International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2016): 79.57 | Impact Factor (2015): 6.391

Global Stability of a Cholera Carrier Epidemic Model with Nonlinear Incidence Functions

Lawal Jibril [1], Sule Amiru[2]

[1]Department of Mathematical Sciences, Federal University Gusau, Zamfara-State, Nigeria

[2]Department of Mathematics, Zamfara State College of Education Maru, Zamfara-State, Nigeria

Abstract: ***A new deterministic susceptible-carrier-infectious-removed-pathogen (SCIRP) cholera epidemic model with combined mass action incidence and saturated incidence rates is proposed. The threshold behavior of the model system is analyzed and establishes that cholera dies out whenever basic reproduction number is less than unity, and the disease would persist in the populations whenever the model basic reproduction number exceeds unity. The global stabilities of the model system are investigated using Lyapunov functional approach and were found to be globally asymptotically stable at both equilibrium states. Numerical simulations and graphical illustrations are presented to support the analytical results found in the study.***

Keywords: Mass action, saturated incidence, Global asymptotically stable, Cholera carrier

1. Introduction

Civil unrest, for instance, religious, ethnic, and political crisis plays a vital role in the destruction of basic services, disruption of water and sanitation system, which forced millions of people to become displaced into an inadequate environment, formation of: refugees, IDPs camps, and overcrowding the neighboring communities or camps [2]. Cholera remains global health challenges for centuries. The disease is transmitted through ingesting contaminated foods, water or vomitus containing the bacterium Vibrio cholera. Cholera is characterized by a short incubation period ranging between 2hours and 5days maximum that leads to death if untreated [1].

The incidence functions such like Mass action law, $\lambda S(t)I(t)$, the standard incidence [14], $\frac{\lambda S(t)I(t)}{N}$ the saturated incidence, $\frac{\lambda S(t)B(t)}{(k+B)}$ as well as separable incidence [14], have been applied to model cholera by many researchers to study the dynamics of the disease [17].

Complexity nature of the disease for both transmissions between human to human and indirect transmission between environments to human popularize usage of saturated incidence rate, as appeared in most literatures related to cholera and its carrier. The justification for using saturated incidence [14] is to observe the inhibition effects that exist in the contacts of infective and that of susceptible individuals in association with the crowding effect of the cholera carrier individuals in the population. The saturation level is reached whenever there is an increase in infective individuals or whenever the pathogen population is high in relations to the severity of the conflicts or the preventive measures by the susceptible individuals in curtailing the spread of the disease [4, 14].

The incidence rates were used in different forms to model cholera, for instance, Capasso and Serio [3] proposed incidence rate in the form, $\frac{kS(t)I(t)}{(1+\alpha I(t))}$ with human to human transmission only; and similar incidence rate was used in the work of [7, 5, 11] that includes both transmission pathways. Codeco [4] formulated a model using incidence rate in the form $\frac{\lambda S(t)B(t)}{(k+B(t))}$ describing the environment to human transmission model only, and similar incidence rate was used in the work of Hartley et al.[8]. In efforts to analyze the basic reproduction number for the 2008-2009 cholera epidemic outbreaks in Zimbabwe [15, 16] Mukandavire et al. reported an incidence function in the form $\beta_h S(t)I(t)+\frac{\lambda S(t)B(t)}{(k+B(t))}$, with both environment to human and human to human transmission pathways, and similar incidence rate is used in Tian and Wang [23].

The study modified the model of Kalajdzieska et al. [11], and is organized into sections as follows: Section 1 briefly discussed the role of incidence rates in the mathematical modeling of cholera disease. Section 2 presents the model formulation and description of the cholera carrier. Qualitative analysis and the model basic properties, as well as global stability behavior, are discussed in section 3. The model is simulated and plotted to support the analytical results obtained in section 4. The conclusion and references are given in section 5.

