

Modelling The Role of Rehydration And Antibiotic Treatment on Reduction of Cholera Mortality

Owade Kennedy Jakob¹, Akinyi Colleta², Owuor George Lawi³

^{1,2,3}Department of Mathematics Masinde Muliro University of Science and Technology

P.O Box ,Kakamega (Kenya)

Corresponding Author: Owade Kennedy Jakob¹

Abstract: Cholera is an infection of the small intestine of humans caused by a gram negative bacterium called *Vibrio cholerae*. It is spread through eating food or drinking water contaminated with faeces from an infected person. It causes rapid dehydration and general body imbalance, and can lead to death since untreated individuals suffer severely from diarrhoea and vomiting. In this paper we formulate a mathematical model to assess the role of rehydration and antibiotic treatment on reduction of cholera mortality. All solutions in our model are positive and bounded hence well posed. The stability analysis of the model has been done. Numerical simulation shows that rehydration and administration of antibiotics play a major role in reducing cholera deaths.

Keywords: Cholera Disease, Role of Rehydration and Antibiotic treatment

Date of Submission: 05-01-20178

Date of acceptance: 16-01-2018

I. Introduction

Cholera is an infection of small intestine caused by a gram-negative bacterium called *Vibrio cholerae*. The dynamics of cholera involve multiple interactions between the human host, the pathogen, and the environment, which contribute to both human to human and indirect environment to humans transmission pathways Mari et al [12]. The bacterium is generally present in the faeces of an infected person for 7 to 14 days, though with treatment, the symptoms do not last long. The bacterium is acquired by humans through eating food or drinking water contaminated by faeces from an infected person. The incubation period of the bacteria is 12 hours to 5 days. During infection the bacteria attach themselves to the intestinal walls where they multiply and produce toxic proteins which cause the intestines to secrete large amounts of fluids. Signs and symptoms include stomach cramps, mild fever, vomiting and watery diarrhoea will lead to death due to dehydration Nelson et al [14].

Diagnosis is done through culture of the stool, agglutination tests are then done for confirmation of the disease. Treatment is based on the severity of dehydration of the patient. Simple oral rehydration solutions containing salts and glucose are used to treat mild to moderate cases. For severe cases, treatment is based on antibiotics that can shorten the cause and diminish the severity of cholera, but it is important to replace the fluids that have been lost through diarrhoea. WHO [16]. The existence of acquired immunity against the cholera disease has been known since very ancient time. Patients recovering cholera are either protected against re-infection with the same *Vibrio cholerae*, or the subsequent episodes are less severe Lavine et al [10]. Prevention and control measures of cholera include improved food safety, provision of safe drinking water, proper sanitation, and strengthening surveillance. Health education is also very important Aryda et al [1].

Several mathematical models of cholera transmission dynamics and protection measures such as vaccination, improved sanitation, water chlorination, and education have been formulated but did not incorporate rehydration and antibiotic treatment, for Emmanuele et al. [6] formulated an SIR-C cholera model to study the dynamics of cholera with control strategy where C denotes the pathogen concentration. Based on their idea, cholera deaths can be reduced by good sanitation and water treatment. Other control strategies like vaccination and curative treatment were not considered in the model. Aryda et al [1] developed and analyzed an SIR model to investigate cholera disease with education and chlorination. They concluded that with no chlorination, the disease free equilibrium is shown to be globally stable and the sensitivity analysis of basic reproduction number shows that it is most sensitive to education, per capita birth and death rate of the bacteria. They also concluded that per capita birth and death rate of the bacteria can be increased by chlorination. The model ignored factors such as environmental factors which may promote disease outbreak among poor communities. The model also ignores the role of rehydration and antibiotic treatment.

II. Model Description and Formulation

2.1 The model

The total human population $N(t)$ is divided into classes of susceptible $S(t)$, infected $I(t)$ and recovered $R(t)$, where $I(t) = I_a + I_b$, I_a represents individuals infected with the bacteria in the intestine only and I_b represents individuals infected with bacteria in both the intestine and the bloodstream.

The total population is given by;

$$N(t) = S(t) + I(t) + R(t) \quad (1)$$

The flow chart diagram for the dynamics of the transmission is given by the figure below.

