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Sensitivity Analysis of Typhoid Fever model with Saturated Incidence rate

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Abstract: In this study, a dynamic model for typhoid fever incorporating protection against infection in the presence of saturated incidence rate is proposed. The existence and uniqueness solution is proved in order to ascertain the existence of the model. Stability analysis of endemic and disease free equilibrium was carried out to investigate the dynamic behavior of the transmission of the disease in a given population. Sensitivity analysis was also carried out to detect the impact of the parameters of the reproductive number and which parameters should focus as a control intervention. Numerical simulation of the model was carried out and the result is presented graphically, the result shows that an increase in the probability of the sources of protection and sociology factor dictate low disease prevalence in a population.

Keywords: Typhoid fever, Saturated Incident Rate, Sensitivity Analysis, Stability.

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1 Introduction

Typhoid fever was so named because its signs and symptoms resemble that of typhus. It is an endemic infectious disease caused by a highly virulent and invasive Salmonella enteric serovar Typhi (S.Typhi) that affects human [25]. Typhoid fever is a communicable disease found only in human and occurs due to systemic infection mainly by salmonilla typhi organism, this disease is declared as a major health concern in many developing countries where safe water supply, food in hygienic and environmental sanitation are the significant challenges [6, 9, 14,15,16,17, 20,21,28,29,30,31]

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, [1,7,10, 11, 13, 26,32, 33].

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The symptoms of this illness include fever, headaches, and general discomfort. As the illness worsens, a high fever (103F or 39.5C) and severe diarrhea appear. The incubation phase lasts for approximately 10–14 days, yet it can occasionally be 3 days too short or extended for 21 days. When S. paratyphi A, B, and occasionally C are the suspects, this illness is less severe. It is endemic in Nigeria, some regions of Africa, the Indian subcontinent, and south-east Asia. According to estimates from 2000, the sickness sickened 21.6 million people worldwide and killed 216,500 people.

Other relative studies like [2,3,4,18,19,23,24,26,27] gave more insight into the modeling of the typhoid fever with different techniques and approaches in order to better understanding the dynamics and evolution of the outbreak in many scenarios. A mathematical model that will assist in establishing the conditions under which the spread of the disease can be controlled and eradicated is urgently needed due to the rising incidence of typhoid fever

The goal of this study is to develop a model that takes into account of Sociology factor, the other parts of the study are divided as follows: section 2 describes the model formulation, section 3 presents some mathematical analysis of the model, section 4 displays the results from the study and the last cover conclusion.

2 Material and Methods

2.1 Model Description

In this study model proposed by [25] was modified to incorporate of saturated incidence rate, the total population is subdivided into the following sub-population classes; (P) Protected class, (S) Susceptible, (I) Infected class, (T) Treatment class. We can see from the diagram above that the recruitment rate into the class of individuals protected against typhoid $\Lambda\alpha$ are moving into protected class. The protected class has the probability of joining a susceptible class by, γ loss of protection rate. $(1-\alpha)\Lambda$ is the recruitment rate into the class of individuals susceptible to typhoid. People can die in the susceptible class due to the cause of natural death μS . And the susceptible class has the probability of being a member of infected class by $(1-v)$ and the rate of treatment (β) from infected to treatment class. People in the infected class can either die due to the cause of disease or as a result of natural death $(\delta+\mu)I$ and if treated, they can be free from the disease at the rate of βI and also, people in the treatment class can still die due to natural death μT .

$$\left.\begin{aligned}
\frac{dP}{dt} &= \alpha\Lambda-(\mu+\gamma)P \\
\frac{dS}{dt} &= (1-\alpha)\Lambda+\gamma P-\frac{\omega S(1-v)I}{1+mI}-\mu S \\
\frac{dI}{dt} &= \frac{\omega S(1-v)I}{1+mI}-(\delta+\beta+\mu)I \\
\frac{dT}{dt} &= \beta I+\mu T
\end{aligned}\right\} \quad (1)$$

