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A Mathematical Model for Cholera Epidemic

Chin, M. J* and Kimbir, A .R.
Department of mathematics/Statistics/Computer Science University of Agriculture Makurdi
PMB 2373, Makurdi, Nigeria
Corresponding Author: Chin,M.J

Abstract: ***Cholera*** *has long been, and continues to be a global challenge. Despite study of the disease for many decades, it continues to be a threat to human existence in the affected regions of the world. It is important to continue to try to understand the disease dynamics and how interaction with environment and human factors contribute to the epidemic behavior. War, lack of good drinking water and inadequate human faeces disposal facilities seems to be the driving force of the epidemic in the affected communities. In other to find a solution to this global challenge, we extended a mathematical model for cholera epidemic by Codeco (2000), by varying the net reproduction rate of humans and allowing for susceptibility of recovered individuals. It is proved that the disease-free equilibrium state of the extended model exists which is locally asymptotically stable under prescribed threshold conditions. Numerical experiments using published data indicated that the disease could be controlled or eradicated, provided that the rate of exposure of individuals to contaminated water and that of pollution of aquatic environment by infected people is sufficiently reduced. This is consistent with results obtained from the stability analysis of the disease-free state of the model.*
Keywords*: Cholera; Transmission; disease-free equilibrium; mathematical models; eradication*

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I. Introduction

Cholera is an acute bacterial infection caused by *vibrio cholerae*, non-invasive bacterium called *vibrio* or the 'comma bacillus' which lives in the small intestine. A disease clinically resembling (but not proved to be) *cholera* was described by early Indian, Greek, and Chinese writers. Alternative names are Asiatic, epidemic, or malignant cholera; the name (in English) is derived from *choler*, the toxin or poison of this bacterium.. *Vibrio cholerae* is a large and very diverse species. It is divided into about 200 serogroups, denoted by 01, 02, 03,…, 0200 of which only 01 and 0139 contain pathogenic members. Most of the strains isolated from cholera patients belong to the 01 serogroup. This serogroup is divided into three serotypes, namely, Inaba, Ogawa, and Hijokima, See Reidl *et al.* (2002). Cholera infection is caused by ingestion of the organism(s) through contaminated water or food (there is no animal reservoir of infection). Snow was the first to hypothesize in 1849 that cholera was transmitted by contaminated water, though this theory was not generally accepted until 1871. It was not until 1883 the waterborne bacteria now called *Vibrio cholerae* was "discovered" by Robert Koch and the bacteria was finally accepted to be the cholera pathogen, see Lipp *et al*. (2002). Recent epidemiological research suggests that an individual's susceptibility to cholera (and other diarrhea infections) is affected by their blood type. Those with type O blood are the most susceptible while those with type AB are the most resistant. Between these two extremes are the A and B blood types with type A being more resistant than type B Swerdlow *et al* (1974) and Haris *et al. (* 2005). The incubation period ranges from a few hours to five days, See Reidl *et al.* (2002) and Lipp *et al.* (2002) . Management consists first and foremost of rapid rehydration, usually by mouth (using an oral rehydration fluid) only in exceptional cases is an intravenous drip (first advocated in the 1831–2 outbreak) necessary. The antibiotics tetracycline and doxycycline reduce the duration of diarrhea and excretion of *V. cholerae;* however, the organism may rapidly become resistant in an epidemic. Co-trimoxazole and furazolidone have also been used in individual cases. Public health measures are required for long-term control, chlorination of water supplies, boiling of water (in households), construction and maintenance of temporary latrines are imperative. Safe feces disposal is also essential. Vaccines have no useful role in an outbreak; individual protection is not above 50–60%. Effective surveillance (with identification of affected cases) is an essential component of cholera control. New vaccines (using genetic engineering techniques) are undergoing clinical trials; they may contribute to effective control of an infection which is likely (given experience in South- Africa, Cameron and Nigeria) to remain of considerable importance in tropical countries for many years to come, see Reidl *et al.* (2002).

Several mathematics models have been proposed and analyzed on the transmission dynamics of cholera . For example Mark *et al (*2006) studied the role of bacteriophage in the control of cholera outbreaks while

Bouma *et al*. (2001) developed a model to assess Ocean surveillance by satellite remote sensing to monitor changes in sea surface temperature(SSH) and sea surface height(SST) .

