Modeling the Endemic Equilibrium of Typhoid Fever

Moffat Chamuchi Nyaboe*, Mose Ong'au Fred** & Johana Kibet Sigey***

*Department of Pure and Applied Mathematics, Jomo Kenyatta University of Agriculture and Technology, Nairobi, KENYA.
E-Mail: chamuchimoffat{at}yahoo{dot}com
**Department of Pure and Applied Mathematics, Jomo Kenyatta University of Agriculture and Technology, Nairobi, KENYA.
E-Mail: moseongau{at}yahoo{dot}com
***Department of Pure and Applied Mathematics, Jomo Kenyatta University of Agriculture and Technology, Nairobi, KENYA.
E-Mail: jksigey{at}jkuat{dot}ac{dot}com

***Abstract*—**In this study, we have proposed and analyzed a nonlinear mathematical model for the spread of typhoid in a population with variable size structure. A threshold parameter is found that completely determines the stability dynamics and outcome of the disease. It is found that if the threshold parameter is less than one the disease free equilibrium is stable and the disease dies out. However, if the threshold parameter is more than one, there exists a unique endemic equilibrium that is locally asymptotically stable. Numerical simulation of the model is also performed by using fourth order Runge - Kutta method. Numerically, it has been found that the system exhibits steady state bifurcation for some parameter values. It is concluded from our analysis that endemic level of infective population increases with the increase in rate of transmission of infection due to infective among susceptible class. The results shall be presented in tabular and graphical form.

***Keywords*—**Basic Reproduction Ratio; Bifurcation; Educated; Exposed; Infective; Persistence; Steady State Equilibrium; Susceptible.

***Abbreviations*—**World Health Organization (WHO).

I. INTRODUCTION

AN infectious disease is one that can be spread or be transmitted form one host to another. Typhoid fever is an infectious disease.

The focus of our study is on infectious disease. An infectious disease that produces long-term asymptotic carriers is the Typhoid fever caused by the bacteria Salmonella typhi. Typhoid fever reached public notoriety at the beginning of the 20th century with the cases of Mr. N. the "milker" in England and Typhoid Mary in the US, Trotter et al., [3]. These individuals infected hundreds of people over the decades while they worked in the food production industry and private homes. Even today, Typhoid fever infects 21 million people and kills 200,000 worldwide every year [6, 18]. Asymptomatic carriers are believed to play an essential role in the evolution and global transmission of typhi, and their presence greatly hinders the eradication of Typhoid fever using treatment and vaccination [11, 12].

Nomenclature

b:	Rate of influx of susceptibles
$,d_4$:	Natural death rates
$,d_3$:	Death rates for I_c and I compartments, respectively, including both natural and disease-caused death
β:	Transmission coefficient for the carrier compartment I
γ:	Transmission coefficient for the symptomatically infected compartment I
α:	Rate at which carriers develop symptoms
π:	Rate of recovery
p:	Probability of a newly infected individual is asymptomatic
v:	Vaccination rate

II. LITERATURE REVIEW

The study of infectious diseases in the past has been focused mainly on their impact on the human population. Although infectious diseases are present in human populations at all times to some degree, the effects of epidemics are the most noticeable and spectacular, WHO [8]. The New World was considered virtually disease-free. Between 1918 and 1921, the Soviet Union incurred about 25 million cases of typhus with a death rate of approximately 10 per cent. From Daniel Bernoulli's analysis of smallpox (1760), mathematical modeling on epidemic models of infectious diseases has been extended greatly in recent year. He was, by a long way, the first to express the proportion of susceptible individuals of an endemic infection in terms of the force of infection (the annual rate of acquiring an infection) and life expectancy. Hundreds of mathematical models have proven particularly powerful in the study of the effects of bacterial, parasitic and viral pathogens Zhao et al., [7]. In the past much understanding has been gained through the use of relatively simple models capturing only the most critical biological mechanisms. One of the most important reasons that developed countries have become as productive as they are today is that the population remains healthy and disease free. This essential task is performed by each country's health department and is carried out by individuals known as epidemiologists Korobeinikov & Maini [2]. Without their efforts and their coordination with others in the medical field, it would be very difficult if not impossible to obtain current information regarding important diseases, methods of transmission, methods of control, and the like Lasalle [5]. Furthermore, information on the incidence or prevalence of diseases and statistics on morbidity and mortality rates, all of which are essential to physicians and other medical personnel to help control and understand diseases, would not be available without the efforts of the epidemiologists [Black, 2004].

