IJST EXPLORE International Journal of Science and Technology
CENTRE OF PROFESSIONAL RESEARCH PUBLICATIONS
ISSN 2049-7318

International Journal of Science and Technology Volume 2 No. 11, November, 2013

A Mathematical Model for the Transmission Dynamics of Cholera with Control Strategy

Ochoche, Jeffrey M.
Department of Mathematics/Statistics/Computer Science,
Federal University of Agriculture, P.M.B. 2373,Makurdi, Nigeria.

ABSTRACT

Cholera is generally a disease of the poor, affecting regions that lack a heightened sense of hygiene and access to safe drinking water. In this research a mathematical model for the control of cholera transmission dynamics using water treatment as a control strategy is proposed. The model is designed by dividing the system into compartments leading to corresponding differential equations. The model is built on the assumption that cholera is contracted only through the ingestion of contaminated water. Conditions are derived for the existence of the disease free and endemic equilibria. We proved that the disease free equilibrium is locally asymptotically stable under prescribed conditions on the given parameters. This means that cholera can be eradicated under such conditions in finite time. Numerical simulations are carried out using parameter values from published data to investigate the effect of transmission parameters on the dynamics of the infection. We simulated cases with no control, weak and strong control. Our results showed that water treatment is an effective method of controlling cholera however cholera cases will continue to be present in the population if the contribution of the each infected person to the aquatic environment and the contact rate with contaminated water is high.

Keywords: *Cholera, Control strategy, Epidemic, Endemic, Mathematical model, Numerical simulation.*

1. INTRODUCTION

Apart from disasters like tsunami and earthquakes, the greatest threat to human race is infectious diseases. The outbreak of infectious disease causes mortality of millions of people as well as expenditure of enormous amount of money in health care and disease control. It is therefore important that adequate attention is paid to stopping the spread of such diseases by using effective control measures. Many infectious diseases are as a direct result of poor hygienic conditions and contact between an infectious person and a susceptible person. Some infectious diseases are air-borne like tuberculosis while others are water – borne like cholera, the subject of our study.

Cholera is an acute, infectious disease characterized by extreme diarrhea and vomiting. It is deadly water – borne disease usually resulting from poor hygiene and untreated water .The cholera bacteria produce a toxin which keeps the human body from absorbing liquids hence dehydration. Untreated individuals may die from severe dehydration within two to three hours. This disease has been the killer of millions worldwide and it is endemic in both Bangladesh and Peru.

Cholera appears to have started on the Indian sub continent [1]. It became endemic by 1831 and spread to Russia. Eastern and central Europe has suffered epidemics and outbreaks through World War 1. The disease spread to the United States via English immigrants in 1832. In 1991 several parts of Africa suffered cholera epidemic.

Every year there is an estimated 3 – 5 million cholera cases and 100, 000 – 120,000 deaths due to cholera. Cholera has a short incubation period of two hours to five hours which enhances the potentiality of explosive pattern of outbreaks.

Though cholera may be life threatening, controlling and preventing the disease is normally straight forward if proper sanitation practices are followed. In developed countries, due to nearly universal advanced water treatment and sanitation practices, cholera is no longer a major health threat. The last major outbreak of cholera in the United States occurred between 1910 and 1911. If good sanitation practices are adhered to, it is usually sufficient to stop an epidemic.

Mathematical modelling provides a unique approach to gain basic knowledge in cholera dynamics [2]. Based on this knowledge effective prevention and intervention strategies can be possibly designed. The model formulation process clarifies assumptions, variables and parameters.

Mathematical models provide results such as thresholds, basic reproduction numbers, contact numbers and replacement numbers [3]. These results can help health workers understand and predict the spread of an epidemic and evaluate potential effectiveness of the different control measures to be used. They can improve our understanding of the relationship between social and biological factors that influence the spread of diseases. Mathematical models and computer simulations are useful experimental tools for building and testing theories, answering specific questions and determining sensitivities to changes in parameter values and estimating key parameter from data.

Several mathematical models of cholera transmission dynamics have been formulated and studied, see for example [1,2,4-6].

Notable among these is the Codeço [1] Model. The model is popular for being the first to explicitly incorporate the environmental component. The Model however does not incorporate control strategy. In reality, once there is an epidemic, public enlightenment follows and control is inevitable. A basic and most fundamental control strategy for cholera is water treatment. It is against this backdrop that we modify the Codeço model to include water treatment as a control strategy.

