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Global Stability for Cholera epidemic model in Nigeria

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ABSTRACT

Cholera is a water and food borne infectious disease caused by the gram-negative bacterium, Vibrio cholera. Its dynamics are highly complex owing to the coupling among multiple transmission pathways and different factors in pathogen ecology. In this paper, we conduct global stability analysis for the deterministic cholera epidemic model. Our model is incorporating both human population and pathogen V.cholerae concentration. This follows a slight modification as compared to previous cholera models. We employ a system of nonlinear ordinary differential equations for our model. The model is analyzed and results presented in this paper make building blocks towards a comprehensive study and deeper understanding of the fundamental mechanism in cholera dynamics.

Keywords: Global Stability, Cholera, Mathematical model.

INTRODUCTION

Cholera is an acute intestinal infectious disease caused by ingestion of contaminated food and water with Vibrio Cholerae bacterium. Among the 200 Serogroup of V.Cholera, it is only V.cholerae 01 and 0139 that are known to be the cause of cholera disease. Vibrio cholerae is a motile gram negative curved-rod bacterium with polar flagellum that causes cholera in human. Cholera is characterized, in its most severe form, by the sudden onset of profuse watery diarrhea and leg pain. It has been found that transmission transpires mostly via absorption of contaminated drinking water or food. Worldwide, almost every year there is an estimated 3-5 million cholera cases and 100, 000-130, 000 deaths due to cholera a year as of 2010[15]. It has a very short incubation period which starts from a few hours to five days. The health of an infected person disintegrates rapidly and death may occur if treatment is not promptly given. Cholera was first discovered in the Indian subcontinent in 1817. The disease reaches all the way through Asian continent in the 1960s, getting in to Africa in 1970 and Latin America in 1991 [4]. In many parts of Africa and Asia the disease is still endemic.

Cholera is a disastrous water-borne infectious disease that is caused by the bacterium vibrio cholera. It is a very serious problem in many developing countries due to inadequate access to safe drinking water supply, improper treatment of reservoirs and improper sanitation. In 2012, WHO reported 245, 393 cholera cases and 3034 death cases across 48 countries in which 67% cases occurred in African countries [16]. In 2005, Nigeria had 4, 477 cases and 174 deaths. There were reported cases of cholera in 2008 in Nigeria in which there were 429 deaths out of 6, 330 cases. Furthermore, 2, 304 cases were reported in Niger State in which 114 were death cases [7]. Also in 2009, Nigeria reported 13, 691 cases and 431 deaths [14]. [12][11] evidenced that recent years have seen a strong trend of cholera outbreak in developing countries, such as in India (2007), Iraq (2008), Congo (2008), Zimbabwe (2008-2009), Haiti (2010), Kenya (2010) and Nigeria (2010).

In Nigeria, outbreaks of the disease have been taking place with ever-increasing occurrence ever since the earliest outbreak in recent times in 1970, and more recently there were outbreaks of Cholera in the Northern parts of Nigeria (WHO 2018) [5]. In summary the United Nation (UN) unit, reports: "despite Nigeria's oil wealth, more than 70% of the country's 126 million people live below the poverty line and cholera outbreaks are common in poor urban areas which lack proper sanitation and clean drinking water" (UN Office for the Coordination of Humanitarian Affairs Integrated Regional Information Networks (IFIN) 2005). In the last few decades,[2],[4],[10], [16],[6],[1],[13],[18],[12] and [9], have designed mathematical models to explore the transmission dynamics and control of the disease. However, there have not been many studies on cholera in Nigeria using mathematical modeling. Hence we decided to apply mathematical model to study the disease without natural recovery.