Guillaume *et al.* (2005) modeled the effect of chlorophyll concentration along the coast on cholera epidemic while Alen *et al.* (2007) proposed an age structure model and used partial differential equations to study the effect of hyper infective and non hyper infective *vibrios* on cholera transmission. According to Bertuzzo (2010) the vehicle of infection (*Vibrio cholerae*) is transported through the network links that are thought of as hydrological connections among susceptible communities while Esben *et al.* (2010), looked at the spread of epidemics in networks, more specific, the spread of *Vibrio cholerae* in river systems.

A mathematical model to simulate the role of aquatic reservoir on the persistent outbreak of cholera epidemic was developed by Codeco (2000), he assumed constant population, the same human and bacteria birth rate n. His work does not reflect a realistic situation because population in developing countries is constantly changing due birth and death process. Nevertheless, the work by Codeco (2000) form the bases of our research work. In order to study the epidemic of cholera in a varying population, we extended the model by Codeco (2000), by varying the net reproduction rate of humans and allowing for susceptibility of recovered individuals. We solved the extended model numerically using Runge-Kutta 4th order method to establish the role of the parameters of the extended model in the control of the cholera epidemic.In this paper we intend to study the effects of high contribution of infected people to aquatic environment and the effect of high rate of exposure to contaminated water.

The rest of the paper will be organized as follows. We provide the mathematical model in section II while the steady state analysis is performed in section III . numerical results are provided in section IV and we draw some conclusion in section V.

II. The Mathematical Model

2.1 The existing model

We begin our model extension by introducing the model by Codeco (2000) which is the motivational paper for this study . First, we present the assumptions of the existing model

2.2 Assumptions of the existing model

The following are assumptions of the existing model:

(i) The population was assumed to be constant.

(ii) The growth rate (n) of the bacteria is the same as that of the human population.

(ii) The only route for infection is the ingestion of contaminated water from non-treated source.

2.3 Variables and Parameters of the existing model

The variables and parameters of the existing model are as defined below.

Variables/ Parameters	Description
S	Number of susceptible
I	Number of infected
H	Total human population
B	Concentration of toxigenic *Vibrio cholerae* in water (cell/ml)
nb	growth rate of *V. Cholerae* in the aquatic environment (day^{-1})
mb	loss rate of *Vibrio cholerae* in the aquatic environment (day^{-1})
r	rate which people recover from cholera (day^{-1})
e	contribution of each infected person to the population of *Vibrio cholerae* in the aquatic environment (cell/m/day^{-1}/person^{-1})
d	Death rate of human (day^{-1})
a	rate of exposure to contaminated water (day^{-1})
K	Concentration of vibrio cholerae in water that yields 50% chance of been infected with cholera(cells/ml)

Based on the above assumptions; parameters and variables by Codeco (2000), the following model equations were derived

$$\frac{dS}{dt} = n(H - S) - a\lambda(B)S \quad (2.3.1)$$

$$\frac{dI}{dt} = a\lambda(B)S - rI - dI \quad (2.3.2)$$

$$\frac{dB}{dt} = B(nb - mb) + eI \quad (2.3.3)$$

$$S(0) = H, I(0) > 0, B(0) = 0 \quad (2.3.4)$$

Experimental studies suggest that it is necessary for a heavy inoculum of *V. cholerae* in order to develop cholera, see Cash *et al.(* 1974). Here this dependence is represented by

$$\lambda(B) = \frac{B}{K+B}$$

where here $\lambda(B)$ is the probability of a person been infected with cholera.

2.4 The extended model

2.4.1 Assumptions of the mode

In addition to the assumptions of Codeco (2000) , we further assumed that

(i) The infected individuals also die naturally.

(ii) The population is assumed to be homogenous and varying as a result of birth.