Ronald Ross (May 13, 1857September 16, 1932) was an English physician. Ross was a pioneer in developing mathematical models for the study of epidemiology.

Anderson Gray McKendrick (September 8, 1876 - May 30, 1943) Scottish physician and epidemiologist was another pioneer in the use of mathematical methods in epidemiology. McKendrick's career as a mathematical epidemiologist began in India. In 1914 he published a paper in which he gave equations for the pure birth process and a particular birth-death process. After his return to Scotland he collaborated with Kermack on a notable series of papers. The first paper (1927) gave the differential equations for a deterministic general epidemic. It is important to mention that modeling is very crucial in epidemiology since in most cases we cannot do experiments. It is possible to mathematically model the progress of most infectious diseases to discover the likely outcome of an epidemic or to help manage them by different control programs. In the early 20th century, mathematical methods were introduced into epidemiology by Ronald Ross [Ross, 1916], Anderson, Gray, McKendrick [McKendrick and Kermack, 1927] and others.

The methods to model the typhoid fever dynamic have to take into consideration its aims and stage of development which finally divide the models into the following categories: Typhoid fever Compartmental and Distributional Models. From the total population, you can take at least two big groups of populations (Variables), a susceptible population and the population with the typhoid infection, in these models each one of these subpopulations are called compartments of susceptible and infected respectively. Changes in population involved in a typhoid transmission model can take place either in discrete steps or as a smooth continuous process. In discrete models, differential equations reflect the change over the whole time step, whereas in continuous models, differential equations are developed to explore changes in one variable with a diminishingly small change in another variable [9, 15].

In recent decades, there have been several investigations of infectious diseases using deterministic mathematical models with or without demographic change.

In particular, Kalajdzievska [9], has studied an infectious disease model with population-dependent death rate using computer simulation.

Guo [4] analyzed an infectious disease model with logistic population growth.

Roumagnac et al., [6] have studied a few models for infectious diseases using various kinds of demographics.

Chamuchi et al., [1] has discussed an epidemic model in which the carrier population is assumed to be constant. But in general the size of the carrier population varies and depends on the natural conditions of the environment as well as on various discharges into it by the human population.

2.1. Statement of the Problem

Typhoid fever continues to be a major public health problem in the in Kenya more specifically in Kisii County. Antibiotic therapy has been the main stay of treating typhoid fever for decades. The emergence of drug-resistant typhoid strain in the last two decades has been a major problem in tackling this scourge. A mathematical model for investigating the impact of drug resistance on the transmission dynamics of typhoid fever is developed. The reproductive number for the model has been computed. Numerical results in this study suggest that when a typhoid outbreak occurs with more drug-sensitive cases than drug-resistant cases, then it may take 10–15 months for symptomatic drug-resistant cases to outnumber all typhoid cases, and it may take an average of 15–20months for non-symptomatic drug-resistant cases to outnumber all drug-sensitive cases.

2.2. Motivation of this Research

The motivations of this research are to:

- Model the transmission dynamics of typhoid for Computational mathematical researchers.

- Optimize a model for reducing typhoid cases in Kenya since it is a developing country without increasing public spending.
- Study the magnitude of public protection since it greatly influences the total number of cases avoided and the value of public treatment cost savings.

2.3. Research Objectives

2.3.1. General Objectives

- It is possible to mathematically model the progress of most infectious diseases to discover the likely outcome of an epidemic or to help manage them by different control programs.

2.3.2. Specific Objectives

The objectives of this research are to:

- Propose a mathematical model for the typhoid fever on transmission dynamics.
- Assess the impact of typhoid fever in Kenya.
- Validate the effect of typhoid fever model with data from Kenya.

2.4. Contributions of this Article

- Change the perception that mathematical models for the typhoid fever which were considered merely speculative and imprecise, but in this study, with the inclusion of explicit elements of biology and behavior in the models, it is possible that they lead to a deeper understanding of the future spread of disease.
- Bring to the attention of other researchers that even though the actual data needed for the models might not be accurate or even available, this modeling is still vital in investigating how changes in the various assumptions and parameter values affect the course of the epidemic. Mathematical modeling helps in the set of conditions by which the extinction of susceptible population is reached.
- Show that the improvements in data capture produce major challenges in developing frameworks capable of utilizing this data to predict the complex patterns of evolution of infectious diseases in increasingly dense and interconnected human populations. This mathematical model and its computer simulation is useful in analyzing the spread and control of typhoid as an infectious diseases.

