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A Deterministic Mathematical Model on Cholera Dynamics and Some Control Strategies

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Abstract: ***The mode of spread and transmission of cholera is very vital in considering appropriate measures for its control. The dynamics of cholera is analysed in this study using a system of four differential equations with two control measures*** τ ***and*** ω***, which are; therapeutic treatment and sanitary measures respectively. A zero Vibrio Cholerae bacteria environment was first assumed and analysed establishing disease free equilibrium state (DFE), which is interpreted as*** $R_0 < 1$***. Epidemic equilibrium state assumed as*** $R_0 > 1$ ***was then obtained after analysing the non-zero Vibrio Cholerae bacteria environment. This established the fact that; measures aimed at reducing Vibrio Cholerae bacteria in the environment will in turn reduce or control cholera.***

Keywords--Vibrio Cholerae, criteria, hygiene, basic reproduction number, concentration of toxigenic, therapeutic treatment, sanitary measures.

INTRODUCTION

Cholera is a severe diarrhoea disease cause by the bacterium Vibrio Cholerae. An infectious disease, cholera affects the absorption of water in the small intestine. It is characterised by vomiting and profuse dehydration through rice-water stool diarrhoea. It is a form of gastroenteritis caused by bacteria called Vibrio Cholerae.

Cholera is transmitted through two broad ways, the environment-to-human and human-to-human. The environment-to-human way of transmitting cholera is meanly through ingesting Vibrio Cholerae bacteria from contaminated food or water. While human-to-human way of cholera transmission is mostly unhygienic contact with cholera patients' faeces, vomit and corpse. Nelson et al (2008) mentioned that; 'in over-crowded location with fail water infrastructure, a great opportunity for fast transmission of Vibrio Cholerae is created, especially when coupled with increased infectivity and culturability of recently shed Vibrio Cholerae which inturn, creates a charged setting for explosive cholera outbreak'.

About one hundred million Vibrio Cholerae bacteria need to be ingested to cause cholera disease in a normal healthy adult. It may not reach that amount in people with lower gastric acidity. Isere and Osemwenkhae (2010) mentioned that: 'experiment suggests that a dose of 10^6 cells /ml is expected to cause cholera outbreak'.

In recent years, cholera outbreaks have been on increase, there are more than 250,000 cases of cholera each year worldwide. Many factors influence cholera outbreak, these include flood, draught and river height. Codeco (2001) 'Flooding and draught are likely to affect cholera dynamic in a complex way. Flooding washes contaminated faeces and sewage in to the river. It can also disrupt water distribution service and aggravate hygiene conditions. Draught on the other hand shortens the availability of potable water, aggravates hygiene condition, by increasing the number of people sharing the same water supply and may increase per capita water contamination'.

Gazi et al (2010) mentioned that 'Increase in ocean chlorophyll concentration, sea surface temperature and river height play a significant role on occurrence of cholera in a community and magnitude of the epidemic'. Hence, any accident or condition that will disrupt hygienic wellbeing of man in a community is a threat. Fastening treatment and other curing measures also help in curtailing the spread of the disease after outbreak. Yoon and Mekalano (2006) "It is tempting to speculate in ORS may have contributed to an active selection of El Tor strains, at least in those patients that were not simultaneously treated with antibiotics". "Because the infectious dose of V. Cholerae is known to be quite high in people with normally acidic contents, transmission via contaminated food may be more important than transmission through contaminated water alone".

Many mathematical models on cholera were developed before now, some of the famous ones are: The Capasso and Paveri-Fontuna mathematical model on cholera that described the dynamics of the 1973 epidemic of cholera in Italy, the model has two equations. Codeco (2001) developed mathematical cholera model with three equations, the first two equations described the susceptible and the infected population and the third equation described the concentration of Vibrio Cholerae bacteria in the environment. Pascaul et al (2002), seem to have generalised the model in Codeco(2001) a fourth equation on the volume of water in which the formative live, is added. A five equations mathematical model on cholera was developed by Hartley et al (2006), it describe the dynamics of the susceptible, infected, recovered or removed human population and the hyper infective and lower infective states of Vibrio Cholerae population.

