Available online at http://scik.org
J. Math. Comput. Sci. 5 (2015), No. 2, 172-187
ISSN: 1927-5307

A MATHEMATICAL MODEL ON THE CONTROL OF CHOLERA: HYGIENE CONSCIOUSNESS AS A STRATEGY

M. JEFFREY OCHOCHE *, E. CHINWENDU MADUBUEZE AND T. BENJAMIN AKAABO
Department of Mathematics/Statistics/Computer science, University of Agriculture, Makurdi, Nigeria

Copyright © 2015 Ochoche, Madubueze and Akaabo. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract. We presented a mathematical model of cholera incorporating hygiene consciousness as a control strategy. We derived the Basic Reproduction number using the next generation approach. We showed that the disease free equilibrium state of the model is locally asymptotically stable if the basic reproduction number is less than unity. Numerical simulations showed that hygiene consciousness could be an effective means of controlling cholera as incrementing the hygiene consciousness parameter translate into a reduction in the disease prevalence.

Keywords: Cholera; Disease modelling; Equilibrium stability; Hygiene.
2010 AMS Subject Classification: 93A30.

1 INTRODUCTION

Events such as human or animal invasions of new ecosystem, global warming, environmental degradation, increased international travel, and changes in economic and social patterns will continue to provide opportunities for new and infectious disease. In particular global warming has resulted to a rise in sea level, the ultimate effect of climate change would be more floods and more water borne diseases like cholera as people become more incline to use unsafe surface water [1].

Cholera is an acute intestinal infection caused by ingestion of food and water contaminated with the bacterium vibrio cholera. It has a short incubation period, from less than one day to five days, and produces an enterotoxin that causes copious, painless, watery diarrhoea that can quickly lead to severe dehydration and death if treatment is not promptly given. Symptoms of severe cholera infection include: Profuse watery diarrhoea, Vomiting and rapid heart rate, also persons with

*Corresponding author
Received May 13, 2014

severe cholera can develop acute renal failure, severe electrolyte imbalance and coma. If untreated, severe dehydration can rapidly lead to shock and death [2]. Cholera has been found to vary with climate fluctuations over long time periods. Temperatures and rainfall anomalies can generate cases of diarrhoea and cholera since floods caused by heavy monsoons can contaminate drinking water with the bacterium.

Profuse diarrhoea produced by cholera patients contains large amount of infections vibrio cholera bacteria that can infect others if ingested, and when these bacteria contaminate water or food it leads to additional case. Infected persons when treated rapidly can recover quickly and there are typically no long term consequences. Cholera can be transmitted from environment – to – human through ingestion of contaminated water and human – to – human through contact with hygienically unconscious individuals who are infected or have had contact with excretes of an infected person.

Cholera outbreak in a region is noted to have caused unjustified panic by other countries which include curtailing or restricting travel from countries where a cholera outbreak is occurring, or import restrictions on certain foods. For example the cholera outbreak in Peru 1991 cost the country US$770 million due to food trade embargoes and adverse effects on tourism. Cholera has the potential to spread widely and can cause many deaths; thus a well-coordinated timely and effective response to outbreaks is paramount. A single case of cholera in a person with no history of overseas travel is considered an outbreak and measures should be taken to initiate a thorough investigation in determining the vehicle and circumstance of transmission and plan control strategies accordingly.

Code φ's work on cholera [3] was the first to explicitly capture the aquatic habitat of the bacterium *v.cholerea* . He explored the role of aquatic reservoir on the persistence of endemic cholera as well as defined minimum condition for the development of epidemic and endemic cholera. His work has been a platform for most authors.

Manju and vinay [4], proposed and analysed a mathematical model which focused on natural birth and immigration for the spread of cholera with carriers in the environment. They stated that all susceptible are affected by carrier population density. The carrier population density is assumed to follow the logistic model growth due to conducive human population density related factors. They showed that the spread of disease increase due to the presence of carrier in the environment, their interaction with susceptible directly as well as indirectly through transport

mechanisms used by carriers.They found that cholera become more endemic due to immigration. They concluded that the spread of cholera disease could increase if the growth of carrier population caused by conducive household discharges increased, and increase in recovered individual would decrease infective human population. Hence, the endemic equilibrium would be locally and asymptotically stable.

Hethcote *et al.* [5], developed some SIS and SIR endemic models with a new class Q of quarantine individuals who have been removed from the infective population either voluntarily or coercively. The models aimed at preventing the infective from mixing with the susceptible in order to infect them. They stated that in an outbreak of like cholera, susceptible become infected, these infected individuals should be transferred to the quarantine class Q and remain there until they are recovered, at which time, they return to the susceptible class. They claimed that disease outbreak will continue to be endemic in the affected areas if quarantine practice is not considered. Thus, duration of staying in the Q class and treatment offered to them were not indicated. Hence, the endemic equilibrium will be asymptotically stable with the use of a quarantine class.

