequality can be solved by separation of variables. Since at $t = 0, S(0) = S^0$, then the complete solution to the differential inequality for the susceptible population is given by

$$S(t) \geq \frac{\Lambda}{\mu_h + \theta} + \left(S^0 - \frac{\Lambda}{\mu_h + \theta}\right) \mathrm{e}^{-(\mu_h + \theta)t} \tag{8}$$

$$\lim_{n\to\infty} \inf \; S(t) \geq 0$$

Using the same principle, the rest of the phase space variables as *t* approaches infinity can be shown to satisfy

$$I(t) \geq I^0 \mathrm{e}^{-(\mu_h + d_1 + \eta_1 + \epsilon_1)t},$$

$$I_c(t) \geq I^0 \mathrm{e}^{-(\mu_h + d_2 + \eta_2 + \epsilon_2)t}$$

$$R(t) \geq R_0 e^{-(\mu_h+\phi)t}$$

From which the limit *inf* of the corresponding state variables can be shown to be non-negative. Using the equation describing the evolution of the pathogen concentration, we have a differential inequality given by

$$\frac{dB}{dt} + (\mu_b - r)B \geq -\frac{B^2}{K} \tag{9}$$

Equation (9) is a Bernoulli type of equation. It is solved by substitution *i.e.* $B = y^{-1}$ to obtain

$$B(t) \geq -\frac{e^{-(\mu_b - r)t}}{AK(\mu_b - r)} \tag{10}$$

2.2.2. Boundedness of Solutions

The model can be separated into two parts which include, the human population T_H and the concentration of the pathogen in the aquatic environment T_B such that $T_H = \{(S(t), I(t), I_c(t), R(t)) \in \mathbb{R}_+^4 : S + I + I_c + R = N\}$ and $T_B = \{B(t) \in \mathbb{R}_+^1\}$ respectively. From Equation (1) the differential inequality of the susceptible population is given by

$$\frac{dS}{dt} = \Lambda + \phi R - (\lambda + \mu_h + \theta)S$$

$$\frac{dS}{dt} + (\mu_h + \theta)S \leq \Lambda + \phi R \tag{11}$$

Using a suitable integrating factor, $I.F = e^{-\mu t}$, the differential inequality (11) can be solved to obtain

$$S(t) \leq \frac{\Lambda}{\mu_h} + e^{-\mu t}\int_0^t \omega R(x) e^{-\mu x} dx \tag{12}$$

Following the theorem of differential inequality by Birkhoff and Rota [21] we obtain

$$\lim_{t\to\infty} \text{Sup}\ \ S(t) \leq \frac{\Lambda}{\mu_h}$$

Therefore, the state variable describing the evolution of the susceptible population is less or equal to the ratio of the recruitment rate and the natural mortality rate. We note also that the total population is given as $N = S + I + I_c + R$. If we take the time derivative of N *i.e.* $\frac{dN}{dt}$ and substitute the Equations (1)-(4) into the resulting expression we obtain

$$\frac{dN}{dt} = \Lambda - \mu_h N - d_1 I - d_2 I_c \tag{13}$$

The solution (12) can be obtained by separating variables and integrating both sides with respect to the corresponding variable. This result into

$$\ln|\Lambda - \mu_h N| \geq -\mu_h t + c$$

where c is a constant of integration. If we exponentiate both sides and assume that the initial total population is N^0, the solution becomes

$$N \le \frac{\Lambda}{\mu_h} - \left(\frac{\Lambda}{\mu_h} - N^0\right) e^{-\mu_h t} \tag{14}$$

therefore,

$$\lim_{t\to\infty} \text{Sup}\ \ N(t) \le \frac{\Lambda}{\mu_h}$$

Since N is the sum of all state space variables, then each of the individual state variables is less or equal to $\frac{\Lambda}{\mu_h}$. Using Equation (5), we assume that the growth rate of the pathogen in linear at a constant rate r. We therefore obtain a differential inequality

$$\frac{dB}{dt} + (\mu_b - r)B \le \epsilon_1 I + \epsilon_2 I_c \tag{15}$$

$$\frac{dB}{dt} + (\mu_b - r)B \le (\epsilon_1 + \epsilon_2)\frac{\Lambda}{\mu_h} \tag{16}$$