2. Model Description

In this section, the study divided the populations into two classes, the first class is the total human populations denoted by $N_H(t)$ and splitting into the following subpopulations as: susceptible individuals, $S(t)$, cholera carrier individuals $C(t)$, infectious individuals, $I(t)$, and the removed individuals, $R(t)$, respectively. The second class is the pathogen population denoted by $P(t)$ or $N_p(t)$. The population of the susceptible individuals, is generated

through new births and inflow of susceptible immigrants at a rate, π. The immigrants that are carrier increases the carrier individuals population at a rate, m, Hence, the cholera carrier transmission model is formulated with these following assumptions, that:

1) The population is considered a homogeneously mixing.
2) Transmission occurred through direct contact of susceptible individuals with infectious or carrier individuals carrying the Vibrio cholera at a rate, $\beta_1 C(t)S(t)$ and, $\beta_2 I(t)S(t)$, respectively.
3) Transmission occurred through indirect contact of susceptible individuals with pathogen populations at a rate, $\dfrac{\beta_3 P(t)S(t)}{(1+\alpha P(t))}$.
4) The contact rate is expressed as, $\left(\beta_1 C(t)+\beta_2 I(t)+\dfrac{\beta_3 P(t)}{(1+\alpha P(t))}\right)S(t)$. The environment to human transmission rate $\dfrac{\beta_3 P(t)S(t)}{(1+\alpha P(t))}$ fluctuates randomly as, α varied to reach a saturation level. The mass action law $\beta_3 P(t)S(t)$ is obtained as the parameter value, α is very small; it indicates that contact between the susceptible and the pathogen population is unlimited. However, as the pathogen population increases, the rate will respond more slowly and tends to minimize the interaction of contacts per unit time than linearly to the increased in the pathogen population and thus, the saturation level is reached.
5) It is assume that all state variables and parameters are positive for all time, $t \geq 0$. The associated variables and parameters are described and estimated in Table 1.

The cholera carrier model equations are given as follows:

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= \pi - \left[\beta_1 C(t)+\beta_2 I(t)+\frac{\beta_3 P(t)}{1+\alpha P(t)}\right]S(t) - \mu S(t)\\
\frac{dC(t)}{dt} &= (1-\sigma)\left[\beta_1 C(t)+\beta_2 I(t)+\frac{\beta_3 P(t)}{1+\alpha P(t)}\right]S(t) - (\mu+\varepsilon-m)C(t)\\
\frac{dI(t)}{dt} &= \varepsilon C(t)+\sigma\left[\beta_1 C(t)+\beta_2 I(t)+\frac{\beta_3 P(t)}{1+\alpha P(t)}\right]S(t) - (\mu+\mu_1+g\gamma)I(t)\\
\frac{dR(t)}{dt} &= g\gamma I(t) - \mu R(t)\\
\frac{dP(t)}{dt} &= d_1 C(t)+d_2 I(t) - (\mu_0 - b)P(t)
\end{aligned}\right\} \quad (1)$$

Since $R(t)$ does not appear in any of the first three equations in system (1), thus, the model system (1) is reduced to

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= \pi - \Gamma S(t) - \mu S(t)\\
\frac{dC(t)}{dt} &= (1-\sigma)\Gamma S(t) - Q_1 C(t)\\
\frac{dI(t)}{dt} &= \varepsilon C(t) + \sigma\Gamma S(t) - Q_2 I(t)\\
\frac{dP(t)}{dt} &= d_1 C(t)+d_2 I(t) - Q_3 P(t)
\end{aligned}\right\} (2)$$

Where, $\Gamma = \beta_1 C(t)+\beta_2 I(t)+\dfrac{\beta_3 P(t)}{1+\alpha P(t)}$,

$Q_1 = \mu+\varepsilon-m, Q_2 = g\mu+\mu_1+g\gamma, Q_3 = \mu_0 - b$

Subject to the initial conditions: $S(0)=S_0, C(0)=C_0, I(0)=I_0, P(0)=P_0$

For convenience, the state variables $S(t), C(t), I(t), R(t)$, and $P(t)$ are re-write as S, C, I, R and P respectively.