We solved the modified model numerically using ode45, an inbuilt function of MATLAB which uses Runge-Kutta method of order 4 to solve non-linear ordinary differential equations. We therefore establish the role of the parameters of the modified model in the control of cholera epidemic. Furthermore we investigated the existence and stability of the disease – Free State of the modified model by linearization approach. We also investigated the existence of the endemic equilibrium state of the modified model.

2. LITERATURE REVIEW

In London England, Dr. John Snow was able to stop a major cholera epidemic by closing the Broad street pump, and thus showed that cholera is a water – borne disease. This discovery paved way for for further studies on chorela.See for example [7 – 8].

Since untreated stools from cholera patients are the primary source of environmental contamination, proper treatment and safe disposal of liquid waste, including patient's excreta and vomit, should be undertaken to prevent contamination and secondary spread of infection. Hand hygiene should be observed at all times, especially after any contact with excreta and before preparing or eating food [9].

Mathematical modelling of cholera began with Capaso and Pavari – Fontana [4] who proposed a simple deterministic model to study a cholera epidemic that occurred in the mediterranean in 1973.

Codeço [1] was the first to build a cholera model that explicitly incorporate the environmental component, i.e the *v.cholerae* concentration in the water supply denoted by B, into a regular SIR system to form a combined environment – to – human (SIR – B) epidemiological model. This model enables a careful study on the complex interaction between human host and environmental pathogen towards a better understanding of the cholera transmission mechanism and as such, it has motivated the development of several other cholera models, see for example [2,6,10].

Building on Codeço's work , Hartley *et al* [10] developed a more general model which took into account the different infective states of *v.cholerae*. The Hartley *et al* model consists of five equations which describes the dynamics of a susceptible, infectious and removed human population and the dynamics of a hyper infective state and lower infective states of *v.cholerae* population . In their model they assumed the total population N, is constant and also assumed a constant birth and death rate b. Infection is caused by ingestion of contaminated water with either B_H (Hyper infective *vibrios*) or B_L (Lower infective *vibrios*).

Mukandavire *et al* [6] built on the Codeço model by incorporating the human – to human factor (i.e interactions between the susceptible and infectives) into the model. They were able to estimate the basic reproduction number (R_o) for the 2008 – 2009 cholera outbreak in Zimbabwe. They presented a model, fitted to the Zimbabwean data that provides insights into the nature of the epidemic in Zimbabwe and, on a broader scale, to control of cholera at a global level. More specifically, they used Zimbabwean data to derive estimates of the basic reproductive number (R_o) of cholera on a regional basis.

Ashleigh *et al* [5] built a SIR compartmental transmission model that characterized the population as susceptible to infection, infected and infectious to others. Recovered or otherwise were removed from risk to further infection. They assumed that cholera could be transmitted through either contaminated water or close contact with infectives but that water borne transmission was a far more important method of transmission. They added a water compartment to the model (what Codeço referred to as aquatic environment).

As a control measure, they introduced vaccination into the model by moving persons from susceptible compartment to recovered (immune) compartment when vaccination is completed. They also explored the relative effect of replacing vaccination (as control) with provision of clean water which reduces the number of persons who are susceptible to infection through contaminated water

With the advances in sanitation systems and availability of portable water, coupled with heightened awareness of personal hygiene, cholera incidence is no longer a problem of advanced countries, also WHO [11] recommend that cholera vaccine should only be used in conjunction with other prevention and control strategies such as improving water quality and sanitation measures in areas where the disease is endemic. It is against this backdrop that we modified the Codeço cholera model (a focal point in contemporary mathematical study of cholera) to include water treatment as a control strategy so as to effectively study the transmission dynamics of cholera.

3. METHODS

In this section, we present the original Codeço model. Using the assumptions of the original model, we also modified the model to incorporate water treatment as a control strategy; furthermore we modified the model to incorporate a different birth and death rate. It should be noted that Tian *et al* [2] also modified same model and incorporate control strategies including water treatment but used the same birth and death rate . Our model seeks to investigate only the effect of water treatment as a control strategy unlike Tian *et al* that incorporated other control strategies like therapeutic treatment and vaccination of newborns.