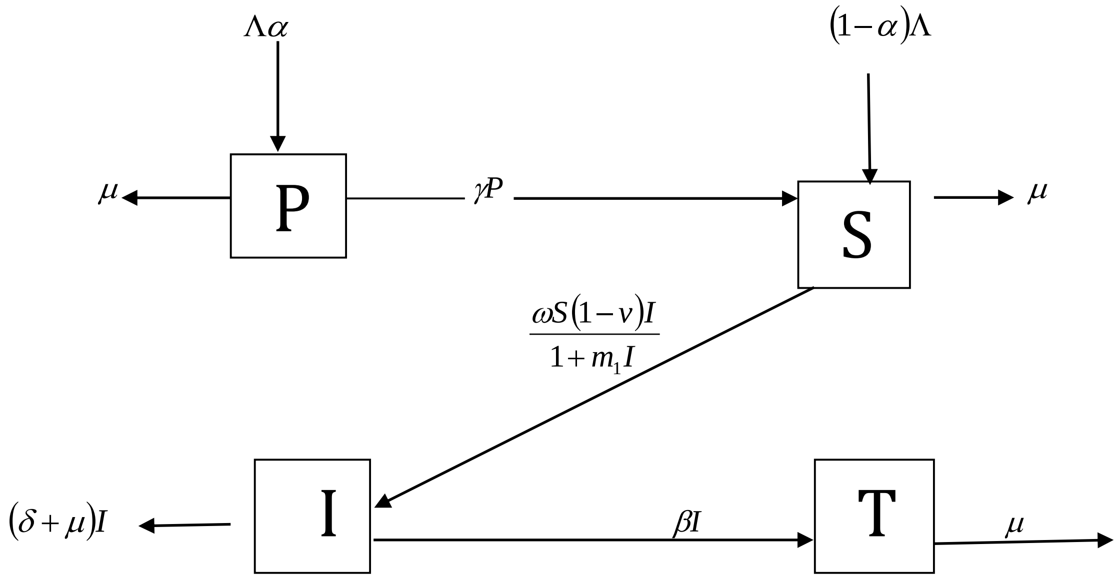

Fig.1: Proposed schematic diagram for the PSIT model with saturated incident rate.

2.2 Existence and Uniqueness Solution of the Model.

Using a Lipschitz criterion, from the equations in (1) above:

$$F_1 = \Lambda\alpha - (\gamma + \mu)P \quad \textbf{(2)}$$

$$F_2 = (1-\alpha)\Lambda + \gamma P - \frac{\omega S(1-v)I}{1+m\ I} - \mu S \quad \textbf{(3)}$$

$$F_3 = \frac{\omega S(1-v)I}{1+mI} - (\delta + \beta + \mu)I \quad \textbf{(4)}$$

$$F_4 = \beta I - \mu T \quad \textbf{(5)}$$

Theorem: Let D denote the region $0 \le z \le R$ then the system of equation in (1) has a unique solution, provided that $\frac{\partial f_i}{\partial f_j}, i, j = 1,2,...,$ are continuous and bounded [9, 8].

Proof.

$\frac{\partial f_i}{\partial f_j}, i, j = 1,2,3,4$ are continuous and bounded in D

Finding the partial derivative of equation (2 – 5), we obtained the following;

$$\left|\frac{\partial f_1}{\partial P}\right| = |\gamma + \mu| < \infty, \left|\frac{\partial f_1}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_1}{\partial I}\right| = 0 < \infty, \left|\frac{\partial f_1}{\partial T}\right| = 0 < \infty$$

$$\left|\frac{\partial f_2}{\partial P}\right| = |\gamma| < \infty, \left|\frac{\partial f_2}{\partial S}\right| = |-\mu| < \infty, \left|\frac{\partial f_2}{\partial I}\right| = \left|-\frac{\omega S(1-v)}{(1+mI)^2}\right| < \infty, \left|\frac{\partial f_2}{\partial T}\right| = 0 < \infty$$

$$\left|\frac{\partial f_3}{\partial P}\right| = 0 < \infty, \left|\frac{\partial f_3}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_3}{\partial I}\right| = \left|\frac{\omega S(1-v)(1+mI) - \omega S(1-v)mI}{(1+mI)^2} - (\delta + \beta + \mu)\right| < \infty, \left|\frac{\partial f_3}{\partial T}\right| = o < \infty$$

$$\left|\frac{\partial f_4}{\partial P}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial I}\right| = |\beta| < \infty, \left|\frac{\partial f_4}{\partial T}\right| = |-\mu| < \infty$$

Obviously, from the equation above the partial derivatives exist, continuous and are bounded. Hence the model has a unique solution.