Model formulation

A mathematical model for cholera transmission is developed by making some relevant modifications to the previous models [2]. Our model is devoid of natural recovery, this is because cholera is a fatal disease, and a large percentage of those who recover, do so because of treatment. Our model on the other hand incorporates treatment, water hygiene and environmental waste management. The model contains five variables which are susceptible, infected, recovered, total human population, and the concentration of vibrio cholera in water. The susceptible population is generated either through birth or through immigration. They acquire infection and move to the infected class at the rate:

$$\lambda = \frac{aB}{K+B}$$

Figure 1: A schematic representation of our model is given below: The model consists of the following system of ordinary differential equations given in (1) to (4):

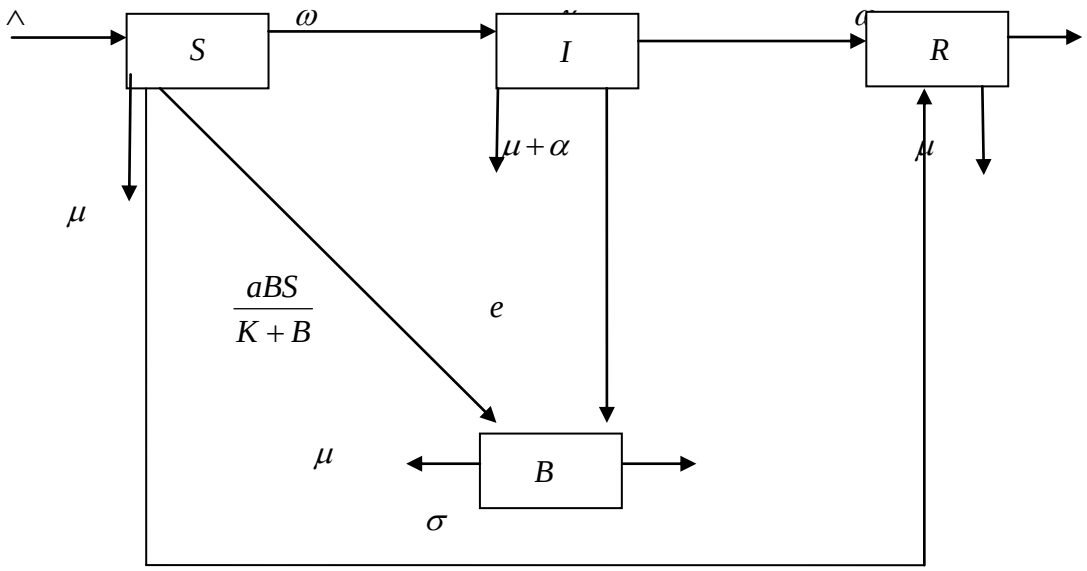

Fig.1 Schematic diagram of controlled cholera transmission model without natural recovery.

Table-1 Variables of the cholera model

Symbol	Description
$S(t)$	Susceptible human population at time t
$I(t)$	Infected human population at time t
$R(t)$	Recovered human population at time t
$B(t)$	The pathogen population at time t
N	Total population of humans

Table-2 Parameters of the cholera model

Symbol	Description
$\wedge$	Recruitment rate
μ	Per capita natural death rate for humans
a	Rate of exposure to contaminated water
K	Concentration of vibrio cholera in water
α	Death rate of V.cholerae in aquatic environment
γ	Natural recovery rate
ω	Loss rate of immunity by recovered individuals
e	Contribution of each infected person to the population of vibrio cholera in the aquatic environment
λ	Force of infection for susceptible

The model consists of the following system of ordinary differential equations given in (1) to (4):

$$\frac{dS}{dt} = \wedge - \frac{aB}{K+B}S - (\omega + \mu)S \quad 1$$

$$\frac{dI}{dt} = \frac{aB}{K+B}S - (\gamma + \mu + \alpha)I \quad 2$$

$$\frac{dB}{dt} = eI - (\alpha + \mu)B \quad 3$$

$$\frac{dR}{dt} = \gamma I + \sigma \wedge - (\omega + \mu)R \quad 4$$

Where N=S+I+B+R

Epidemic dynamics

Existence of disease-free equilibrium point (DFE) for the model is given by

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dB}{dt} = \frac{dR}{dt} = 0 \quad 5$$