The solution to this equation can be obtained by using a suitable integrating factor to obtain

$$B(t) \le \frac{(\epsilon_1 + \epsilon_2)\Lambda}{\mu_h(\mu_b - r)} + Ae^{(\mu_b - r)t} \tag{17}$$

therefore,

$$\lim_{t\to\infty} \text{Sup}\ \ B(t) \le \frac{(\epsilon_1 + \epsilon_2)\Lambda}{\mu_h(\mu_b - r)}$$

The domain of biological significance of the system (1)-(5) is

$$T = \left[S, I, I_c, R, B(t) \ge 0 : S + I + I_c + R \le \frac{\Lambda}{\mu_h}, B(t) \le \frac{(\epsilon_1 + \epsilon_2)\Lambda}{\mu_h(\mu_b - r)}\right]. \tag{18}$$

The domain T is positively invariant under the flow induced by the system (1)-(5). Therefore, the system (1)-(5) is biologically meaningful and it is feasible to analyze the model in the domain T.

2.3. The Basic Reproduction Number, R_0

The basic reproduction number denoted by R_0 is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness Diekmann *et al.* [22]. 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_0. Furthermore, stability of equilibria can be analyzed using R_0. If $R_0 < 1$ it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_0 < 1$, every infectious individual will cause more than one secondary infection and hence the disease will invade the population. A

large number of R_0 may indicate the possibility of a major epidemic. For the case of a model with a single infected class, R_0 is simply the product of the infection rate and the mean duration of the infection. In more complicated epidemics we compute the basic reproduction number, R_0 using the next generation operator approach by Van den Driessche and Watmough [23]. We calculate the basic reproduction number by using the next generation operator method on the system Equation (1)-(5). The basic reproduction number is obtained by taking the largest (dominant) eigenvalue (spectral radius)

$$FV^{-1} = \left[\frac{\partial \mathcal{F}_i(E_0)}{\partial x_j}\right]\left[\frac{\partial \mathcal{V}_i(E_0)}{\partial x_j}\right]^{-1} \tag{19}$$

where F_i is the rate of appearance of new infection in compartment i, V_i is the transfer of infections from one compartment i to another and E^0 is the disease-free equilibrium. From system equation of the system (1)-(4), we re-write the equations with infectious classes, I, I_c and B. This leads to the system

$$\frac{\mathrm{d}I}{\mathrm{d}t} = p\lambda S + \alpha I_c - \left(\mu_h + d_1 + \eta_1 + \epsilon_1\right) I$$

$$\frac{\mathrm{d}I_c}{\mathrm{d}t} = \left(1-p\right)\lambda S - \left(\alpha + \mu_h + d_2 + \eta_2 + \epsilon_2\right) I_c$$

$$\frac{\mathrm{d}B}{\mathrm{d}t} = r\left(1 - \frac{B}{K}\right) B + \epsilon_1 I + \epsilon_2 I_c - \mu_b B$$

Jacobian at diseases free point (E_0) is computed and found to be

$$J\left(E_0\right) = \begin{bmatrix} \dfrac{p\tau BS}{K+B} + p\beta IS + p\gamma I_c S \\ \dfrac{\left(1-p\right)\tau BS}{K+B} + \left(1-p\right)\beta IS + \left(1-p\right)\gamma I_c S \\ 0 \end{bmatrix} \tag{20}$$

from which we obtain:

$$\mathcal{F}_i = \begin{bmatrix} \dfrac{p\tau BS}{K+B} + p\beta IS + p\gamma I_c S \\ \dfrac{\left(1-p\right)\tau BS}{K+B} + \left(1-p\right)\beta IS + \left(1-p\right)\gamma I_c S \\ 0 \end{bmatrix} \tag{21}$$

$$\mathcal{V}_i = \begin{bmatrix} \left(\mu_h + d_1 + \eta_1 + \epsilon_1\right) I \\ \left(\mu_h + d_2 + \eta_2 + \epsilon_2\right) I_c \\ \mu_b B - \epsilon_1 I \epsilon_2 I_c + r\left(1 - \dfrac{B}{K}\right) B \end{bmatrix} \tag{22}$$