2.3 Model Solution

In the absence of disease infection in the population, $I = 0$.Solving the (1) gives disease – free equilibrium

$$(P, S, I, T) = \left(\frac{\Lambda\alpha}{(\gamma + \mu)}, 0,0,0\right)$$

Also, in the presence of disease infection in the population, $I \neq 0$. Solving (1) gives Endemic equilibrium.

$$E_* = (P_*, S_*, I_*, T_*)$$

Where;

$$P_* = \frac{\Lambda\alpha}{(\gamma + \mu)}, \quad S_* = \frac{\mu^2 + (\gamma + (-\alpha + 1)\Lambda m + \beta + \sigma)\mu + \gamma(\Lambda m + \beta - \sigma)}{(\gamma + \mu)(\mu m - \varpi(v-1))}$$

$$I_* = \frac{-\mu^3 + (-\beta - \gamma - \sigma)\mu^2 + (\Lambda(v-1)(\alpha-1)\varpi - \gamma(\beta + \sigma))\mu - \gamma\Lambda\varpi(v-1)}{(\gamma + \mu)(\mu m - \varpi(v-1))(\mu + \beta + \sigma)}$$

$$T_* = -\frac{(\mu^3 + (\beta + \gamma + \sigma)\mu^2 + (-\Lambda(v-1)(\alpha-1)\varpi + \gamma(\beta + \sigma))\mu + \gamma\Lambda\varpi(v-1))\beta}{(\gamma + \mu)(\mu m - \varpi(v-1))\mu(\mu + \beta + \sigma)}$$

2.4 Basic Reproductive Number (R_0)

The basic reproductive number is very vital and it play an important role to the effort required to eliminate infectious [2,5,19,23,24]. In order to compute the basic reproduction number, next generation matrix method was employed: Let $G = FV^{-1}$

Where F is the rate of new infectious, V is the rate of transfer of infection from one compartment to another. From the proposed model in (1);

$$F = \frac{\omega S(1-v)I}{1+mI} \tag{8}$$

and $V = (\beta + \mu + \sigma)I$ **(9)**

Taking derivative of (8) and (9) respectively with respect to I, we obtained F as;

$$F = \frac{\omega\Lambda(\gamma + \mu - \alpha\mu)(1-v)}{\mu(\gamma+\mu)} \tag{10}$$

Recall from D.F.E,

$$S = \frac{\Lambda(\gamma + \mu - \alpha\mu)}{\Lambda(\gamma+\mu)}$$

Also obtained; $V = (\delta + \beta + \mu)$ **(11)**

Thus;

$$V^{-1} = \frac{1}{(\delta + \beta + \mu)} \tag{12}$$

Hence, $FV^{-1} = \dfrac{\omega\Lambda(\gamma + \mu - \alpha\mu)(1-v)}{\mu(\gamma+\mu)}\left(\dfrac{1}{\delta + \beta + \mu}\right)$

Therefore,

$$R_0 = \frac{\omega\Lambda(\gamma + \mu - \alpha\mu)(1-v)}{\mu(\gamma+\mu)(\delta + \beta + \mu)} \tag{13}$$

2.5 Local Stability of Disease Free Equilibrium

Proposition 1. If $R_0 < 1$, then the disease free equilibrium E_0 is locally asymptotically stable.

Proof.

Considering Linearization method, the resulting characteristic equation of system (1) is $|A - \lambda I| = 0$

If $R_0 < 1$, then the disease free equilibrium E_0 is locally asymptotically stable.

$$|A - \lambda I| = \begin{pmatrix} -(\gamma+\mu) & 0 & 0 & 0 \\ \gamma & -\mu & -\dfrac{\varpi\Lambda(-\alpha\mu + \gamma + \mu)(1-v)}{\mu(\gamma+\mu)} & 0 \\ 0 & 0 & \dfrac{\varpi\Lambda(-\alpha\mu + \gamma + \mu)(1-v)}{\mu(\gamma+\mu)} - (\sigma + \beta + \mu) & 0 \\ 0 & 0 & \beta & -\mu \end{pmatrix}$$

Computing the Trace and the determinant of the matrix above, thus the trace at DFE, is given by;

$$\tau\left(J_{E_0}\right) = -(\gamma+\mu) - 2\mu + \frac{\varpi\Lambda(-\alpha\mu+\gamma+\mu)(1-v)}{\mu(\gamma+\mu)} - (\sigma+\beta+\mu) \quad \textbf{(14)}$$

