Stability Analysis of a Cholera Model

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Abstract: **In this paper, a cholera model has been formulated. Using Lipchitz condition, the efficacy of the model was ascertaine by testing for the existence and uniqueness of the solution of the model. The disease free equilibrium (DFE), the endemic equilibrium (EE) and the local asymptotic stability of the DFE were also conducted. Using the next generation matrix approach, the basic reproduction number, R_0 was derived. The DFE was proved to be locally asymptotically stable when $R_0 < 1$.**

Keywords: **existence and uniqueness of solution, Lipchitz condition, Cholera disease, local stability**

Table 1: Description of parameters for the model

Parameter	Description
b	Per capital birth rate of humans
μ	Per capital natural death rate of humans
a	Rate of exposure to contaminated water
K	Concentration of vibrio cholera in water
η	Natural recovery
τ	Recovery due to treatment
c	Rate of compliance with water hygiene
β	Rate of compliance with environmental sanitation
δ	Cholera-induced death rate
m	Growth rate of vibrio cholera in the aquatic environment
ω	Loss rate of immunity by recovered individuals
e	Contribution of each infected person to the population of vibrio cholera in the aquatic environment

I. INTRODUCTION

Cholera is an acute diarrhoeal disease caused by *Vibro Cholerae* bacterium. It has continued to be a global threat to public health and a key indicator of lack of social development (Adagbada *et al.*, 2012). Based on a report by the Nigeria Centre for Disease Control (NCDC) in 2019, there have been seven pandemics of Cholera worldwide, the last of which began in Indonesia in 1961, with an estimate of between 1.3 to 4.0 million cases and 21,000 to 143,000 deaths globally due to Cholera every year. The World Health Organization (WHO) reported that only 5 - 10% of the actual cases have been reported especially in low and middle income countries probably due to poor surveillance systems and inadequate disease notification system. The transmission of the bacteria is majorly through ingestion of contaminated food or water as a result of inadequate access to clean water and poor sanitation, especially in peri-urban slums where basic infrastructures are not available. Severe cases of Cholera can lead to death within few hours due to dehydration and the fertility ratio can be up to 50% especially in people without access to treatment which drops to about 1% with adequate treatment. People with low immunity such as malnourished children or people living with HIV are at a greater risk of death if infected [NCDC, 2019]. In Nigeria, Cholera occurs annually mostly during the rainy season, especially in areas with poor sanitation with the first series of the outbreak reported between 1970 and 1990. Major epidemics also occurred in 1992, 1995-1996 and 1997. The Federal ministry of health reported 37,289 cases and 1434 deaths between January and October 2010. NCDC reported 42,466 suspected cases including 830 deaths with a fatality rate of 1.95% from 20 out of 36 states from January to October 2018.

Dynamics of Cholera model have been studied by many authors. For instance, Capasso and Serio (1978) introduced an incidence rate in the form $KSI/(I+\propto I)$ with human to human transmission model only. Codeco (2001) proposed an incidence form of $aSB/(K+B)$ with environment to human transmission model with the incorporation of pathogen concentration into the Cholera model. Liao and Wang (2012) generalized Codeco (2001) models by incorporating the theory of Volterra-Lyapunov stable matrices into the classic method of Lyapunov functions. The model of Liao and Wang (2012) was improved by Cui *et al.* (2014) in two ways. Firstly, they added a parameter d to describe the rate of disease-related death and secondly, they proposed a proportion of the vaccination in susceptible individuals. Several mathematical models for the transmission of Cholera have been proposed by Capasso and Paveri-Fontana (1979), Capone *et al.* (2015), Mwasa and Tchuenche (2011) and Shuai *et al.* (2012). In 2014, Isere *et al.* examined two formulated model, one without the control terms and the other with control terms that are time-dependent. They carried out simulations of the model to see the effect of those controls in the population dynamics of the disease. Also in 2014, Sulayman *et al.* presented and analysed a mathematical model for the control of Cholera in Nigeria with modifications as compared to previous Cholera models. They incorporated treatment, water hygiene and environmental sanitation in controlling the disease. Lemos-Palao *et al.* (2018) proposed a SITRV(Susceptible-Infectious-Treated-Recovered-Vaccinated) type model which included a class of bacteria concentration. They modified the model of Lemos-Palao et al. (2017) by adding a vaccination class and considering different kinds of Cholera's treatment. Chin and Kimbir (2018) extended a mathematical model by Codeco (2001) by varying

the net reproduction rate of humans and allowing for susceptibility of recovered individuals.