III. METHODOLOGY

Mathematical models have been used for centuries to develop a better understanding of systems in order to control or optimize results. A wide range of applications include everything from radar development to production rates within factories to the spread of disease.

Epidemic models are mathematical models concerned with the spread of infectious diseases.

Models are created to study treatment and infection rates in order to optimize our ability to predict quarantine and control disease.

We assume that all parameters in the model are nonnegative and that $b > 0; d_i > 0; i =$

Governing Equations

3.1. A General Epidemic Model with Asymptomatic Carriers

We formulate an compartmental model where and R represent the susceptible, carrier, symptomatically infectious and removed classes, respectively. A susceptible individual can be infected through direct contact with an infected individual can become a carrier with probability p, or shows disease symptoms with probability $1 - p$. We assume that the rate of transmission for carriers is higher than the rate ¥ of symptomatically infected individuals due to the fact that they are more likely to be unaware of their condition, and therefore continue with their regular behaviors.

Based on our assumptions and the transfer diagram, we can derive the following system of differential equations that govern our model [5, 13].

$$\left.\begin{array}{rl} & S(\beta I \quad \gamma I)- \\ & pS(\beta I \quad \gamma I)-(d \quad \alpha)I \\ =(1 \quad p)S(\beta I & \gamma I)-(d \quad \pi)I \end{array}\right\} \qquad (1)$$

We note that disease carriage is different from disease latency in that individuals in the carrier state are infectious while those in the latent period are not. This model is thus different from the traditional SEIR models that incorporate disease latency. In the special case when $\beta = 0$, the I_c class is not infectious and can be considered as latent, and our model becomes a modified SEIR model in which new infections can be either latent or infectious. While both $I_c$and$I$ are infectious, our model is different from the differential infectivity models considered since new infections from I_c or I may enter either compartment with certain probability. In this model we incorporate demography and disease-caused death. We also allow carriers to become symptomatic over their life time. Our model is different from the carrier model in that we allow new infections to be either symptomatic or asymptomatic with certain probabilities.

3.2. Feasible Region and Equilibria

From (1) we have that $(d \quad \theta)S$, and thus $(t)\frac{}{(\quad \theta)}$ along each solution. Also from (1) we see that

$\bar{d}N$

Where $\bar{d}= min\{d_1, d_2, d_3, d_4\}$. Therefore $\lim sup_{t\to\infty} N(t)$ $b/\bar{d}$. The equation for R can be omitted in our analysis as R does not appear in the other equations. This shows that the model can be studied in the feasible region.

$$\{(S \quad I) \qquad \text{———} \qquad \bar{d}\}$$

It can be verified that Γ is positively invariant with respect to (1). Once the dynamics of (S, I_c, I) are understood, those of Rcan then be determined from the equation R'

R. The first step in our analysis is to find equilibria (S^*, I_c^*, I^*) from equations

$$\left.\begin{array}{r} (\beta I_c^* + \gamma I^*) \\ (\beta I_c^* + \gamma I^*) - (d_2 + \alpha) I_c^* \\ (1 - p) S^* (\beta \qquad) \quad (d \quad \pi) I \end{array}\right\} \qquad (2)$$

Model (1) always has a disease-free equilibrium $\left(\text{——}\right), 0$. An endemic equilibrium $(S \quad)$ satisfies $(S \ , I \quad)$. From the equilibrium equation we can show that a unique exists with

$$\frac{(d_3 + \pi)(d_2 + \alpha)}{(d_2 + \alpha)\gamma + p(\pi - d_2\gamma)}$$

For P^* to exist in the feasible region Γ, it is necessary and sufficient that $0 < S^* \le \frac{b}{d + \theta}$ or equivalently, —— ≥ 1. Define

$$\text{———} \quad \text{——} \frac{+(d \quad \alpha)\gamma \quad p(\pi \quad \gamma)}{(d \quad \pi)(d \quad \alpha)} \qquad (3)$$

Then R_0 is a threshold parameter that determines the number of equilibria. We will show in Section 3 that $R_0$is the basic reproduction number.

If $R_0 \le 1$ then P_0 is the only equilibrium in Γ; if $R_0 > 1$, then there are two equilibria, P_0 and a unique endemic equilibrium $P*$. Rewrite in (3) as

$$[(1 \quad p)\gamma \text{——} \quad p(\beta \text{———} \qquad \text{———} \quad \text{——})] \text{——} \qquad (4)$$

In the following, we show that R_0 is the basic reproduction number, namely, it represents the average number of secondary infections caused by a single infective in an entirely susceptible population during its entire infectious period.