Simplifying we have;

$$\tau\left(J_{E_0}\right) = -(\gamma+\mu) - 2\mu + (R_0-1)(\sigma+\beta+\mu) \quad \textbf{(15)}$$

And the determinant of the matrix is obtained as;

$$Det\left(J_{E_0}\right) = -(\gamma+\mu)\left[\mu^2\left(\frac{\varpi\Lambda(-\alpha\mu+\gamma+\mu)(1-v)}{\mu(\gamma+\mu)} - (\sigma+\beta+\mu)\right) - 0\right] \quad \textbf{(16)}$$

Simplifying (16) we obtained,

$$Det\left(J_{E_0}\right) = (\gamma+\mu)\mu^2(1-R_0) \quad \textbf{(17)}$$

The Trace (τ) stable provided and the determinant is positive with the same condition. Thus, the disease free equilibrium is asymptotically stable provided the $R_0 < 1$.

2.6 Local Stability of Endemic Equilibrium

Proposition 2. The endemic equilibrium E_* of the model (1) is locally asymptotically stable if $R_0 > 1$.

Proof.

Let the endemic equilibrium $E_* = (S_*, I_*, R_*)$, using linearization method by setting

$$X = S - S_*,\ Y = P - P_*,\ Z = I - I_*,\ M = T - T_* \quad \textbf{(18)}$$

Then the resulting jacobian matrix of system of equation (1) is

$$\begin{pmatrix} -\gamma-\mu & 0 & 0 & 0 \\ \gamma & \frac{-\omega(1-v)I}{1+m_1 I} - \mu & \frac{-\omega S(1-v)}{1+m_1 I} + \frac{\omega S(1-v)m_1 I}{(1+m_1 I)^2} & 0 \\ 0 & \frac{\omega(1-v)I}{1+m_1 I} & \frac{\omega S(1-v)}{1+m_1 I} - \frac{\omega S(1-v)m_1 I}{(1+m_1 I)^2} - \delta - \beta - \mu & 0 \\ 0 & 0 & \beta & -\mu \end{pmatrix} \quad \textbf{(19)}$$

Solving the determinant of the characteristic equation (19), the following characteristic polynomial is obtained;

$$H(\lambda)=\lambda^4+a_0\lambda^3-a_1\lambda^2+a_2\lambda-a_3 \tag{20}$$

Where,

$$a_1=\left(\frac{\omega S(1-v)}{1+mI}-\frac{\omega S(1-v)m_1 I}{(1+mI)^2}-\delta-\beta-\frac{\omega(1-v)I}{1+mI}-3\mu\right)(\gamma+\mu)$$
$$+\left(2\mu+\frac{\omega(1-v)I}{1+mI}\right)\left(\frac{\omega S(1-v)}{1+mI}-\frac{\omega S(1-v)mI}{(1+mI)^2}-\delta-\beta-\mu\right)-3\mu^2$$

$$a_2=\left(\left(\frac{\omega S(1-v)}{1+mI}-\frac{\omega S(1-v)m_1 I}{(1+mI)^2}-\beta-\delta-\mu\right)\left(2\mu+\frac{\omega(1-v)I}{1+mI}\right)-3\mu^2\right)(\gamma+\mu)$$
$$-\left(\frac{\omega S(1-v)}{1+mI}-\frac{\omega S(1-v)mI}{(1+mI)^2}-\beta-\delta-\mu\right)\left(\mu^2+2\mu\frac{\omega(1-v)I}{1+mI}\right)-\frac{\omega S(1-v)mI}{(1+mI)^2}-\frac{\omega S(1-v)}{1+mI}$$

$$a_3=\left(\mu^2+2\mu\frac{\omega(1-v)I}{1+mI}\right)\left(\frac{\omega S(1-v)}{1+mI}-\frac{\omega S(1-v)m_1 I}{(1+mI)^2}-\delta-\beta-\mu\right)(\gamma+\mu)+\left(\frac{\omega S(1-v)}{1+mI}+\frac{\omega S(1-v)m_1 I}{(1+mI)^2}\right)\mu$$

Considering Routh – Hurwitz criteria which stated that all characteristics root must have negative real part. If $a_3>0, a_3a_2-a_1>0$ and $a_1(a_3a_2-a_0)-a_3^2a_0>0$, then (20) has roots with negative real parts. Hence, endemic equilibrium is locally asymptotically stable.