Proper excreta disposal is referred to as a good sanitation practice. It is a cost-effective way of controlling cholera dynamics.. According to WHO, Pit latrine is an acceptable form of excreta disposal in rural Africa. World Bank Technology Advisory Group said efficacy of other alternative facilities depended on how it has been put to use. In Global Water Supply and Sanitation Assessment 2002 Report, only sewage, septic tanks with soak-aways, pure-flush latrines and pit latrines are accepted as improved technological way of excreta Disposal. Good sanitation with the use of latrines can greatly reduce cholera as noted by [6], who attributed a median reduction in incidence of 36 per cent to sanitation. Thus, latrine owners must be hygienically conscious.

Curtis and Cairncross [7], carried out a study on promoting hygiene practices by washing one's hands with soap to reduce the effects of cholera. In their research they stated that hand washing with soap and intervention to promote it could reduce diarrheal morbidity. In [8] it was suggested that the integration of water supply, sanitation and hygiene promotion can provide more effective result on the reduction of cholera; in addition, it is highly cost-effectiv

Many cholera models have been formulated and studied; see [9 - 14]. To the authors' knowledge, none has considered the effects of hygiene as a main intervention strategy. In this paper we developed a mathematical model incorporating hygiene consciousness as a control strategy.

2.0 THE CHOLERA MODEL

2.1 Model Formulation

One difficulty in studying cholera dynamics is the coupling between its multiple transmission pathways which involve both direct human to human and indirect human to human mode and which lead to combined human environment epidemiological model [15]. We formulated our model thus; the total human population N is divided into four mutually exclusive compartments namely; $S(t)$ corresponding to susceptible individuals, $I(t)$, is the number of infected individuals, $C(t)$ standing for hygiene conscious individuals and $R(t)$ denoting the number of recovered individuals.

The pathogen is capture in compartment B, where B represents concentration of vibrios in water. Below is tabular description of the state variables.

Table 1: State variables of the cholera model

Symbol	Description
$S\ (t)$	susceptible individuals
$I\ (t)$	infectious persons
$C\ (t)$	Hygiene conscious individuals
$R\ (t)$	Recovered individuals

In this model, μ denotes the per capital natural human death rate, β_1 and β_2 are the rate of ingesting vibrios from contaminated water and through human to human interaction respectively. π is the recruitment rate of susceptible individuals through births and immigration [9]. Further, m is the death rate caused as a result of the diseases, δ is the decay rate of vibrios. Susceptible are assumed to experience a natural recovery rate at parameter r, ξ represents the contribution of each infected person to the aquatic environment. Improved hygiene practices will lead to recovery rate ρ of hygiene conscious individuals. Finally, α stands for the rate at which infected individual become hygienically conscious, thus the incidence consist of two parts, one is due to environment to human transmission, the other represent human to human transmission. The environment to human transmission is modelled by non-linear function $\beta_1 \frac{B}{K+B}$ with K being the pathogen concentration that yields 50% chances of catching cholera [9]. This incidence

represents a logistic response to the increase in B. The human transmission is modelled linearly as $\beta_2 I$.

Table 2: Parameters of the cholera model

Symbol	Description
π	Recruitment rate of human population
μ	Per capital natural human death rate
β_1	Rate of ingesting *vibrios* from contaminated water
β_2	Contact rate with *vibrios* through human to human interaction
m	Death rate as result of cholera disease.
α	Rate at which individuals become Hygiene Conscious
δ	Decay rate of *vibrios*
r	Natural recovery rate
ξ	Contribution of each infected person to the aquatic environment
ρ	Recovery rate of hygiene conscious individuals
k	Pathogen concentration in water

The model transfer diagram is as shown below:

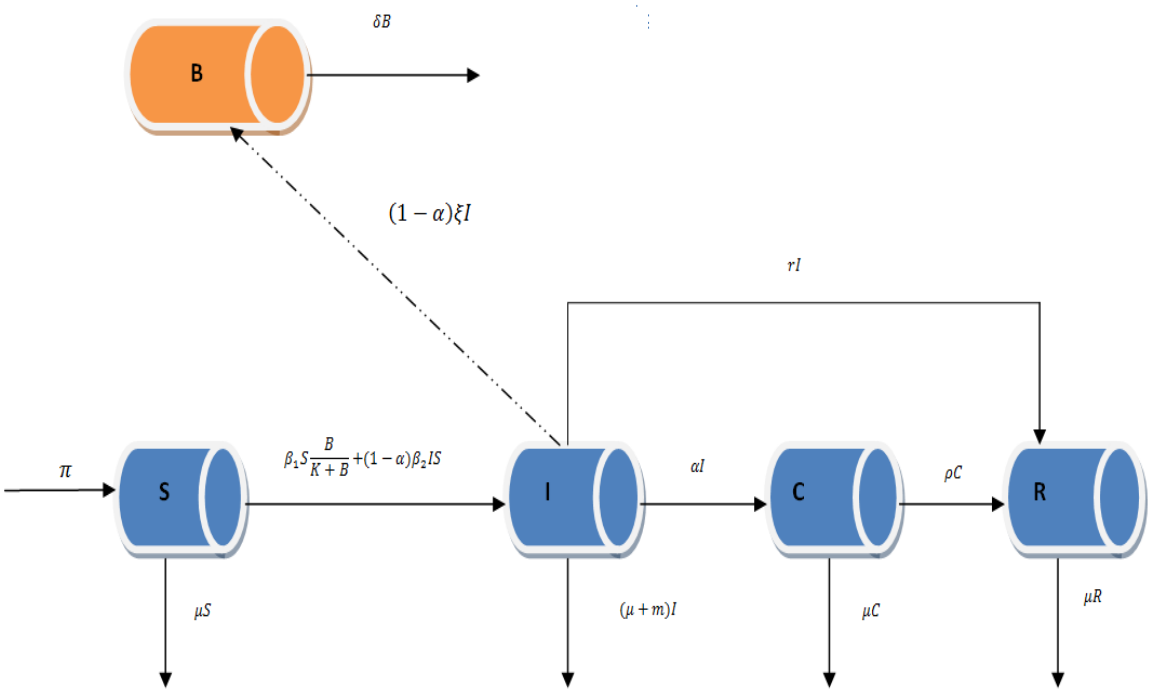


Figure 1: The model transfer diagram

Assume that the infection confers permanent immunity, so that individuals can move from *S* class to *I* class and then either directly to the *R* class or to *C* class and then to the *R* class as presented in fig. 3.1 above. The model equation is as shown below using a set of ordinary differential equations.

$$\frac{dS}{dt} = \pi - \beta_1 S \frac{\mathrm{B}}{\mathrm{K}+\mathrm{B}} - (1-\alpha)\beta_2 \mathrm{SI} - \mu \mathrm{S}$$

$$\frac{dI}{dt} = \beta_1 S \frac{\mathrm{B}}{\mathrm{K}+\mathrm{B}} + (1-\alpha)\beta_2 \mathrm{SI} - (\mathrm{r}+\mathrm{m}+\mu+\alpha)\mathrm{I}$$

$$\frac{dC}{dt} = \alpha I - (\rho + \mu)C \qquad (2.1)$$

$$\frac{dR}{dt} = \rho C + rI - \mu R$$

$$\frac{dB}{dt} = (1-\alpha)\xi I - \delta B$$

2.1.1 Invariant Region

Since the model deals with people, it is assumed that both the state variables and parameters are positive, also, *N (t) > 0* for all $t \geq 0$

Let *N* which is the total number of individuals under consideration be $N_1 = S + I + C$.

Differentiating N_1 we have;

$$\frac{dN_1}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dC}{dt}$$

Expanding the equation, we have;

$$\frac{dN_1}{dt} = \pi - \mu S - (r+m+\mu)I - (\rho+\mu)C \text{ or}$$

$$\frac{dN_1}{dt} = \pi - \mu N_1 - (r+m)I - \rho C \qquad (2.2)$$

It is clearly seen that in the absence of cholera disease, there is no recovery and death rate caused by the disease. That is $r = 0, m = 0$ and $\rho = 0$, hence 3.2 becomes;

$$\Rightarrow \frac{dN_1}{dt} \leq \pi - \mu N_1 \qquad (2.3)$$

Applying Birkhoff and Rota's theorem on a differential inequality, we have;

$$\frac{dN_1}{\pi - \mu N_1} \leq dt \qquad (2.4)$$

Integrating the above equation on both sides and applying the initial value conditions we have.

$$\pi - \mu N_1 \geq \left(\pi - \mu N_1(0)\right) e^{-\mu t} \pi \qquad (2.5)$$

Or

$$-\mu N_1 \geq \left(\pi - \mu N_1(0)\right)e^{-\mu t} - \pi$$

$$N_1 \leq -\left(\frac{\pi-\mu N_1(0)}{\mu}\right)e^{-\mu t} + \frac{\pi}{\mu}$$

That is $N_1 \leq \frac{\pi}{\mu} - \left(\frac{\pi-\mu N_1(0)}{\mu}\right)e^{-\mu t}$ (2.6)

As t $\rightarrow \infty$ we have;

$$N_1 \leq \frac{\pi}{\mu} \Rightarrow N_1 \rightarrow \frac{\pi}{\mu} \quad (2.7)$$

Since the solution is bounded, it enters the region.

$$Q = \left\{(S, I, C) \in R^3_+, \ N_1 \leq \frac{\pi}{\mu}\right\}$$

Using the last equation of (3.1)

$\frac{dB}{dt} = (1-\alpha)\xi I - \delta B.$ Let N = N_B, We have

$\frac{dN_B}{dt} = (1-\alpha)\xi I - \delta N_B.$

But $I < \frac{\pi}{\mu}$ Hence

$$\frac{dN_B}{dt} \leq \frac{\pi}{\mu}(1-\alpha)\xi - \delta N_B \quad (2.8)$$

Integrating (2.8) on both sides;

$$N_B(t) \leq \frac{\xi\pi}{\mu(1-\alpha)}\left[1 - Me^{-\delta(t)}\right] \quad (2.9)$$