(iii). The birth and death rates are different

(iv). The human and bacteria birth rates are also different

2.5 Variables and parameters of the extended model equations

In addition to the variables and parameters of the existing model , we state the variables and parameters of the extended model

Variables/ Parameters	Description
$N_H(t)$	Total human population at time t.
α	disease induce death rate (day^{-1}).
μ	natural death rate of the human population (day^{-1}).
ω	human population birth rate (day^{-1}).
$N_H(t)$	S + I
γ	net growth rate of *Vibrio cholerae* in the aquatic environment ($\gamma = nb - mb$) (day^{-1}).

2.6 The extended model equations

From the assumptions, we obtain the extended model equations, which are set of three ordinary differential equations.

$$\frac{dS}{dt} = \omega N_H - a\lambda(B)S + rI - \mu s \quad (2.6.1)$$

$$\frac{dI}{dt} = a\lambda(B)S - rI - \mu I - \alpha I \quad (2.6.2)$$

$$\frac{dB}{dt} = \gamma B + eI \quad (2.6.3)$$

III. Stability Analysis of the Extended Model

3.1 The Basic Reproduction number (R_0)

R_0, the basic reproductive number, is defined as the average number of secondary infections produced when one infected individual is introduced into a host population where the rest of the population is susceptible. R_0 is the threshold parameter that determines the existence and local stability of the disease-free equilibrium of a compartmental infectious disease model, van den Driessche and Watmough (20002). If R_0 < 1, there exists a locally asymptotically stable equilibrium. In biological terms, it means that on average of an infected individual produces less than one new infected individual over the course of its infectious period. Hence the infection cannot persist, and the model will eventually reach a locally stable disease-free equilibrium . Conversely, if $R_0 > 1$, the disease-free equilibrium is locally unstable, and the infection will persist because each newly infected individual will spread the disease to at least one susceptible individual on average. The R_0 expression for the extended model using the 'Next Generation Method' is as shown below. Detailed explanation and proofs of the method were developed by van den Driessche and Watmough,(2002).

Consider the extended model equations:

$$\frac{dS}{dt} = \omega N_H - \frac{aSB}{K+B} + rI - \mu S \quad (2.6.1)$$

$$\frac{dI}{dt} = \frac{aSB}{K+B} - rI - \mu I - \alpha I \quad (2.6.2)$$

$$\frac{dB}{dt} = B(nb - mb) + eI \quad (2.6.3)$$

We let

$F_i = \begin{pmatrix} \frac{B}{K+B}as \\ 0 \end{pmatrix}$, $V_i = \begin{pmatrix} (r+\mu+\alpha)I & 0 \\ -eI & (mb-nb)B \end{pmatrix}$

$F = \begin{pmatrix} 0 & \frac{a}{K}S^* \\ 0 & 0 \end{pmatrix}$, $V = \begin{pmatrix} r+\mu+\alpha & 0 \\ -e & mb-nb \end{pmatrix}$

We now compute our V^{-1} as follows

$|V| = (mb-nb)(r+\mu+\alpha) - 0$

$|V| = (mb-nb)(r+\mu+\alpha)$

$V^{-1} = \frac{1}{(mb-nb)(r+\mu+\alpha)} \begin{pmatrix} mb-nb & 0 \\ e & r+\mu+\alpha \end{pmatrix}$

$V^{-1} = \begin{pmatrix} \frac{1}{r+\mu+\alpha} & 0 \\ \frac{e}{(mb-nb)(r+\mu+\alpha)} & \frac{1}{(mb-nb)} \end{pmatrix}$

$FV^{-1} = \begin{pmatrix} 0 & \frac{a}{K}S^* \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{r+\mu+\alpha} & 0 \\ \frac{e}{(mb-nb)(r+\mu+\alpha)} & \frac{1}{(mb-nb)} \end{pmatrix}$

$FV^{-1} = \begin{pmatrix} \frac{as^*}{Kb(m-n)(r+\mu+\alpha)} & \frac{as^*}{Kb(m-n)} \\ 0 & 0 \end{pmatrix}$

therefore ,

$R_o = \frac{aS^*}{kb(m-n)(r+\mu+\alpha)}$ (3.1.1)

3.2 The Existence of the Disease –free Equilibrium state of the Extended model

To establish the existence of the disease free equilibrium state of the extended model we equate the left hand side of equations (2.6.1)- (2.6.3) to zero .

