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Mathematical Model Analysis of The Spread of Typhoid Fever with Carriers and Bacteria

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Abstract:- Typhoid fever is a disease that spreads through food or water that has been contaminated by bacteria. This study aims to establish mathematical models of the spread of Typhoid Fever disease and analyze the stability of disease-free equilibrium points. Based on the next-generation matrix method obtained a global asymptotic stable equilibrium point if it has a base reproductive number of less than one. If it is the other way around, then the disease will spread.

Keywords:- *Typhoid Fever, Basic Reproductive Numbers, Disease-Free Equilibrium Point, Stability.*

I. INTRODUCTION

Typhoid fever is an infectious disease caused by a bacterial infection of salmonella Typhi. These bacteria live in the intestines of cold and hot-blooded animals but cannot multiply outside the host's digestive tract. These bacteria can live for several weeks or even a few years in water and in soils that have the appropriate humidity, pH, and temperature.

The disease can lead to death. Transmission of this disease through oral-fecal or oral urine that is one can be infected by consuming food or water that has been contaminated with feces or urine containing bacteria [1]. The incubation period is usually 10-14 days, but some are three days to 21 days depending on the number of bacteria entering [2] so that although symptoms have not yet arisen, people who already have bacteria in their body can transmit it to other individuals. [3] Moffat modeled the spread of typhoid fever by noting the carrier's effects on the spread of the disease.

In this paper, it is assumed by giving vaccines to vulnerable subpopulations, and then the individual will be immune to the disease, in other words, immediately healed (recovered). Like Moffat, Steady Mushayabasa in [2] also modeled the transmission of typhoid fever by treating the carrier subpopulation and being infected. This paper by Steady Mushayabasa [4] was developed by analyzing the model by involving vulnerable subpopulations, carriers, infected and cured. In addition, vaccine effects are also given on vulnerable subpopulations. This model by S Mushayabasa is a model developed from [5]. [2]-[5] The discussion focused on the spread of diseases with carrier effects and vaccine administration, whereas the role of Salmonella typhi bacteria in the transmission of this disease was very large.

The spread of these bacteria occurs not only from infected individuals or carriers but is also caused by bacteria resed in the environment that are directly related to vulnerable subpopulations, carriers, infected or cured. Therefore, this paper will discuss the transmission of typhoid fever disease involving carriers and bacteria.

II. THEORY

A. Differential Equation System

Given a system of differential equations

$$\dot{x} = G(t, x) \qquad (2.1)$$

with $x \in \mathbb{R}^n, t \in \mathbb{R}, \dot{x} = \frac{dx}{dt} : D \subset \mathbb{R}^{n+1} \to \mathbb{R}^n$ is a continuous function in *D*. The system of differential equations (2.1) is said to be autonomous system equations if the variable *t* otherwise implicitly, whereas if the variable *t* explicitly expressed, then the system (2.1) is said to be the system of non-autonomous differential equations. Autonomous system equations can be written in the form:

$$\dot{x} = G(x) \qquad (2.2)$$

If the system (2.2) can be written:

$$\begin{aligned} \dot{x}_1 &= a_{11}x_1 + \cdots + a_{1n}x_n \\ &\vdots \\ \dot{x}_n &= a_{n1}x_1 + \cdots + a_{nn}x_n \end{aligned} \qquad (2.3)$$

with *aij* are real numbers, then the system (2.2) is an autonomous linear differential equation system. If the system (2.2) can be written as the form (2.3), then the system (2.2) is an autonomous system of nonlinear differential equation.

B. Equilibrium and Stability

Definition 2.1 (Equilibrium Point)
Point $x^* \in \mathbb{R}^n$, is called the equilibrium point of the system of equations

$\dot{x} = G(x), x \in \mathbb{R}^n if$ it satisfies the equation $G(x^*) = 0$.

Definition 2.2 (Hyperbolic Equilibrium Point)
Point $x^* \in \mathbb{R}^n$ called the hyperbolic equilibrium point of the equation

$\dot{x} = G(x), \quad if$ it satisfies the equation $G(x^*) = 0$ and the matrix

$$\frac{\partial G}{\partial x}(x^*) = \begin{bmatrix} \frac{\partial G_1}{\partial x_1}(x^*) & \cdots & \frac{\partial G_1}{\partial x_n}(x^*) \\ \vdots & \ddots & \vdots \\ \frac{\partial G_n}{\partial x_1}(x^*) & \cdots & \frac{\partial G_n}{\partial x_n}(x^*) \end{bmatrix}$$

not having a real part eigenvalues zero.