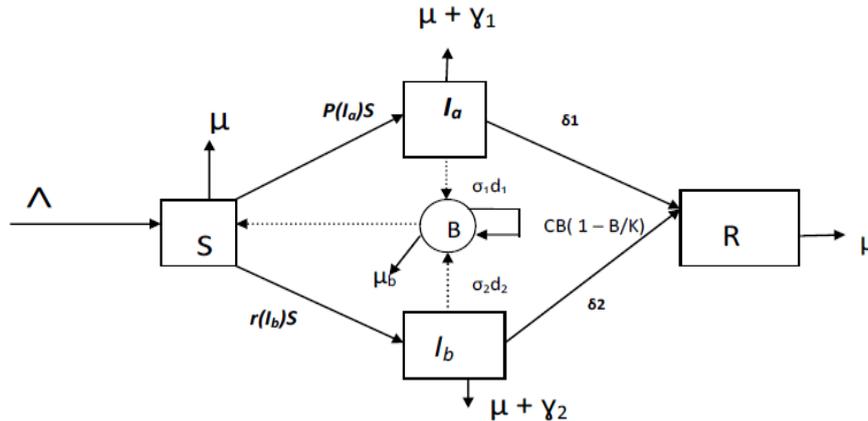


Figure 1: The model flow diagram

The system of differential equations describing the dynamics of the model is as follows;

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \left[\frac{\alpha B}{K+B}\right]I_a S - \left[\frac{\alpha B}{K+B}\right]I_b S - \mu S \\ \frac{dI_a}{dt} &= \left[\frac{\alpha B}{K+B}\right]I_a S - (\mu + \delta_1 + \gamma_1)I_a \\ \frac{dI_b}{dt} &= \left[\frac{\alpha B}{K+B}\right]I_b S - (\mu + \delta_2 + \gamma_2)I_b \\ \frac{dR}{dt} &= \delta_1 I_a + \delta_2 I_b - \mu R \\ \frac{dB}{dt} &= C\left(1 - \frac{B}{K}\right)B + d_1 I_a \sigma_1 + d_2 I_b \sigma_2 - \mu_b B \end{aligned}$$

In our model, there is a decrease in human population through natural death at a rate μ or as a result of the infection either in the intestine only γ_1 or the infection both in the intestine and the bloodstream γ_2 .

When rehydration is done the bacteria shed rate as a result of the infection in the intestine only reduces at the rate σ_1 and the bacteria shed rate as a result of the infection in both the intestine and the bloodstream is reduced at the rate σ_2 due to rehydration and administration of antibiotics. Recovery rate as a result of rehydration is δ_1 and recovery rate due to rehydration and administration of antibiotics is δ_2 , B is the concentration of *Vibrio* in the environment, K the carrying capacity of *Vibrio* where, $K > 0$.

The effective contact rate of the bacteria given by σ and the probability of susceptible to catch Cholera defined by the term $\frac{B}{K+B}$. The model will take an assumption that infected individuals only acquire the bacteria from the environment. The pathogen population grows logistically and the bacteria enter the pathogen reservoir of *Vibrio cholera* at the rate

$CB(1 - \frac{B(t)}{K})$, proportional to bacteria density in this class, where $C > 0$ is the per capita growth rate for *Vibrio cholerae*, d_1 is the bacteria shed rate due to rehydration only and d_2 is the bacteria shed rate due to both rehydration and administration of antibiotics such that $d_1 = d_2$ and bacteria death rate is given by μ_b .

Suppose that the initial condition for the System 2 takes the form:
 $S(t_0) = S(0); I(t_0) = (I_a; I_b) = I(0) = 0; R(t_0) = R(0); t_0 = 0$ (3)

III. Model Analysis

Since in our model we are studying human population, all solutions for the System of Equation (2) are all positive and bounded hence well posed.

3.1 The basic reproduction number, R_0

The basic reproduction number R_0 is defined as the average number of secondary infections due to a single infectious individual introduced in a fully susceptible population. If $R_0 < 1$ it means the disease is contained in the population and $R_0 > 1$ means the disease is persistent in the population.

The constant R_0 determined by the method of Next Generation matrix approach Van [15] is;

$$R_0 = \frac{\Lambda \alpha B}{\mu(K + B)(\mu + \delta_1 + \gamma_1)}$$

3.2 Existence of Disease Free Equilibrium (DFE) point

Disease Free Equilibrium is defined as the state at which no cholera disease is present in the population. Therefore the

$$\text{DFE } E^0(S^0, I_a^0, I_b^0, R^0, B^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right).$$

3.3 Local Stability of the Disease Free Equilibrium (DFE)

The stability of equilibrium point is related to the basic reproduction number R_0 of the model.