4. MODEL FORMULATION

The variables and parameters of the Codeço model are defined below:

$S(t)$= Number of susceptible individuals at time t
$I(t)$= Number of infected individuals at time t
$B(t)$= Concentration of v.cholerae in water at time t
H = Initial human population
n = Human birth and death rate
a = Rate of exposure to contaminated water
k = Concentration of *v.cholerae* in water that yields 50% chance of catching Cholera
r = Rate at which people recover from cholera
c = *Vibrios* growth rate
d = *Vibrios* loss rate
e = Contribution of each infected person to the population of *v.cholerae* in the aquatic environment

The Codeço model for cholera transmission is given below.

$$\frac{dS}{dt} = n(H - S) - a\lambda(B)S, \quad (1)$$

$$\frac{dI}{dt} = a\lambda(B)S - rI, \quad (2)$$

$$\frac{dB}{dt} = B(c - d) + eI, \quad (3)$$

$$S(0) = H, I(0) > 0, B(0) > 0$$

The first equation describes the dynamics of the susceptibles of constant size *H*. susceptible individuals are renewed at a rate *n*. Renewal may occur as a result of birth or loss of acquired immunity (cholera does not confer a lifelong immunity). Susceptible people may become infected at a rate $a\lambda(B)$ where *a* is the rate of contact with untreated water and $\lambda(B)$is the probability of such a person to contract cholera.

The second equation describes the dynamics of infected people in the community. This category includes not only cholera cases but also those with asymptomatic and mild infections.
The third equation describes the dynamics of the pathogenic *v.cholerae* in the aquatic reservoir, in this case, the untreated water consumed by the population.

5. THE MODIFIED MODEL

The modified model follows the same assumptions as in the Codeço model and uses the same variables.

$$\frac{dS}{dt} = bH - \mu S - \frac{aB}{K+B} S, \quad (4)$$

$$\frac{dI}{dt} = \frac{aB}{K+B} S - (\gamma + \mu) I, \quad (5)$$

$$\frac{dB}{dt} = eI - (w - p)B, \quad (6)$$

Where *w* is death rate of *vibrios* as a result of water treatment.

$$S(0) = H, I(0) > 0, B(0) > 0$$

The model is partitioned into two human populations, *S(t)* and $I(t)$ and a *vibrios* population *B(t).* We assure that as a result of water treatment, the *vibrios* are dying at a rate *w* as captured in (3.6). *p* in (3.6) is equal to c – *d* in the original model which is the net death rate of *vibrios.b* is the birth rate of the human population and μ is their natural death rate. γ is the recovery rate from cholera.

The incidence rate which determines the rate of new infection is given by $\frac{aB}{K+B}$. *e* is the rate at which each infected person is contaminating the aquatic reservoir i.e passing *vibrios* into drinking water.

EXISTENCE OF THE DISEASE FREE RQUILIBRUIM STATE

We now discuss the existence and stability of the equilibrium states of the modified model. At the equilibrium state $\frac{ds}{dt}$, $\frac{dI}{dt}$, $\frac{dB}{dt}$ all vanish. Therefore, equating the right hand sides of the model equations (4), (5) and (6) to zero we have,

$$bH - \mu S - \frac{aB}{K+B} S = 0, \quad (7)$$

$$\frac{aB}{K+B} S - (\gamma + \mu)I = 0, \quad (8)$$

$$eI - (w - p)B = 0, \quad (9)$$

At the Disease Free State (DFE), there are no infectives, that is

I = 0 . Substituting this into (9) we have
$-(w - p)B = 0$, therefore $B = 0$ provided that $w - p \neq 0$
Substituting $B = 0$ into (7) we have
$S = \frac{bH}{\mu}$.

Hence we have the following theorem.

THEOREM 1

There exist a disease free equilibrium state given by $E_0 = (\frac{bH}{\mu}, 0, 0)$

STABILITY OF THE DISEASE FREE EQUILIBRIUM STATE

We shall use the linearization approach to prove that the disease free equilibrium state is locally asymptotically stable.