$\frac{dS}{dt} = 0$, $\frac{dI}{dt} = 0$, $\frac{dB}{dt} = 0$.

therefore,

$0 = \omega(S-I) - \frac{aSB}{K+B} + rI - \mu S$ (3.2.1)

$0 = \frac{aSB}{K+B} - rI - \mu I - \alpha I$ (3.2.2)

$0 = \gamma B + eI$ (3.2.3)

Theorem 3.1: Given $\gamma, e, r, \mu, \alpha, a, k, \omega, > 0$. If $\omega = \mu$, then there exists a DFE state $(S, I, B) = (S^*, 0, 0,)$ where S^* is arbitrary.

Proof:

Substituting $I = 0$, $B = 0$, *in* (3.2.1), (3.2.2) and (3.3.3) *and* solving simultaneously, we have

$0 = \omega S - \mu S$ or $S(\omega - \mu) = 0$

from the hypothesis of the theorem, $\omega = \mu$, therefore

$S = S^* \neq 0$. With $I = B = 0$,

3.3 Stability Analysis of the disease-free Equilibrium State of the Extended model

In this section, we discussed the stability of the disease-free equilibrium state of the extended model. We therefore refer to the equations (3.2.1), (3.2.2) and (3.2.3)

Let

$f_1 = \omega(S+I) - \left(\frac{aB}{K+B} - \mu\right)S + rI$ (3.3.1)

$f_2 = \frac{aSB}{K+B} + rI - \mu I - \alpha I$ (3.3.2)

$f_3 = \gamma B + eI$ (3.3.3)

Theorem 3.2

Given $\gamma, e, r, \mu, \alpha, a, K, \omega, > 0$. If $\gamma < r+\mu+\alpha$ and $\omega = \mu$, then the disease-free equilibrium state is locally asymptotically stable.

Proof:

Differentiating (3.3.1) , (3.3.2) *and* (3.3.3) partially with respect to S, I and B.

gives the following

from (3.3.1) we have

$\frac{\partial f_1}{\partial S} = \omega - (\mu + \frac{aB}{K+B})$, $\frac{\partial f_1}{\partial I} = \omega + r$, $\frac{\partial f_1}{\partial B} = -\frac{aSK(1+B)}{(k+B)^2}$

From (3.3.2) we have,

$\frac{\partial f_2}{\partial S} = \frac{aB}{K+B}$, $\frac{\partial f_2}{\partial I} = -r - \mu - \alpha = -(r + \mu + \alpha)$, $\frac{\partial f_2}{\partial B} = \frac{aSK(1+B)}{(K+B)^2}$

From (3.3.3) we have,
$\frac{\partial f_3}{\partial S} = 0$, $\frac{\partial f_3}{\partial I} = e$, $\frac{\partial f_3}{\partial B} = \gamma$

therefore the Jacobian matrix is

$$J_{(S,I,B)} = \begin{pmatrix} \omega - (\mu + \frac{aB}{K+B}) & \omega + r & -\frac{aSK(1+B)}{(K+B)^2} \\ \frac{aB}{K+B} & -(r + \mu + \alpha) & \frac{aSK(1+B)}{(K+B)^2} \\ 0 & e & \gamma \end{pmatrix} \quad (3.3.4)$$

The Jacobian matrix J_0 at the disease-free equilibrium state (S , I , B) = (S^*, 0 ,0) is given as

$$J_0 = \begin{pmatrix} \omega - \mu & \omega + r & -\frac{aS^*}{K} \\ 0 & -(r + \mu + \alpha) & \frac{aS^*}{K} \\ 0 & e & \gamma \end{pmatrix}$$

The corresponding characteristic equation at the D F E S is

$$\begin{vmatrix} (\omega - \mu) - \lambda & \omega + r & -\frac{aS^*}{K} \\ 0 & -(r + \mu + \alpha + \lambda) & \frac{aS^*}{K} \\ 0 & e & \gamma - \lambda \end{vmatrix} = 0$$

$\Phi_0(\lambda) = \big((\omega - \mu) - \lambda\big)\big(-(r + \mu + \alpha + \lambda)\big)(\gamma - \lambda) - \frac{aeS^*}{K} = 0$