Definition 2.3 (Stability of Equilibrium Point)
Let x^* is equilibrium point of the system $\dot{x} = G(x)$, and x^0 is starting point,

1. x^* is said to be stable if, for every $\varepsilon > 0$, there is $\delta(\varepsilon) > 0$, so every $x^0 \in \mathbb{R}^n$ with $\|x^0 - x^*\| < \delta$, the solution $\varphi(t, x^0)$ of $\dot{x} = G(x)$through the poin x^0 at $t = 0$ satisfy the inequality$\|\varphi(t, x^0) - x^*\| < \varepsilon$ for every $t \geq 0$.
2. x^* is said to be asymptotically stable, if x^* stable and there is $r > 0$, such that $\|\varphi(t, x^0) - x^*\| \to 0$ at $t \to \infty$for every x^0 that satisfied $\|x^0 - x^*\| < r$
3. x^* is said unstable, if there is $\eta > 0$ such that every $\delta > 0$ there is x^0 with $\|x^0 - x^*\| < \delta$ and $t_{x^0} > 0$ such that $\|\varphi(t_{x^0}, x^0) - x^*\| > \eta$.

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III. FORMATION OF TYPHOID FEVER MATHEMATICAL MODEL

A. Variables and Parameters

S is the proportion of vulnerable individuals (susceptible) at the time of *t*. *C* is the proportion of individual carriers at the time of *t*. *I is the p*roportion of infected individuals at the time of *t*. *R* is the proportion of individuals recovering from the disease (recovered) at the time of *t* and B is the proportion of Salmonella typhi bacteria that are in the environment at the time of *t*. The parameters of the model are shown in the table.

Table 1. Parameters

Parameters	Decription
α	Birth rate
β_1	The rate of subpopulation interaction S with I
β_2	The rate of subpopulation interaction with S with C
γ	Natural mortality rate
θ	Rate of death due to disease
κ	Subpopulation interaction rate B withsubpopulation$C$
τ_1	Rate of bacterial addition from subpopulation C
τ_2	Rate of bacterial addition from subpopulation I
μ	Rate of subpopulation healing I
σ_1	Rate of bacterial reduction due to being carried into subpopulation S
σ_2	Rate of bacterial reduction due to being carried into subpopulation C
ρ	Contact rate of subpopulations S and bacteria in the environment
ω	Rate of bacterial reduction due to environmental control programs
η	The rate at which the subpopulation Cchanges to I

B. Assumtions

1. The human population is divided into subpopulation of vulnerable individuals(S), subpopulation of infected individuals(I), subpopulation of individuals who appear cured but infected with bacteria and transmitted or called Carrier(C), and subpopulation of cured individuals(R), so$N = S + I + C + R = 1$.
2. There are births that enter the S subpopulation.
3. There are natural deaths in all subpopulations with a mortality rate γ.
4. There were deaths from the disease in subpopulation I with a mortality rate of θ.
5. Subpopulation S can contract the disease due to direct interaction with subpopulation C with the rate of interactionβ_2, dand can be infected due to direct interaction with subpopulation I with the rate of interaction β_1. In addition, Subpopulation S can contract the disease due to direct interaction with salmonella typhi bacteria in the environment at the rate of interaction.ρ.
6. Subpopulation C can be subpopulated I with the rate of η. This subpopulation is assumed to interact directly with existing batteries in the environment with the rate of interactionκ.
7. Subpopulation S before infected will be the carrier first.
8. Subpopulation can be cured with a treatment, and immune to disease.
9. It is assumed that infected and cured subpopulations do not interact directly with bacteria present in the environment, thus causing bacteria to simply get carried away in subpopulationS and C at consecutive rates of $\sigma_1$andσ_2. In addition, bacteria will be reduced due to the existence of environmental control programs with a reduced rate of ω.
10. Bacteria in the environment increase because there are bacteria emitted by subpopulations I and C with a rate of increase in bacteria of $\tau_2$andτ_1.

C. Modelling

Based on the assumptions above, the transfer diagram can be created as follows.