Where M = constant, as $t \rightarrow \infty$, N_B will be

$$N_B \leq \frac{\xi\pi}{\mu(1-\alpha)} \Rightarrow N_B \rightarrow \frac{\xi\pi}{\mu(1-\alpha)}$$

Now the solution enters the region

$$Q_B = B \in R_+, N_B \leq \frac{\xi\pi}{\mu(1-\alpha)}$$

The general set for the model equation will enter the region

$$Q^1 = \left\{(S, I, C,) \in R^3_+; B \in R_+; \ S, I, C, B \geq 0; \ N_1 \leq \frac{\pi}{\mu}; \ N_B \leq \frac{\xi\pi}{\mu(1-\alpha)}\right\}$$

This shows a positive invariant set of the model, it also indicates the biological explanation of the model system in the domain being considered above.

2.1.2 Positivity of solutions

Lemma 1 *Let the initial data be S(0) ≥ 0, I(0) ≥ 0, C(0) ≥ 0 and B(0) ≥ 0 belong to the domain Q, then the solution set { S(t), I(t), C(t), B(t) } of the model is positive for all t ≥ 0.*

Proof

Considering the first equation of the model system (2.1) we obtain

$$\frac{dS}{dt} = \pi - \beta_1 S\frac{B}{K+B} - (1-\alpha)\beta_2 SI - \mu S$$

$$\frac{dS}{dt} \geq -\mu S \qquad (3.10)$$

$$\int \frac{dS}{S} \geq -\int \mu dt \Rightarrow S(t) \geq S(0)e^{-\mu t} \geq 0$$

From the second equation;

$$\frac{dI}{dt} = \beta_1 S\,\frac{\mathrm{B}}{\mathrm{K}+\mathrm{B}} - (1-\alpha)\beta_2 \mathrm{SI} - (\mathrm{r}+\mathrm{m}+\mu+\alpha)\mathrm{I}$$

$$\frac{dI}{dt} \geq -(r+m+\mu)I \qquad (2.11)$$

Integrating both sides;

$$\int \frac{dI}{I} \geq -\int (r+m+\mu)dt \Rightarrow I(t) \geq I(0)e^{-(r+m+\mu)t} \geq 0$$

Using the third equation

$$\frac{dC}{dt} = \alpha I - (\rho+\mu)C$$

$$\frac{dC}{dt} \geq -(\rho+\mu)C \qquad (2.12)$$

Integrating both sides;

$$\int \frac{dC}{C} \geq -\int (\rho+\mu)dt \Rightarrow C(t) \geq C(0)e^{-(\rho+\mu)t} \geq 0$$

From the fifth equation;

$$\frac{dB}{dt} = (1-\alpha)\xi I - \delta B.$$

$$\frac{dB}{dt} \geq -\delta B \qquad (2.13)$$

Integrating both sides gives;

$$\int \frac{dB}{B} \geq -\int \delta dt \Rightarrow B(t) \geq B(0)e^{-\delta(t)} \geq 0$$

Therefore, the solution set for all the four equations is positive in Q for all t ≥ 0.

2.2 Model Analysis

Here we shall investigate the existence and stability of equilibria of model (2.1)

2.2.1 Existence and stability of Equilibrium points

Let $E(S^*, I^*, C^*, B^*)$ be the equilibrium points of the model. Solving the right-hand side of the model system (3.1) by equating it to zero, we get the following relevant equilibria.

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dB}{dt} = 0 \tag{2.14}$$

Consequently, equation (3.14) results to the following equations;

$$\begin{aligned} 0 &= \pi - \beta_1 S \frac{\mathrm{B}}{\mathrm{K}+\mathrm{B}} - (1-\alpha)\beta_2 \mathrm{SI} - \mu \mathrm{S} \\ 0 &= \beta_1 S \frac{\mathrm{B}}{\mathrm{K}+\mathrm{B}} + (1-\alpha)\beta_2 \mathrm{SI} - (\mathrm{r}+\mathrm{m}+\mu+\alpha)\mathrm{I} \\ 0 &= \alpha I - (\rho + \mu)C \\ 0 &= (1-\alpha)\xi I - \delta \end{aligned} \tag{2.15}$$

2.2.2 The Disease-free Equilibrium Points E_0

Disease free equilibrium points (DFE) in this context is a situation where there is no cholera outbreak. Hence the following variables will be I* = 0, C* = 0, B* = 0.

At this point, only equation one remains with the reduced form.

$$0 = \pi - \mu S$$

$$\Rightarrow -\pi = -\mu S$$

$$\Rightarrow S^* = \frac{\pi}{\mu}$$

Therefore, *E_0 = (S*, 0, 0, 0).*

Or $E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$.