Table 1: Description of Variables, Parameters and their Values used in the Model Simulations

Variables	Description of variables
$S(t)$	Susceptible Population
$C(t)$	Carrier Population
$I(t)$	Symptomatic Population
$R(t)$	Removed Population
$P(t)$	Pathogen Population

Parameters	Description of Parameters	Value	Sources
π	Birth rate	0.6	Estimate
σ	Cholera carrier symptomatic	0-1	Varied
μ	Natural death	0.2	Estimate
β_1	Force of infection in susceptible	$1.5\beta_2$	[10]
β_2	Force of infection in carrier	0.05	[11]
β_3	Force of infection in pathogen	0.05	[11]
m	Immigrants with carrier	0-1	Varied
μ_1	Death due to V. Cholera	0.015	[24]
μ_0	Death of Vibrios	0.02	Estimate
b	The Growth of V.cholera	0.01	[24]
$g\gamma$	Clearance rate due to treatment	0.2	[9]
d_1	V. cholera Contribution	0-1	Varied
d_2	V. cholera Contribution	0.01	[13]
α	Saturation constant	0.02	[11]

3. Analysis of the Model

This section explored dynamical features of the model system (1) for the invariant region, stability and equilibria analysis.

3.1 Invariant region

The aim is to test for the suitable feasible region that all state variables and parameters are non-negative.

Lemma 1. The closed set

$$\Phi = \left\{(S,C,I,R,P)\in \mathrm{R}^5_+ : S+C+I+R \leq \frac{\pi}{\mu}; P \leq \frac{\pi(d_1+d_2)}{\mu(\mu_0-b)}\right\},$$ is positively-invariant and attracting with respect to the model system (1).

Proof. Adding the total human population assuming to be, $N_H(t) = S+C+I+R$. The differential equations in model system (1) yields

$$\frac{dN_H(t)}{dt}=\frac{dS}{dt}+\frac{dC}{dt}+\frac{dI}{dt}+\frac{dR}{dt}$$

With simplification we get

$$\frac{dN_H}{dt}\le \pi-\mu N_H-\mu_1 I+mC \qquad (3)$$

Setting $C=I=0$ in (3) due to the absence of carrier and cholera infection, it follows that

$$\frac{dN_H}{dt}\le \pi-\mu N_H$$

Using the standard comparison Theorem [20],

$$N_H(t)\le N(0)e^{-\mu t}+\frac{\pi}{\mu}\left[1-e^{-\mu t}\right] \qquad (4)$$

As, $t\to\infty$, in (4) the human population size, N_H approaches $\frac{\pi}{\mu}$. Thus, the limit set of the model system (1) enter the plane, Φ. Similarly, the pathogen population and assuming the total pathogen population is, $N_P(t)$. With simplification and applying standard comparison theorem, we obtained, $N_P(t)=\frac{\pi(d_1+d_2)}{\mu(\mu_0-b)}\left[1+e^{(-\mu_0-\psi)t}\right]$, and as, $t\to\infty$, the pathogen population N_P approaches $\frac{\pi(d_1+d_2)}{\mu(\mu_0-b)}$. Hence, it is clearly observed that both human and pathogen populations of the model system (1) enter the positively-invariant region in, R^5_+. Obviously, it is sufficient to consider the model system (1) in the region. Thus, the model system (1) can be regarded as epidemiologically and mathematically well-posed.

3.2 Stability and Equilibria Analysis

In this section model system (1) equilibria and stability analysis are presented.

Lemma 2. The model system (1) has two equilibria:

i) The disease-free equilibrium (*DFE*) given by

$$E_0=\left(S^0,C^0,I^0,P^0\right)=\left(\frac{\pi}{\mu},0,0,0\right)$$

The nature of stability of the model system (1) at, *DFE* , E_0, is computed using the next generation operator matrix method about the basic reproduction number R_0. The R_0, describes the average number of new infections produced by a typical infectious individual in a community. Therefore, to compute the basic reproduction number, R_0 for the model system (1) we describe the non-negative matrix, F to denote the new appearance of the disease in individuals and V denote the transfer of the disease by other means. Thus

$$F=\begin{bmatrix}(1-\sigma)\beta_1 S & (1-\sigma)\beta_2 S & (1-\sigma)\beta_3 S\\ \sigma\beta_1 S & \sigma\beta_2 S & \sigma\beta_3 S\\ 0 & 0 & 0\end{bmatrix},$$ And

$$V=\begin{bmatrix}Q_1 & 0 & 0\\ -\varepsilon & Q_2 & 0\\ -d_1 & -d_2 & Q_3\end{bmatrix}$$

The basic reproduction number of the model system (1) is given by

$$\mathrm{R}_0=\rho\left(FV^{-1}\right)=\frac{[\sigma(Q_1\beta_3Q_3+\beta_3Q_1d_2)+(1-\sigma)(\beta_1Q_2Q_3+\beta_2\delta Q_3+\beta_3\varepsilon d_2+\beta_3Q_2d_1)]\pi}{\mu Q_1Q_2Q_3}$$

Where

ρ Is the dominant eigen-value (spectral radius) of the next generation matrix, FV^{-1}. Thus, Theorem 1 established the following result.