The first roots of $\Phi_0(\lambda)$ is given as

$\lambda_1 = \omega - \mu$ (3.3.5)

or

$\Phi_0(\lambda) = \lambda^2 + \lambda(r + \mu + \alpha - \gamma) - \gamma(r + \mu + \alpha) - \frac{aeS^*}{K} = 0$ (3.3.6)

From (3.3.5) by theorem 3.2,

$\lambda_1 = 0$

The remaining two roots are obtained from (3.3.6) as follows

$Let\ B = (r + \mu + \alpha - \gamma),\ \ C = -\gamma(r + \mu + \alpha) - \frac{aeS^*}{K}$

then,

$\lambda^2 + B\lambda - C = 0$ (3.3.7)

Using the quadratic formula,

$\Phi_0(\lambda) = \frac{-B \pm \sqrt{(B)^2 - 4C}}{2}$

we get

$$-\frac{B}{2} \pm \frac{\sqrt{(B)^2-4C}}{2} \leq \frac{-B}{2} \pm \frac{\sqrt{B^2}}{2} \quad (C > 0)$$

$$\Phi_0(\lambda) \leq \frac{-B}{2} \pm \frac{B}{2}$$

$therefore$,

$$\lambda_2 \leq 0$$

or

$$\lambda_3 \leq -B \quad .$$

that is

$$\lambda_3 \leq -(r + \mu + \alpha - \gamma)$$

$$\lambda_3 \leq \gamma - r - \mu - \alpha$$

From the hypothesis of Theorem.3.2

$$\lambda_3 < 0$$

IV. Numerical Experiments

We solve the system (2.3.1)-(2.3-3) with a view of studying five distinct situations. The simulation will run for a period of 20 years. The five situations will be as follow

(i) A disease- free state (cholera)

(ii) Very high contribution (infected people urinating or defecating in the source of drinking water) of infected people in the aquatic environment and very high rate of exposure to contaminated water.

(iii) High contribution (infected people urinating or defecating in the source of drinking water) of infected people in the aquatic environment and high rate of exposure to contaminated water

(iv) Low rate of exposure to contaminated water and low contribution of infected people in the aquatic environment

(V). Very low rate of exposure to contaminated water and very low contribution of infected people in the aquatic environment.

In all the numerical experiments we use the values in the table 4.2.

Table 4.2: Table of Parameter Values For The Numerical Experiments.

Parameter values \ Experiments	1	2	3	4	5	6
S(0)	1000	1000	1000	1000	1000	1000
I(0)	0.000	100.0	100.0	100.0	100.0	100.0
B(0)	0.000	0.002	0.002	0.002	0.002	0.002
ω	0.500	0.500	0.500	0.500	0.500	0.500
N_H	1100	1100	1100	1100	1100	1100
a	0.000	1.000	0.500	0.003	0.0025.	0.0001
K	0.0001	100.0	100.0	100.0	100.0	100.0
r	0.000	0.020	0.020	0.020	0.020	0.020
μ	0.021	0.021	0.021	0.021	0.021	0.021
α	0.000	0.048	0.048	0.048	0.048	0.048
γ	0.000	0.033	0.033	0.033	0.033	0.033

e	0.000	100.0	50.00	25.00	3-000	1.000

4.1.1 A disease- free state (cholera)

First we will look at a case where there is no disease ($e = 0.000$ and $a = 0.000$). The result shown in figure 2 shows that the population will grow normally