This paper presents a stability analysis of the model of Sulayman *et al*. (2014) by testing for the existence and uniqueness of the solution of the model and proving that the DFE is locally asymptotically stable when $R_0 < 1$ and that the disease will always die out.

II. DESCRIPTION AND FORMULATION OF THE MODEL

This model comprises of four compartments namely: The Susceptible (S) which is generated either through birth or immigration, the Infected (I), the Recovered (R) and the concentration of vibrio cholera in contaminated water (B). It incorporates treatment, water hygiene and environmental sanitation. The susceptible population acquire infection and move to the infected class at the rate $a(1-c)B/(K+B)$ where a is the rate of exposure to contaminated water, c is the rate of compliance with water hygiene, K is the concentration of vibrio cholera that yields 50% chance of catching cholera (Codeco, 2001) and B is the concentration of vibrio cholera in contaminated water. Thus the following system of ordinary differential equations describe the model:

$$\frac{ds}{dt} = bN - \frac{a(1-c)B}{K+B}S + \omega R - \mu S \quad (1)$$

$$\frac{dI}{dt} = \frac{a(1-c)B}{K+B}S - (\eta + \tau + \mu + \delta)I \quad (2)$$

$$\frac{dR}{dt} = (\eta + \tau)I - (\omega + \mu)R \quad (3)$$

$$\frac{dB}{dt} = e(1-\beta)I - mB \quad (4)$$

where

$$N = S + I + R.$$

2.1 Existence and Uniqueness of solution

To ascertain the existence and uniqueness of the mathematical model represented by the system of equations (1) – (4), Lipchitz condition shall be used. Let the system be given as follows:

$$P_1 = bN - \frac{a(1-c)B}{K+B}S + \omega R - \mu S \quad (5)$$

$$P_2 = \frac{a(1-c)B}{K+B}S - (\eta + \tau + \mu + \delta)I \quad (6)$$

$$P_3 = (\eta + \tau)I - (\omega + \mu)R \quad (7)$$

$$P_4 = e(1-\beta)I - mB \quad (8)$$

Theorem 1 (Derrick and Groosman, 1976): Let the region

$$|t - t_0| \le a, \| x - x_0 \| \le 1, x = (x_1, x_2, \dots \dots . x_n), x_0 = (x_{10}, x_{20}, \dots \dots . x_{n0})$$

be denoted by D and suppose that $f(t,x)$ satisfies the Lipchitz condition:

$$\|f(t,x_1) - f(t,x_2)\| \le k\|x_1 - x_2\| \quad (9)$$

whenever the pair (t,x_1) and (t,x_2) belong to D, where k is a positive constant. Then, there is a constant $\delta \ge 0$ such that there exists a unique continuous vector of $x(t)$ of the system in the interval $t - t_0 \le \delta$.

Theorem 2: Let D denote the region $0 \le \propto \le D$, then equations (5) to (8) have a unique solution.

Based on theorems 1 and 2, and taking to consideration the model equations (5) - (8), it is required that

$$\frac{\partial p_i}{\partial x_j}, i, j, = 1,2, \dots,$$

be continuous and bounded in D. Our interest is in the region $0 \le \propto \le D$ and so we look for the bounded solution in the region whose partial derivatives satisfy $p \le \propto \le 0$, where $\propto$ and δ are positive constants. We show that

$$\frac{\partial p_i}{\partial x_j}, i, j, = 1,2,3,4$$

are continuous and bounded in D. For p_1,

$$\left|\frac{\partial p_1}{\partial S}\right| = \left|-\left(\frac{a(1-c)B}{k+B} + \mu\right)\right| < \infty, \quad \left|\frac{\partial p_1}{\partial I}\right| = 0 < \infty, \left|\frac{\partial p_1}{\partial R}\right| = |\omega| < \infty,$$

$$\left|\frac{\partial p_1}{\partial B}\right| = \left|-\left(k\frac{a(1-c)}{(k+B)^2}S\right)\right| < \infty.$$