Theorem 1 The *DFE* , E_0 of the model system (1) is locally asymptotically stable (LAS), if, $\mathrm{R}_0<1$ and unstable if $\mathrm{R}_0>1$.

ii) The endemic equilibrium point (*EEP*) E^* of the model system (1) is given by the following results, $E^*=\left(S^*,C^*,I^*,P^*\right)$

Where

$$S^*=\frac{\pi}{\Gamma+\mu},\ C^*=\frac{\Gamma\pi L_3}{Q_1(\Gamma+\mu)},\ I^*=\frac{\Gamma\pi L_2}{Q_1Q_2(\Gamma+\mu)},\ P^*=\frac{\Gamma\pi L_1}{Q_1Q_2Q_3(\Gamma+\mu)}$$

Similarly, the endemic equilibrium E^* can be expressed in terms of R_0 to establish the following results

$$S^{**}=\frac{\pi}{\mu\mathrm{R}_0},\ C^{**}=\frac{\mu(\mathrm{R}_0-1)\pi L_3}{\mu\mathrm{R}_0Q_1},\ I^{**}=\frac{\mu(\mathrm{R}_0-1)\pi L_2}{Q_1Q_2\mu\mathrm{R}_0},\ P^{**}=\frac{\mu(\mathrm{R}_0-1)\pi L_1}{Q_1Q_2Q_3\mu\mathrm{R}_0}$$

With

$$L_1=d_1Q_2(1-\sigma)+d_2(\varepsilon(1-\sigma)+Q_1\sigma),\ L_2=\varepsilon(1-\sigma)+\sigma Q_1,\ L_3=(1-\sigma)$$

$$\Gamma=\beta_1C+\beta_2I+\frac{\beta_3P}{1+\alpha P},\ Q_1=\mu+\varepsilon-m,\ Q_2=\mu+\mu_1+g\gamma,\ Q_3=\mu_0-b$$

The following result from (ii) is summarized in Lemma 3.

Lemma 3. If $\mathrm{R}_0<1$, then the point E^* does not exist and there exists a unique endemic equilibrium if and only if $\mathrm{R}_0>1$..

3.3. Global Stability of DFE and EE

Special case

With reference to [14, 15] .

Theorem 2. The *DFE* of the model system (1) is globally asymptotically stable (GAS) whenever, $\mathrm{R}_0\le 1$.

Proof. Consider the Lyapunov function

$$L_1=\left(\frac{\beta_1Q_2Q_3+\beta_3d_1Q_2+\varepsilon(\beta_2Q_3+\beta_3d_2)}{Q_1Q_2}\right)C+\left(\frac{\beta_2Q_3+\beta_3d_2}{Q_3}\right)I+(\beta_3)P$$

With the Lyapunov derivative given by

$$\dot{L}_1=\left(\frac{\beta_1Q_2Q_3+\beta_3d_1Q_2+\varepsilon(\beta_2Q_3+\beta_3d_2)}{Q_1Q_2}\right)\dot{C}+\left(\frac{\beta_2Q_3+\beta_3d_2}{Q_3}\right)\dot{I}+(\beta_3)\dot{P} \qquad (5)$$

Substituting model system (1) into (5)

$$\dot{L}_1=\frac{\beta_1Q_2Q_3+\beta_3d_1Q_2+\varepsilon(\beta_2Q_3+\beta_3d_2)}{Q_1Q_2}\left[\left(\beta_1(1-\sigma)C+\beta_2(1-\sigma)I+\frac{\beta_3(1-\sigma)P}{1+\alpha P}\right)S-Q_1C\right]$$
$$+\frac{\beta_2Q_3+\beta_3d_2}{Q_3}\left(\left[\beta_1\sigma C+\beta_2\sigma I+\frac{\beta_3\sigma P}{1+\alpha P}\right]S+\delta C-Q_2I\right)+\beta_3(d_1C+d_2I-Q_3P)$$