2.2.3 The Basic Reproduction Number, R_o

The basic reproduction number denoted by R_0, defined as the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness, we shall calculate R_0 using the next generation operator approach as described by Van den Driessche and Watmough [16]. The infected population is captured in the following system of equations.

$$\frac{dI}{dt} = \beta_1 S\frac{B}{K+B} + (1-\alpha)\beta_2 SI - (r+m+u+\alpha)$$
$$\frac{dC}{dt} = \alpha I - (\rho+\mu)C \quad (2.16)$$
$$\frac{dB}{dt} = (1-\alpha)\xi I - \delta B$$

From (2.16) we define F_i and V_i as

$$F_i = \begin{bmatrix} \beta_1 S\frac{B}{K+B} + (1-\delta)\beta_2 SI \\ 0 \\ 0 \end{bmatrix} \quad (2.17)$$

And

$$V_i = \begin{bmatrix} (r+m+\mu+\alpha)I \\ (\rho+\mu)C - \alpha I \\ \delta B - (1-\alpha)\xi I \end{bmatrix} \quad (2.18)$$

We obtain the partial derivative of (3.17) with respect to *I, C, B*, and evaluating at disease-free point we have;

$$F = \begin{bmatrix} (1-\alpha)\frac{\beta_2\pi}{\mu} & 0 & \frac{\beta_1\pi}{\mu k} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (2.19)$$

Similarly the Jacobian matrix of *V* is found by partial differentiation of (3.18) to give;

$$V = \begin{bmatrix} (r+m+\mu+\alpha) & 0 & 0 \\ -\alpha & (\rho+\mu) & 0 \\ -(1-\alpha)\xi & 0 & \delta \end{bmatrix} \quad (2.20)$$

Note: the determinant of *V* is $(r+m+\mu+\alpha)(\rho+\mu)\delta$

We now find the inverse of *V*, that is;

$$V^{-1} = \begin{bmatrix} \frac{1}{(r+m+\mu+\alpha} & 0 & 0 \\ \frac{\alpha}{(r+m+\mu+\alpha)(\rho+\mu)} & \frac{1}{(\rho+\mu)} & 0 \\ \frac{(1-\alpha)\xi}{(r+m+\mu+\alpha)\delta} & 0 & \frac{1}{\delta} \end{bmatrix} \quad (2.21)$$

We further compute FV^{-1}, which gives;

$$FV^{-1} = \begin{bmatrix} \frac{(1-\alpha)\beta_2\pi}{\mu(r+m+\mu+\alpha)} + \frac{(1-\alpha)\xi(\beta_1\pi)}{k\mu(r+m+\mu+\alpha)\delta} & 0 & \frac{\beta_1\pi}{\mu k}\frac{1}{\delta} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (2.22)$$

R_0 is the spectral radius of FV^{-1}

Hence; $R_o = \frac{(1-\alpha)\beta_2\pi}{\mu(r+m+\mu+\alpha)} + \frac{(1-\alpha)\xi\beta_1\pi}{\mu k(r+m+\mu+\alpha)\delta}$ (2.23)

3. LOCAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

To achieve the local stability of the disease-free equilibrium, we will linearize the model system (2.1), and compute its Jacobian matrix. This Jacobian matrix can be determined by partially differentiating each of the equations of (2.1) with respect to the state variables S, I, C, B. System (2.1) can be redefined using $x_1,\ x_2,\ x_3,\ x_4$ to represent the state variables.

Then we have;

$$x_1 = \pi - \beta_1 S\frac{B}{K+B} - (1-\alpha)\beta_2 SI - \mu S$$

$$x_2 = \beta_1 S\frac{B}{K+B} + (1-\alpha)\beta_1 SI - (r+m+\mu+\alpha)I \quad (3.1)$$

$$x_3 = \alpha \mathrm{I} - (\rho+\mu)\mathrm{C}$$

$$x_4 = (1-\alpha)\xi I - \delta B$$

The Jacobian matrix (D) for $x_1,\ x_2,\ x_3,\ x_4$ is as shown below.

$$\mathrm{D} = \begin{bmatrix} -\frac{\beta_1 B(K+B)}{(K+B)} - (1-\alpha)\beta_2 I - \mu & -(1-\alpha)\beta_2 S & 0 & -\left(\frac{K\beta_1 S}{(K+B)^2}\right) \\ \frac{\beta_1 B(K+B)}{(K+B)} + (1-\alpha)\beta_2 I & (1-\alpha)\beta_2 S - (r+m+\mu+\alpha) & 0 & \frac{K\beta_1 S}{(K+B)^2} \\ 0 & \alpha & -(\rho+\mu) & 0 \\ 0 & (1-\alpha)\xi & 0 & -\delta \end{bmatrix} \quad (3.2)$$

At the disease-free equilibrium point E_o (3.25) will become;

$$D = \begin{bmatrix} -\mu & -(1-\alpha)\beta_1\frac{\pi}{\mu} & 0 & \frac{\beta_1\pi}{\mu k} \\ 0 & -(r+m+\mu+\alpha) + (1-\alpha)\beta_2\frac{\pi}{\mu} & 0 & \frac{\beta_1\pi}{\mu k} \\ 0 & \alpha & -(\rho+\mu) & 0 \\ 0 & (1-\alpha)\xi & 0 & -\delta \end{bmatrix} \quad (3.3)$$

The Disease-free equilibrium point E_o is locally asymptotically stable if all the real parts of eigenvalues of the Jacobian matrix (3.26) are all negative. Otherwise it is unstable.