This study aim at portraying effective measures to curtail spreading of cholera after its outbreak in a community, synthesised the level at which the measures must reach for it to be more effective.

MODEL FORMULATIONS

This study concentrates on the dynamics of cholera and the control measures with their effectiveness in curtailing and finally eradicating cholera disease. It is a modification of Tian et al (2010), who added three control strategies: (vaccine, therapeutic treatment and water sanitation), to the notable deterministic cholera model by Codeco (2001).

The basic assumptions are: In many African countries (Nigeria inclusive) there are fair or poor healthcare facilities or infrastructures, the worse is obtainable in the rural areas.

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Vaccines as delicate drugs are very expensive in terms of storage and distribution, coupled up with its other problems such as its life span (the maximum time it can take to wane out), rate of coverage and getting enough amounts for full coverage.
Hence, vaccine as a control strategy as in Tian et al (2010), is dropped in this study and therapeutic treatment (such as hydration therapy and administering antibiotic drugs) and sanitary measures is adopted. The therapeutic treatment is applied to infected people at the rate τ, so that τI individuals per time are removed from the infected people and added to the recovered people. Sanitary measures that leads to death and reduction of vibrio cholerae bacteria in food and water consumed by people at the rate ω. All individuals in the community are susceptible. The modified system is:

$$\frac{dS}{dt} = np - \frac{\alpha BS}{k+B} - nS \quad (1)$$
$$\frac{dI}{dt} = \frac{\alpha BS}{k+B} - (\gamma - \tau)I \quad (2)$$
$$\frac{dB}{dt} = \varepsilon I - (g - l + \omega)B \quad (3)$$
$$\frac{dR}{dt} = (\gamma - n + \tau)I - nR \quad (4)$$

Equation (1) describes the dynamics of susceptible in the community of size p. The birth and death rate of the susceptible individuals is represented by n, np is the rate of recruitment into susceptible class α is the rate of exposure to contaminated food and water, $\frac{\alpha BS}{k+B}$ is the probability of susceptible catching cholera. S is the susceptible and B is the concentration of toxigenic Vibrio Cholerae bacteria in food and water.

Equation (2) describes the dynamics of infected people in the community, their number increases as susceptible become infected and decreases as the infected recovers or die from the disease. Measures to curtail the spread of the disease, such as hygiene and total sanitation reduced the amount of Vibrio Cholerae bacteria in the environment.

Equation (3) describes the dynamics of pathogenic Vibrio Cholerae in the environment, comprising the contaminated food or water consumed by people and unhygienic handling of cholera patients and their waste products. The last equation describes the dynamics of effect of treatment or lack of treatment to the population of infected people. S-I-B-R makes up the compartments of the model or its state variables. S is the susceptible population, I is the infected population, B is the concentration of toxogenic Vibrio Cholerae bacteria in food and water and finally R is the recovered population.

Concentration of Vibrio Cholerae in food and water that yields 50% chance of catching cholera disease k, γ is the rate, at which infected people recovered from cholera disease, g is the growth rate of vibrio Cholerae bacteria in the environment; l is the loss rate of Vibrio Cholerae in the environment. Contribution of each infected person to the population of Vibrio Cholerae in the environment is ε. A schematic diagram of the model is as follows:

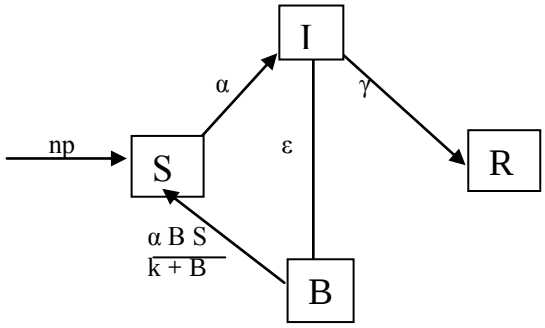

Figure 1 Diagrammatic representation of the model.

