Mathematical modeling of typhoid fever disease incorporating unprotected humans in the spread dynamics.

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Abstract

Typhoid fever is an endemic infectious disease that can be classified as enteritis disease, *Salmonella Jyphi* is causative agent of typhoid fever in humans and it's transmitted through food and water contaminated with faeces and urine of an infected person [3]. The disease is endemic in developing countries where there is unsafe water supply, poor food hygiene and also wanting environmental sanitation . Incubation period is 7 to 14 days. General symptoms and effects of typhoid are the following headache, stomachache, Joint ache, backache, and muscle pain, loss of appetite, vomiting, diarrhea, rashes and fever. According to World Health Organization an estimated 17 million illness cases of typhoid fever were reported per year worldwide resulting to 0.6 million deaths annually[4,5]. In this study we have develop a deterministic mathematical model for spread dynamics of typhoid fever disease incorporating unprotected humans. The model result into a system of ordinary differential equations which are used for interpretations and comparison to the qualitative solutions in study the spread dynamics of typhoid fever. The model incorporating Susceptible, unprotected,

Infectious and Recovered humans which are analyzed mathematically. The existence of steady states of the mathematical model is determined. More so we have determined positivity of a solution and finally computed the basic reproductive number using next generation matrix.

Key words: Basic reproduction number, invariant region, positivity of solution, Mathematical model, Disease Free Equilibrium, Endemic equilibrium point.

Introduction

Typhoid fever is an endemic infectious disease that can be classified as enteritis diseases, and it is caused by presence of bacterium called Salmonella Typhi in the human body. The disease is a common infectious disease in human beings and it's transmitted through food and water contaminated with faeces and urine of an infected person [3]. The disease is endemic in developing countries where it continuously causes illness and death. This is brought about by unsafe water supply, poor food hygiene and also wanting environmental sanitation. Incubation period is 7 to 14 days. General symptoms and effects of typhoid are the following; headache, stomachache, Joint ache, backache, muscle pain, loss of appetite, vomiting, diarrhea, rashes and fever. According to World Health Organization an estimated 17 million illness cases of typhoid fever were reported per year worldwide resulting to 0.6 million deaths annually[4,5]. Typhoid fever is an endemic disease that is classified as an enteritis disease. The disease is caused by a bacterium called Salmonella Typhi. It is a common infectious disease in human beings and is transmitted through food and water contaminated with faeces and urine of an infected person [3]. The disease is endemic in developing countries where it continuously causes illness and death. This is contributed by unsafe water supply, poor food hygiene and wanting environmental sanitation. According to World Health Organization an estimated 17 million illness cases of typhoid fever were reported per year worldwide resulting to 0.6 million deaths annually[4,5].

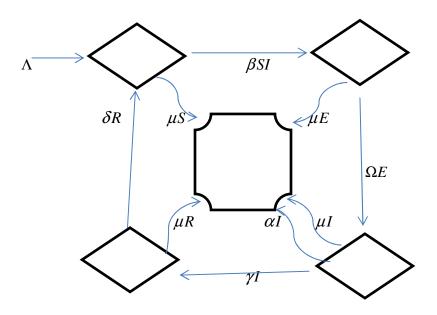
1. Description and model formulation

We formulated a deterministic model for spread dynamics of typhoid fever that considers human population at time t. The model is divided into four compartments as follows. Susceptible(S), Unprotected (E), Infective (I) and Recovered(R). The model has the following flow. $S \rightarrow E \rightarrow I \rightarrow R \rightarrow S$. We use the following parameters in our model.(i) μ is the natural death rate (ii) α is the disease induced death rate.(iii) Λ human recruitment rate (birth). (iv) β disease interaction rate .(v) Ω unprotected symptoms showing rate(vi) γ Infective recovery rate and finally(vii) δ this is the rate at which recovered humans loses temporary immunity obtained through treatment and get the disease back again. All the compartments are positive in the feasible region φ where $\{S, E, I, R\} \in \varphi \subset R_{+}^{4}$. All

the solutions are also bounded in φ such that $0 \le N \le \frac{\Lambda}{\mu}$. Thus the model is

epidemiologically well posed in the region φ .

The following flow chart shows various compartments in the model.