The Jacobian matrix is therefore given by

$$J = \begin{pmatrix} -\mu - \frac{aB}{k+B} & 0 & -\frac{aS}{K+B} + \frac{aBS}{(k+B)^2} \\ \frac{aB}{k+B} & -(\gamma+\mu) & \frac{aS}{K+B} - \frac{aBS}{(k+B)^2} \\ 0 & e & -(w-p) \end{pmatrix}$$

At the DFE, when S = H , I = 0 and B = 0, we have

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\frac{abH}{K\mu} \\ 0 & -(\gamma+\mu) & \frac{abH}{k\mu} \\ 0 & e & -(w-p) \end{pmatrix}$$

The corresponding characteristic equation is

$$\left| J(E_0) - \lambda I \right| = \begin{vmatrix} -\mu - \lambda & 0 & -\frac{abH}{K\mu} \\ 0 & -(\gamma+\mu) - \lambda & \frac{aH}{K} \\ 0 & e & -(w-P) - \lambda \end{vmatrix} = 0$$

or

$$(-\mu - \lambda)\left[(-(\gamma+\mu) - \lambda)(-(w-p) - \lambda) - \frac{eabH}{k\mu}\right] = 0$$

Thus $\lambda_1 = -\mu$

λ_2 , λ_3 are found from the roots of:

$$\lambda^2 + \big((w-p) + (\gamma+\mu)\big)\lambda + (w-p)r - \frac{eabH}{k\mu} = 0$$

That is,

$$\lambda_{2,3} = \frac{-(w-p+(\gamma+\mu)) \pm \sqrt{(w-p+(\gamma+\mu))^2 - 4\left[(w-p)(\gamma+\mu) - \frac{eabH}{k\mu}\right]}}{2}$$

or

$$\lambda_{2,3} = \frac{-(w-p+(\gamma+\mu))}{2} \pm \frac{\sqrt{(w-p+(\gamma+\mu))^2 - 4(w-p)(\gamma+\mu)\,(1-Ro)}}{2} \qquad (10)$$

where $R_0 = \frac{eabH}{k\mu(w-p)(\gamma+\mu)}$

Suppose $w - p < 0$ if $R_0 < 1$ we have,

$$4(w-p)(\gamma+\mu)\,(1-Ro) > 0$$

therefore, $(w-p+(\gamma+\mu))^2 - 4(w-p)(\gamma+\mu)\,(1-Ro) < (w-p+(\gamma+\mu))^2$

$$\Rightarrow \sqrt{(w-p+(\gamma+\mu))^2 - 4(w-p)(\gamma+\mu)\,(1-Ro)} \leq (w-p+(\gamma+\mu))$$

Hence from 10

$$\lambda_{2,3} \leq \frac{-(w-p+(\gamma+\mu))}{2} \pm \frac{(w-p+(\gamma+\mu))}{2}$$

$$\lambda_2 \leq \frac{-(w-p+(\gamma+\mu))}{2} + \frac{(w-p+(\gamma+\mu))}{2} = 0$$

$$\lambda_3 \leq \frac{-(w-p+(\gamma+\mu))}{2} - \frac{(w-p+(\gamma+\mu))}{2} < 0$$

Thus if $R_0 < 1$ then all the roots are negative and we have the following theorem

THEOREM 2

Given $R_0 < 1$, the DFE of the modified model is locally asymptotically stable.

EXISTENCE OF ENDEMIC EQUILIBRIUM STATE

We shall now study the existence of the endemic equilibrium state of the modified model.

At equilibrium state $\frac{ds}{dt} = \frac{dB}{dt} = \frac{dI}{dt} = 0$. Therefore, equating the right hand sides of the model equations (4) – (6) to zero we have,

$$bH - \mu S - \frac{aB}{K+B} S = 0 \qquad (11)$$

$$\frac{aB}{K+B} S - (\gamma+\mu)I = 0 \qquad (12)$$

$$eI - (w-p)B = 0 \qquad (13)$$

From (13)

$$eI - (w-p)B = 0$$
$$eI = (w-p)B$$
$$B = \frac{eI}{(w-P)} \qquad (14)$$

From (12)

$$\frac{aBS}{K+B} - (\gamma+\mu)I = 0$$
$$S = \frac{(K+B)}{aB}(\gamma+\mu)I \qquad (15)$$

From (11)

$$bH - \mu S - \frac{aB}{K+B} S = 0$$
$$bH - S\left(\mu + \frac{aB}{K+B}\right) = 0$$

Substituting S in (15), we have

$$bH - \frac{K+B}{aB}(\gamma+\mu)I\left(\mu + \frac{aB}{K+B}\right) = 0$$

Or

$$bH - (\gamma + \mu)I\left(\frac{k+B}{aB}\mu + 1\right) = 0$$

Substituting B in (14) we have

$$bH - (\gamma + \mu)I\left[\frac{\left(K+\frac{eI}{(w-p)}\right)}{a\frac{eI}{(w-p)}}\mu + 1\right] = 0$$