Using $\frac{S}{1+\alpha P} \le S \le \frac{\pi}{\mu}$ with further simplification to obtain

$\dot{L}_1 \le Q_3(\beta_1 C + \beta_2 I + \beta_3 P)(R_0 - 1)$

Thus, $\dot{L}_1 = 0$ only when, $C = I = P = 0$. On substituting $C = 0$, $I = 0$ and $P = 0$ into the model system (1) shows that $S \to \frac{\pi}{\mu}$ as $t \to \infty$. Obviously, the largest compact invariant set in $\{(S, C, I, P) \in \Omega : \dot{L}_1 \le 0\}$ is the singleton set $\{\varepsilon_0\}$. Thus, the global stability of $\{\varepsilon_0\}$ when $R_0 \le 1$ follows from LaSalle's invariance principle that every solution to the model system (1) with initial conditions in that set converges to the *DFE* as, $t \to \infty$.

Theorem 3. (Global stability of the EEP) The endemic equilibrium point E^* of model system (1) is globally asymptotically stable if and only if $R_0 > 1$.

Proof. Consider the non-linear Lyapunov function of Goh-Volterra type

$$L_2 = \left[S - S^* - S^* ln\frac{S}{S^*}\right] + \left[C - C^* - C^* ln\frac{C}{C^*}\right] + \frac{(\beta_2 Q_3 + \beta_3 d_2)S^*}{Q_2 Q_3}\left[I - I^* - I^* ln\frac{I}{I^*}\right] + \frac{\beta_3 S^*}{Q_3}\left[P - P^* - P^* ln\frac{P}{P^*}\right]$$

With Lyapunov derivatives

$$\dot{L}_2 = \left[1 - \frac{S^*}{S}\right]\dot{S} + \left[1 - \frac{C^*}{C}\right]\dot{C} + \left[\frac{\beta_2}{Q_2} + \frac{\beta_3 d_2}{Q_2 Q_3}\right]S^*\left[1 - \frac{I^*}{I}\right]\dot{I} + \frac{\beta_3 S^*}{Q_3}\left[1 - \frac{P^*}{P}\right]\dot{P} \quad (6)$$

With substitution model system (1) into equation (6)

$$\dot{L}_2 = \left[1 - \frac{S^*}{S}\right](\pi - [\beta_1 C + \beta_2 I + \beta_3 P]S - \mu S) + \left[1 - \frac{C^*}{C}\right]([\beta_1 C + \beta_2 I + \beta_3 P]S - Q_1 C) + S^*\left(\frac{\beta_2}{Q_2} + \frac{\beta_3 d_2}{Q_2 Q_3}\right)\left[1 - \frac{I^*}{I}\right](\varepsilon C - Q_2 I) + \frac{\beta_3 S^*}{Q_3}\left[1 - \frac{P^*}{P}\right](d_1 C + d_2 I - Q_3 P)$$

Using

$\pi = (\beta_1 C^* + \beta_2 I^* + \beta_3 P^*)S^* + \mu S^*$, $Q_1 C^* = (\beta_1 C^* + \beta_2 I^* + \beta_3 P^*)S^*$

, $Q_2 I^* = \varepsilon C^*$, $P^* = \frac{d_1 C^*}{Q_3} + \frac{d_2 I^*}{Q_3}$

With simplifications

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + [\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]S^* - [\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]\frac{S^{*2}}{S} - [\beta_1 C + \beta_2 I + \beta_3 P]\frac{SC^*}{C} + [\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]S^* + \frac{(\beta_2 Q_3 + \beta_3 d_2)S^*}{Q_2 Q_3}\left[Q_2\left(I^* - \frac{CI^{*2}}{C^* I}\right)\right] + \frac{\beta_3 S^*}{Q_3}\left[P^*\left(-\frac{d_1 C}{P} - \frac{d_2 I}{P}\right)\right] + \beta_3 S^* P^*$$

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + 2[\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]S^* - [\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]\frac{S^{*2}}{S} - [\beta_1 C + \beta_2 I + \beta_3 P]\frac{SC^*}{C} + (\beta_2 + \frac{\beta_3 d_2}{Q_3})S^* I^* - (\beta_2 + \frac{\beta_3 d_2}{Q_3})\frac{S^* CI^{*2}}{CI^*} - \beta_3 S^* P^*\left[\frac{d_1 C}{Q_3 P} + \frac{d_2 I}{Q_3 P}\right] + \beta_3 S^* P^*$$