So equation (1-4) take the form

$$0 = \wedge - \frac{aB}{K+B}S - (\omega + \mu)S \quad 6$$

$$0 = \frac{aB}{K+B}S - (\gamma + \mu + \alpha)I \quad 7$$

$$0 = eI - (\alpha + \mu)B \quad 8$$

$$0 = \gamma I + \sigma \wedge - (\omega + \mu)R \quad 9$$

From 8

$$B = \frac{eI}{(\alpha + \mu)} \quad 10$$

Substitute 10 into 7

Let $E(S^0, I^0, B^0, R^0)$ be the equilibrium point of the mode l(1-4)

The equilibrium points can be derived by setting the right hand side equal to zero That is:

$$a\frac{\left(\frac{eI}{\alpha+\mu}\right)s}{K+\frac{eI}{\alpha+\mu}}-(\gamma+\mu+\alpha)I=0 \quad 11$$

Either $$a\frac{\left(\frac{eI}{\alpha+\mu}\right)s}{K+\frac{eI}{\alpha+\mu}}-(\gamma+\mu+\alpha)=0 \quad 12$$

OR $$I=0 \quad 13$$

Substitute 13 into 8 we get

$$B=\frac{0}{\mu+\alpha}=0 \quad 14$$

From 6

$$S=\frac{\wedge}{\mu+\vee} \quad 15$$

And from 4

$$R=\frac{\gamma I+\sigma\wedge}{\omega+\mu} \quad 16$$

The DFE state is thus given by

$$\left(S^0,I^0,B^0,R^0\right)=\left[\frac{\wedge}{\mu+\vee},\,0,\,0,\,\frac{\gamma I+\sigma\wedge}{(\omega+\mu)}\right] \quad 17$$

This shows the state where there is no cholera infection and is known as the disease free equilibrium.

The effective Reproduction Number, (R_E)

The basic reproduction number denoted by R_0, is the expected number of secondary cases produced, in a completely susceptible population, by a typical infective individual. It is one of the most useful threshold parameters, which characterize mathematical problems concerning infectious diseases [6, 7].

If R_0<1, this implies that, on average an infected individual produces less than one new infected individual during the infectious period and the infection can be wiped out. Conversely, if R_0>1, then, each infected individual produces, on average, more than one new infection, and the disease are spread in the population. For a single infected compartment, R_0 is simply the product of the infection rate and the mean duration of the infection. But for complicated models, this simple

definition of R_0 is insufficient. We therefore compute the basic reproduction number R0, using the next generation operator approach by [5]

This method is described as follows:

Assume that there are n compartments so that the first m compartments correspond to infected individuals. Let $F_i(x)$ be the rates of appearance of new infections in compartment i. $V_i^+(x)$ be the rate of transfer of individuals into compartment I

$Vi = v^-{}_i(x) - v^+(x)$

One other important step is to obtain the disease-free equilibrium point x_0. We then compute matrices F and V which are mxm matrices, where m represents the infected classes, defined by $f = \left[\frac{\partial f_i}{\partial x_j},(x_0)\right]$ and

$V = \left[\frac{\partial v_i}{\partial x_j},(x_0)\right] with \le i, j \le m$ and F is nonnegative and V is a nonsingular M-matrix (a matrix with inverse, belonging to the class of positive matrices). Since F is We define f_1 & V_1 as follows

$$f_1 = \begin{bmatrix} \frac{aBs}{K+B} \\ eI \\ 0 \\ 0 \end{bmatrix}$$

by all other means, other than the epidemic. $V_i^-(x)$ be the transfer of individuals out of the compartment I.

The disease transmission model consists of the system of equations.

$xi = f_i(x) = f_i(x) - v_i(x)$

Where

nonnegative and V is nonsingular, then v^{-1} is non-negative and also fv^{-1} is non-negative. We then compute fv^{-1} matrix, defined as the next generation matrix Diekmann et al., (1990).

The basic reproductive number (reproduction ratio) R_0 is then defined as $p(fV^{-1})$ where $p(A)$ the spectral radius of matrix A is, (or the maximum modulus of the Eigen-values of A). By using the method described above, we establish local stability of the basic model using the basic reproduction number (R_0).