2.7 SENSITIVITY ANALYSIS OF R_0

Adopting [16,19] the sensitivity indices of the parameters of R_0 was calculated using the normalized forward sensitive index (NFSI), mathematically defined as:

$$\gamma_\theta^{R_0}=\frac{\partial R_0}{\partial\theta}\times\frac{\theta}{R_0}$$

Table 2.1: Parameters and Sensitivity indices of R_0 Parameter and indices of sensitivity analysis

S/N	1	2	3	4	5	6	7	8
Parameter	v	ω	Λ	α	β	μ	γ	δ
Indices	-0.25	1	1	-3.9215	-0.7792	0.005	0.0156	-0.0043

Table 2.1 presents the values of sensitivity indices for the parameter values of the model, sensitivity analysis depend on the parameters value, it can be seen from the table 2.1 and concluded that the most sensitive parameters to the basic reproductive number, R_0 of the model proposed in equation (1) are

β, v, ω, α *and* Λ, any increment in the values of β *and* v will decrease R_0 by 77.92% and 25% respectively also an increase in the value of the ω *and* Λ will decrease R_0 by 100%.

3 RESULT AND DISCUSSION FOR THE NUMERICAL SIMULATION

Table 3.1 Parameter values of the model

Parameter description	symbol	Value	Source
Recruitment rate	Λ	0.005	Assumed
Adjustment parameter	α	0.8	Nthiri
Natural death	μ	0.025	Assumed
Disease induced mortality rate	δ	0.005	Assumed
Loss of protection rate	γ	0.001	Assumed
Rate of treatment	β	0.9	Assumed
Contact rate of infection	ω	0.0002-0.2	Assumed
Probability of the success of protection against typhoid.	v	$0<v<1$	Assumed
Economic factor	m	0.8	Assumed

3.1 NUMERICAL SIMULATION:

We used the numerical software (MAPLE) to plot the graph.

From figure. 3.1, it can be seen that as the contact rate is decreases, the susceptible population is increases. At day 50, contact rate ω is 0.2 later decreases to 0.02 and also to 0.002 and further decreases to 0.0002, the susceptible population increases from 2 to 28 and also to 42 respectively. This could be as a result of awareness or improvement on sociological factors.

Figure. 3.2 depict graph of infected against time in days. It can be seen that as the contact rate is decreases, the infected population is decreases. At day 3, contact rate ω is 0.2 which further decreases to 0.0002. This shows that susceptible population avoids a physical contact with infected individuals, this could also be as a result of the impact of medial campaign on transmission of the disease.

Figure. 3.3 show the graph of sociological factor (m) against time in days, it can be seen from the graph that as the sociology factor (m) is increasing, the susceptible is reducing. At day 3, the sociology factor (m) is 0.8 which further increases to 0.2.

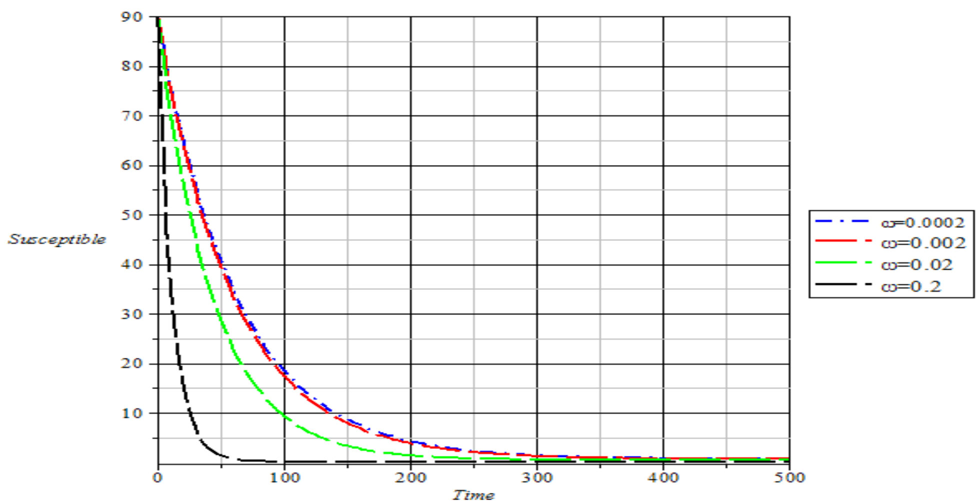

Figure 3.1: Graph of Susceptible with varying value of contact rate and other variable remain constant.