The stability of equilibrium point is related to the basic reproduction number R_0 of the model.

Proposition 1. For any time $t \geq 0$, the disease free equilibrium $E^0 = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ of system (3.3) is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

Proof. The J_{DFE} for equation (2) yields

$$J_{DFE} = \begin{pmatrix} -\mu & -R_0(\mu + \delta_1 + \gamma_1) & -R_0(\mu + \delta_1 + \gamma_1) & 0 & 0 \\ 0 & (\mu + \delta_1 + \gamma_1)(R_0 - 1) & 0 & 0 & 0 \\ 0 & 0 & (R_0 - 1)\mu + R_0(\delta_1 + \gamma_1) - (\delta_2 + \gamma_2) & 0 & 0 \\ 0 & \delta_1 & \delta_2 & -\mu & 0 \\ 0 & d_1\sigma_1 & d_2\sigma_2 & 0 & C - \mu_b \end{pmatrix} \quad (4)$$

The matrix has eigenvalues; $\lambda = -\mu, (\mu + \delta_1 + \gamma_1)(R_0 - 1), (R_0 - 1)\mu + R_0(\delta_1 + \gamma_1) - (\delta_2 + \gamma_2), -\mu, C - \mu_b$. For local asymptotic stability, all real parts of λ should be negative. The eigenvalue $\lambda = -\mu, C - \mu_b$ have negative real parts, this shows that the DFE is locally asymptotically stable hence $C > \mu_b$. The roots $\lambda = \{(\mu + \delta_1 + \gamma_1)(R_0 - 1), (R_0 - 1)\mu + R_0(\delta_1 + \gamma_1) - (\delta_2 + \gamma_2)\}$ are negative if and only if $R_0 < 1$. If $R_0 < 1$, the DFE is locally asymptotically stable and if $R_0 > 1$, the DFE is unstable. \square

3.4 Global Stability of the Disease Free Equilibrium (DFE)

In this section, the global asymptotic stability of the DFE of the model (2) is explored.

Theorem 1. The DFE of the system of equation (2) is globally stable whenever $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. It follows that $S = N' - I_a - I_b - R$ at the steady state. The proof is based on Comparison Theorem Lakshmikanthan[11] to prove the global stability. Using the Comparison Theorem, we have;

$$\begin{pmatrix} I'_a \\ I'_b \end{pmatrix} = (F - V) - \mathcal{F} \begin{pmatrix} I_a \\ I_b \end{pmatrix}$$

such that $\{I_a(t), I_b(t)\} \rightarrow (0, 0)$ as $t \rightarrow \infty$ hence;

$$\begin{pmatrix} I'_a \\ I'_b \end{pmatrix} = (F - V)$$

where;

$$F - V = \begin{pmatrix} (\mu + \delta_1 + \gamma_1)(R_0 - 1) & 0 & 0 & 0 \\ (\mu + \delta_1 + \gamma_1)R_0 & -(\mu + \delta_2 + \gamma_2) & 0 & 0 \\ \delta_1 & \delta_2 & -\mu & 0 \\ d_1\sigma_1 & d_1\sigma_2 & 0 & -\mu_b \end{pmatrix} \quad (5)$$

It follows that the linearized differential equation of system (2) is stable whenever $R_0 < 1$. Consequently by Comparison Theorem Lakshmikanthan[11], Substituting $I_a = I_b = R = B = 0$ into equation (2), $S(t) \rightarrow S(0)$ as $t \rightarrow \infty$. We therefore conclude that $I_a = I_b = R = B = 0$ as $t \rightarrow \infty$. It follows that the DFE is globally asymptotically stable whenever $R_0 < 1$. \square

3.5 Local Stability of the Endemic Equilibrium (EE) points

Theorem 2. *The endemic equilibrium $E^*(S^*, I_a^*, I_b^*, R^*)$ of system (2) is locally asymptotically stable whenever $R_0 > 1$.*

Proof. The endemic equilibrium point is determined under two cases.