When a single infective is introduced into the population, with probability $1 - p$ it is a non-carrier, hence makes γeffective contacts per unit time. This is multiplied by the average infectious period $\frac{1}{d + \pi}$ for non-carriers; with probability p the infective is a carrier, and hence makes effective contacts per unit time during the average period —— it remains a carrier. This number should be augmented by the number of infections —— caused by this infective after it becomes a non-carrier, with probability to survive the carrier stage. Therefore, the expression in the big square brackets in (4) is the per capita average number of secondary infections. This number multiplied by the number of susceptibles at the disease-free equilibrium, $\frac{b}{d + \theta}$ gives R_0.

The carriers in our system can have a great effect on R_0. The parameters β, α, and $p$are all related to the carrier class and all appear in the basic reproductive number. It is straightforward from (3) that R_0 increases as β increases. This agrees with the intuition that higher transmissibility increases the basic reproduction number.

To see the effect of p on R_0 we note

$$\text{——}=[-\text{——} \quad \text{——} \quad \text{————}]\text{——}$$

$$\frac{b(d \quad \alpha)}{\text{——}}\beta[-\text{——} \ ]$$

and thus

$$\text{——} \qquad \text{if} \qquad \text{——} \qquad (5)$$

We see that a greater probability to develop carriage will increase the basic reproduction number under the condition (5).

We can also analyze the effect of diagnosis rate α on R_0. Straightforward computation gives

$$\text{——} \quad p[-\frac{}{(d \quad \alpha)^2} + \frac{}{\pi}\frac{}{(d \quad \alpha)^2}] \text{———}$$

$$\frac{}{(d \quad \alpha)^2(d \quad \theta)}\beta[-\text{———}\gamma]$$

and thus —— if the same condition (5) holds.

From these analysis we see that parameter p and α have opposite effects on R_0: while a higher probability p of carriage increases R_0, a higher diagnosis rate α of carriage decreases R_0. The latter aspect can be a very useful control strategy and will be further explored in Section 6 through numerical simulations. Biologically, condition (5) only requires that the transmissibility β of carriers is not too small compared to that of the symptomatically infected. This is likely to hold for many diseases with carriers since carriers can unknowingly infect many people.

3.3. Stability of the Disease-Free Equilibrium

To examine the local stability of the disease-free equilibrium P_0 we evaluate the Jacobian matrix at

$$\left(\text{———}, 0, \right)$$

$$J(P_0)$$

$$\begin{bmatrix} \text{——} & \gamma(\text{———}) \\ b\beta\left(\text{———}\right) - & \text{——} \\ (1 \quad p)\beta \text{——} & (1 \quad p)\gamma\left(\text{———}\right) - \end{bmatrix}$$

We have the following stability result that shows R_0 is a sharp threshold.

P_0 is locally asymptotically stable if R_0 <1 and is unstable if R_0 >1.

One eigenvalue of $J(P_0)$ is $\lambda_1 = -(d_1 + \theta) < 0$. The other two eigenvalues λ_2, λ_3 are eigenvalues of the 2 matrix

$$\begin{bmatrix} b\beta\left(\text{———}\right) & \text{——} \\ (1 \quad p)\beta \text{——} & (1 \quad p)\gamma\left(\text{———}\right) \end{bmatrix}$$

We want to show, when R_0 <1, that the Routh-Hurwitz conditions hold, namely, tr(*A*) <0 and det (*A*) >0. Simple calculations show that

$$tr(A) = (d \quad \alpha)\left[\frac{b\beta\left(\quad\right)}{(d \quad \alpha)} \quad 1\right]$$

$$(d \quad \pi)\left[\frac{(1 \quad p)\gamma}{\quad} \quad 1\right]$$

Using our assumption that $\left[\frac{}{(\quad \alpha)}\right.$ $\frac{}{(d \quad \alpha)(d \quad \pi)}$ $\frac{(1-p)\gamma}{}$ $\left. 1\right]$ we have

$\frac{}{}$ and $\frac{(1 \quad p)\gamma}{}$

Therefore

$\frac{}{}$ 0 and $\frac{(1 \quad p)\gamma}{}$

This shows that $tr(A)$ Now we calculate

$$\mathrm{t}(A) = \left[p\beta \frac{}{} - (d \quad \alpha)\right]\left[(1-p)\gamma \frac{}{} \quad (d \quad \pi)\right] - \left[(1 \quad p\beta)\frac{}{} \quad \alpha\right]p\gamma \frac{}{}$$

$$(d \quad \alpha)(d \quad \pi) - (d \quad \pi)p\beta \frac{}{}$$

$$(1 \quad p)\gamma \frac{}{}$$

$$(d \quad \alpha)(d \quad \pi)[1 \quad]$$

Therefore, $det(A) > 0$ if and only if $R_0 < 1$. This proves the proposition.