Partial differentiation of F_i and V_i with respect to I, I_c and B gives

$$F = \begin{bmatrix} p\beta S^0 & p\gamma S^0 & \dfrac{p\tau S^0}{K} \\ (1-p)\beta S^0 & (1-p)\beta S^0 & \dfrac{(1-p)\tau S^0}{K} \\ 0 & 0 & 0 \end{bmatrix} \tag{23}$$

$$V = \begin{bmatrix} (\mu_h + d_1 + \eta_1 + \epsilon_1) & 0 & 0 \\ 0 & (\mu_h + d_2 + \eta_2 + \epsilon_2) & 0 \\ -\epsilon_1 & -\epsilon_2 & \mu_b - r \end{bmatrix} \tag{24}$$

V^{-1} was computed and found to be:

$$V^{-1} = \frac{1}{a_1 a_2 (\mu_b - r)} \begin{bmatrix} a_2(\mu_b - r) & \alpha(\mu_b - r) & 0 \\ 0 & a_1(\mu_b - r) & 0 \\ \epsilon_1 a_2 & a\epsilon_2 & a_1 \end{bmatrix}$$

Lastly FV^{-1} was calculated and results is:

$$FV^{-1} = \frac{1}{a_1 a_2 (\mu_b - r)} \begin{pmatrix} p\beta S^0 a_2(u_b - r) + \dfrac{p\tau S^0 \epsilon_1 a_2}{K} & p\gamma a_1 S^0 (u_b - r) + \dfrac{p\tau S^0 f}{K} & \dfrac{p a_1 \tau S^0}{K} \\ (1-p)\beta S^0 a_2(u_b - r) + \dfrac{(1-p)\tau S^0 \epsilon_1 a_2}{K} & (1-p)\gamma a_1 S^0 (u_b - r) + \dfrac{(1-p) a_1 \tau S^0 f}{K} & \dfrac{(1-p) a_1 \tau S^0}{K} \\ 0 & 0 & 0 \end{pmatrix}$$

The eigenvalues of FV^{-1} was calculated as follows:

$$\det \begin{pmatrix} \lambda - \left[p\beta S^0 a_2(u_b - r) + \dfrac{p\tau S^0 \epsilon_1 a_2}{K} \right] & p\gamma a_1 S^0 (u_b - r) + \dfrac{p\tau S^0 f}{K} & \dfrac{p a_1 \tau S^0}{K} \\ (1-p)\beta S^0 a_2(u_b - r) + \dfrac{(1-p)\tau S^0 \epsilon_1 a_2}{K} & \lambda - \left[(1-p)\gamma a_1 S^0 (u_b - r) + \dfrac{(1-p) a_1 \tau S^0 f}{K} \right] & \dfrac{(1-p) a_1 \tau S^0}{K} \\ 0 & 0 & \lambda \end{pmatrix} = 0$$

let

$$Z_1 = p\beta S^0 a_2(u_b - r) + \frac{p\tau S^0 \epsilon_1 a_2}{K}$$

$$Z_2 = p\beta S^0 \alpha(u_b - r) + p\gamma a_1 S^0 (u_b - r) + \frac{p\tau S^0 f}{K}$$

$$Z_3 = \frac{p\tau S^0}{K}$$

$$Z_4 = (1-p)\beta S^0 a_2(u_b - r) + \frac{(1-p)\tau S^0 \epsilon_1 a_2}{K}$$

$$Z_5 = (1-p)\gamma a_1 S^0 (u_b - r) + \frac{(1-p) a_1 \tau S^0 f}{K}$$

$$Z_6 = \frac{(1-p) a_1 \tau S^0}{K}$$

We get

$$\begin{vmatrix} \lambda - Z_1 & Z_2 & Z_3 \\ Z_4 & \lambda - Z_5 & Z_6 \\ 0 & 0 & \lambda \end{vmatrix} = 0$$