18

$$V_1 = \begin{bmatrix} (\gamma+\mu+\alpha)I \\ (\alpha+\mu)B \\ (\mu+\omega)S - \dfrac{aBs}{K+B} + \wedge \\ (\omega+\mu)R - \sigma\wedge + \gamma I \end{bmatrix} \quad 19$$

We then obtain the partial derivatives of (18) and (19) with respect to (B,I) and by substituting the disease-free equilibrium point (17). We obtain a 2x2 matrix since there are two infected classes, B and I.

$$F = \begin{pmatrix} \dfrac{dF}{dB} & \dfrac{dF}{dI} \\ \dfrac{dF}{dB} & \dfrac{dF}{dI} \end{pmatrix}_I^B \quad N(t) \le \frac{\pi}{\mu}$$

$$F = \begin{bmatrix} 0 & \dfrac{(aBs)(\sigma\wedge)}{K+B(\omega+\mu)} \\ 0 & \dfrac{(eI)(\sigma\wedge)}{(\omega+\mu)} \end{bmatrix} \quad 20$$

And $V = \begin{pmatrix} \dfrac{dF}{dB} & \dfrac{dF}{dI} \\ \dfrac{dF}{dB} & \dfrac{dF}{dI} \end{pmatrix}_I^B$

$$V = \begin{bmatrix} (\gamma+\mu+) & 0 \\ (\alpha+\mu)\mathrm{B} & \end{bmatrix} \quad 21$$

Taking the inverse of (21) yields

$$V^{-1} = \begin{pmatrix} \dfrac{1}{\gamma+\mu+\alpha} & \\ \dfrac{B}{(\alpha+\mu)(\gamma+\mu+\alpha)} & \dfrac{1}{(\alpha+\mu+)} \end{pmatrix} \quad 22$$

The product of (20) and (22) gives

$$FV^{-1} = \begin{pmatrix} \dfrac{(\mathrm{aBs})(\sigma\wedge)}{(\mathrm{K}+\mathrm{B})(\omega+\mu)(\gamma+\mu+\alpha)(\alpha+\mu)\mathrm{B}} & \dfrac{(\mathrm{aBs})(\sigma\wedge)}{(\mathrm{K}+\mathrm{B})(\omega+\mu)(\alpha+\mu)\mathrm{B}} \\ \dfrac{\left[(\mathrm{eI})(\sigma\wedge)\right]}{(\mu+\omega)(\gamma+\mu+\alpha\ )(\mu+\alpha)\mathrm{B}} & \dfrac{\left[(\mathrm{eI})(\sigma\wedge)\right]}{(\mu+\omega)(\alpha+\mu)\mathrm{B}} \end{pmatrix} \quad 23$$

From (23), we calculate the Eigen values to determine our effective reproduction number of our model i.e. $\left|FV^{-1}-\lambda I\right|=0$

$$\begin{pmatrix} \dfrac{\left[\ (aBs)(\sigma\wedge)\right]B}{(\mathrm{K}+B)(\gamma+\mu+\alpha)(\omega+\mu)(\alpha+\mu)B}-\lambda & \dfrac{(\mathrm{aBs})(\sigma\wedge)}{(\mathrm{K}+\mathrm{B})(\omega+\mu)(\alpha+\mu)\mathrm{B}} \\ \dfrac{(\mathrm{e}\,I)(\sigma\wedge)}{(\mu+\omega)(\gamma+\mu+\alpha)(\mu+\alpha)}B & \dfrac{(\mathrm{e}\,I)(\sigma\wedge)}{(\mu+\omega)(\mu+\alpha)\mathrm{B}}-\lambda \end{pmatrix}=0 \quad 24$$

Evaluating (24) yields

$$\left(\frac{(eI)(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B}-\lambda\right)\left(\frac{(aBs)(\sigma\wedge)}{(K+B)(\omega+\mu)(\gamma+\mu+\alpha)(\alpha+\mu)}B-\lambda\right)-$$

$$\left(\frac{(aBs)(\sigma\wedge)}{(K+B)(\omega+\mu)(\alpha+\mu)B}\right)\left(\frac{(eI)(\sigma\wedge)}{(\omega+\mu)(\gamma+\mu+\alpha)(\alpha+\mu)}\right)=0 \quad 25$$

$$\left(\frac{(eI)(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B}\right)\frac{(eI)(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B}$$

$$\left(\frac{(aBs)(\sigma\wedge)}{K+B(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)}\right)-\frac{(aBs)(\sigma\wedge)}{K+B(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)}$$

$$+\lambda^2-\left(-\frac{(aBs)(\sigma\wedge)}{K+B(\omega+\mu)(\alpha+\mu)B}\right)\quad\left(\frac{(\mathrm{eI})(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)}\right)=0 \quad 26$$