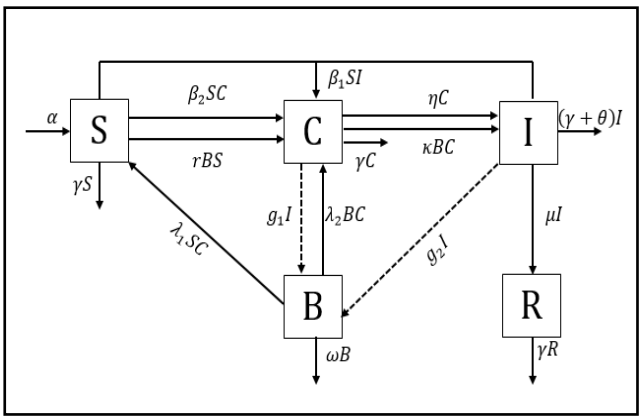

Figure 1. Diagram Transfer

Based on the transfer diagram above, the model can be arranged as follows.

1. There are births of all subpopulations that go into the S subpopulation with a α birth rate. S subpopulation is infected because there is direct contact with subpopulation I at a rate of $\beta_1 SI$ SI and direct contact with carrier subpopulation at a rate of $\beta_2 SC$. In addition, S subpopulation can be infected directly because there is contact between the subpopulation of S and bacteria in the environment with a contact rate of ρSB. In subpopulation S the mortality rate is γS. Obtained models in subpopulation S are:

$$\frac{dS}{dt} = \alpha - \beta_1 SI - \beta_2 SC - \gamma S - \rho BS$$

2. The rate of increase in the number of individuals from subpopulation C is $\beta_2 SC$. Subpopulation C increases due to direct contact of S subpopulation and bacteria by ρSB, and increases due to direct contact with subpopulation I of $\beta_1 SI$. Subpopulation C can be subpopulated I with rate η is ηC. The C subpopulation is reduced due to the interaction between bacteria and the C subpopulation which causes the C subpopulation to become infected with a reduced rate of κBC. In addition, the presence of natural mortality from this subpopulation namely γC also reduces the subpopulation of C. So obtained models on subpopulation C are:

$$\frac{dC}{dt} = \beta_1 SI + \beta_2 SC - \gamma C + \rho SB - \eta C - \kappa BC$$

3. The change of subpopulation C to population I causes an increase in subpopulation I by ηC. This subpopulation will also increase due to the interaction between bacteria and carriers as large as κBC. Subpopulation I is reduced due to natural death and death from disease at the rate (γ+θ)I. Subpopulation I will be reduced to R subpopulation (cured) along with the administration of treatment in subpopulation I at a rate of μI, so that the model obtained in subpopulation I is:

$$\frac{dI}{dt} = \kappa BC - (\gamma + \theta)I + \eta C - \mu I$$

4. The subpopulation of R increased due to the administration of treatment in subpopulation I which is μI, and there is a natural death of γR, so that the model obtained in the R subpopulation is:

$$\frac{dR}{dt} = \mu I - \gamma R$$

5. SubpopulationB will increase due to the presence of bacteria derived from subpopulation C with an increase rate of $\tau_1 C$, the addition of subpopulation I by $\tau_2 I$. Bacteria will be reduced due to environmental control at a rate of ωB, and reduced because it is brought to subpopulation S, I and C at successive rates $\sigma_1 BS$, $\sigma_2 BC$, so that models for subpopulation B are obtained:

$$\frac{dB}{dt} = \tau_1 C + \tau_2 I - \omega B - \sigma_1 BS - \sigma_2 BC$$

Obtained mathematical model of the spread of typhoid fever disease with carriers and bacteria as follow:

$$\frac{dS}{dt} = \alpha - \beta_1 SI - \beta_2 SC - \gamma S - \rho BS$$

$$\frac{dC}{dt} = \beta_1 SI + \beta_2 SC - \gamma C + \rho SB - \eta C - \kappa BC$$

$$\frac{dI}{dt} = \kappa BC - (\gamma + \theta)I + \eta C - \mu I$$

$$\frac{dR}{dt} = \mu I - \gamma R$$

$$\frac{dB}{dt} = \tau_1 C + \tau_2 I - \omega B - \sigma_1 BS - \sigma_2 BC$$

with $S(0) > 0, I(0) \geq 0, C(0) \geq 0, R(0) \geq 0, B(0) \geq 0$.