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + 2[\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]S^* - [\beta_1 C^* + \beta_2 I^* + \beta_3 P^*]\frac{S^{*2}}{S} - [\beta_1 C + \beta_2 I + \beta_3 P]\frac{SC^*}{C} + \beta_2 S^* I^* + \frac{\beta_3 d_2 S^* I^*}{Q_3} - \frac{\beta_2 S^* CI^{*2}}{C^* I} - \frac{\beta_3 d_2 S^* CI^{*2}}{Q_3 C^* I} - \frac{\beta_3 S^* P^*}{Q_3}\left[\frac{C}{P}\left(\frac{Q_3 P^*}{C^*} - \frac{d_2 I^*}{C^*}\right) + \frac{I}{P}\left(\frac{Q_3 P^*}{I^*} - \frac{d_1 C^*}{I^*}\right)\right] + \beta_3 S^* P^*$$

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_1 C^* S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_2 I^* S^*\left[3 - \frac{S^*}{S} - \frac{SIC^*}{S^* I^* C} - \frac{I^* C^*}{C^* I}\right] + \beta_3 P^* S^*\left[3 - \frac{S^*}{S} - \frac{SPC^*}{S^* P^* C} - \frac{P^* C}{C^* P}\right] + \frac{\beta_3 d_2 S^* I^*}{Q_3}\left[1 - \frac{CI^*}{C^* I} - \frac{P^* I}{PI^*}\right] + \frac{\beta_3 d_2 S^* I^*}{Q_3}\frac{P^* C}{C^* P} + \frac{2\beta_3 d_2 S^* I^*}{Q_3} - \frac{2\beta_3 d_2 S^* I^*}{Q_3} + \frac{\beta_3 d_2 S^* I^* PC^*}{Q_3 P^* C} - \frac{\beta_3 d_2 S^* I^* PC^*}{Q_3 P^* C}$$

Further simplification yields

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_1 C^* S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_2 I^* S^*\left[3 - \frac{S^*}{S} - \frac{SIC^*}{S^* I^* C} - \frac{I^* C^*}{C^* I}\right] + \beta_3 P^* S^*\left[3 - \frac{S^*}{S} - \frac{SPC^*}{S^* P^* C} - \frac{P^* C}{C^* P}\right] + \frac{\beta_3 d_2 S^* I^*}{Q_3}\left[3 - \frac{CI^*}{C^* I} - \frac{P^* I}{PI^*} - \frac{PC^*}{P^* C}\right] + \frac{\beta_3 d_2 S^* I^*}{Q_3}\left(\sqrt{\frac{P^* C}{C^* P}} - \sqrt{\frac{PC^*}{P^* C}}\right)^2$$

$$= \mu S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_1 C^* S^*\left[2 - \frac{S^*}{S} - \frac{S}{S^*}\right] + \beta_2 I^* S^*\left[3 - \frac{S^*}{S} - \frac{SIC^*}{S^* I^* C} - \frac{I^* C^*}{C^* I}\right] + \beta_3 P^* S^*\left[3 - \frac{S^*}{S} - \frac{SPC^*}{S^* P^* C} - \frac{P^* C}{C^* P}\right] + \frac{\beta_3 d_2 S^* I^*}{Q_3}\left[\left(\sqrt{\frac{P^* C}{C^* P}} - \sqrt{\frac{PC^*}{P^* C}}\right)^2 + 3 - \frac{CI^*}{C^* I} - \frac{P^* I}{PI^*} - \frac{PC^*}{P^* C}\right] \le 0$$

Obviously, the arithmetic mean exceeds the geometric mean, and then it follows that

$$2 - \frac{S^*}{S} - \frac{S}{S^*} \le 0,\quad 2 - \frac{S^*}{S} - \frac{S}{S^*} \le 0,\ 3 - \frac{S^*}{S} - \frac{SIC^*}{S^* I^* C} - \frac{I^* C^*}{C^* I} \le 0,$$

$$3 - \frac{S^*}{S} - \frac{SPC^*}{S^* P^* C} - \frac{P^* C}{C^* P} \le 0,\ \left(\sqrt{\frac{P^* C}{C^* P}} - \sqrt{\frac{PC^*}{P^* C}}\right)^2 \le 0,\ 3 - \frac{CI^*}{C^* I} - \frac{P^* I}{PI^*} - \frac{PC^*}{P^* C} \le 0$$

Hence, $\dot{L}_2 \le 0$.