From 26 we obtain

$$\lambda^2 - \frac{(\text{eI})(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B}\lambda - \frac{(aBs)(\sigma\wedge)}{K+B(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)}\lambda = 0 \quad 27$$

$$\lambda^2 - \left\{\frac{(eI)(\sigma\wedge)(\gamma+\mu+\alpha)(\text{aBs})(\sigma\wedge)}{(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)(K+B)}\right\}\lambda = 0 \quad 28$$

$$\lambda\left(\lambda - \frac{\{(eI)(\sigma\wedge)(\gamma+\mu+\alpha)+(\text{aBs})(\sigma\wedge)\}}{(\omega+\mu)(\alpha+\mu)B(\gamma+\mu+\alpha)(K+B)}\right) = 0 \quad 29$$

From 29 either

$$\lambda_1\left(\frac{\{eI\sigma\wedge(\gamma+\mu+\alpha)+aBs\sigma\wedge\}}{\omega+\mu(\alpha+\mu)B(\gamma+\mu+\alpha)(K+B)}\right)$$

$and\ \lambda_2 = 0$

Clearly, λ_1 is the dominant Eigen-value and therefore becomes the effective reproduction number (RE) of the model (1) - (4).

$$\therefore\ RE = \left(\frac{\{eI\sigma\wedge(\gamma+\mu+\alpha)+aBs\sigma\wedge\}}{\omega+\mu(\alpha+\mu)B(\gamma+\mu+\alpha)(K+B)}\right) \quad 30$$

LOCAL STABILITY OF THE DISEASE FREE EQUILIBRIUM

In order to obtain conditions for the local stability of the disease free equilibrium state, we re-write equations (1-4) as follows:

$$f_1 = \wedge - \frac{aBS}{K+B} - (\mu+\omega)S \quad 31$$

$$f_2 = \frac{aBS}{K+B} - (\gamma+\mu+\alpha)I \quad 32$$

$$f_3 = eI - (\alpha+\mu)B \quad 33$$

$$f_4 = \gamma I + \sigma\wedge - (\omega+\mu)R \quad 34$$

We now obtain the partial derivatives of f_1, f_2, f_3, f_4 with respect to S,B,I and R.

$$\frac{\partial f_1}{\partial S} = -\mu - \omega\,,\ \ \frac{\partial f_1}{\partial B} = \frac{a}{K},\ \ \frac{\partial f_1}{\partial I} = 0,\ \ \frac{\partial f_1}{\partial R} = 0$$

$$\frac{\partial f_2}{\partial S} = \frac{aB}{K+B},\ \ \frac{\partial f_2}{\partial B} = \frac{as}{K},\ \ \frac{\partial f_2}{\partial I} = (\gamma + \mu + \alpha),\ \ \frac{\partial f_2}{\partial R} = 0$$

$$\frac{\partial f_3}{\partial S} = 0,\ \frac{\partial f_3}{\partial B} = \alpha + \mu,\ \frac{\partial f_3}{\partial I} = e,\ \frac{\partial f_3}{\partial R} = 0$$

$$\frac{\partial f_4}{\partial S} = 0,\ \frac{\partial f_4}{\partial B} = 0,\ \frac{\partial f_4}{\partial I} = \gamma,\ \frac{\partial f_4}{\partial R} = \omega + \mu$$

We substitute the above partial derivatives into the Jacobian matrix below

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial B} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial B} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial B} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial B} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \end{bmatrix} \qquad 35$$