Clearly, $-\mu$ and $-(\rho+\mu)$ are eigenvalues the remaining two eigenvalues will be found from a 2×2 matrix reduced from (3.26) as shown below;

$$D_1 = \begin{bmatrix} -(r+m+\mu+\alpha)+(1-\alpha)\beta_2\frac{\pi}{\mu} & \frac{\beta_1\pi}{\mu k} \\ (1-\alpha)\xi & -\delta \end{bmatrix}$$

The stability of D is completely determined by Routh-Hurwitz criteria

(1) Trace of $D_1 < 0$

(2) Det $D_1 > 0$

Applying these criteria, we have;

Trace $D_1 = (1-\alpha)\beta_2\frac{\pi}{\mu} - (r+m+\mu+\alpha) - \delta < 0$

Det $D_1 = (r+m+\mu+\alpha)\delta - (1-\alpha)\beta_2\frac{\pi}{\mu}\delta - \frac{\beta_1\pi}{\mu k}(1-\alpha)\xi > 0$

This is simplified to

$$1 - \left(\frac{(1-\alpha)\beta_2\pi}{\mu(r+m+\mu+\alpha)} + \frac{\beta_1\pi(1-\alpha)\xi}{\mu k(r+m+\mu+\alpha)\delta}\right) > 0$$

which leads to

$1 - R_0 > 0 \quad \Rightarrow \quad R_0 < 1$

At this point it is clearly seen that all the eigenvalues are negative. Hence, E_0 is locally asymptotically stable.

Theorem 1: *the disease-free equilibrium of the model (3.1) is locally asymptotically stable if R_0 is < 1 and unstable if $R_0 > 0$.*

4. NUMERICAL SIMULATION

Using the parameter values in table 3, we simulated the model equations to investigate the effect of hygiene consciousness in the transmission dynamics of the diseases. Results of the simulation (which was done in MATLAB) are presented in the figures below.

Table 3: Parameter values	
Parameter	**Nominal value**
π	200
μ	0.02
β_1	0.5
β_2	0.03
m	0.04
α	0.3
δ	0.228
r	0.2
ξ	10
ρ	0.38
k	1000000