Case I:

$$\begin{aligned}
 I_b^* &= 0 \\
 S^* &= \frac{K+B}{\alpha B}(\mu + \delta_1 + \gamma_1) \\
 R^* &= \frac{\delta_1[\Lambda(\frac{\alpha B}{K+B}) - \mu(\mu + \delta_1 + \gamma_1)]}{(\frac{\alpha B}{K+B})[\mu(\mu + \delta_1 + \gamma_1) - \alpha\delta_1]} \\
 I_a^* &= \frac{\mu[-\Lambda(\frac{\alpha B}{K+B}) - \mu(\mu + \delta_1 + \gamma_1)]}{(\frac{\alpha B}{K+B})[\mu(\mu + \delta_1 + \gamma_1) - \alpha\delta_1]} \\
 B^* &= \frac{K(C - \mu_b) \pm \sqrt{K^2(C - \mu_b)^2 - 4CKd_1I_a^*\sigma_1}}{2C}
 \end{aligned}$$

Writing I_a^* in terms of R_0 yield;

$$I_a^* = \frac{\mu[(-\mu R_0(\mu + \delta_1 + \gamma_1) - \mu(\mu + \delta_1 + \gamma_1))]}{(\mu + \delta_1 + \gamma_1)(\frac{\mu R_0}{\Lambda})[\mu(\mu + \delta_1 + \gamma_1) - \delta_1]}$$

Case II:

$$\begin{aligned}
 I_a^* &= 0 \\
 S^* &= \frac{K+B}{\alpha B}(\mu + \delta_2 + \gamma_2) \\
 R^* &= \frac{\delta_2[-\Lambda(\frac{\alpha B}{K+B}) + \mu(\mu + \delta_2 + \gamma_2)]}{(\frac{\alpha B}{K+B})[\alpha\delta_2 - \mu(\mu + \delta_2 + \gamma_2)]} \\
 I_b^* &= \frac{\mu[-\Lambda(\frac{\alpha B}{K+B}) + \mu(\mu + \delta_2 + \gamma_2)]}{(\frac{\alpha B}{K+B})[\delta_2 - \mu(\mu + \delta_2 + \gamma_2)]} \\
 B^* &= \frac{K(C - \mu_b) \pm \sqrt{K^2(C - \mu_b)^2 - 4CKd_2I_b^*\sigma_2}}{2C}
 \end{aligned}$$

Writing I_b^* in terms of R_0 yield;

$$I_b^* = \frac{\mu[(-\mu R_0(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_2 + \gamma_2))]}{(\mu + \delta_1 + \gamma_1)(\frac{\mu R_0}{\Lambda})[\delta_2 - \mu(\mu + \delta_2 + \gamma_2)]}$$

Substituting the endemic states in the linearized matrix of system of equation (2) when $I_a^* = 0$ yield;

$$J_{EE} = \begin{pmatrix} (-\frac{\alpha B}{K+B})I_b^* - \mu & (-\frac{\alpha B}{K+B})S^* & (-\frac{\alpha B}{K+B})S^* & 0 \\ 0 & (\frac{\alpha B}{K+B})S^* - (\mu + \delta_1 + \gamma_1) & 0 & 0 \\ (\frac{\alpha B}{K+B})I_b^* & 0 & (\frac{\alpha B}{K+B})S^* - (\mu + \delta_2 + \gamma_2) & 0 \\ 0 & \delta_1 & \delta_2 & -\mu \end{pmatrix} \tag{6}$$

Substituting the endemic states in matrix (6) and determining the eigenvalues when $I_a^* = 0$ yield; $\lambda_1 = -\mu$, $\lambda_2 = (\mu + \delta_2 + \gamma_2) - (\mu + \delta_1 + \gamma_1)$ and $\lambda_{3,4} = \frac{1}{2}[-(\frac{\mu(-\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_2 + \gamma_2)}{\delta_2 - \mu(\mu + \delta_2 + \gamma_2)})] \pm \frac{1}{2}\sqrt{[(\frac{\mu(-\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_2 + \gamma_2)}{\delta_2 - \mu(\mu + \delta_2 + \gamma_2)})^2 - 4(\mu + \delta_2 + \gamma_2)(\frac{\mu(\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_2 + \gamma_2)}{\delta_2 - \mu(\mu + \delta_2 + \gamma_2)})]}$

The endemic equilibrium (EE) of system of equation (2) locally asymptotically stable for $\lambda_1 = -\mu$ and for $\lambda_2 = (\mu + \delta_2 + \gamma_2) - (\mu + \delta_1 + \gamma_1)$ if and only if $(\mu + \delta_1 + \gamma_1) > (\mu + \delta_2 + \gamma_2)$. From $\lambda_{3,4}$ we have;

$$\begin{aligned} & \left(\frac{\mu[(\mu R_0)(\mu + \delta_1 + \gamma_1) - \mu(\mu + \delta_2 + \gamma_2)]}{\mu(\mu + \delta_2 + \gamma_2) - \delta_2}\right) > 0 \\ & \frac{\mu^2}{\mu - \frac{\delta_2}{(\mu + \delta_2 + \gamma_2)}} \left\{ \frac{R_0(\mu + \delta_1 + \gamma_1)}{(\mu + \delta_2 + \gamma_2)} - 1 \right\} > 0 \\ & \left\{ \frac{R_0(\mu + \delta_1 + \gamma_1)}{(\mu + \delta_2 + \gamma_2)} - 1 \right\} > 0 \end{aligned}$$