The model dynamics results to four differential equations as shown equation1.

$$\frac{dS}{dt} = \Lambda + \delta R - \beta SI - \mu S$$

$$\frac{dE}{dt} = \beta SI - \Omega E - \mu E$$

$$\frac{dI}{dt} = \Omega E - \gamma I - \alpha I - \mu I$$

$$\frac{dR}{dt} = \gamma I - \delta R - \mu R$$
(1)

2. Disease free equilibrium point and endemic equilibrium point

The disease free equilibrium of the model is obtained by setting

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

In absence of disease

$$E=0, I=0, R=0$$

Setting the right hand side of equations of system 1 to zero we have

$$\Lambda + \delta R - \beta SI - \mu S = 0.$$

$$\beta SI - \Omega E - \mu E = 0$$

$$\Omega E - \gamma I - \alpha I - \mu I = 0$$

$$\gamma I - \delta R - \mu R = 0$$
(2)

Using equation 1 and 2 then equating E,I and R to zero.

Making S the subject

$$\Lambda + \delta R - \beta SI - \mu S = 0.$$

$$\Lambda - \mu S = 0$$

$$S^{*} = \frac{\Lambda}{\mu}$$

Hence model has a disease free equilibrium given by

$$(S^* E^* I^* R^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right) \tag{3}$$

The basic reproductive number (R_0) which is average number of secondary infections caused by one infectious individual introduced in a completely susceptible population is obtained using next generation matrix as

$$R_0 = \frac{\beta S\Omega}{(\Omega + \mu)(\alpha + \mu + \gamma)} \text{ where at disease free equilibrium } R_0 = \frac{\beta \Lambda\Omega}{\mu(\Omega + \mu)(\alpha + \mu + \gamma)}.$$

Theorm1

If $\Omega < \frac{\mu^2 (\alpha + \mu + \gamma)}{\beta \Lambda - u (\alpha + \mu + \gamma)}$, there disease free equilibrium will be stable and typhoid disease will not have a hand in the population.

Proof

When R < 1; this means that $\frac{\beta \Lambda \Omega}{\mu(\Omega + \mu)(\alpha + \mu + \gamma)} < 1$.

Making Ω the subject, $\Omega < \frac{\mu^2(\alpha + \mu + \gamma)}{\beta \Lambda - u(\alpha + \mu + \gamma)}$

Disease free equilibrium point therefore is locally asymptotically stable if the basic reproduction number (R_0) less than one $(R_0 < 1)$ and unstable if the basic reproduction number is greater than $(R_0 > 1)$.

3. Endemic equilibrium point

Endemic equilibrium E_2^* ; disease exists. Evaluating the state variables of equations of the system 2, the endemic equilibrium points are in this form

$$E_{2}^{*} = \left\{S^{**}, E^{**}, I^{**}, R^{**}\right\}$$
Where
$$S^{**} = \frac{\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)}{\Omega\beta}$$

$$E^{**} = \frac{\left(\gamma + \alpha + \mu\right)\left(\delta + \mu\right)\left\{\Lambda\Omega\beta - \mu\left\{\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\}\right\}}{\beta\Omega\left\{\left(\delta + \mu\right)\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\} - \gamma\Omega\delta}$$

$$I^{**} = \frac{\left(\delta + \mu\right)}{\beta} \cdot \left\{\frac{\Lambda\Omega\beta - \mu\left\{\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\}}{\left\{\left(\delta + \mu\right)\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\} - \gamma\Omega\delta}$$

$$R^{**} = \frac{1}{\beta}\left\{\frac{\left\{\gamma\Lambda\Omega\beta - \gamma\mu\left\{\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\}\right\}}{\left\{\left(\delta + \mu\right)\left(\Omega + \mu\right)\left(\gamma + \alpha + \mu\right)\right\} - \gamma\Omega\delta}$$
(4)

4. Stability of endemic equilibrium

Proof: By the use of lyapunov function defined by LaSalle [1976]

$$L(S^{**}, E^{**}, I^{**}, R^{**}) = (S - S^{**} - S^{**} \ln(\frac{S^{**}}{S}) + \left(E - E^{**} - E^{**} \ln(\frac{E^{**}}{E}) + I - I^{**} - I^{**} \ln(\frac{I^{**}}{I}) + R - R^{**} - R^{**} \ln(\frac{R^{**}}{R}) + I - I^{**} + I - I^{**} \ln(\frac{I^{**}}{I}) + R - R^{**} - R^{**} \ln(\frac{R^{**}}{R}) + I - I^{**} + I - I^{*} + I - I + I - I^{*} + I - I + I + I + I + I + I + I + I +$$

computing the derivative of L along the solutions of the system is directly:

$$\frac{dL}{dt} = \left(\frac{s - s^{**}}{s}\right)\frac{dS}{dt} + \left(\frac{E - E^{**}}{E}\right)\frac{dE}{dt} + \left(\frac{I - I^{**}}{I}\right)\frac{dI}{dt} + \left(\frac{R - R^{**}}{R}\right)\frac{dR}{dt}$$
(5)