For p_2,

$$\left|\frac{\partial p_2}{\partial S}\right| = \left|\left(\frac{a(1-c)B}{k+B}\right)\right| < \infty, \left|\frac{\partial p_2}{\partial I}\right| = |-(\eta + \tau + \mu + \delta)| < \infty$$

$$\left|\frac{\partial p_2}{\partial R}\right| = 0, \left|\frac{\partial p_2}{\partial B}\right| = \left|-\left(k\frac{a(1-c)}{(k+B)^2}S\right)\right| < \infty.$$

For p_3,

$$\left|\frac{\partial p_3}{\partial S}\right| = 0, \quad \left|\frac{\partial p_3}{\partial I}\right| = |\eta + \tau| < \infty, \quad \left|\frac{\partial p_3}{\partial I}\right| = |\omega + \mu| < \infty, \quad \left|\frac{\partial p_3}{\partial I}\right| = 0$$

For p_4,

$$\left|\frac{\partial p_4}{\partial S}\right| = 0, \quad \left|\frac{\partial p_4}{\partial I}\right| = |e(1-ß)| < \infty, \quad \left|\frac{\partial p_4}{\partial S}\right| = 0, \quad \left|\frac{\partial p_4}{\partial S}\right| = |-m| < \infty.$$

Since these partial derivatives exist, continuous and are bounded, then the model (5) to (8) has a unique solution as stated by theorem 2.

2.2 Equilibrium States of the Model

The disease free equilibrum of the model equations (1) to (4) can be obtained by setting:

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

we have,

$$bN - \frac{a(1-c)B}{K+B}S + \omega R - \mu S = 0 \quad (10)$$

$$\frac{a(1-c)B}{K+B}S - (\eta + \tau + \mu + \delta)I = 0 \quad (11)$$

$$(\eta + \tau)I - (\omega + \mu)R = 0$$

$$e(1-\beta)I - mB = 0$$

In the absence of disease, $I = 0,\ R = 0,\ B = 0$ and hence, the model has a disease free equilibrium:

$$(S, I, R, B) = \left[\frac{bN}{\mu}, 0, 0, 0\right].$$

To calculate the endemic equilibrium, S I R B is set not equal to zero. Thus, by solving the system of equation (1) - (4), the endemic equilibrium states are obtained as follows:

$$S^* = \frac{y_2(Neby_4y_5 - km\omega y_3 + kmy_2y_4)}{ey_5(\mu y_2y_4 - \omega y_1y_3 + y_1y_2y_4)}$$

$$I^* = \frac{y_4(Neby_1y_5 - km\mu y_2)}{ey_5(\mu y_2y_4 - \omega y_1y_3 + y_1y_2y_4)} \quad (15)$$

$$R^* = \frac{y_3(Neby_1y_5 - km\mu y_2)}{ey_5(\mu y_2y_4 - \omega y_1y_3 + y_1y_2y_4)} \quad (16)$$

$$B^* = \frac{y_4(Neby_1y_5 - km\mu y_2)}{m(\mu y_2y_4 - \omega y_1y_3 + y_1y_2y_4)} \quad (17)$$

where

$$y_1 = a(1-c), \quad y_2 = \eta + \tau + \mu + \delta, \quad y_3 = \eta + \tau, y_4 = \omega + \mu \quad \text{and} \quad y_5 = e(1-\beta).$$