IV. ANALYSIS

D. Disease-free equilibrium points

Disease-free equilibrium points are obtained when I=0 and C=0 and B=0.

$$\frac{dS}{dt} = 0, \frac{dI}{dt} = 0, \frac{dC}{dt} = 0, \frac{dR}{dt} = 0, \frac{dB}{dt} = 0$$

$$-\beta_1 SI - \beta_2 SC - \gamma S - \rho BS = 0 \quad (1)$$

$$\beta_1 SI + \beta_2 SC - \gamma C + \rho SB - \eta C - \kappa BC = 0 \quad (2)$$

$$\kappa BC - (\gamma + \theta)I + \eta C - \mu I = 0 \quad (3)$$

$$\mu I - \gamma R = 0 \quad (4)$$

$$\tau_1 C + \tau_2 I - \omega B - \sigma_1 BS - \sigma_2 BC = 0 \quad (5)$$

If substituted values when I=0 and C=0 and B=0 in the equation (1) – (5) are obtained:

$$\alpha - \gamma S = 0 \quad (6)$$

$$\mu I - \gamma R = 0 \quad (7)$$

So from (6) we have $S = \frac{\alpha}{\gamma}$ and from (7) we have $R = 0$. thus obtained a point of bacteria-free equilbrium i $E_0 = \left(\frac{\alpha}{\gamma}, 0,0,0,0\right)$.

E. Disease-Free Equilibrium Point Stability Analysis

Known points of disease-free equilibrium are $E_0 = \left(\frac{\alpha}{\gamma}, 0,0,0,0\right)$.In the model of transmission of typhoid fever there are two infected subpopulations, namely subpopulation C and I, which will both be used to calculate the $\Re_0$.

If$\mathcal{F}$is a matrix representing the growth rate of new infections (increasing the number of infected subpopulations and carriers) and $\mathcal{V}$is a matrix representing the displacement of individuals in infected and carrier subpopulations. Note that

$$\frac{dC}{dt} = \beta_1 SI + \beta_2 SC + \rho SB - (\gamma C + \eta C + \kappa BC)$$

$$\frac{dI}{dt} = \kappa BC + \eta C - ((\gamma + \theta)I + \mu I)$$

From the equation above, it can be formed:

$$x'_i = \mathcal{F} - \mathcal{V}, \qquad (8)$$

with

$$\mathcal{F} = \begin{pmatrix} \mathcal{F}_C \\ \mathcal{F}_I \end{pmatrix} = \begin{pmatrix} \beta_1 SI + \beta_2 SC + \rho SB \\ \kappa BC + \eta C \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} \mathcal{V}_C \\ \mathcal{V}_I \end{pmatrix} = \begin{pmatrix} \gamma C + \eta C + \kappa BC \\ (\gamma + \theta)I + \mu I \end{pmatrix}$$

In the condition (8), $\mathcal{F}(E_0) = 0$ dan $\mathcal{V}(E_0) = 0$.

Next will be determined $F$and$V$:

$$F = \begin{pmatrix} \frac{\partial \mathcal{F}_C}{\partial C}(E_0) & \frac{\partial \mathcal{F}_C}{\partial I}(E_0) \\ \frac{\partial \mathcal{F}_I}{\partial C}(E_0) & \frac{\partial \mathcal{F}_I}{\partial I}(E_0) \end{pmatrix} = \begin{pmatrix} \beta_2 \frac{\alpha}{\gamma} & \beta_1 \frac{\alpha}{\gamma} \\ \eta & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \frac{\partial \mathcal{V}_C}{\partial C}(E_0) & \frac{\partial \mathcal{V}_C}{\partial I}(E_0) \\ \frac{\partial \mathcal{V}_I}{\partial C}(E_0) & \frac{\partial \mathcal{V}_I}{\partial I}(E_0) \end{pmatrix} = \begin{pmatrix} \gamma + \eta & 0 \\ 0 & \gamma + \theta + \mu \end{pmatrix}$$

Lemma 2.1

If F non negativeand V is a non singular matrix, then $\Re_0 = \rho(FV^{-1}) < 1$*if and only if the eigen value of the* $(F - V)$*have a negative value real part..*

In the model above, F is a non-negative matrix and V is$(\gamma + \eta)(\gamma + \theta + \mu) \neq 0$.

$$F - V = \begin{pmatrix} \beta_2 \frac{\alpha}{\gamma} & \beta_1 \frac{\alpha}{\gamma} \\ \eta & 0 \end{pmatrix} - \begin{pmatrix} \gamma + \eta & 0 \\ 0 & \gamma + \theta + \mu \end{pmatrix} = \begin{pmatrix} \beta_2 \frac{\alpha}{\gamma} - \gamma - \eta & \beta_1 \frac{\alpha}{\gamma} \\ \eta & -(\gamma + \theta + \mu) \end{pmatrix}$$

Eigen value of$F - V$is obtained from the equation of characteristics:

$$(\lambda - \beta_2 \frac{\alpha}{\gamma} + \gamma + \eta)(\lambda + (\gamma + \theta + \mu) - \beta_1 \frac{\alpha}{\gamma}\eta = 0$$

Because the eigen value of $F - V$have negative roots, then$\rho(FV^{-1}) < 1$.