Definition of symbols as used in this study: Susceptible are all the individual in the community. The individuals are either born in the community or immigrated into it at the rate np. Susceptible S becomes infected with cholera at the rate α. Infected I contribute to the population of Vibrio Cholerae at a rate ε, infected people either die from cholera disease or recover at a rate γ. Recovered people become susceptible again, since it is assumed that no immunity after infection. Chance of catching cholera as result of contact with contaminated food or water and other means is $\frac{\alpha BS}{k+B}$.

Since B is not human population, total size of the population $P = S + I + R$ is a constant. Adding up the equations of human population

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \frac{d(S+I+R)}{dt}$$
$$= np - \frac{\alpha BS}{k+B} - nS + \frac{\alpha BS}{k+B} - (\gamma + \tau)I + (\gamma + \tau)I - nI - nR$$
$$= np - nS - nI - nR$$
$$= n(p - S - I - R)$$
$$= 0$$

The feasible region is:

$$\Omega = \left\{(S,I,B,R) \middle| S \geq 0, I \geq 0, R \geq 0, 0 \leq S + I + R \leq p, 0 \leq B \leq \frac{\varepsilon p}{g - l + \omega}\right\}$$

As by Tian et al (2010). Hence the region Ω is positively invariant for the system (1)-(4).
Setting $h = \gamma + \tau$ and $q = g - l + \omega$ for convinience.

EQUILIBRIUM STATES OF THE MODEL

The Disease Free Equilibrium State

In a cholera disease free community, we assumed there is no vibrio cholerae bacteria in food and water consumed by people; $B = 0$, no body is infected by cholera hence nobody recovered, this implies $I = R = 0$. From equation (1), when $B = I = R = 0$ and as in Neuhauser (2004),

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dB}{dt} = \frac{dR}{dt} = 0, \Rightarrow p = S \text{ and}$$
$$np - \frac{\alpha BS}{k+B} - nS = 0 \quad (5)$$
$$\frac{\alpha BS}{k+B} - hI = 0 \quad (6)$$
$$\varepsilon I - qB = 0 \quad (7)$$
$$(h - n)I - nR = 0 \quad (8)$$

The disease free equilibrium state is then:

$$D_0 = (p, 0, 0, 0) \quad (9)$$

The Jacobian of the system (1)-(4) is then given by:

$$J=\begin{bmatrix} -n-\frac{\alpha B}{k+B} & 0 & \frac{\alpha S}{k+B}-\frac{\alpha BS}{(k+B)^2} & 0 \\ \frac{\alpha B}{k+B} & -h & -\frac{\alpha S}{k+B}+\frac{\alpha BS}{(k+B)^2} & 0 \\ 0 & \varepsilon & -q & 0 \\ 0 & h-n & 0 & -n \end{bmatrix} \quad (10)$$

Substituting the $DFE\ D_0$ as in equation (9) into equation (10), the following is obtained

$$J(D_O)=\begin{bmatrix} -n & 0 & \frac{\alpha p}{k} & 0 \\ 0 & -h & -\frac{\alpha p}{k} & 0 \\ 0 & \varepsilon & -q & 0 \\ 0 & h-n & 0 & -n \end{bmatrix}$$

$$Det(J(D_O)-\lambda I)=\begin{vmatrix} -n-\lambda & 0 & \frac{\alpha p}{k} & 0 \\ 0 & -h-\lambda & -\frac{\alpha p}{k} & 0 \\ 0 & \varepsilon & -q-\lambda & 0 \\ 0 & h-n & 0 & -n-\lambda \end{vmatrix}$$

$$(-n-\lambda)^2(-h-\lambda)(-q-\lambda)+\frac{\alpha p\varepsilon}{k}=0 \quad (11)$$