$$\lambda\left[\left(\lambda - Z_5\right)\left(\lambda - Z_1\right) - Z_2 Z_5\right] = 0$$

$$\lambda\left(\lambda^2 - \left(Z_1 + Z_5\right)\lambda + \left(Z_1 Z_5 - Z_2 Z_4\right)\right) = 0$$

it can be shown that $Z_1 Z_5 - Z_2 Z_4 = 0$

$$\lambda^2\left(\lambda - \left(Z_1 + Z_5\right)\right) = 0 \tag{25}$$

$$\lambda = 0, 0, Z_1 + Z_5 \tag{26}$$

thus the maximum eigenvalues is

$$Z_1 + Z_5$$

which gives

$$R_e = Z_1 + Z_5$$

therefore

$$R_e = p\beta S^0 a_2\left(\mu_b - r\right) + a_1\left(1-p\right)\gamma S^0\left(\mu_b - r\right) + \frac{p\tau S^0 \epsilon_1 a_2}{K} + \frac{\left(1-p\right)\tau S^0 a_1 \epsilon_2}{K} \tag{27}$$

$$= \frac{S^0}{a_1 a_2\left(\mu_b - r\right)}\left(\left(\mu_b - r\right)\left[p a_2 \beta + a_1\left(1-p\right)\gamma\right] + \frac{\tau}{K}\left[a_2 p\epsilon_1 + \left(1-p\right)a_1\epsilon_2\right]\right) \tag{28}$$

$$= \frac{S^0 p}{a_1\left(\mu_b - r\right)}\frac{\tau}{K}\left(\beta\left(\mu_b - r\right) + \frac{\tau\epsilon_1}{K}\right) + \frac{\left(1-p\right)S^0}{a_2\left(\mu_b - r\right)} \tag{29}$$

$$= \left(\frac{S^0 p}{a_1}\beta + \frac{S^0 p\tau}{a_1\left(\mu_b - r\right)K}\epsilon_1\right) + \left(\frac{\left(1-p\right)S^0}{a_2}\gamma + \frac{\tau}{K}\frac{\left(1-p\right)S^0\gamma}{a_2\left(\mu_b - r\right)}\epsilon_2\right) \tag{30}$$

This can be written as

$$R_e = \left(R_{01} + R_{03}\right) + \left(R_{02} + R_{04}\right)$$

where

$$R_{01} = \left(\frac{S^0 p}{a_1}\right) \tag{31}$$

$$R_{02} = \frac{S^0\left(1-p\right)\gamma}{a_2} \tag{32}$$

$$R_{03} = \left(\frac{S^0 p\tau}{a_1\left(\mu_b - r\right)K}\right)\epsilon_1 \tag{33}$$

$$R_{04} = \frac{S^0\left(1-p\right)\tau}{a_2\left(\mu_b - r\right)K}\epsilon_2 \tag{34}$$

$$S^0 = \frac{\left(\mu_h + \phi\right)\Lambda}{\mu_h\left(\mu_h + \theta + \phi\right)} \tag{35}$$

$$a_1 = \mu_h + d_1 + \eta_1 + \epsilon_1 \tag{36}$$

$$a_2 = \mu_h + d_2 + \eta_2 + \epsilon_2 \tag{37}$$

Thus R_e is the effective reproduction number (basic reproduction number with controls). The terms $\frac{1}{a_1}$ and $\frac{1}{a_2}$ indicate the maximum time an individual is expected to stay in compartments I and I_c respectively. The reproduction number consists of four terms which characterize the contribution from the different pathways to new infections with typhoid.

3. Results and Discussion

In this study our objective is to model transmission dynamics of typhoid fever via the direct and indirect paths, we want to analyze what happens to the system when control measures like vaccination, treatment and water sanitation either effected or not. Furthermore we want to know the role of carriers and symptomatic individuals to this dynamical system. Various graphical representations have been generated with the help of MATLAB which will support our analytical results. Since, most of the parameters were not readily available; it was found convenient to pick from other sources and unavailable data were estimated. In order to perform simulations, baseline values of parameters from Table 1 presented before were used.