Next we will find$\rho(FV^{-1})$, that is by first calculating the inverse of the V matrix i.e.:

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma + \eta} & 0 \\ 0 & \frac{1}{\gamma + \theta + \mu} \end{pmatrix}$$

and

$$FV^{-1} = \begin{pmatrix} \beta_2 \frac{\alpha}{\gamma} & \beta_1 \frac{\alpha}{\gamma} \\ \eta & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\gamma + \eta} & 0 \\ 0 & \frac{1}{\gamma + \theta + \mu} \end{pmatrix}$$

$$= \begin{pmatrix} \beta_2 \frac{\alpha}{\gamma(\gamma + \eta)} & \beta_1 \frac{\alpha}{\gamma(\gamma + \theta + \mu)} \\ \frac{\eta}{\gamma + \eta} & 0 \end{pmatrix}$$

It will then be calculated $\rho(FV^{-1})$. Previously, eigen values would be calculated from FV^{-1}obtained from the following characteristic equations.

$$\lambda\left(\lambda - \beta_2 \frac{\alpha}{\gamma(\gamma + \eta)}\right) - \beta_1 \frac{\alpha\eta}{\gamma(\gamma + \theta + \mu)(\gamma + \eta)} = 0$$

$$\lambda^2 - \beta_2 \frac{\alpha}{\gamma(\gamma + \eta)}\lambda - \beta_1 \frac{\alpha\eta}{\gamma(\gamma + \theta + \mu)(\gamma + \eta)} = 0$$

$$\lambda_{1,2} = \frac{\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)} \pm \sqrt{\left(\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)}\right)^2 + 4(\beta_1 \frac{\alpha\eta}{\gamma(\gamma+\theta+\mu)(\gamma+\eta)})}}{2}$$

Thus polished

$$\Re_0 = \frac{\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)} + \sqrt{\left(\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)}\right)^2 + 4(\beta_1 \frac{\alpha\eta}{\gamma(\gamma+\theta+\mu)(\gamma+\eta)})}}{2} < 1.$$

Theorem 2.1

Disease-free equilibrium point E_0*is asymptotic stable locally if*$\Re_0 < 1$..

Based on the above theorem, then$E_0 = \left(\frac{\alpha}{\gamma}, 0,0,0,0\right)$stable asymptotic local

If –A is a non singular matix, meets the*(8), and*$\Re_0 < 1$, *then the global asymptotic stable disease-free equilbrium point.*

It will then be investigated whether the matricess $-A = -(F - V)$is a non singular matrix.

Note that:

$$-A = -\begin{pmatrix} \beta_2 \frac{\alpha}{\gamma} - \gamma - \eta & \beta_1 \frac{\alpha}{\gamma} \\ \eta & -(\gamma + \theta + \mu) \end{pmatrix}$$

$$= \begin{pmatrix} -\beta_2 \frac{\alpha}{\gamma} + \gamma + \eta & -\beta_1 \frac{\alpha}{\gamma} \\ -\eta & \gamma + \theta + \mu \end{pmatrix}$$

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$$\det(-A) = \begin{vmatrix} -\beta_2\frac{\alpha}{\gamma} + \gamma + \eta & -\beta_1\frac{\alpha}{\gamma} \\ -\eta & \gamma + \theta + \mu \end{vmatrix}$$

$$= \left(-\beta_2\frac{\alpha}{\gamma} + \gamma + \eta\right)(\gamma + \theta + \mu) - \beta_1\frac{\alpha\eta}{\gamma}$$

$$= -\frac{\alpha}{\gamma}(\beta_2\gamma + \beta_2\theta + \beta_2\mu + \eta\beta_1) + \eta\gamma + \eta\theta + \eta\mu + \gamma^2 + \gamma\theta + \gamma\mu$$

$\neq 0$.