P_0 is globally asymptotically stable in the feasible region if $R_0 \leq 1$.

To prove the global asymptotic stability of P_0 we use the method of Lyapunov functions. Define

$$\left[\frac{}{} \quad \frac{}{(d \quad \pi)(d \quad \alpha)} \quad \frac{}{} I\right]$$

Then

$$\frac{}{} \quad \left[\frac{}{} \quad \frac{}{(d \quad \pi)(d \quad \alpha)}\right] I \quad \frac{}{}$$

$$\left[\frac{}{} \quad \frac{}{(d \quad \pi)(d \quad \alpha)} \quad \frac{(1 \quad p)\gamma}{}\right] S(\beta \quad \gamma I)$$

$$(\beta \quad \gamma I)$$

$$\left[\frac{}{} \quad 1\right](\beta \quad \gamma I).$$

Using $\frac{}{}$ we know.

$$\frac{}{} \quad (R \quad 1)(\beta \quad \gamma I)$$

So $\frac{}{}$ if 1. Furthermore, $\frac{}{}$

or and $\frac{}{}$.

Therefore the largest invariant set in the closure of where $\frac{}{}$ is the singleton $\{P_0\}$. By LaSalle's Invariance Principle [10], P_0 is globally asymptotically stable in Γ, completing the proof.

3.4. Stability of the Endemic Equilibrium

If $P_0 > 1$, then P^* is globally asymptotically stable with respect to the interior of Γ.

To study the global stability of the endemic equilibrium, we make use of a Lyapunov function V of form

$$V(S, I_c, I) = x_1(S - S^* \ln S) + x_2(I_c - I_c^* \ln I_c) \quad (6)$$

$$(I \quad I)$$

Where $x_1, x_2, x_3 > 0$ are constants to be specified. Note that V has a global minimum at $P^*(S^*, I_c^*, I^*)$ and $V(S, I_c, I) - V(P^*)$ is positive definite. We show that suitable constants x can be chosen such that the Lyapunov derivative of V is negative definite with respect to P^*. Direct calculation and applying the identity $b = d_1 S^* + \theta S^* + \beta I_c^* + \gamma I_c^* S^*$ lead to

$$\frac{}{} \quad \left(S \quad \frac{}{} \right) \quad \left(I \quad \frac{}{} \right) \quad (I \quad \frac{}{} ')$$

$$\left[b \quad (d \quad \theta)S \quad (\beta \quad \gamma I)S \quad \frac{}{} \quad (d \quad \theta)S \right.$$

$$\left. (\beta \quad \gamma I)S^*\right]$$

$$\left[(1 \quad p)(\beta \quad \gamma I)S \quad (d \quad \alpha)I \right.$$

$$\left. (1 \quad p)\frac{}{} \quad (1 \quad p)\frac{}{} \quad (d \right.$$

$$\left. \alpha)I_c^*\right]$$

$$\left[p(\beta \quad \gamma I)S \quad (d \quad \pi)I \right.$$

$$\left. \frac{}{} \quad \frac{}{} \quad (d \quad \pi)I^*\right]$$

$$\left[x_1(d \quad \theta)S^*(2 \quad \frac{}{} \quad \frac{}{})\right]$$

$$[x_1(\beta \quad) \quad (d \quad \alpha)I$$

$$(d \quad \pi)I^*]$$

$$\left[x \frac{(\beta \quad)S}{} \quad (1 \quad p)\beta S \right.$$

$$(1 \quad p)\frac{}{} \quad \left(p\frac{}{}\right)$$

$$\left. \frac{}{}\right].$$

Positive constants x_1, x_2, and x_3 are chosen as

$$\frac{(d_3 + \pi)(\beta}{(d \quad \alpha)(d \quad \pi)} \quad (7)$$

$$\overline{(d_3 + \pi)}$$

It can be verified that they satisfy relations

$$\left.\begin{array}{l} (1 - p) \\ (d \quad \pi) \\ (d \quad \alpha) \end{array}\right\} \quad (8)$$

