

## Endemicity of cholera in Nigeria: A mathematical model to investigate its nature

<sup>1</sup>J. E Osemwenkhae, <sup>2</sup>A. O. Isere and <sup>3</sup>D. U. Okuonghae

<sup>1,3</sup>Department of Mathematics, University of Benin, Nigeria

<sup>2</sup>Department of Mathematics and Statistics, Ambrose Alli University, Nigeria.

### Abstract

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*This work investigates cholera as a disease using mathematical models with emphasis on its endemic nature. The focal point is to investigate the persistent endemic nature of cholera in Nigeria using mathematical model. We found that, there can be no backward bifurcation because there existed only one positive endemic equilibrium. In other words, it is not possible for multiple endemic equilibria to exist if the reproduction number is less than one. Even when reproduction number is greater than one, only a single endemic equilibrium is shown to exist. There was however a transcritical (forward) bifurcation explaining the existence of a single endemic equilibrium.*

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### Keywords

Cholera, mathematical model, endemic equilibrium, reproduction number, backward bifurcations.

## 1.0 Introduction

Cholera epidemic was first presented formally in Snow's seminal work in 1855 where he associated cholera with contaminated water supply – see Codeco [1]. Over a century later, Cockburn and Cassanos [2] published an article on epidemiology of endemic cholera in Asia. Then McCormack et al [3] investigated cholera in a rural community of Pakistan. In Nigeria, outbreaks of the disease have been occurring with increasing frequency since the first outbreak in 1970 – see Epstein [4]. Most of the available literature lay credence to the fact that *V. cholerae* 01 was the main causative agent of cholera and much effort was directed towards its control.

A lot of epidemiological surveillances on cholera epidemics have been published – see for example Hustin and Luby [5] and Lawoyin et al [6], but of particular interest to us is the mathematical model formulated by Capasso and Paveri-Fontana [7] to describe the 1973's cholera epidemics in Italy. This model was a system of two ordinary differential equations, considering dynamics of the infected and that of the toxigenic *V. cholerae* in an aquatic reservoir. Codeco [1] extended Capasso and Paveri-Fontana [7] model to a system of three ordinary differential equations by including the susceptible class. Our model tends to include the carrying capacity of the organism by using a logistic growth approach that incorporates demographic factors and examine show these will affect the endemic nature of cholera in Nigeria.

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<sup>2</sup>Corresponding author:

<sup>1</sup>Telephone: 08037378121, 08071066580

<sup>2</sup>e-mail: abednis@yahoo.co.uk

## 2.0 Model formulations

A careful observation however, shows that bacteria population growth is fitted excellently with a logistic equation – see Britton [8]. Since the probability of catching cholera depends on the concentration of *V. cholerae* in aquatic environment, it behooves us to know the carrying capacity of the aquatic environment. The logistic term accommodates a carrying capacity for the aquatic environment hosting the bacteria. Basically, the growth term for the bacteria population used in Codeco [1] suggests that the population growth of bacteria is linear. In this work, we propose a model that incorporates demographic factors. In view of the foregoing, we proposed a model by using the logistic growth approach in line with Britton [8] and Appendix I.

Let  $S(t)$ ,  $I(t)$ ,  $B(t)$  be the number of susceptible individuals, the infected and the concentration of the toxigenic *V. cholerae* in water at given time  $t$  respectively.

Let  $\Lambda$  be the recruitment rate into the susceptible class, which could include immigrants and / or newborns that are uninfected. We assume that  $\mu$  is the per capita natural human death rate; then  $1/\mu$  is an average lifespan of individuals in the total human population  $N$ . Let ' $a$ ' be the per capita exposure rate to contaminated water ( $\text{day}^{-1}$ ). Let  $\theta(B)$  be the probability of any one exposed to contaminated water and food to catch cholera. This is dependent on the concentration of the toxigenic *V. cholerae* in water. Let  $d$  be the per capita cholera related death rate ( $\text{day}^{-1}$ ). We assume that  $\beta$  is the rate of recovering from cholera,  $r$  is the growth rate of *V. cholerae* in the aquatic environment.  $K$  is the carrying capacity of *V. cholerae* in the aquatic reservoir,  $k$  is the concentration of *V. cholerae* in water that yields 50% chance of catching cholera and  $e$  is the per capita contribution of the infected to the population of *V. cholerae*. The model is:

$$\frac{dS}{dt} = \Lambda - a\theta(B)S - \mu S \quad (2.1a)$$

$$\frac{dI}{dt} = a\theta(B)S - (\mu + d + \beta)I \quad (2.1b)$$

$$\frac{dB}{dt} = rB(1 - B/K) - nB + eI \quad (2.1c)$$

$$S(0) = N, I(0) > 0, B(0) = 0 \quad (2.2)$$

Given the model in (2.1a – 2.1c) above, Isere (2009 – see Appendix I) showed that the disease free equilibrium (DFE) becomes stable if the rate of exposure of people to contaminated water (the transmission rate) and the rate of contribution of the infested to *V. cholerae* is less than the concentration of *V. cholerae* in water that yields 50% chance of catching cholera. That is if the basic reproduction ratio,  $R_0$ , is less than unity, where

$$R_0 = \left( \frac{ae\Lambda}{k \mu (n-r)(\mu + d + \beta)} \right) \quad (2.3)$$

Hence, if  $R_0 < 1$ , the DFE is asymptotically stable.

If  $R_0 < 1$ , the disease dies out, but if  $R_0 > 1$ , it remains endemic in the population. If  $R_0 > 1$ , a disease outbreak can easily occur (the equilibrium point under consideration becomes unstable the infection spreads). For a detailed understanding of (2.1a – 2.1c) and proof of (2.3) see Appendix I).

Suppose we define  $R_0$  as  $R_0 = \frac{ea}{k(n-r)(d + \beta + \mu)} S_0$  where  $S_0 = \Lambda / \mu$  (asymptotic

population size). But  $S_c$  is the critical number of susceptible pool above which an outbreak occurs with the introduction of infective into the community. This happens when  $R_0 = 1$  (i.e. the threshold value). For this value of  $R_0$

$$S_c = \frac{k(n-r)(\mu+d+\beta)}{ae} \quad (2.4)$$

We observe from (2.4) that  $S_c$ , increases proportionally to  $k$ ,  $(n-r)$  and the removal rates  $(\mu+d+\beta)$  as well the net loss rate of the V. cholerae in water. It decreases as the contamination of water 'a' and the contribution of the infected 'e' to the population of the organism is increased.

### 3.0 Endemic cholera

Endemicity refers to a situation whereby a disease seems to be locally persistent in a community over a long period of time. In such situation, the community is vulnerable. Since  $S_0 > S_c$ , the introduction of an infective in the community starts a cholera outbreak. In this case, however, cholera does not vanish after the first peak – see Codeco [1].

Setting the derivatives in (2.1a-2.1c) to zero and solving algebraically, we obtain the endemic equilibrium, in terms of  $\lambda^*$ , the incidence evaluated at the endemic equilibrium

$$S^* = \frac{\Lambda}{\lambda^* + \mu} \quad (3.1)$$

Let 
$$\lambda = a\theta(B), I^* = \frac{\lambda^* S^*}{\mu + d + \beta} \quad (3.2)$$

Setting (2.1c) to zero result in a quadratic equation which gives the solution (the positive one) as  $rB \left(1 - \frac{B}{K}\right) - nB + eI = 0$

$$rB - \frac{rB^2}{K} - nB + eI = 0 \quad (3.3)$$

$$(r-n)B - \frac{rB^2}{K} + eI = 0 \quad (3.4)$$

Let  $\frac{r}{K} = \varepsilon$  and  $z = r - n$ ,  $\varepsilon B^2 - zB - eI = 0$ . We would expect  $r \ll K$  for this problem.

$$\varepsilon B^2 - zB - eI = 0, \varepsilon \rightarrow 0, zB + eI = 0$$

$$B^* = B^* = \frac{-eI^*}{r-n} = \frac{eI^*}{n-r} \quad (3.5)$$

Substituting (3.1) into (3.2), we have

$$I^* = \frac{\lambda^* \Lambda}{(\lambda^* + \mu)(\mu + d + \beta)} \quad (3.6)$$