From λ_2 given by

$$(\mu + \delta_2 + \gamma_2) - (\mu + \delta_1 + \gamma_1)$$

Such that

$$(\mu + \delta_1 + \gamma_1) > (\mu + \delta_2 + \gamma_2)$$

We have

$$R_0 > \left\{ \frac{(\mu + \delta_1 + \gamma_1)}{(\mu + \delta_2 + \gamma_2)} \right\}$$

It implies that

$$(\mu + \delta_1 + \gamma_1) > (\mu + \delta_2 + \gamma_2) \approx 1$$

As a result $R_0 > 1$. Therefore for $R_0 > 1$, EE is locally asymptotically stable.

Theorem 3. for the system of equation (2) endemic equilibrium is locally asymptotically stable if and only if $R_0 > 1$ for $I_b^* = 0$.

Proof. substituting the endemic states in the linearized jacobian matrix of system of equation (2) when $I_b^* = 0$ yield.

$$J_{EE} = \begin{pmatrix} (-\frac{\alpha B}{K+B})I_a^* - \mu & (-\frac{\alpha B}{K+B})S^* & (-\frac{\alpha B}{K+B})S^* & 0 \\ (\frac{\alpha B}{K+B})I_a^* & (\frac{\alpha B}{K+B})S^* - (\mu + \delta_1 + \gamma_1) & 0 & 0 \\ 0 & 0 & (\frac{\alpha B}{K+B})S^* - (\mu + \delta_2 + \gamma_2) & 0 \\ 0 & \delta_1 & \delta_2 & -\mu \end{pmatrix} \quad (7)$$

Substituting the endemic states in matrix (7) and determining the eigenvalues when $I_b^* = 0$ yield; $\lambda_1 = -\mu$, $\lambda_2 = (\mu + \delta_1 + \gamma_1) - (\mu + \delta_2 + \gamma_2)$ and $\lambda_{3,4} = \frac{1}{2}[-(\frac{\mu(-\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_1 + \gamma_1)}{\mu(\mu + \delta_2 + \gamma_2) - \delta_1}) - \mu] \pm \frac{1}{2}\sqrt{[(\frac{\mu(-\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_1 + \gamma_1)}{\mu(\mu + \delta_2 + \gamma_2) - \delta_1}) - \mu]^2 - 4((\mu + \delta_2 + \gamma_2)(\frac{\mu(\mu R_0)(\mu + \delta_1 + \gamma_1) + \mu(\mu + \delta_1 + \gamma_1)}{\mu(\mu + \delta_2 + \gamma_2) - \delta_1}))}$

Therefore endemic equilibrium (EE) is locally asymptotically stable for $\lambda_1 = -\mu$ and for $\lambda_2 = (\mu + \delta_1 + \gamma_1) - (\mu + \delta_2 + \gamma_2)$ if and only if $(\mu + \delta_2 + \gamma_2) > (\mu + \delta_1 + \gamma_1)$. From $\lambda_{3,4}$ we have;

$$\left(\frac{\mu[(\mu R_0)(\mu + \delta_1 + \gamma_1) - \mu(\mu + \delta_1 + \gamma_1)]}{\delta_1 - \mu(\mu + \delta_1 + \gamma_1)}\right) > 0$$

$$\left(\frac{\mu^2(\mu + \delta_1 + \gamma_1)[R_0 - 1]}{\delta_1 - \mu(\mu + \delta_1 + \gamma_1)}\right) > 0$$

Dividing through by

$$(\mu + \delta_1 + \gamma_1)$$

We have

$$\frac{\mu^2}{\frac{\delta_1}{(\mu + \delta_1 + \gamma_1)} - \mu} [R_0 - 1] > 0$$

It implies that $R_0 > 1$. For $R_0 > 1$, EE is locally asymptotically stable. This ends the proof. □

4 Numerical Simulation

In this section, we use Matlab software to illustrate the numerical simulations describing the theoretical results for the system of equation (2). The parameters values used in the simulation are either obtained from literature or estimated. The parameter values have been varied to better understand how rehydration and antibiotics use reduce cholera mortality.