That is:

$$J = \begin{bmatrix} -\mu - \omega & \frac{a}{K} & 0 & 0 \\ \frac{aB}{K+B} & \frac{aS}{K} & \gamma + \mu + \alpha & 0 \\ 0 & \alpha + \mu & e & 0 \\ 0 & 0 & \gamma & \omega + \mu \end{bmatrix} \qquad 36$$

$$J(E_0) = \begin{bmatrix} -\mu - \omega & K & 0 & 0 \\ \frac{aB}{K+B} & \frac{a\wedge}{(\mu + \wedge)K} & \gamma + \mu + \alpha & 0 \\ 0 & e + \mu & -\lambda + e & 0 \\ 0 & 0 & \gamma & -\lambda + \omega + \mu \end{bmatrix} \qquad 37$$

From 37 we obtain

$$-(\lambda+\mu+\omega)\left(\frac{\lambda-a\wedge}{(\mu+\vee)K}\right)\left[-\lambda^2+(e+\omega+\mu)\lambda+(e+\mu+\gamma+\mu+\alpha)e-\lambda+\omega+\mu-e\frac{aB}{K+B}e\gamma\right]=0\ 3$$

With $\lambda_1=-(\mu+\omega)$, $\lambda_2=\frac{a\wedge}{(\mu+\vee)K}$,

We obtain $\lambda_3 \& \lambda_4$ from the quadratic below

$$\lambda^2-(\omega+\mu)\lambda+e+\lambda+\omega+\mu+e\frac{aB}{K+B}+e\gamma-e+\mu+\gamma+\mu+\alpha=0 \qquad 39$$

From 39 we obtain

$$\lambda_3,\lambda_4=\frac{1}{2}\lambda+\frac{1}{2}\mu\pm\frac{1}{2}\sqrt{2\omega+2\omega\mu+\mu^2-\mu 2+2\mu e+e^2}$$

It is not clear whether λ_3 *and* λ_4 are less or greater than zero. We therefore, conclude that the disease free equilibrium state is locally asymptotically stable if λ_3 *and* λ_4 are less than zero and unstable if otherwise.

$$\frac{dZs}{di}=x\left(Zs-Z_{DFEs}\right)+x_1z_i$$
$$\frac{dZi}{dt}=x_2z_i$$

Where Zs is the vector representing the non-transmitting compartments and Zi is the vector representing the transmitting compartments. The DFE is globally

$Z_i=(B,I), Zs=(S,I,B,R)$

Global stability of the disease-free equilibrium E_o

Analyzing the global stability of the disease free equilibrium point we use (8) approach

We write model system 1-4 in the form

asymptotically stable if A has real negative eigenvalues and X_2 is a Metzler matrix.

From system 1-4 we have

$$Zs - Z_{DFEs} = \begin{bmatrix} S - \dfrac{\wedge}{\mu + \vee} \\ R - 1 + \dfrac{\wedge}{\mu + \vee} \end{bmatrix} \quad 40$$

We check if the non-transmitting compartments has real negative eigenvalues and that X_2 is a Metzler matrix

From equation 1-4 the non-transmitting compartments are:

$$x = \begin{bmatrix} \dfrac{as}{K} & \gamma + \mu + \alpha & 0 \\ \alpha + \mu & e & 0 \end{bmatrix} \quad 41$$

$$x_1 = \begin{bmatrix} \dfrac{a}{K} & 0 \\ 0 & \gamma \end{bmatrix} \quad 42$$

$$x_2 = \begin{bmatrix} \dfrac{as}{K} & \gamma + \mu + \alpha \\ \alpha + \mu & e \end{bmatrix} \quad 43$$

Our direct computation shows that the eigenvalues of x are real and negative.

Thus, the DFE is globally asymptotically stable.

This implies that the system $\dfrac{\partial Z_i}{\partial t} = x\left(Zs - Z_{DFEs}\right) + x_1 Z_i$ is globally asymptotically stable at DFE .
More so, since $0 \le i < 1$we have, $\left(1 - i\right) > 0$and this implies x_2 a Metzler matrix.

The disease-free equilibrium point is globally asymptotically stable in Ω if $R_e < 1$and unstable if $R_e > 1$.