$$\Rightarrow \lambda_1=-n, \lambda_2=-n \text{ and } (-h-\lambda)(-q-\lambda)+\frac{\alpha p\varepsilon}{k}=0$$

$$\lambda^2+(h+q)\lambda+hq+\frac{\alpha p\varepsilon}{k}=0 \quad (12)$$

Using Routh-Hurwitz criteria, as in Tian et al (2010), Liao and Wang (2011) and Korn & Korn (2000), setting $A_1=h+q$ and $A_2=hq+\frac{\alpha p\varepsilon}{k}$. All the roots of polynomial equation (12) are negative or have negative real part if $A_1A_2>0$. This implies that the roots of equation (12) are all negative if $(h+q)\left(hq+\frac{\alpha p\varepsilon}{k}\right)>0$.

Since all the roots of equation (11) are negative, the disease free equilibrium will be stable if, $khq(h+q)+\alpha p\varepsilon(h+q)>0$

Similarly if, $khq(h+q)+\alpha p\varepsilon(h+q)>0$

$$\Rightarrow p<\frac{hkq}{\alpha\varepsilon} \quad (13)$$

Dividing equation (13) throughout by p and taking the reciprocal we have $1>\frac{\alpha p\varepsilon}{hkq}$

Then $R_0=\frac{\alpha p\varepsilon}{hkq}<1$

R_0 is a key parameter obtained and used to determine the stability of equilibrium point in epidemic models. It denotes the basic reproduction number, which is the expected number of secondary cases produced in a completely susceptible population by a typical infective individual. When $R_0<1$ the equilibrium stability of the model is stable, when $R_0>1$ it is unstable. From parameters of this model $R_0<1$, therefore the disease free equilibrium point $D_0=(p,0,0,0)$ is locally asymptotically stable.

The epidemic equilibrium state

From equation (7) $\varepsilon I=qB$

$$\Rightarrow B^*=\frac{\varepsilon I}{q} \quad (14)$$

Also from equation (5) $np=\left(\frac{\alpha B+n(k+B)}{k+B}\right)S$

$$\Rightarrow S=\frac{np(k+B)}{\alpha B+n(k+B)} \quad (15)$$

Substituting equation (14) into equation (15)

$$S^*=\frac{np(kq+\varepsilon I)}{\alpha\varepsilon I+n(kq+\varepsilon I)} \quad (16)$$

From equation (6) $\frac{\alpha BS}{k+B}=hI$

$$\Rightarrow I=\frac{\alpha BS}{h(k+B)} \quad (17)$$

Substituting equation (14) and (16) into equation (17), it gives

$$I^*=\frac{\alpha\varepsilon np-hknq}{\varepsilon h(\alpha+n)} \quad (18)$$

Finally from equation (8) $(h-n)I=nR$

$$\Rightarrow R^*=\frac{(h-n)I}{n} \quad (19)$$

From equation (18) infected people at epidemic state is given by I* and this I* > 0 because at epidemic state infective are available

$\therefore I^*=\frac{\alpha\varepsilon np-hknq}{\varepsilon h(\alpha+n)}>0$, but $\frac{\alpha\varepsilon np-hknq}{\varepsilon h(\alpha+n)}>0$

$\Rightarrow p>\frac{hkq}{\alpha\varepsilon}$

From equation (13)

$p<\frac{hkq}{\alpha\varepsilon} \Rightarrow R_0=\frac{\alpha p\varepsilon}{hkq}<1$,

but now $p>\frac{hkq}{\alpha\varepsilon}$

This implies that $R_0=\frac{\alpha p\varepsilon}{hkq}>1$. It has been established when R_0 is greater than one epidemic equilibrium state is stable, $R_0>1$ is obtained. To confirm the stability of epidemic equilibrium state, equation (10) is used, so that, substituting B^* for B, $S^*$for S and replacing $\frac{\alpha B^*}{k+B^*}>0$ by M and $\frac{\alpha S^*k}{(k+B^*)^2}>0$ by N.