It can be seen from Figures 2(a)-(d) that sanitation, vaccination, treatment of both symptomatic and asymptomatically infected individuals respectively, are observed to reduce the severity of the disease if such parameters are increased.

Table 1. Parameters and their description.

Parameter	Value	Description	Source
Λ	10^6	Constant human recruitment rate	[10] [13]
μ_b	0.4/year	Mortality rate for bacteria, including phage degradation	Estimated
μ_h	0.167/year	Natural human mortality rate	Estimated
d_1	0.15/year	Disease induced death rate	[2]
d_2	0.6/year	Disease induced death rate	Estimated
β	0.02/year	Effective contact rate between individuals (contact sufficient)	Estimated
τ	0.7/year	Per capita contact rate for humans and contaminated water	Estimated
η_1	0.04/year	Recovery rate of infectious humans	[4]
η_2	0.05/year	Recovery rate of infectious humans	Estimated
ϵ_1	0.5/year	Bacteria shed rate into the water supply by infectious human	Estimated
ϵ_2	0.4/year	Bacteria shed rate into the water supply by infectious human	Estimated
ϕ	0.33/year	Per capita rate at which recovered humans are susceptible	Estimated
θ	0.4/year	Per capita rate at which susceptible humans are vaccinated	[10]
r	0.01/year	(Maximum) per capita growth rate for S. typhi bacteria	[10]
K	100/year	Carrying capacity for S. typhi	Estimated
p	0.8/year	Proportion of infected individuals who are symptomatic	Estimated