Or

$$bH - (\gamma + \mu)I\left[\left(\frac{K((w-p))}{aeI} + \frac{1}{a}\right)\mu + 1\right] = 0$$

Or

$$bH - \frac{(\gamma+\mu)IK(w-p)\mu}{aeI} - \frac{(\gamma+\mu)I\mu}{a} - (\gamma + \mu)I = 0$$

$$bH - \frac{(\gamma+\mu)K(w-p)\mu}{ae} - (\gamma + \mu)I\left(\frac{\mu}{a} + 1\right) = 0$$

$$\Rightarrow bHea - (\gamma + \mu)K(w-p)\mu - (\gamma + \mu)Iea\left(\frac{\mu}{a} + 1\right) = 0$$

$$\Rightarrow bHea - (\gamma + \mu)K(w-p)\mu - (\gamma + \mu)Ie\,(\mu + a) = 0$$

Therefore,

$$I = \frac{bHea - (\gamma+\mu)K(w-p)\mu}{(\gamma+\mu)e(\mu+a)}$$

For I to be > 0, $bHea > (\gamma + \mu)K(w-p)\mu$

or $\frac{bHea}{(\gamma+\mu)K(w-p)\mu} > 1$

or $R_0 > 1$, where $R_0 = \frac{bHea}{(\gamma+\mu)K(w-p)\mu}$

We have proved the following theorem.

THEOREM 3

A positive endemic equilibrium state exist if $R_0 > 1$

6. NUMERICAL EXPERIMENTS

We solved the equations of the modified model numerically. Plots of the numerical solution are used to investigate the effect of some parameters on the infective population component. In order to carry out the task, the model parameters are assigned specific values as in the Codeço model. We used the inbuilt MATLAB function ode45 to solve the model equations. The function solves systems of ordinary differential equations using the fourth order Runge – Kutta method.

The results obtained from numerical experiments are presented below.

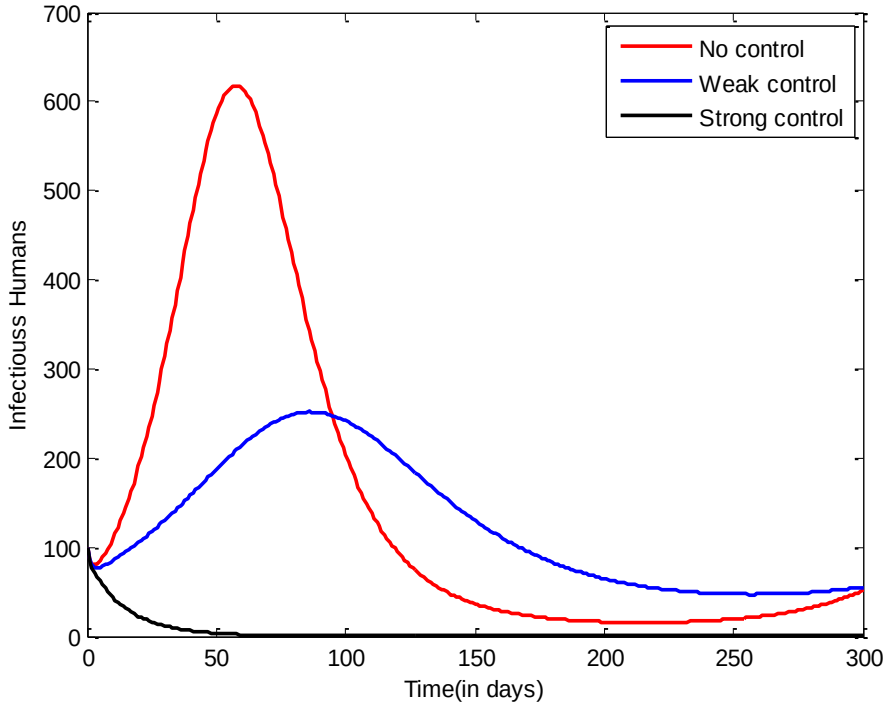

Fig. 1: Graph of infected human population against time with a case of average exposure (a = 0.5) to contaminated water and low contribution of each infected person (e = 10) to the aquatic environment. With $w = 0, w = 0.1$ *and* $w = 0.5$ for no control, weak and strong control respectively

Here we compared the effect of control on the population of infected humans using the modified model when the rate of exposed humans to contaminated water is low (a = 0.5) and the rate of contribution of each infected person to the aquatic environment is low

(e = 10). We see from Fig. 1 that strong control yields the best result although moderate control could also reduce the infection significantly.