We re-group terms in $\frac{}{}$ such that $\frac{}{}$, where

$$(d \quad \theta)S^*\left(2 \quad \frac{}{} \quad \frac{}{}\right),$$

$$(\beta \quad) \quad (d \quad \alpha)I \quad (d \quad \pi)I$$

$$\frac{(\beta \quad)S}{} \quad (1 \quad p)\beta S$$

$$\frac{(1 \quad p)\gamma}{} \quad \frac{}{} \quad \frac{}{}$$

We see that $V_1 \leq 0$ from the inequality $x + \frac{1}{x} \geq 2$ for all $x > 0$ and that $V_1 = 0$ if and only if $S = S^*$. We are left to show that $V_2 + V_3 \leq 0$. We begin by examining V_2. Using the values for x_1, x_2, and x_3 in (7), relations in (8), and the equilibrium relation

$$(pd_2 + \alpha)I_c^* = (1-p)(d_3 + \pi)I^*, \tag{9}$$

we can rewrite V_2 as

$$\frac{2(1 \quad p)x}{\frac{3(1 \quad p)\alpha}{(pd_2 + \alpha)}} \tag{10}$$

Similarly, we can rewrite V_3 as

$$\left[- \quad (1 \quad p)\beta \quad \frac{(1 \quad p)x}{}\right] \left[- \quad \frac{}{}\right] \left[- \quad \frac{(1 \quad p)\gamma}{} \quad \frac{}{}\right] \frac{(1 \quad p)x}{} \quad \frac{}{}\Bigg]$$

Where

$$\frac{(1 \quad p)\alpha}{(pd_2+\alpha)(1-p)x_2}, \qquad \frac{(1 \quad p)\beta}{(pd_2+\alpha)(1-p)x}$$

Write $V_3 = V_a + V_b + V_c + V_d$, with each term representing the expression enclosed in a pair of big square brackets. We will estimate each term in V_3 by applying the inequality

$$\frac{}{} \quad (a \quad)^{1/n} \text{ for } \quad 0.$$

We obtain

$$(1 \quad p)\beta \quad \frac{(1 \quad p)x}{} \quad 2\sqrt{(x_2(1 \quad p))^2(\beta \quad)} \quad 2(1 \quad p)\beta \tag{11}$$

And

$$\frac{}{} \quad 2\sqrt{(x \ p)^2(\gamma I \quad)} \tag{12}$$

Similarly,

$$\frac{(1 \quad p)\gamma}{} \quad \frac{}{} \quad \frac{(1 \quad p)x}{} \quad 3[(x_2(1 \quad p)) \quad (\gamma \quad)^2]^- \quad \frac{3(1 \quad p)\alpha}{(p \quad \alpha)} \tag{13}$$

And

$$-(1 \quad y)\frac{(1 \quad p)\gamma}{} \quad \frac{}{} \quad (1 \quad y)\frac{(1 \quad p)\gamma}{} \quad \frac{}{} \quad 4[(x_2(1 \quad p))^2(p \quad)^2(1 \quad y) \quad]^- \tag{14}$$

Therefore, (11) - (14) imply

$$-2(1-p)x \ \beta \quad \frac{3(1 \quad p)\alpha}{(pd_2 + \alpha)} \tag{15}$$

It follows from (10) and (15) that $V_2 + V_3 \leq 0$ and thus — 0. Furthermore,

— 0 if and only if $V_1 = 0$ and $V_2 + V_3 = 0$. Using (10) - (15), we can show that

— $\Leftrightarrow (S, I_c, I) = (S, I_c^*, I^*)$, and thus $\frac{dV}{}$ is negative definite with respect to P^*. The global stability of P^* follows from the classical stability theorem of Lyapunov.

IV. NUMERICAL SIMULATION

To substantiate the above analytical findings, the model is studied numerically. The system of differential equation is integrated using fourth order Runge-Kutta method under the following set of compatible parameters, which satisfy the stability conditions.

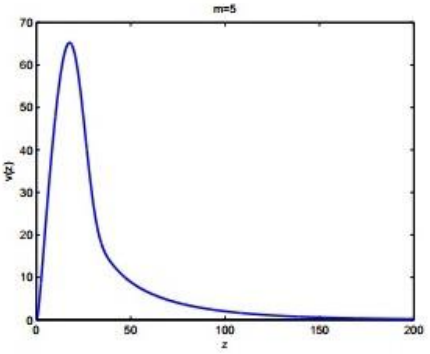

Figure 2: The figure shows variation of susceptible population with time for different values of transmission coefficient of infection due to exposed class.