CONCLUSION

Nigeria is endemic to Cholera and has being rated as one of the countries in West Africa with the high transmission rate of cholera, still all hope is not lost because from our model, we got the the Reproduction number is less than one $R_0 < 1$,which simply implies that cholera can still be eradicated from the country.

Gradually the infection will surely die off since then $R_0 < 1$,the model is globally asymptotically stable at DFE. This means that irrespective of the initial population the disease can carefully eradicated provided $R_0 < 1$.

REFERENCES

1.Bertuzzo, E., S. Azaele, A. Maritan, M. Gatto, I. Rodriguez-Iturbe and A. Rinaldo, 2008. On the space-time evolution of a cholera epidemic. Water Resource. Res., 44(1): W01424.

2. Capasso,V. and S.L. Paveri-Fontana, 1979. A mathematical model for the 1973 cholera epidemic in the European Mediterranean region. Rev. Epidemiol. Sante, 27(2): 121.

3.CIA (Central Intelligence Agency), 2011. World Factbook. Retieved from: http://www.cia.gov/ library/publications/the world factbook/geos/ni.html.

4.Codecco, C.T., 2001. Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir. BMC Infect. Dis., 1(1).

5.Epstein, P.R., 1993. Algal blooms in the spread and persistence of cholera. Biosystems, 31(2): 209- 221.

6.Jensen, M.A., S.M. Faruque, J.J. Mekalanos and B.R. Levin, 2006. Modeling the role of bacteriophage in the control of cholera outbreaks. P. Natl. Acad. Sci. USA, 103(12): 4652-4657.

7.NBS (National Bureau of Statistics), 2009. Social Statistics in Nigeria. Retrieved from: http://www. nigerianstat.gov.ng/pages/latestreleases.

8.N. Nyerere, L. S. Luboobi and Y. Nkansah-Gyekye.Bifurcation and Stability analysis of the dynamics of Tuberculosis model incorporating, vaccination, Screening and treatment. *Communications inMathematical biology and* Neuroscience, Vol. 2014 (2014), Article ID 3.

9.Misra, A.K. and V. Singh, 2012. A delay mathematical model for the spread and control of water borne diseases. J. Theorem. Biol., 301: 49-56.

10.Pascual, M., M.J. Bouma and A.P. Dobson, 2002. Cholera and climate: Revisiting the quantitative evidence. Microbes. Infect., 4: 237-245.

11.Sulayman Fatima, Isthrinayagy Krihnarajah, M.Z.A.M Jaffar, Mohd Bakri Adam: A mathematical model for the control of cholera in Nigeria. Research Journal of Environmental and Earth science 6(6):321-325,2014.

12.Tian, J.P. and J. Wang, 2011. Global stability for cholera epidemic models. Math. Biosci., 232(1): 341.

13.Tien, J.H. and D.J.D. Earn, 2010. Multiple transmission pathways and disease dynamics in a waterborne pathogen model. Bull. Math. Biol., 72: 1506-1533.

14.WHO (World Health Organization), 2012. Global Task on Cholera Control. Cholera Country Profile:Nigeria.Retrievedfrom: http://www.who.int/ cholera/countries/NigeriaCountryProfile2011.pdf.

15.WHO (World Health Organization), 2013a. Cholera 2012 wkly. Epidemiol. Rec., 88(31): 321-336.

16.WHO (World Health Organization), 2013b. Media Center: Cholera. Retrieved from:http://www.who. int/media centre/factsheets/fs107/en.

17.Hartley, D.M., J.B. Morris and D.L. Smith, 2006. Hyper infectivity: A critical element in the ability of V. cholerae to cause epidemics. Plos Med., 3(1): e7.

18.Hove-Musekwa, S.D., F. Nyabadza, C. Chiyaka, P. Das, A.Tripathi and Z. Mukandavire, 2011. Modeling and analysis of the effects of malnutrition in the spread of cholera. Math. Comput. Model., 53(9):1583-1595.

19.HethcoteH. Zhien.M and Shengbing,L.(2002). Effect of quarantine in six endemic models for infectious disease. Mathematical Bioscience 180:141.160