III. ESTIMATION OF BASIC REPRODUCTION NUMBER

The basic reproduction number (R_0) is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness (Diekmann et al., 1990). Biologically, R_0 captures the power of the disease to invade the population or not. $R_0 < 1$ implies that every infectious person will cause less than one secondary infection and the disease will eventually die out while $R_0 > 1$ implies that every infected individual will cause more than one secondary infection and hence invades the population. By the next generation matrix approach, R_0 is calculated by taking the spectral radius also known as the dominant eigenvalue of the matrix FV^{-1} at the DFE i.e. $\rho(FV^{-1})$ defined by

$$F = \frac{\partial F_i(x_0)}{\partial x_j} \quad \text{and} \quad V = \frac{\partial V_i(x_0)}{\partial x_j}$$

where F is the Matrix of new infections, V is the Matrix of transmission, ρ is Spectra radius or dominant eigenvalue, and x_0 is the Disease free equilibrium. Therefore, for the model equations (1) to (4) we have:

$$F = \begin{bmatrix} 0 & \frac{ky_1S}{(k+B)^2} \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} y_2 & 0 \\ -y_5 & m \end{bmatrix}, \quad (12)$$

$$V^{-1} = \begin{bmatrix} \frac{1}{y_2} & 0 \\ \frac{y_5}{y_2m} & \frac{1}{m} \end{bmatrix} \quad (13)$$

$$\Rightarrow FV^{-1} = \begin{bmatrix} \frac{ky_1y_5S}{(k+B)^2y_2m} & \frac{ky_1S}{(k+B)^2m} \\ 0 & 0 \end{bmatrix}.$$

At the DFE, $(S, I, R, B) = \left[\frac{bN}{\mu}, 0, 0, 0\right]$, thus

$$FV^{-1} = \begin{bmatrix} \frac{y_1y_5N}{\mu ky_2m} & \frac{y_1bN}{\mu km} \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad R_0 = \rho(FV^{-1}) = \frac{y_1y_2bN}{\mu ky_2m}. \quad (14)$$

IV. LOCAL STABILITY OF THE DFE

Theorem 3: The disease free equilibrium is locally asymptotically stable if $R_0 \leq 1$

Proof: The Jacobian matrix of the system (1) to (4) is given by:

$$J=\begin{bmatrix} -\left(\frac{y_1 B}{k+B}+\mu\right) & 0 & \omega & -\frac{k y_1 S}{(k+B)^2} \\ \frac{y_1 B}{(k+B)^2} & -y_2 & 0 & \frac{k y_1 S}{(k+B)^2} \\ 0 & y_3 & -y_4 & 0 \\ 0 & y_5 & 0 & -m \end{bmatrix}.$$

At DFE, $(S, I, R, B)=\left[\frac{bN}{\mu},0,0,0\right]$

$$J=\begin{bmatrix} -\mu & 0 & \omega & -\frac{y_1 bN}{\mu k} \\ 0 & -y_2 & 0 & \frac{y_1 bN}{\mu k} \\ 0 & y_3 & -y_4 & 0 \\ 0 & y_5 & 0 & -m \end{bmatrix}$$

Reducing J to upper triangular matrix we have:

$$J=\begin{bmatrix} -\mu & 0 & \omega & -\frac{y_1 bN}{\mu k} \\ 0 & -y_2 & 0 & \frac{y_1 bN}{\mu k} \\ 0 & 0 & -y_4 & \frac{y_1 y_3 bN}{\mu k y_2} \\ 0 & 0 & 0 & -m+\frac{y_1 y_5 bN}{\mu k y_2} \end{bmatrix}$$

The characteristic equation of the above matrix is obtained by $|J-\lambda I|=0,\ i.e$

$$J^*=\begin{vmatrix} -\mu-\lambda_1 & 0 & \omega & -\frac{y_1 bN}{\mu k} \\ 0 & -y_2-\lambda_2 & 0 & \frac{y_1 bN}{\mu k} \\ 0 & 0 & -y_4-\lambda_3 & \frac{y_1 y_3 bN}{\mu k y_2} \\ 0 & 0 & 0 & -m+\frac{y_1 y_5 bN}{\mu k y_2}-\lambda_4 \end{vmatrix} = 0$$

Yielding

$$(-\mu-\lambda_1)(-y_2-\lambda_2)(-y_4-\lambda_3)\left(\frac{-m\mu k y_2+y_1 y_5 bN}{\mu k y_2}-\lambda_4\right) = 0.$$