Substituting the equations of system 1 in equation 5, the equation becomes

$$\frac{dL}{dt} = \left[\left(\frac{s - s^{**}}{s} \right) \Lambda + \delta R - \left(\beta I + \mu \right) S \right] + \left[\left(\frac{E - E^{**}}{E} \right) \beta S I - \left(\Omega + \mu \right) E \right] + \left[\left(\frac{I - I^{**}}{I} \right) \Omega E - \left(\gamma + \alpha + \mu \right) I \right] + \left[\left(\frac{R - R^{**}}{R} \right) \gamma I - \left(\delta + \mu \right) R \right]$$
(6)

Expanding equation 6, it produces

$$\frac{dL}{dt} = \Lambda + \delta R - (\beta I + \mu) S - \Lambda \frac{S^{**}}{S} - \delta R \frac{S^{**}}{S} + (\beta I + \mu) S^{**} + \beta S I - (\Omega + \mu) E - \beta S I \frac{E^{**}}{E} + (\Omega + \mu) E^{**} + \Omega E - (\gamma + \alpha + \mu) I - \Omega E \frac{I^{**}}{I} + (\gamma + \alpha + \mu) I^{**} + \gamma I - (\delta + \mu) R - \gamma I \frac{R^{**}}{R} + (\delta + \mu) R^{**}$$

Eurther simplification result to

Further simplification result to

$$\frac{dL}{dt} = \left[\Lambda + \delta R + (\beta I + \mu)S^{**} + \beta SI + (\Omega + \mu)E^{**} + \Omega E + (\gamma + \alpha + \mu)I^{**} + \gamma I + (\delta + \mu)R^{**}\right]$$

$$+ \left[-(\beta I + \mu)S - \Lambda \frac{S^{**}}{S} - \delta R \frac{S^{**}}{S} - (\Omega + \mu)E - \beta SI \frac{E^{**}}{E} - (\gamma + \alpha + \mu)I - \Omega E \frac{I^{**}}{I} - (\delta + \mu)R - \gamma I \frac{R^{**}}{R}\right]$$
Or
$$\frac{dL}{dt} = \left[\Lambda + \delta R + (\beta I + \mu)S^{**} + \beta SI + (\Omega + \mu)E^{**} + \Omega E + (\gamma + \alpha + \mu)I^{**} + \gamma I + (\delta + \mu)R^{**}\right]$$

$$- \left[(\beta I + \mu)S + \Lambda \frac{S^{**}}{S} + \delta R \frac{S^{**}}{S} + (\Omega + \mu)E + \beta SI \frac{E^{**}}{E} + (\gamma + \alpha + \mu)I + \Omega E \frac{I^{**}}{I} + (\delta + \mu)R + \gamma I \frac{R^{**}}{R}\right]$$

From equation it's clear that ; $\frac{dL}{dt} = A - B$. Where *A* are the positive terms and *B* are the negative ones, such that;

$$A = \Lambda + \delta R + (\beta I + \mu) S^{**} + \beta SI + (\Omega + \mu) E^{**} + \Omega E + (\gamma + \alpha + \mu) I^{**} + \gamma I + (\delta + \mu) R^{**}$$

$$B = (\beta I + \mu) S + \Lambda \frac{S^{**}}{S} + \delta R \frac{S^{**}}{S} + (\Omega + \mu) E + \beta SI \frac{E^{**}}{E} + (\gamma + \alpha + \mu) I + \Omega E \frac{I^{**}}{I} + (\delta + \mu) R + \gamma I \frac{R^{**}}{R}$$
If $A < B$ then $\frac{dL}{dt} \le 0$

$$\frac{dL}{dt} = 0 \text{ Only if } S = S^{**}, E = E^{**}, I = I^{**}, R = R^{**}$$

The largest invariant set in $\{(S, E, I, R) \in \varphi : \frac{dL}{dt} = 0\}$ is a singleton E_2^* . Where E_2^* is the endemic equilibrium. Therefore, the endemic equilibrium is globally asymptotically stable in the invariant region φ if A < B [1,2].

Conclusion.

From our finding if $\Omega < \frac{\mu^2 (\alpha + \mu + \gamma)}{\beta \Lambda - u (\alpha + \mu + \gamma)}$, there disease equilibrium will be stable and typhoid

disease will not have a hand in the population. However if $\Omega > \frac{\mu^2(\alpha + \mu + \gamma)}{\beta \Lambda - \mu(\alpha + \mu + \gamma)}$,

then disease will be dependent on prevailing circumstances. We also performed numerical simulations to determine the changes in various compartments with time using MATLAB ode solve software. There is direct variation relationship between the unprotected and infectious compartments, therefore the unprotected humans contribute significantly to the spread dynamics of typhoid fever disease.

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