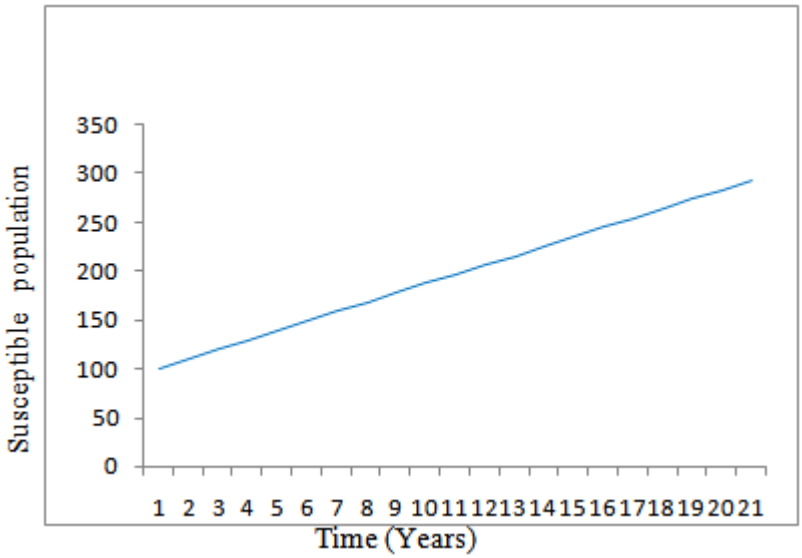

Figure 2: Graph of the susceptible population against time in a disease-free Population.

4.1.2 Very high Contribution (infected people urinating, defecating or vomiting in the source of drinking water) of infected people in the aquatic environment and very high rate of Exposure to Contaminated water.

Secondly, we look at a case where there is very high contributions of infected people in aquatic envorinment ($e = 100.0$) and very high rate of exposure to contaminated water($a = 1.000$). The result shown on figure 3 shows that the disease will ravege the community

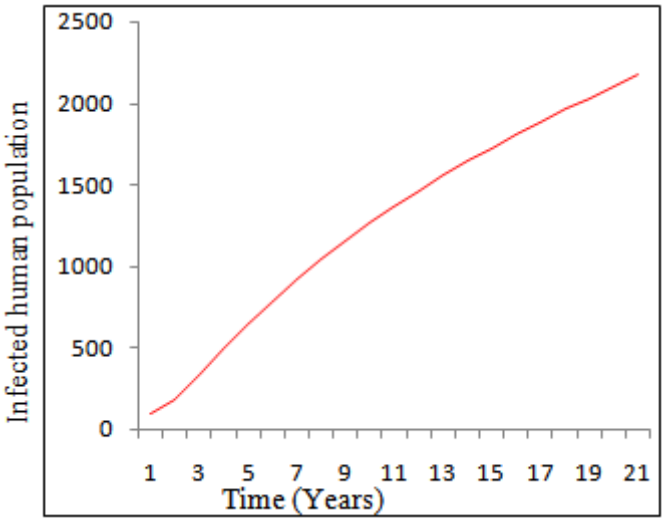

Fig 3: Graph of the infected population against time in a case of very high contributions of infected people in aquatic envorinment and very high rate of exposure to contaminated water.

4.1.3 **High contribution (infected people urinating, defecating or vomiting in the source of drinking water) of infected people in the aquatic environment and high rate of Exposure to Contaminated water**

Next we look at the case where there is high contribution of infected people in the aquatic environment($e = 50.00$) and high rate of exposure to contaminated water ($a = 0.500$). Result shown on figure 4 shows that

the rate of infection is not much as when we had very high contributions of infected people in aquatic envorinment and very high rate of exposure to contaminated water.

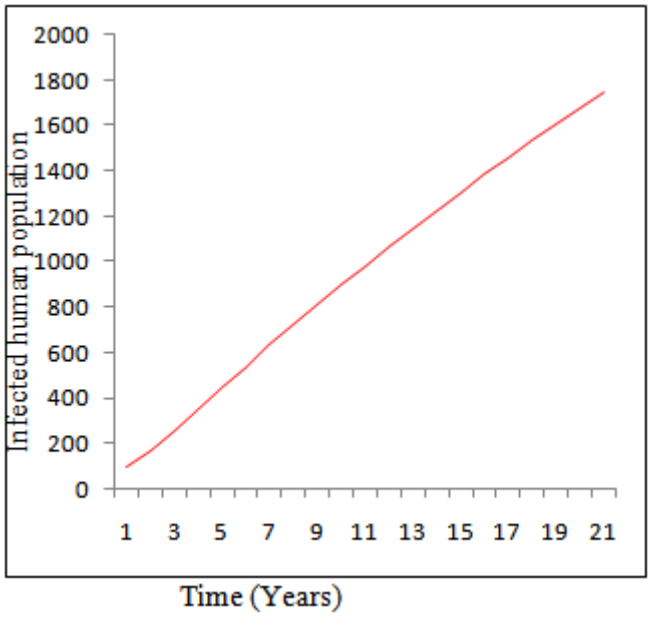

Fig 4: Graph of the infected population against time in a case of high contribution of infected people and high rate of exposure to contaminated water.