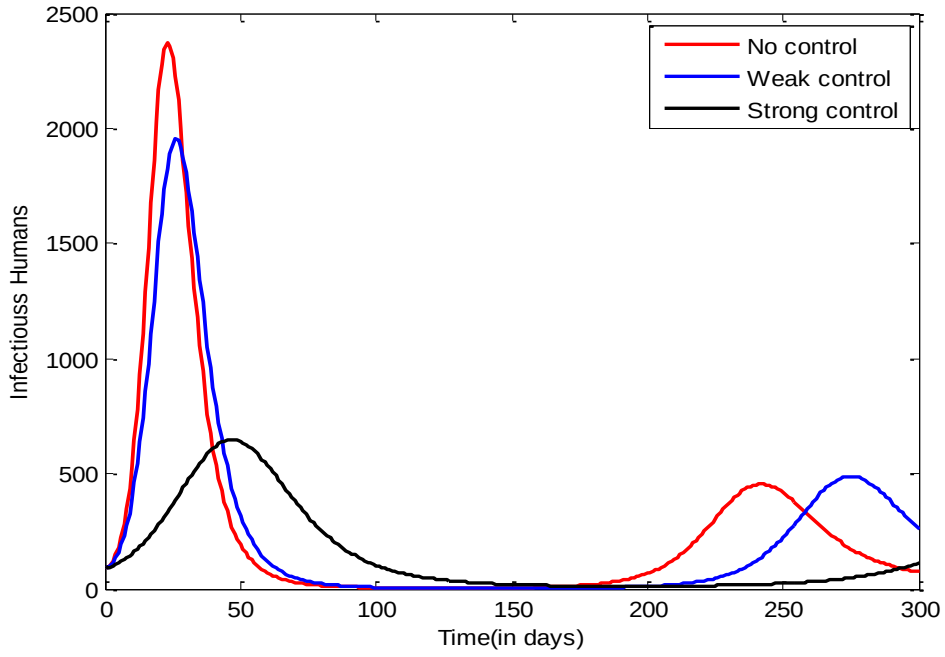

Fig. 2: Graph of infected human population against time with a case of high exposure (a = 0.8) to contaminated water and an increase in the contribution of each infected person (e = 15) to the aquatic environment. With $w = 0, w = 0.1$ *and* $w = 0.5$ for no control, weak and strong control respectively.

We increased the contact rate with contaminated water and the contribution of each infected person to the aquatic environment. Once again strong control yields the best result.