But 
$$\lambda = \frac{aB}{k+B} \quad (3.7)$$

Substituting (3.5) into (3.7), we have

$$\lambda^* = \frac{\frac{aeI^*}{n-r}}{k + \frac{eI^*}{n-r}} = \frac{aeI^*}{n-r} = \frac{aeI^*}{k(n-r) + eI^*} \quad (3.8)$$

$$\text{or } \lambda^* = \frac{ae\lambda^*\Lambda}{k(n-r)(\lambda^* + \mu)(\mu + d + \beta) + e\lambda^*\Lambda}$$

$$ae\lambda^*\Lambda = \lambda^*k(n-r)(\lambda^* + \mu)(\mu + d + \beta) + \lambda^{*2}e\Lambda$$

$$\lambda^{*2}e\Lambda + \lambda^*k(n-r)(\lambda^* + \mu)(\mu + d + \beta) - \lambda^*ae\Lambda = 0$$

$$\lambda^{*2}e\Lambda + \lambda^*k(n-r)(\lambda^*\mu + \lambda^*d + \lambda^*\beta + \mu^2 + \mu d + \mu\beta) - \lambda^*ae\Lambda = 0$$

$$\lambda^{*2}e\Lambda + \lambda^*k(n-r)(\lambda^*\mu + \lambda^*d + \lambda^*\beta) + \lambda^*k(n-r)(\mu^2 + \mu d + \mu\beta) - \lambda^*ae\Lambda = 0$$

$$\lambda^{*2}e\Lambda + \lambda^{*2}k(n-r)(\mu + d + \beta) + \lambda^*[k\mu(n-r)(\mu + d + \beta) - ae\Lambda] = 0$$

$$\lambda^{*2}[e\Lambda + k(n-r)(\mu + d + \beta)] + \lambda^*[k\mu(n-r)(\mu + d + \beta) - ae\Lambda] = 0$$

which implies  $\lambda^* = 0$  or  $\lambda^* = \frac{aR_0 - a}{R_0 + a} = \frac{a(R_0 - 1)}{R_0 + a}$ . Endemic equilibrium exists only if  $R_0 > 1$ . If

$R_0 = 1$ , it reduces to the DFE'. This shows that we have a unique endemic equilibrium. There is a transcritical bifurcation at the point  $R_0 = 1$  when the DFE loses its stability and the endemic equilibrium then exists when  $R_0 > 1$ . If  $R_0 < 1$ , endemic equilibrium ceases to exist.

#### 4.0 Conclusion

We found that, there can be no backward bifurcations because; there existed only one positive endemic equilibrium. In other words, it is not possible for multiple endemic equilibria to exist if the reproduction number is less than one. Even when reproduction number is greater than one, only a single endemic equilibrium is shown to exist. There was however a transcritical (forward) bifurcation explaining the existence of a single endemic equilibrium.

#### 5.0 Acknowledgement

The proof given in Appendix I of this work is an abridged version of part of the seminar work given by A. O Isere (one of the authors) in March, 2009 to the Department of Mathematics, University of Benin, Nigeria.

#### Appendix I

With the model given in (2.1a) – (2.1c) in this work we easily see that the equilibrium solutions for which the system will no longer change is only when the rate of change are equated to zero. On the other hand local stability analysis helps us to determine the behaviour of the different populations near the equilibrium solutions. To achieve this, we compute the linearization of the system, which we obtained from the Jacobian matrix, J, of the system. Hence

$$J = \begin{pmatrix} -\mu - \frac{aB}{k+B} & 0 & \frac{aBS}{(k+B)^2} - \frac{aS}{k+B} \\ \frac{aB}{k+B} & -(\mu+d+\beta) & \frac{aBS}{(k+B)^2} + \frac{aS}{k+B} \\ 0 & e & r - \frac{2rB}{K} - n \end{pmatrix}$$

A community becomes cholera-free at the point  $(S_0, I_0, B_0) = (\Lambda/\mu, 0, 0)$ . We refer to this point as the Disease-Free Equilibrium (DFE). All individuals are susceptible. There are neither infective nor immune individuals nor toxigenic bacteria in the water. The expected population size at this state will simply be the solution of  $\frac{ds}{dt} = \Lambda - \mu S$  to give  $S(t) = \Lambda/\mu + (S_i - \Lambda/\mu)e^{-\mu t}$  where  $S_i$  is the initial number of susceptible individuals in the population. It is easy to see that as  $t \rightarrow \infty, S \rightarrow \Lambda/\mu$ , which is the asymptotic population size. Hence the entire population will comprise wholly of susceptible individuals.