Table 1: Parameter values for the Cholera Model

Parameters	Range/Value	Source
Λ	10^4	Estimated
K	$10^6 \text{ cells ml}^{-1}$	[2]
α	0.05	Varies
μ	0.0068	[5]
δ_1	0.2 day^{-1}	[13]
δ_2	0.25 day^{-1}	assumed
γ_1	0.015 day^{-1}	[9]
γ_2	0.025 day^{-1}	assumed
σ_1	0.6	[13]
σ_2	0.75	assumed
μ_b	0.33 day^{-1}	[4]
C	0.73 day^{-1}	[13]
d_1	$10 \text{ cells per ml day}^{-1}$	[17]
d_2	$10 \text{ cells per ml day}^{-1}$	assumed

4.1 Simulation Results

Figure (2) below shows the bacteria population growth curve in absence of disease. This shows that in absence of disease at $t \geq 0$, the bacteria population grows logistically to the carrying capacity and the disease free equilibrium is globally asymptotically stable as shown in theorem (1). This implies that no rehydration and antibiotic is given to individuals since there is no infection, as a result there is no influence to the stability of the disease free equilibrium.

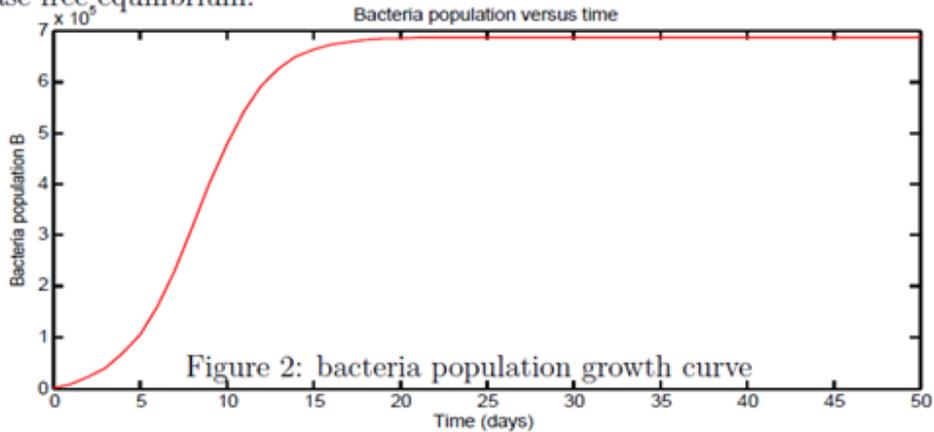


Figure (3) below shows the infection curve for cholera model with rehydration of individuals infected with the bacteria in the intestine only. Initially, there is a sharp increase in the number of those infected with the bacteria in the intestine only. This shows that even though they have been rehydrated, they are not cured immediately. Though it is clearly seen that after the first day, the infection level starts reducing with rehydration hence there is recovery.

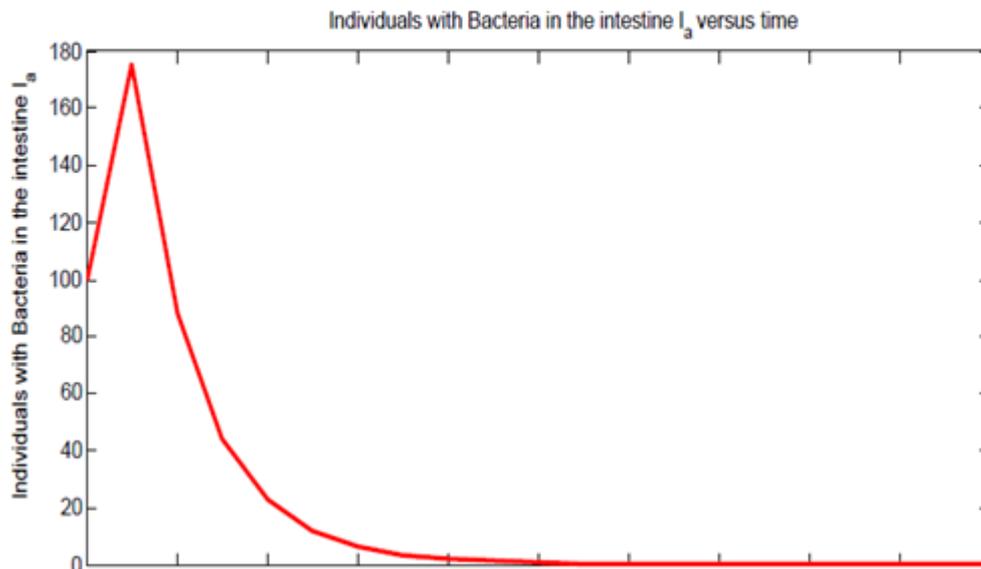


Figure 3: A graph showing how the number of individuals infected with bacteria in the intestine only varies with time in days after rehydration