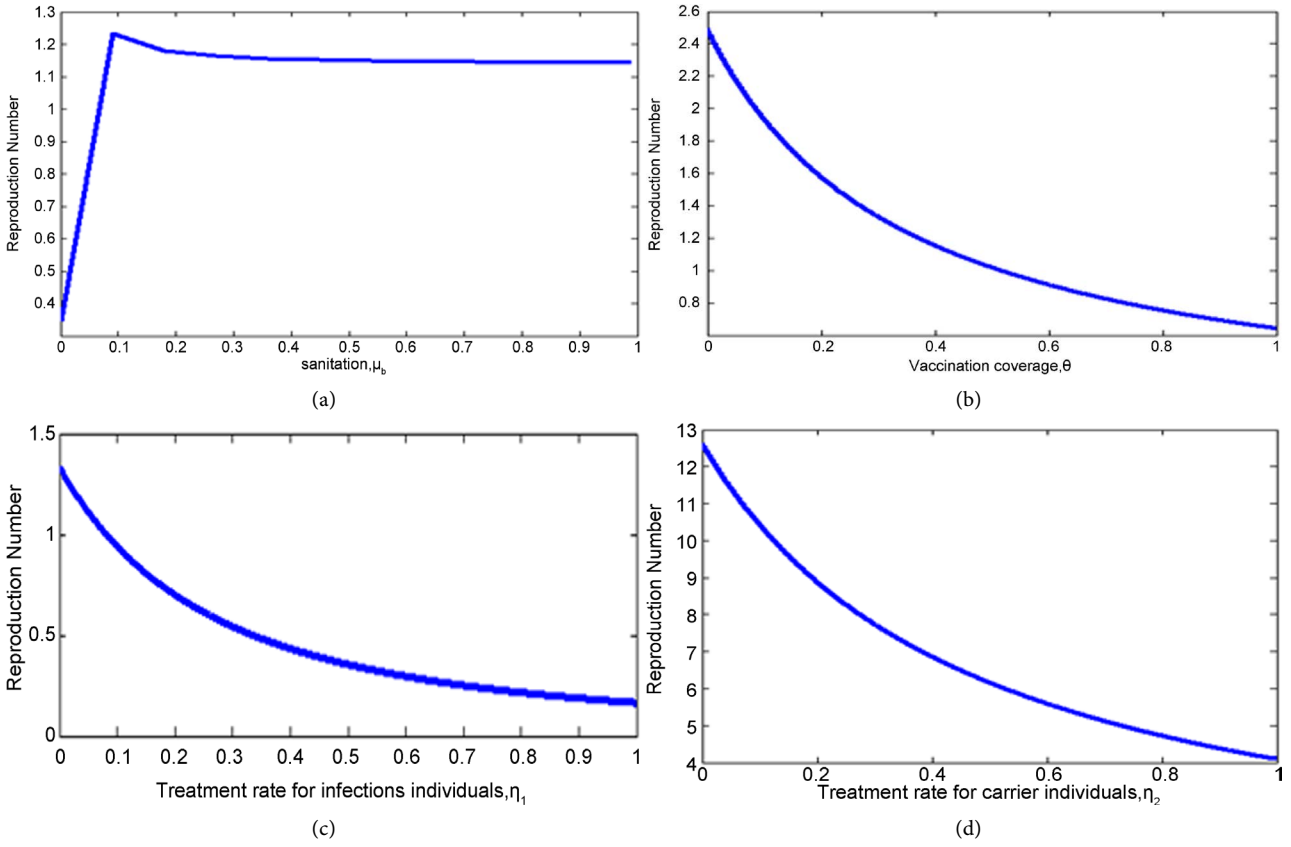

Figure 2. (a)shows the effect of sanitation effort to the effective reproduction number, (b) shows the effect of variation of vaccination coverage on the effective reproduction number, (c) shows effect of variation of therapeutic treatment of symptomatic infectious individuals on the effective reproduction number and (d) shows effect of variation of therapeutic treatment of asymptomatic (carrier) infectious individuals on the effective reproduction number.

Recovery of infected individuals has a twofold benefit in the fight against the infection; 1) it leads to reduction the likelihood getting new infections through direct person to person transmission, and 2) a negligible amount of the pathogen would be shed into the aquatic reservoir greatly reducing the infection risk, most especially for the community that may have direct contact with a potentially contaminated water source. In addition, recovering individuals acquire some immunity to the disease which only wanes over a reasonable period of time hence reducing the susceptible. On the other hand, it can be seen from **Figures 3(a)-(e)** that increased discharge of fresh bacteria into the aquatic environment by either the symptomatic or asymptomatic individuals, increased probability of contracting bacteria from the environment. Likewise, high personal contact rate with either the symptomatic or asymptomatic individuals increase the risk of contracting typhoid fever. The worst case scenario in case of epidemic outbreak may be experienced when the level of hygiene is poor; maximizing person-to-person contact rate and when there is no accesses to clean water; which maximizes disease transmission through contact with the contaminated reservoir. If immunization of the susceptible population alone does not bring about typhoid elimination, then measures to reduce per case or per carrier infectivity, such as improved sanitation or hand washing with soap, might be considered instead