Now, what will happen if a small number of infective come into this community? Will the disease Free State be achieved? Therefore, we carry out the stability analysis for the steady state to obtain:

$$J_0 = \begin{pmatrix} -\mu & 0 & -\frac{a\Lambda}{k\mu} \\ 0 & -(\mu+d+\beta) & \frac{a\Lambda}{k\mu} \\ 0 & e & r-n \end{pmatrix}$$

with eigenvalues as;  $\lambda_1 = \mu, \lambda_2 = \frac{-k\mu(d-r+n+\beta+\mu) + \sqrt{k\mu(4ae\Lambda + k\mu(d+r-n+\beta+\mu)^2)}}{2k\mu}$  and

$$\lambda_3 = \frac{-k\mu(d-r+n+\beta+\mu) - \sqrt{k\mu(4ae\Lambda + k\mu(d+r-n+\beta+\mu)^2)}}{2k\mu}$$

For  $\lambda_2$  to be negative, this implies that

$$\sqrt{k\mu(4ae\Lambda + k\mu(d+r-n+\beta+\mu)^2)} < k\mu(d-r+n+\beta+\mu) \quad (*)$$

If (\*) is satisfied then the DFE will be asymptotically stable. The disease dies out with time.

That is, if the rate of exposure of people to contaminated water (the transmission rate) and the rate of contribution of the infested to V. cholerae is less than the concentration of V. cholerae in water that yields 50% chance of catching cholera. This DFE becomes asymptotically stable.

From (\*) above, we observe that

$$4k^2\mu^2(n-r)(d+\beta+\mu) \left( \frac{4k\mu ae\Lambda}{4k^2\mu^2(n-r)(\mu+d+\beta)} - 1 \right) < 0$$

Since  $(n-r)$  is the net loss rate with  $(n-r) > 0$ , then  $\left( \frac{4k\mu ae\Lambda}{4k^2\mu^2(n-r)(\mu+d+\beta)} - 1 \right) < 0$ .

Hence, if  $R_0 < 1$ , the DFE is asymptotically stable, where  $R_0 = \frac{ae\Lambda}{k\mu(n-r)(d+\beta+\mu)}$ .

### ***References***

- [1] Codeco T. C (2001): Endemic and epidemic dynamics of cholera: The role of the aquatic reservoir. BMC infectious diseases – available at [www.biomedcentral.com](http://www.biomedcentral.com).
- [2] Cockburn T. A and Cassanos J. G (1960). Epidemiology of endemic cholera. Public Health Report, 75, 791 – 803.
- [3] McCormack W. M, Mosley W. H, Fahimuldin M and Benanson A. S (1969): Epidemic Cholera in Rural East Pakistan. American Journal of Epidemiology, 89(4) : 393.
- [4] Epstein PR (1993): Algal blooms in the spread and persistence of cholera. Biosystems, 31:209-221
- [5] Hustin Y and Luby S (2008): A large cholera outbreak in Kano City, Nigeria; the importance of hand washing with soap and the danger of street vended water. UN's IRIN\* humanitarian unit.
- [6] Lawoyin T.O, Ogunbode W.A ,Olumide E.A.A. and Onadeko M.O (2004): Outbreak of cholera in Ibadan, Nigeria. European journal of Epidemiology, springer Netherlands, **15**, 365-368.
- [7] Capasso V. and Paveri – Fontana S .L (1979): A mathematical model for the 1973 cholera epidemic in the European Mediterranean region. Rev Epidem et Sante Pub, 27: 121 – 132.
- [8] Britton F. N (2003): The Essential Mathematical Biology. Springer-verlag London