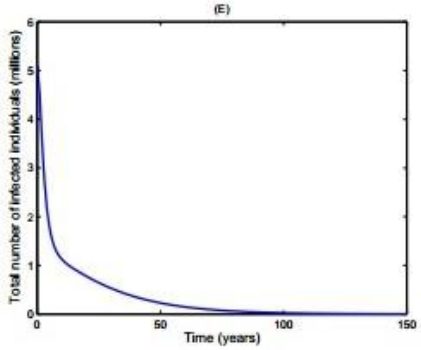

Figure 3: The figure shows variation of infective population with time and it is found that both the infective population decreases with the increase in time and then become constant at their equilibrium level.

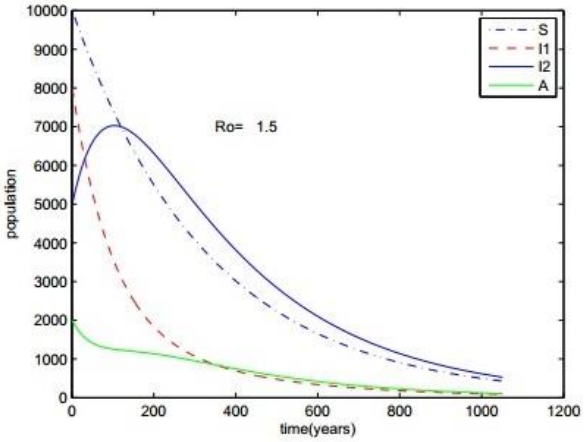

Figure 4: From the graphs it is found that if $R_0 > 1$, there exists a unique endemic equilibrium which is locally asymptotically stable.

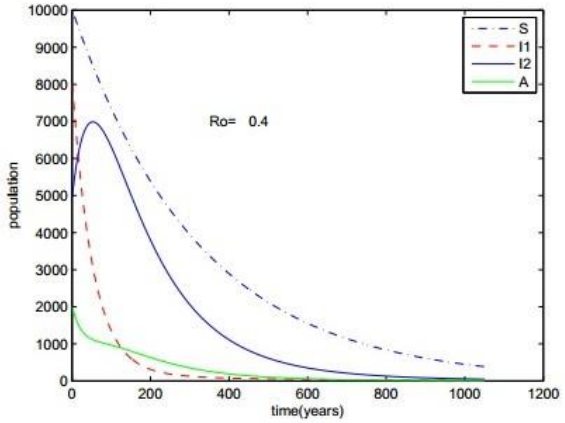

Figure 5: It is found that if R_0<1 the disease free equilibrium is stable and the disease dies out.

V. CONCLUSIONS

It is concluded that endemic level of infective population increases with the increase in rate of transmission of infection from infective to susceptible class, which can further be enhanced if transmission of infection occurs from latent hosts during incubation period. However, for a particular value of disease transmission coefficient exposed and infective population die out. Variation of infective equilibrium size of the population with basic reproduction number is determined and it is found that endemic infective level first rises with the increase in basic reproduction ratio and then becomes constant. In this study model for typhoid is a carrier-dependent disease caused by direct contact of susceptibles with infectives as well as by carriers is proposed and analyzed. It is assumed that both the human and the carrier populations are growing logistically. The density of carrier population is further assumed to increase with the increase in the cumulative density of discharges by the human population into the environment. Equilibrium analysis is presented for both the cases and it is seen that the local stability of the nontrivial equilibria in both the cases is guaranteed only under certain conditions. By computer simulation it is shown that under local stability conditions, the nontrivial equilibrium appears to be globally stable in both the cases. It is concluded from the analysis that if the growth of carrier population caused by conducive household discharges increases, the spread of the typhoid fever rate. Also when the growth rate of the human population increases due to demographic changes, the infectious disease spreads even further and becomes more endemic.

REFERENCES

[1] N.M. Chamuchi, J.K. Sigey, J.A. Okello & J.M. Okwoyo (2014), "SIICR Model and Simulation of the Effects of Carriers on the Transmission Dynamics of Typhoid Fever in KISII Town Kenya", *The SIJ Transactions on Computer Science Engineering & its Applications (CSEA), The Standard International Journals*, Vol. 2, No. 3, Pp. 109–116.

[2] A. Korobeinikov & P.K. Maini (2004), "A Lyaponov Function and Global Properties for SIR and SEIR Epedimiological Models with Nonlinear Incidence", *Mathematical Biosciences and Engineering*, Vol. 1, No. 1, Pp. 57–60.