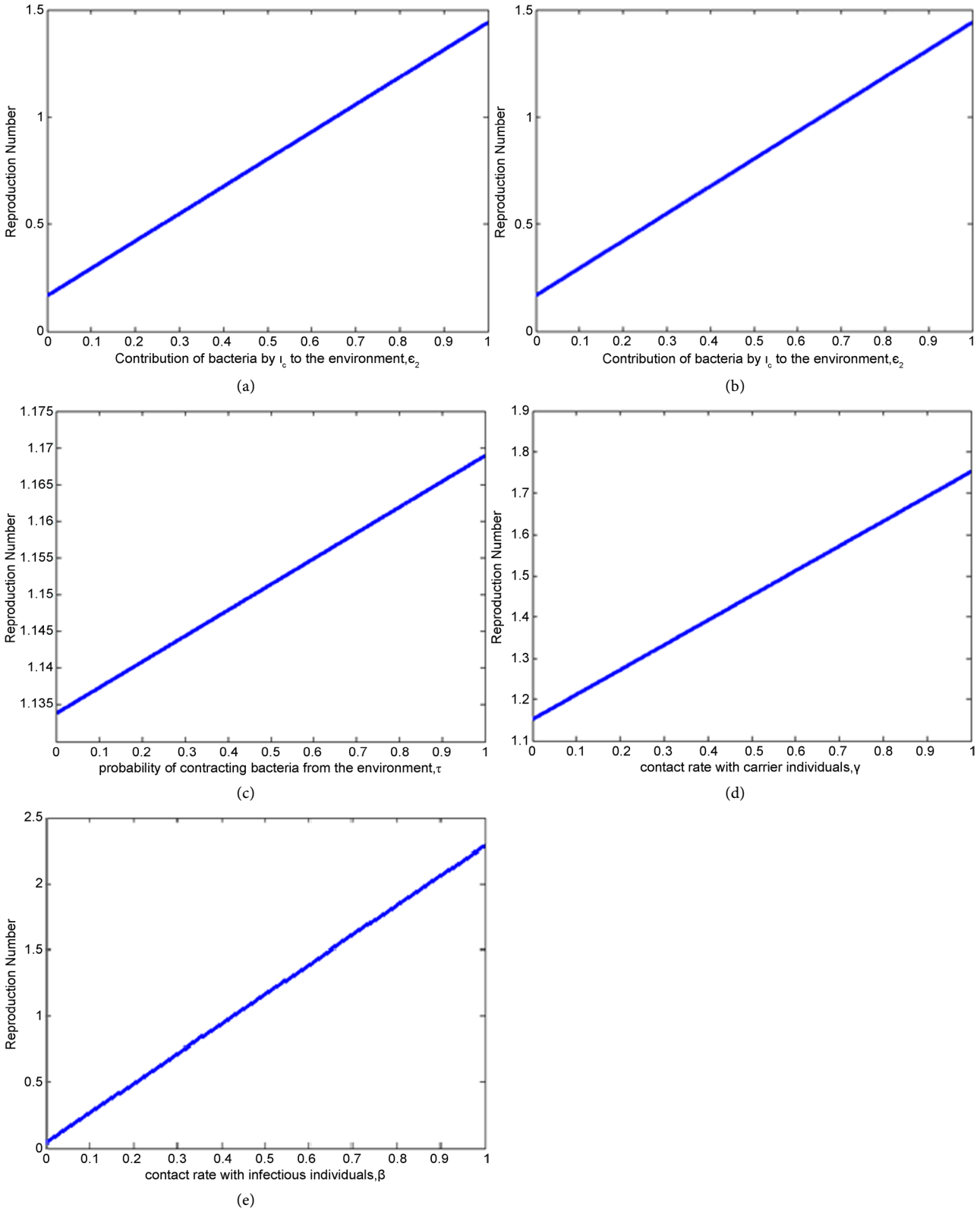

Figure 3. (a) shows the effect of infectious individuals polluting the environment with salmonela bacteria to the effective reproduction number, (b) shows the effect of carriers individuals polluting the environment with salmonela bacteria to the effective reproduction number, (c) shows probability of catching bacteria from the environment, (d) shows effect of variation of contact rate with carrier individuals on the effective reproduction number, (e) shows effect of variation of contact with infectious individual on the effective reproduction number.

of or in conjunction with vaccination [24] [25]. The multi- compartment models suggest such a reduction in effective contact rates could lead to important reduction in prevalence [3] [26] [27]. This is consistent with Briscoe's analytical model [28].

4. Conclusion

In this paper, a deterministic model which incorporates person-to-person contact rate and person-environment was presented and analyzed. Important mathematical features of the models such as the threshold for the epidemic, steady states, positivity and boundedness of solutions as well as the region of biological significance were determined. The model was shown to have a disease free equilibrium which is both locally and globally asymptotically stable when the reproduction number is less than unity. This disease free equilibrium is unstable when the disease threshold is greater than unity. The model has a unique endemic equilibrium for $R_e > 1$. In general, vaccination coverage, therapeutic treatment and water sanitation have been shown to play a pivotal role of diminishing the outbreak when they are encouraged in the community. On the other hand, it has been found beneficial to minimize contact with typhoid patients, avoid spoiling water sources with feces, and people should use Latrines. We acknowledge the fact that this work may have shortfalls as follows. The model does not take into account education campaigns. However, education is recommended as a preventive measure for typhoid. Proposed improvements of the model include consideration of a combination of hygiene, vaccination, education campaigns and biological control with salmonella specific bacteriophage.

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