Figure (4) below shows the infection curve for cholera model of individuals infected with the bacteria in both the intestine and the bloodstream. Initially, there is a sharp increase in the number of those infected. This shows that even though they have been rehydrated and antibiotics given, they do not start recovering immediately. Though the curve clearly shows that they start recovering after the first day of treatment. As expected that the level is supposed to be slightly lower when both rehydration and antibiotic is given, that is not the case. This could be due to large number of bacteria presence to the infected individuals at this level. The curve also shows that the disease still remains endemic.

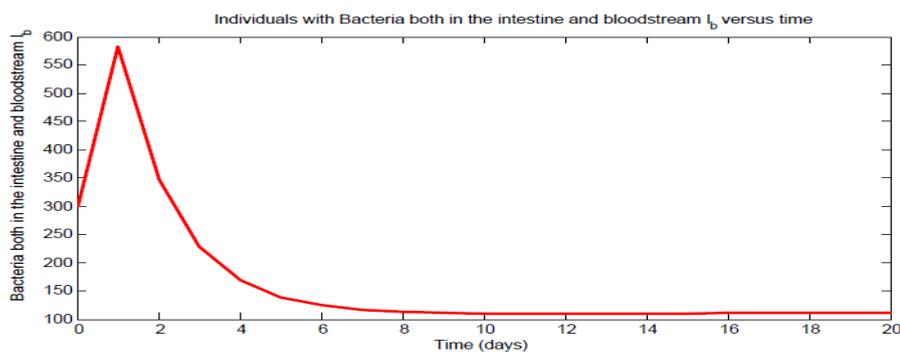


Figure (5) below shows that rehydrating those infected with the bacteria in the intestine only (I_a) and rehydrating and administering antibiotics to those infected with the bacteria in both the intestine and the bloodstream (I_b) reduces cholera deaths hence they recover and the curve shows an increase in number of recovered individuals as time increases since they acquire temporal immunity.

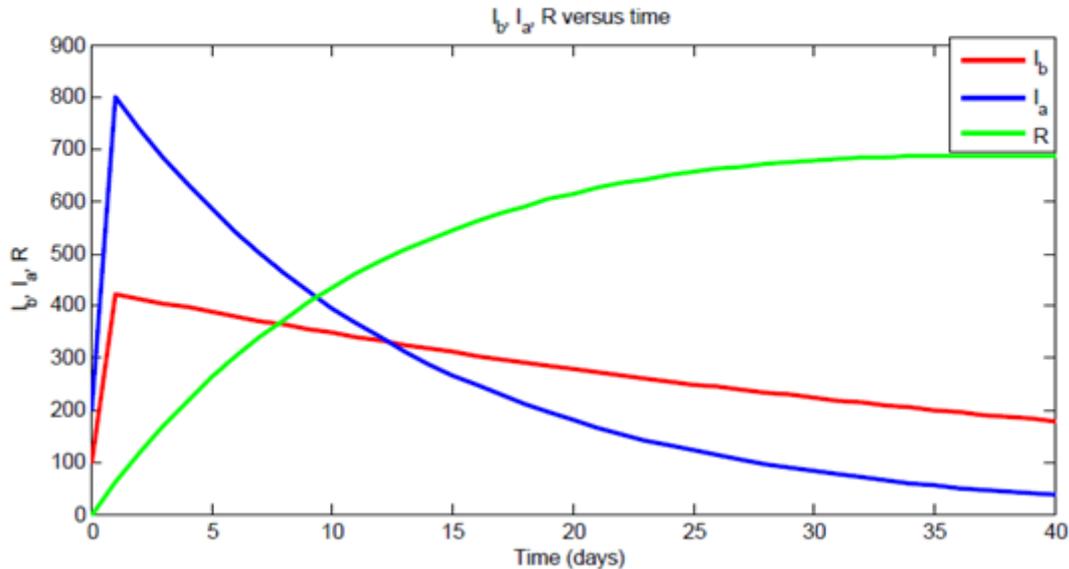


Figure 5: a graph showing the relationship between I_a , I_b and R against time in days

5 Conclusions

This paper is focused on the role of rehydration and antibiotic treatment in reduction of cholera mortality. In this section we therefore make conclusions in relation to the objectives of the study.