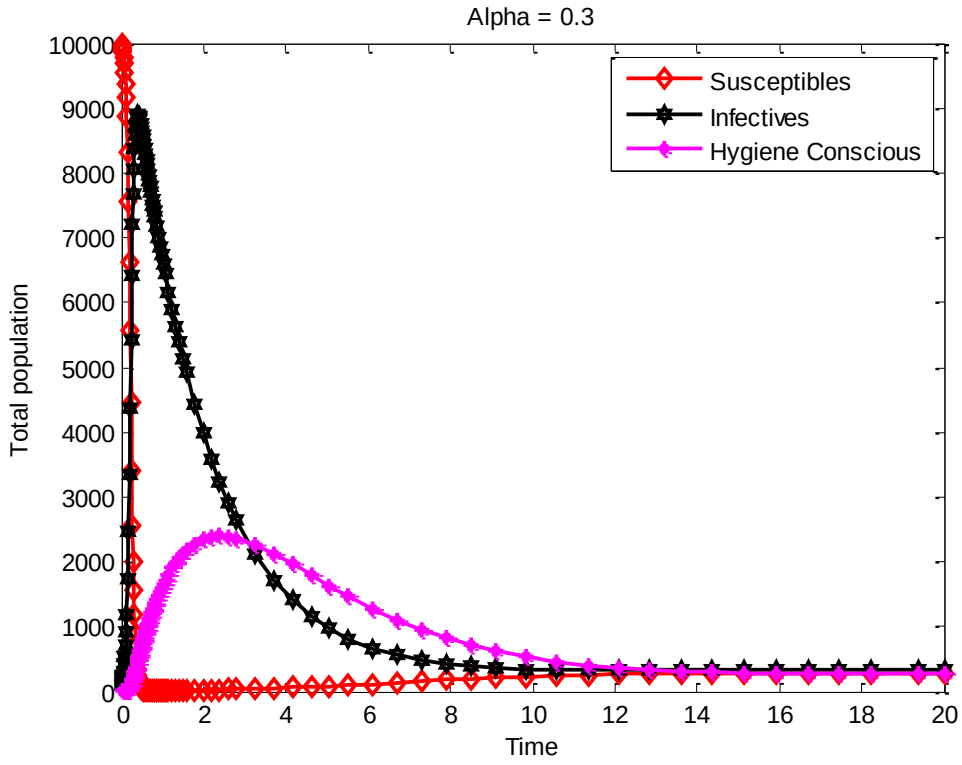


Figure 2: Simulation of model equations when $\alpha = 0.3$

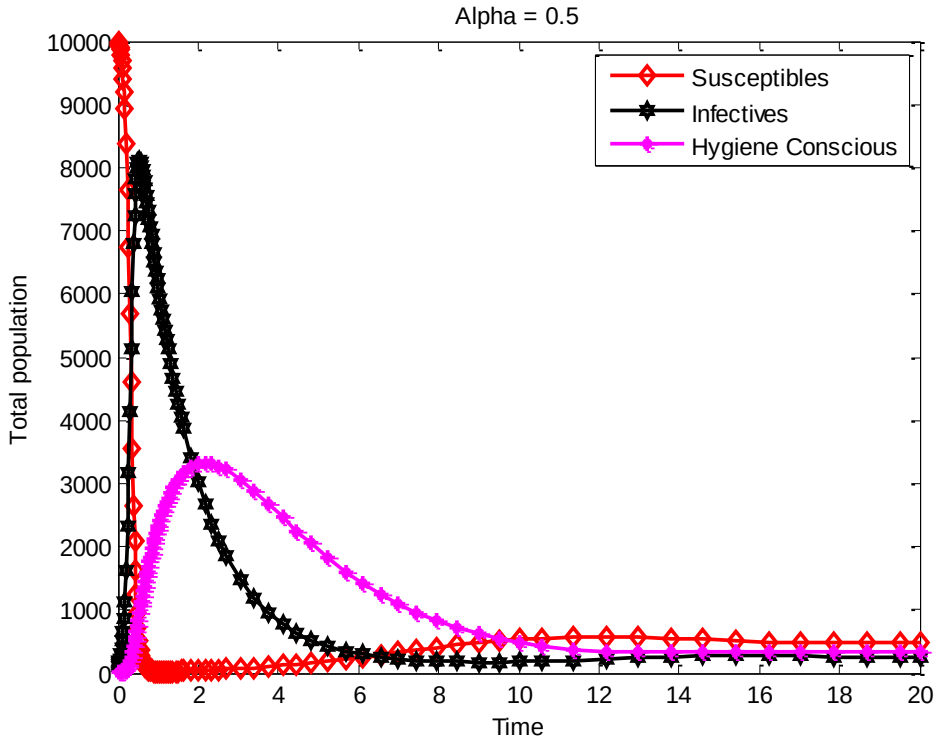


Figure 3: Simulation of model equations when $\alpha = 0.5$

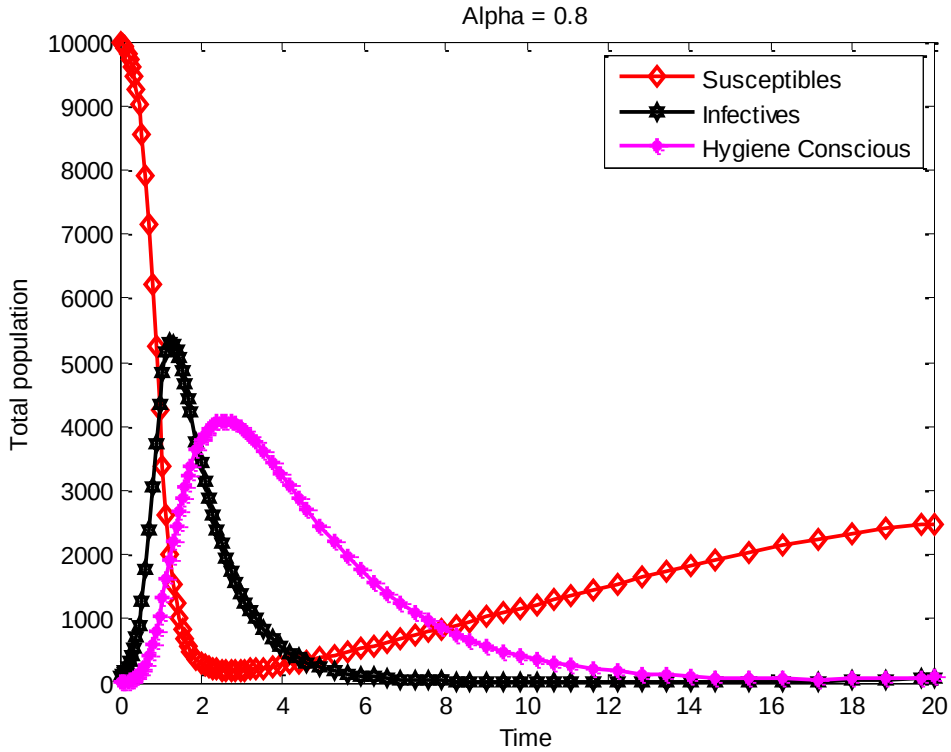


Figure 4: Simulation of model equations when $\alpha = 0.8$

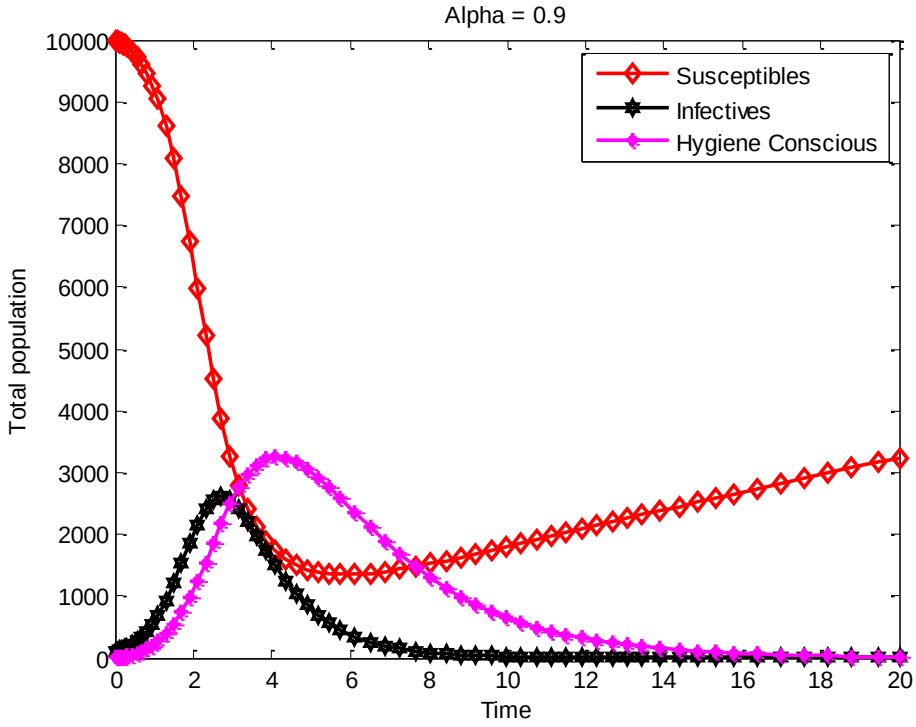


Figure 5: Simulation of model equations when $\alpha = 0.9$

5.0 CONCLUSION

In this paper, we proposed a mathematical model of cholera using hygiene consciousness as a control strategy. The model was found to be positively invariant. We derived the basic reproduction and proved that the disease free equilibrium is locally asymptotically stable. Numerical simulations (figures 2 – 5) show that increasing hygiene consciousness reduces the prevalence of the disease. We reiterated that cholera can be controlled in a hygiene conscious community.

Conflict of Interests

The authors declare that there is no conflict of interests.

REFERENCES

[1] Climate change cell, *Climate change and its impact on transmission dynamics of cholera.* Climate DoE, MoEF; Component 4b, CDMP, MoFDM. Dhaka, (2009).

[2] WHO, *Global Task forces on cholera control,* Media centre fact sheet Nº 107. Available at www.who.int/Mediacentre/factsheets/fs107/en/Accessed 10 July, 2013, (2012).

[3] Codeço C.T. Endemic and epidemic dynamics of cholera; the role of the aquatic reservoir. *BMC infectious Diseases*; 1 (2001): 1.

[4] Agarwal M. and Verma V. Modelling and Analysis of the Spread of an Infectious Disease Cholera with Environmental Fluctuations. *International journal of Applications and Applied mathematics*; 7(1) (2012), 406-425.