[3] C.L. Trotter, N.J. Gay & W.J. Edmunds (2005), "Dynamic Models of Meningococcal Carriage, Disease, and the Impact of Serogroup C Conjugate Vaccination", *American Journal of Epidemiology*, Vol. 162, No. 1, Pp. 89–100.

[4] H. Guo (2005), "Global Dynamics of a Mathematical Model of Tuberculosis", *Canadian Applied Math Quarterly*, Vol. 13, Pp. 313–323.

[5] J.P. LaSalle (1976), "The Stability of Dynamical Systems", *CBMS-NSF Regional Conference Series in Applied Mathematics*, *SIAM*, Philadelphia,.

[6] P. Roumagnac, F.X. Weill, C. Dolecek, S. Baker, S. Brisse, N.T. Chinh, T.A. Hong Le, C.J. Acosta, J. Farrar, G. Dougan & M. Achtman (2006), "Evolutionary History of Salmonella Typhi", *Science*, Vol. 314, No. 5803, Pp. 1301– 1304.

[7] S. Zhao, Z. Xu & Y. Lu (2000), "A Mathematical Model of Hepatitis B Virus Transmission and its Application for Vaccination Strategy in China", *International Journal of Epidemiology*, Vol. 29, Pp. 744–752.

[8] WHO (2008), "Fact Sheet on Hepatitis B", Available from: http://www.who.int/mediacentre/factsheets/fs204/en/index.html

[9] D. Kalajdzievska (2006), "Modeling the Effects of Carriers on the Transmission Dynamics of Infectious Diseases", *M.Sc. Thesis*, *University of Alberta*.

Chamuchi Nyaboe Moffat. He was born on 23rd March, 1982 in Cherenganyi, Kitale town, Western part of Kenya. He holds a Bachelor of Education Science (option in Mathematics and chemistry); specialized in Mathematics from Kenyatta University and a Master of science degree in Applied Mathematics from Jomo Kenyatta University of Agriculture and Technology, Kenya and he is currently pursuing a Doctor of philosophy degree in Applied Mathematics in Jomo Kenyatta University of Agriculture and Technology, Kenya. He has much interest in the study of the Mathematical Modeling of infectious diseases and their resistance to humans and their respective applications to Mathematics and engineering.

Affiliation: Jomo Kenyatta University of Agriculture and Technology, (JKUAT), Kenya.
Tel: +254-721971936
He has much interest in the study of modeling and Epidemiology and their respective applications to engineering.

Mose Ong'au Fred. Fred was born on 1st November, 1980 in Kisii county, Nyanza province, Kenya. He holds a Bachelor of Education Second Honours (Upper Division) degree in Mathematics & Physics from Kenyatta University and a Master of science degree in Applied Mathematics from Jomo Kenyatta University of Agriculture and Technology, Kenya and he is currently pursuing a Doctor of philosophy degree in Applied Mathematics in Jomo Kenyatta University of Agriculture and Technology, Kenya.
Affiliation: Jomo Kenyatta University of Agriculture and Technology, (JKUAT), Kenya.
Tel: +254-721366652
He has much interest in the study of modeling and Epidemiology and their respective applications to engineering.

Johana Kibet Sigey. Sigey holds a Bachelor of Science degree in mathematics and computer science first class honors from Jomo Kenyatta University of Agriculture and Technology, Kenya, Master of Science degree in Applied Mathematics from Kenyatta University and a PhD in applied mathematics from Jomo Kenyatta University of Agriculture and Technology, Kenya.
Affiliation: Jomo Kenyatta University of Agriculture and Technology, (JKUAT), Kenya.
Tel: +254-722795482
He is currently the director, Jkuat, Kisii CBD. He has been the substantive chairman - department of pure and applied mathematics – Jkuat (January 2007 to July- 2012). He holds the rank of senior lecturer, in applied mathematics pure and applied Mathematics department – Jkuat since November 2009 to date. He has published 12 papers on heat transfer in respected journals.
Teaching experience: 2000 to date- postgraduate programmes: (jkuat) Master of Science in applied mathematics Units: complex analysis I and ii, numerical analysis, fluid mechanics, ordinary differential equations, partial differentials equations and Riemannian geometry. Supervision of postgraduate students: Doctor of philosophy: thesis (6 completed, 9 ongoing) and Masters of science in Applied Mathematics: (12 completed, 8 ongoing).