We have presented a cholera epidemiological model assessing the role of rehydration and antibiotic treatment in reduction of cholera mortality. There is only environmental to human transmission pathway. The basic reproduction number R_0 plays a crucial role in determining the epidemic and endemic dynamics. We have shown that for $R_0 < 1$, the DFE is locally asymptotically stable and unstable for $R_0 > 1$. The DFE have also been shown to be globally stable when $R_0 < 1$. This means that given any perturbation the disease free equilibrium remains stable.

Endemic equilibrium (EE) is also shown to be locally asymptotically stable when $R_0 > 1$. Simulation results shows that rehydration plays a major role in reducing cholera deaths when the bacteria is in the intestine only and when rehydration is done and antibiotics given to individuals infected with the bacteria in both the intestine and the bloodstream, cholera deaths are reduced though it still remains endemic with both rehydration and antibiotic administration.

References

- [1] A. Aryda, M. Abubakar, M. Tchuenche, *Modelling cholera disease with education and Chlorination*, Applied Mathematics **21**(4)(2014), 56-72.
- [2] C. Codeco, *Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir*, BMC Infectious diseases, **1**(2001),1.
- [3] Center for Health Protection, *Epidemiology, prevention and control of cholera in Hong Kong*, CHC,(2011), 4-5.
- [4] F. Capone, F. De Ctalis, R. De Luka, *Influence of diffusion on the stability of equilibria in a reaction diffusion system modelling cholera dynamics*.Bioscience **71**(2015),1107-1131.
- [5] Central Intelligence Agency, *World Fact book Report*, Kenya(2016).
- [6] L. Emmanuel, Obiri, W. Agyeil, W. Obeng, *Modelling Cholera Dynamics with control Strategy in Ghana*, Mathematical Bioscience, **1**(2015), 30-41.
- [7] Global task force on cholera control, *Cholera outbreak: assesing the outbreak response and improving preparedness*, WHO,(2004), 42-43.
- [8] A. Gordon, L. Amanda, R.K. Sheryle, *Three cases of Bacteremia caused by V. cholerae O1,7(4)*(2001).
- [9] Hove-Musekwa, F. Nyabandza, Chiyaka, Z. Mukandavire, *Modelling and analysis of the effect of malnutrition in the spread of cholera*, Math Comp Model,**53**(2011), 1583-1595
- [10] M.M. Lavine, J.B. Kaper, D. Herrington ,G. Losonsky, *Immunobiological Relationships among new cholera toxins*.(1981).
- [11] V. Lakshmikanthan, S. Leela, A.A. Martynyuk,*Stability analysis of nonlinear systems*, Marcel Dekker Inc, New York(1989).
- [12] L. Mari, E. Bertuzzo, L. Righetto , R. Casagrandi , M. Gatto, I. Rodriguez and A. Rinaldo, *Modelling cholera epidemics, the role of waterways, human mobility and sanitation*, Journal of the royal society interface, **9**(2012), 376-388.
- [13] A. Mwasa, JM. Tchuenche, *Mathematical analysis of cholera mode with public health intervention*. Biosystem **105**(2011), 190-200.
- [14] E. Nelson, J. Harris, J. Morris, S. Calderwood and A. Camilli, *Cholera transmission, the host, pathogen and bacteriophage dynamics*, Nature Rev. Microbiology, **7**(2009), 693-702.
- [15] D.P Van den and J. Watmough, *Reproduction number and sub-threshold endemic equilibria for compartmental models of disease transmission*. Math.Biosci. **180**(2002), 29-48.
- [16] World Health Organization, *Cholera vaccines: WHO position paper*, **13**(2010),117-128.
- [17] J. Wang and C. Modnak, *Modelling cholera dynamics with controls*,Canadian Applied Mathematics Quarterly, **19**(3)(2011), 256-272.
- [18] World Health Organization, *web page: www.who.org*(2014)

Owade Kennedy Jakob "Modelling The Role of Rehydration And Antibiotic Treatment on Reduction of Cholera Mortality." IOSR Journal of Mathematics (IOSR-JM), vol. 14, no. 1, 2018, pp. 11-21.