and taking $D^*=(S^*,I^*,B^*,R^*)$ as the epidemic equilibrium, the Jacobian matrix becomes:

$$J(D^*)=\begin{bmatrix} -n-M & 0 & M-N & 0 \\ M & -h & -M+N & 0 \\ 0 & \varepsilon & -q & 0 \\ 0 & h-n & 0 & -n \end{bmatrix}$$

$$Det\left(\lambda I-j(D^*)\right)=(-n-M-\lambda)\{(-h-\lambda)(-q-\lambda)(-n-\lambda)-(-M+N)(-n-\lambda)\varepsilon\}+(M-N)[M\varepsilon(-n-\lambda)]=0$$

$$\Rightarrow(-n-\lambda)[(-n-M-\lambda)\{(-h-\lambda)(-q-\lambda)-(-M+N)\varepsilon\}]+(M-N)M\varepsilon=0$$

$$\Rightarrow \lambda_1=-n,$$
$$(-n-M-\lambda)\{(-h-\lambda)(-q-\lambda)-(-M+N)\varepsilon\}+(M-N)M\varepsilon=0$$

After expansion, it gives:

$$\lambda^3+(h+M+n+q)\lambda^2+(hq+nq+nh+M\varepsilon-N\varepsilon+Mh+Mq)\lambda+(hnq+\varepsilon Mn-\varepsilon nN+hMq)=0$$

Using Routh-Hurwitz criteria as in Tian et al (2010), Liao and Wang (2011) and Korn & Korn (2000), setting;

$A_0=1$

$A_1=h+M+n+q$

$A_2=hq+nq+nh+M\varepsilon-N\varepsilon+Mh+Mq$

$A_3=hnq+\varepsilon Mn-\varepsilon nM+hMq$

The sufficient and necessary conditions for stability are:

$A_1>0, A_2>0, A_3>0$ and $A_1A_2-A_0A_3>0$

The basic reproduction number R_0 was defined by Fraser et al (2004) as: "The number of secondary infections generated by a primary infection in susceptible population and which also measures the basic transmissibility of an infectious agent. $R_0>0$ need to be satisfied, for an epidemic to expand in the

early stage of spread more than one secondary case has to be generated by the primary case". From the models' parameters the above four conditions are satisfied, hence the epidemic equilibrium state is stable.

In the cholera outbreak, measuring the rates of the parameters used in this model, substituting them to obtain

$A_0 = 1$,

$A_1 = h + M + n + q$,

$A_2 = hq + nq + nh + M\varepsilon - N\varepsilon + Mh + Mq$,

$A_3 = hnq + \varepsilon Mn - \varepsilon nM + hMq$ and confirming that $A_1 > 0, A_2 > 0, A_3 > 0$ and $A_1A_2 - A_0A_3 > 0$ are established from the obtained values, it interprets the stability of epidemic equilibrium state. Hence, this model can be applied to estimate sufficient rates of sanitary and therapeutic measures the rate to be used/applied to over-come the cholera outbreak.

Stability of epidemic equilibrium state implies that introduction of infected people with cholera in the community will lead to cholera epidemic in it, if strong measures are not taken. The strong measures include high sanitary measures and high therapeutic measures denoted by τ and ω respectively. If pegged with a rates 0.54 and 0.669 respectively or less than this, the stability of the epidemic equilibrium state will more stable. On the other hand, if the rates are higher than 0.54 and 0.669, the epidemic equilibrium state will be unstable. The bigger the values, the more unstable the epidemic equilibrium state will be.

CONCLUSION

In any outbreak of cholera, there is need to quarantine infected people, quickly treat them with therapeutic measures (such as dehydration and treatment with antibiotics). There is also high need to carefully handle contact with diarrhoea and vomit from infected people and anti-germs sprays used in such environment. Applying all possible measures to prevent ingestion of Vibrio Cholerae bacteria through water, food and the entire environment is mandatory.

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