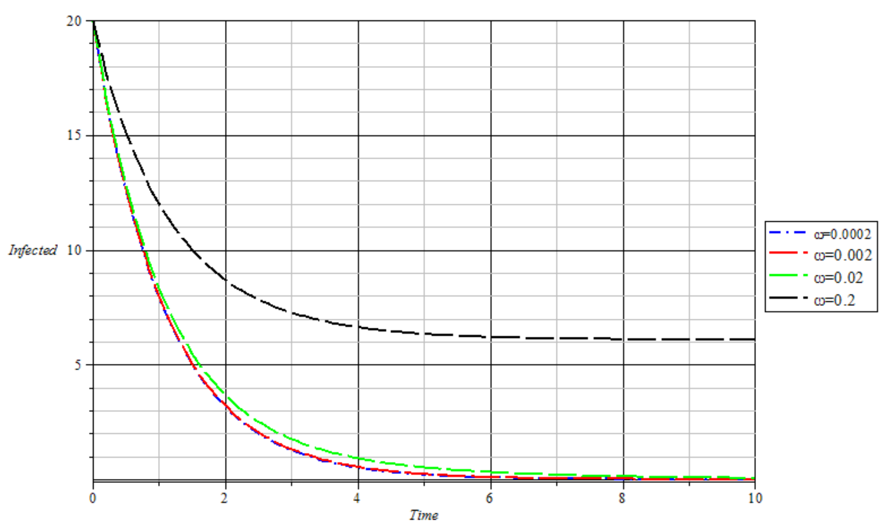

Figure 3.2 : Graph of Infected with varying value of contact rate and other variable remain constant.

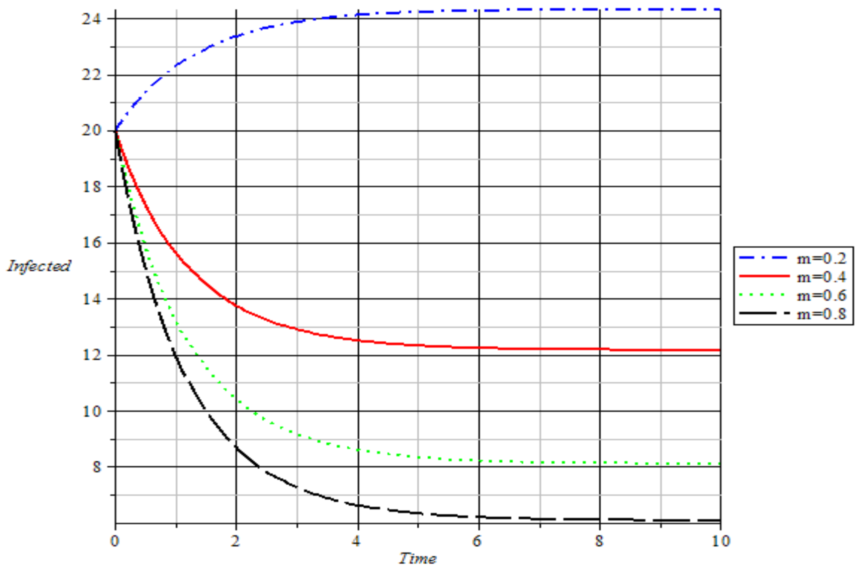

Figure 3.3: Graph of Infected with varying value of sociological factor (m) and other variable remain constant.

4 Conclusions

In this study, we discussed a dynamic model for typhoid fever with the focus on saturated incidence rate. We conclude that effective control of typhoid fever prevents rapid progression to infection especially in scarce resource setting where treatment is not readily available. We proved existence and uniqueness solution in order to ascertain the existence of the model. It was also found that whenever the basic reproduction number is less than one, that is $R_0 < 1$, the disease free equilibrium point is locally asymptotically stable and unstable whenever the basic reproduction number is greater than one, that is $R_0 > 1$. The sensitivity analysis carried out on the reproductive number (R_0) suggests that an increases in v and β has a strong impact in reducing the prevalence rate. However, the study affirmed that the provision of well sanitized environment and supply of clean water will reduce the probability of infection, hence resulting in reduction of the contact rate.

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