[5] Hethcote H. W., Zhien M. and Shengbing L. Effects of Quarantine in six endemic models for infectious disease, *Math Biosci;* 180 (2002), 141-160.

[6] Esrey S.A., Potash J.B., Roberts L. and C. Shift, Effect of Improved Water Supply and Sanitation on Ascariasis, Diarrheal,Dracuncuhasis, and Trachoma. *Bulletin of the World Health Organisation.* (1991).

[7] Curtis V. and Cairncross S. Effect of Washing Hands with Soap in Diarrheal Risk in the Community: A systematic Review. *Lancet Infectious Disease;* 3(5) (2003).

[8] Cairncross S. And Valdmanis V. *Water Supply, Sanitation, and Hygiene Promotion*; NCBI Bookshelf; A service of the National Library of Medicine, National Institutes of Health, (2006).

[9] Codeço C.T. Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir, *BMC Infect. Dis.*, 1(1), (2001).

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

[11] Hartley, D.M., Morris J.G. Jr. and Smith, D.L. Hyperinfectivity: A critical Eelement in the ability of V. cholerae to cause epidemic, *Plos Medicine*, 3(2006), 63 – 69.

[12] Mukandavire Z., Liao S., Wang J., Gaff H., Smith D.L. and Morris, J.G. Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, *PNAS* Early edition, 108 (2010), 8767-8772.

[13] Wang J. and Liao S. A generalized cholera model and epidemic/endemic analysis. *Taylor & Francis, Online;* 6(2) (2010), 568-589.

[14] Wang J. and Liao S. Stability analysis and application of a mathematical cholera model. *Mathematical Bioscience and Engineering, 8(3)* (2011), 733-752.

[15] Tian J.P.and Wang J., ‘’Global Stability for Cholera Epidemic Models’’. *Math. Bio-sc,* 232(2011), 31-41

[16] Van Den Driessche P.and Watmough J. Reroduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission,*Mathematical Biosciences*, 180 (2002), 29-48.