Obtained -A is a non singular matrix. So, the equilibrium point $E_0 = \left(\frac{\alpha}{\gamma}, 0,0,0,0\right)$*global asymptotic stable*.

F. Endemic Equilibrium Point

Endemic equilibrium points are obtained when$I \neq 0$, $C \neq 0$, and$B = 0$

$$\frac{dS}{dt} = 0, \frac{dI}{dt} = 0, \frac{dC}{dt} = 0, \frac{dR}{dt} = 0, \frac{dB}{dt} = 0$$

Note that:

$$\alpha - \beta_1 SI - \beta_2 SC - \gamma S = 0 \quad (9)$$

$$\beta_1 SI + \beta_2 SC - \gamma C - \eta C = 0 \quad (10)$$

$$-(\gamma + \theta)I + \eta C - \mu I = 0 \quad (11)$$

$$\mu I - \gamma R = 0 \quad (12)$$

From equation (11) we have:

$$I = \frac{\eta}{(\gamma+\theta+\mu)}C \quad (13)$$

Substitution of equations (13) to equations (10), obtained:

$$\left[\left(\beta_2 + \frac{\beta_1\eta}{(\gamma+\theta+\mu)}\right)S - (\eta + \gamma)\right]C = 0 \quad (14)$$

Because $C \neq 0$, hence the equation (14) obtained:

$$S^* = \frac{(\eta+\gamma)(\gamma+\theta+\mu)}{(\beta_2(\gamma+\theta+\mu)+\beta_1\eta)} \quad (15)$$

Substitution $S$and$I$in equation (13) dand (15) to equation (9), obtain:

$$C^* = \frac{\alpha\beta_1\eta+\alpha(\gamma+\theta+\mu)(\beta_2-\gamma(\gamma+\eta))}{(\beta_1\eta+\beta_2(\gamma+\theta+\mu))(\eta+\gamma)} \quad (16)$$

Substitution Cin equation (16) to equation (13), obtain:

$$I^* = \frac{\eta}{(\gamma+\theta+\mu)} \cdot \frac{\alpha\beta_1\eta+\alpha(\gamma+\theta+\mu)(\beta_2-\gamma(\gamma+\eta))}{(\beta_1\eta+\beta_2(\gamma+\theta+\mu))(\eta+\gamma)} \quad (17)$$

Because $R = \frac{\mu}{\gamma}I$, so from the equation (13) we have:

$$R^* = \frac{\alpha\mu\beta_1\eta^2}{\gamma(\gamma+\theta+\mu)\left(\beta_1\eta+\beta_2(\gamma+\theta+\mu)\right)(\eta+\gamma)} + \frac{\alpha\eta(\beta_2-\gamma(\gamma+\eta))}{\gamma(\beta_1\eta+\beta_2(\gamma+\theta+\mu))(\eta+\gamma)} \quad (18)$$

The endemic equilibrium$E_1 = (S^*, C^*, I^*, R^*, 0)$with $S^*, C^*, I^*,$ andR^*consecutively filled by (11) - (14). Endemic equilibrium pointE_1, exist if $\beta_2 \geq \gamma(\gamma + \eta)$.

G. Numerical Simulation

Based on the requirement $\Re_0 < 1$,*be given the following parameters:*

α	= 0,2	τ_2	= 0,00005
β_1	= 0,002	μ	= 0,5
β_2	= 0,003	σ_1	= 0,0000025
γ	= 0,1	σ_2	= 0,0006
θ	= 0,002	ρ	= 0,00001
κ	= 0,0002	ω	= 0,003
τ_1	= 0,0001	η	= 0,005

Next by taking the starting value of S = 0.5; C = 0.1; I = 0.1; R = 0.3 and B = 0.05 obtained images change the proportion of subpopulationS S, C, I, R and B against the following time.