4.1.4 Low rate of Exposure to Contaminated water and low contribution of infected people in the aquatic environment

Next we considered a situation where a and e are further reduced $(a = 0.003\ and\ e = 25.00)$. The result shown on figure 5 shows that the rate of infection will slow down.

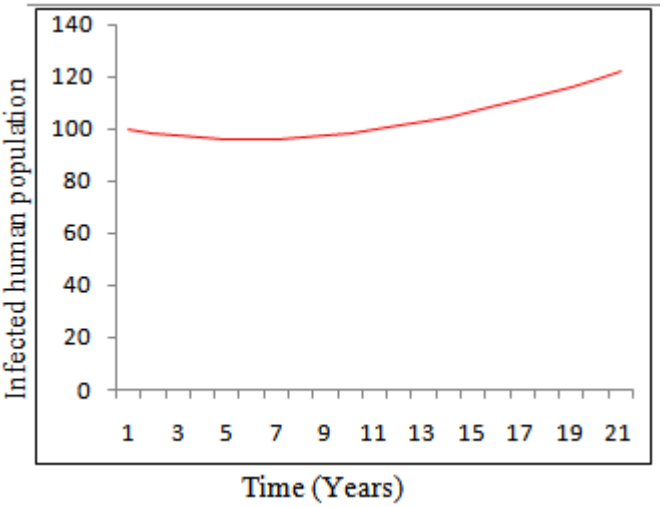

Fig 5: Graph of the infected population against time in a case of low rate of exposure to contaminated water and low contribution of the infected people in the aquatic enviroment.

4.5 Very low rate of Exposure to Contaminated water and very low Contribution of infected people in the aquatic environment.

Finally , we examine a situation where a and e are very low $(e = 1.000\ and\ a = 0.0001)$. the result shown on figure 7 shows that cholera will disapear from the community in a finite time.

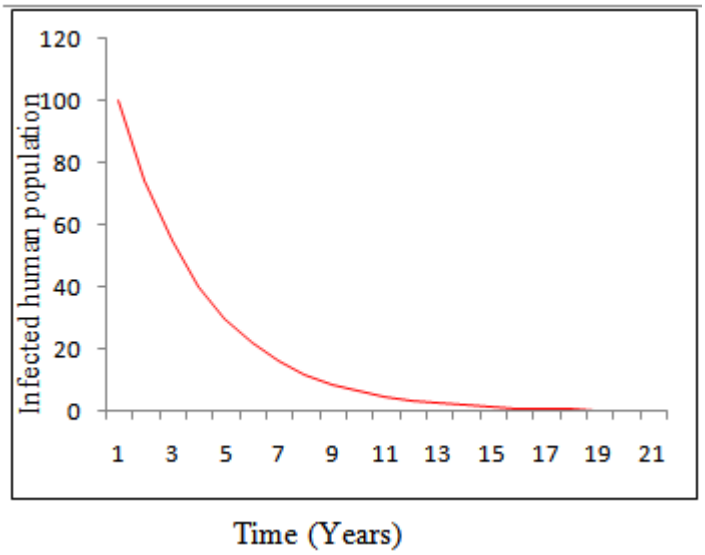

Fig 6: Graph of the infected population against time in a case of very low rate of exposure to contaminated water and very low contribution of the infected people in the aquatic environment.

V. Conclusion

In this research work we extended the model of Codeco (2000) by varying the net reproduction rate of humans and allowing for susceptibility of recovered individuals. It is proved that the disease-free equilibrium state of the extended model exists which is locally asymptotically stable under prescribed threshold conditions. Numerical experiments using published data indicated that the disease could be controlled or eradicated, provided that the rate of exposure of individuals to contaminated water and that of pollution of aquatic environment by infected people is sufficiently reduced. This is consistent with results obtained from the stability analysis of the disease-free state of the extended model.

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