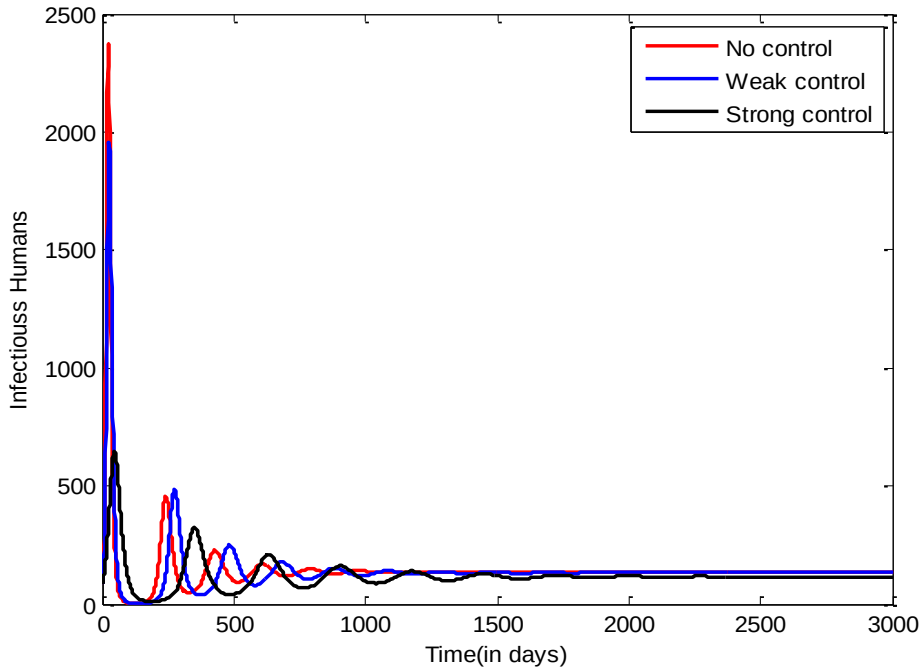

Fig. 3: Extended graph of infected human population against time with a case of high exposure (a = 0.8) to contaminated water and an increase in the contribution of each infected person (e = 15) to the aquatic environment. With $w = 0, w = 0.1$ *and* $w = 0.5$ for no control, weak and strong control respectively.

We now extended the graph in Fig 2. to investigated the long effect of control. As seen in fig 3. Cholera cases are maintained in the population. We therefore conclude that if the contact rate with contaminated water is high in the presence of increased contribution of each infected person to the aquatic environment then cholera will persist in the population even with water treatment as a control strategy. The persistent cholera cases here however do not necessarily imply an epidemic. It could just be mild waves (i.e few cases of cholera).

7. CONCLUSION AND RECOMMENDATIONS

In this research we modified the model by Codeço [1] to incorporate water treatment as a control strategy.

In our findings, there exists a disease free equilibrium state. We showed that if $R_0 < 1$, then the disease free equilibrium state is locally asymptotically stable, which implies that the disease could be eradicated under this condition in finite time. We also showed that an endemic equilibrium state exist if $R_0 > 1$.

Also it is found that the control has more effect and significance if the contribution of each infected person to the aquatic environment and the rate of exposure to contaminated water are sufficiently reduced.

Based on our findings, we recommend that proper education and sensitization be given to the public by relevant authorities and NGO's of the dangers of open defecation and urinating in source of drinking water. This will reduce the contribution of each infected person to the aquatic reservoir (parameter e).

Also, we recommend that the Government should provide portable water to the populace in order to discourage drinking of untreated water. This will reduce the rate of exposure to contaminated water (parameter a).

REFERENCES

[1]. C.T. Codeço. Epidemic and Endemic Cholera: the role of the Aquatic Reservoir. *BMC Infectious Diseases* 1(1),2001. Available from http://www.biomedcentral.com/1471-2334/1/1

[2]. P.T. Tian, S. Liao, J. Wang. *Dynamical analysis and control strategies in modeling cholera*. A monograph, 2010.

[3]. F. Benyah. *Epidemiological Modelling and Analysis*, Paper presented at 13th Edward A Bouchet/Abdus Salam workshop, University of Ghana, Legon, Accra, 9-13 July, 2007.

[4]. V. Capaso, S.L. Pavari-Fontana. A Mathematical Model for the 1973 Cholera Epidemic in the European Mediterranean region. *Rev Epidém Santé Pub*, 27:121 – 132,1979.

[5]. R.T.Ashliegh, J. Tien, M.Eisenberg, J.D.David, M. Junling, N.F. David, Cholera Epidemic in Haiti, 2010: Using a Transmission Model to Explain Spatial Spread of Disease and Identify Optimal Control Interventions**,** ***A****nnals of internal medicine*. 154:593-601, 2011.

[6]. Z. Mukandavire, S. Liao, J. Wang, H.Gaff, D.L. Smith , J.G. Morris. Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, *PNAS Early edition*, 2010.

[7]. T.A. Cockburn , J.G. Cassanos. Epidemiology of endemic cholera. *Public Health Rep*, 75:791 – 803,1960.

[8]. P.R. Epstein. Algal blooms in the Spread of and Persistence of Cholera. *Biosystems*, 31:209 – 221,1993

[9]. WHO . Guidelines for cholera control. Geneva: World Health Organization; 1993 [Available from: http://whqlibdoc.who.int/publications/1993/924154449X.pdf]

[10]. D.M. Hartley, J.G Morris, D.L. Smith. Hyperinfectivity: A critical element in the ability of V. cholerae to cause epidemic, *PLoS MEDICINE*, 3:63 – 69, 2006.

[11]. WHO. *International travel and Health 2011*. Geneva: World Health Organization, 2011 [Available from: http://www.who.int/ith/chapters/ith2011chap6.pdf]