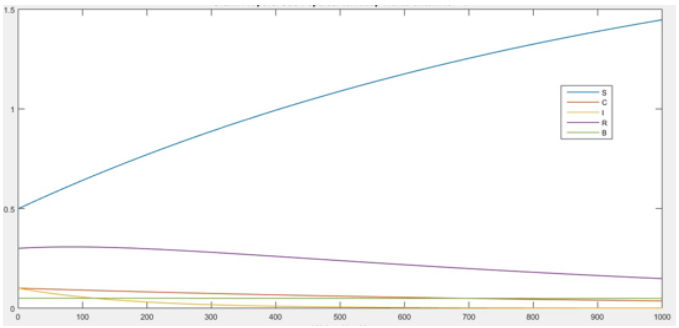

Figure 2. Graph changes the proportions of subpopulations S, C, I, R and B to *t*when $\Re_0 < 1$

Figure 2 indicates a change in the proportion of subpopulations S, C, I, R and B to time. It is seen that for a long period of time, the subpopulation of C, I, R and B will decrease and become zero, while the population of S will increase. This means that if the parameter meets the $\Re_0 < 1$, then typhoid fever will be able to be controlled. In other words, typhoid fever disease will disappear from the population if the rate of interaction of vulnerable individuals with infected individuals, birth rate and rate of change of carrier individuals become infected greater than the rate of natural death, rate of death due to disease, rate of healing and rate of interaction of vulnerable individuals with carrier individuals.

Based on the requirement $\Re_0 \geq 1$be given the following parameters:

α	= 0,5	τ_2	= 0,00005
β_1	= 0,9	μ	= 0,2
β_2	= 0,3	σ_1	= 0,0000025
γ	= 0,2	σ_2	= 0,0006
θ	= 0,6	ρ	= 0,00001
κ	= 0,0002	ω	= 0,003
τ_1	= 0,0001	η	= 0,8

Next by taking the starting value of S = 0.5; C = 0.1; I = 0.1; R = 0.3 and B = 0.05 obtained images change the proportion of subpopulationS S, C, I, R and B against the following time.

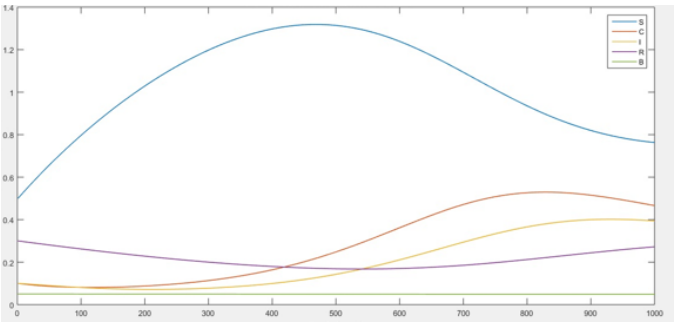

Figure 3. Graph changes the proportions of subpopulations S, C, I, R and B to *t* when$\Re_0 \geq 1$

Figure 3 shows the change in the proportion of subpopulations S, C, I, R and B to time. It is seen that for a long period of time, the S subpopulation will decrease, the subpopulation of C and I will increase for a certain period of time, the R subpopulation in a certain period of time will decrease and then will increase after a certain period of time. Subpopulation B for a certain period of time will be constant. This means that if the parameter meets the$\Re_0 \geq 1$, then typhoid fever can not be controlled.

V. CONCLUSION

1. In the model of the spread of typhoid fever, basic reproductive numbers are obtained, namely:

$$\Re_0 = \frac{\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)} + \sqrt{\left(\beta_2 \frac{\alpha}{\gamma(\gamma+\eta)}\right)^2 + 4(\beta_1 \frac{\alpha\eta}{\gamma(\gamma+\theta+\mu)(\gamma+\eta)})}}{2}$$

2. If$\Re_0 < 1$, then the point of disease-free equilibrium$E_0 = \left(\frac{\alpha}{\gamma}, 0,0,0,0\right)$,global asymptotic stable, which means that for a long period of time, typhoid fever will disappear from the population.
3. Endemic equilibrium point that is $E_1 = (S^*, C^*, I^*, R^*, 0)$, with

$$S^* = \frac{(\eta-\gamma)(\gamma+\theta+\mu)}{(\beta_2(\gamma+\theta+\mu)+\beta_1\eta)}$$

$$C^* = \frac{\alpha}{(\eta-\gamma)} + \frac{\gamma(\gamma+\theta+\mu)}{(\beta_2(\gamma+\theta+\mu)+\beta_1\eta)}$$

$$I^* = \frac{\alpha}{(\eta-\gamma)(\gamma+\theta+\mu)} - \frac{\gamma\eta}{(\beta_2(\gamma+\theta+\mu)+\beta_1\eta)}$$

$$R^* = \frac{\alpha\mu\eta}{\gamma(\eta-\gamma)(\gamma+\theta+\mu)} - \frac{\mu\eta}{(\beta_2(\gamma+\theta+\mu)+\beta_1\eta)}$$

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