Jacobian stability technique states that if all eigenvalues of a system are less than zero, then the system is locally asymptotically stable. Therefore, the eigenvalues are negative

$$\lambda_1=-\mu<0,\ \lambda_2=-y_2<0,\qquad \lambda_3=-y_4<0$$

except λ_4 which is negative if

$$\frac{-m\mu k y_2+y_1 y_5 bN}{\mu k y_2}<0 \Rightarrow y_1 y_5 bN < m\mu k y_2$$
$$\Rightarrow \frac{y_1 y_5 bN}{m\mu k y_2}<1 \Rightarrow R_0<1.$$

V. CONCLUSION

In this paper, the stability analysis of a Cholera model has been presented. Based on theorems 1 and 2, the efficacy of the cholera model has been ascertained, thus confirming the existence and uniqueness of the solution of the model. The equilibrium states of the model, namely the DFE and EE have also been established. Using the next generation matrix approach, the basic reproduction number, R_0 has been derived. The DFE has been proved to be locally asymptotically stable when $R_0<1$. This indicates that the disease will always die out.

REFERENCES

[1] Adagbada A. O., Adesida S. A., Nwaokorie F. O., Niemogha M. and Coker A.O. (2012). Cholera epidemiology in Nigeria: an overview, The Pan African Medical Journal, doi: 10.11604/pamj.02/07/2012.12.59.

[2] Capasso V. and Paveri-Fontana S. L. (1979). A mathematical model for the 1973 cholera epidemic in the European Mediterranean region, Rev Epidemiol Sante Pub., 27, 121-132.

[3] Capasso V. and Serio G. (1978). A generalization of the kermack-mckendrick deterministic epidemic model, Mathematical Biosciences, 42 (1-2), 43-61.

[4] Capone F. De Cataldis V. and De Luca R. (2015). Influence of diffusion on the stability of equilibria in a reaction-diffusion system modelling cholera dynamic, J. Math. Biol., 71, 1107-1131.

[5] Chin M. J. and Kimbir A. R. (2018). A mathematical model for cholera epidemic, IOSR Journal of Mathematics, 14(1), 6-15.

[6] Codeco C. T. (2001). "Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir", BMC Infectious Diseases, 1(1), 1-14.

[7] Cui J., Wu Z. and Zhou X. (2014). Mathematical analysis of a cholera model with vaccination, Journal of Applied Mathematics, 2014, 1-16.

[8] Derrick N. R. and Groosman S. L. (1976). Differential equation with application, Addison Wesley Publishing Company Inc., Philippines.

[9] Diekmann O., Heesterbeek J. A. and Metz J. A. (1990). On the definition and computation of basic reproduction ratio R_0 in models for infectious disease in heterogeneous populations, J. Math. Biology, 28(4), 365-382.

[10] Isere A. O., Osemwenkhae J. E. and Okuonghae D. (2014). Optimal control model for the outbreak of cholera in Nigeria, African Journal of Mathematics and Computer Science research, 7(2), 24-30.

[11] Lemos-Palao A. P., Silva C. J. and Torres D. F. M. (2017). An epidemic model for cholera with optimal control treatment, J. Compt. Appl. Math., 318, 168-180.

[12] Lemos-Palao A. P., Silva C. J. and Torres D. F. M. (2018). A cholera mathematical model with vaccination and the biggest outbreak of world's history, AIMS Mathematics, 3(4), 448-463.

[13] Liao S. and Wang J. (2012). Global stability analysis of epidemiological models based on Volterra-Lyapunov stable matrices, Chaos, Solitons and Fractals, 45(7), 966-977.

[14] Mwasa A. and Tchuenche J. M. (2011). Mathematical analysis of a cholera model with public health interventions, Biosystems, 105, 190-200.

[15] Nigeria Centre for Disease Control (NCDC), 2019. Background, transmission, symptoms, diagnosis and treatment of cholera disease. www.ncdc.gov.ng

[16] Shuai Z., Tien J.H. and Driessche P.V. (2012). Cholera models with hyper-infectivity and temporary immunity, B. Math. Biol., 74, 2423-2445.

[17] Sulayman F., Isthrinayagy K., Jaffar M.Z.A.M. and Mohd B.A. (2014). A mathematical model for the control of cholera in Nigeria, Research Journal of Environmental and Earth Sciences, 6(6),321-325.