Thus, by the Lyapunov stability criteria and LaSalle invariance principle every results of the model system (1) $\dot{L}_2 \le 0$, if $\dot{L}_2 = 0$ holds only when $S = S^*$, $C = C^*$, $I = I^*$ and, $P = P^*$. Obviously, the largest compact invariant set in $\left\{(S, C, I, P) \in \Omega : \dot{L}_2 = 0\right\}$ is the endemic equilibrium point as $t \to \infty$ for $R_0 > 1$. Hence, the *EEP* is globally asymptotically stable (GAS) in, Ω.

4. Numerical Simulations and Discussions

This section presents the numerical simulations and graphical Illustrations that demonstrated the analytical results of the proposed cholera carrier model. This is achieved by using the set of parameter values given in Table 1 and whose sources are mainly from literatures as well as assumptions. The model is simulated to establish the results in Theorem 1 and 2 for the threshold criteria about basic reproduction number which determines global dynamics of the model system (1) for the disease to be eliminated in the population or it will persist in the population. Thus, on setting the initial conditions for the human and pathogen populations' size as follows, so that

$$N(t) = S(t) + C(t) + I(t) + R(t) = 3$$

$S(0) = 1.80$; $C(0) = 0.6$; $I(0) = 0.45$; $R(0) = 0.15$; $P(0) = 15$

$S(0) = 2.30$; $C(0) = 0.5$; $I(0) = 0.15$; $R(0) = 0.05$; $P(0) = 10$

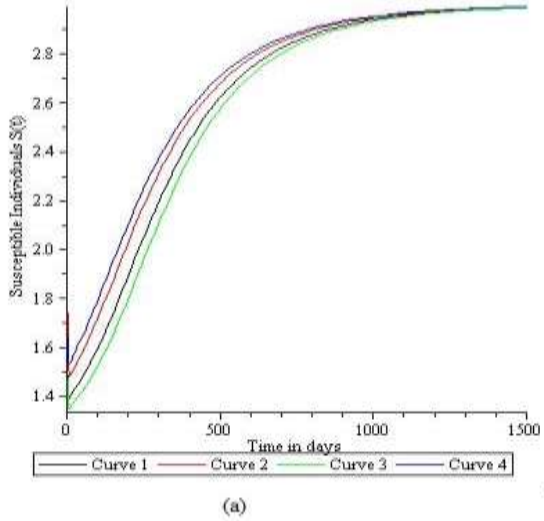
$S(0) = 1.89$; $C(0) = 0.75$; $I(0) = 0.26$; $R(0) = 0.1$; $P(0) = 20$

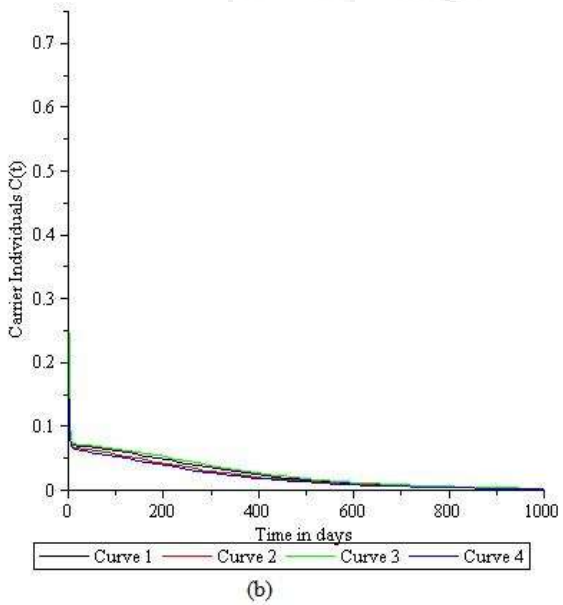
$S(0) = 1.95$; $C(0) = 0.35$; $I(0) = 0.65$; $R(0) = 0.05$; $P(0) = 08$

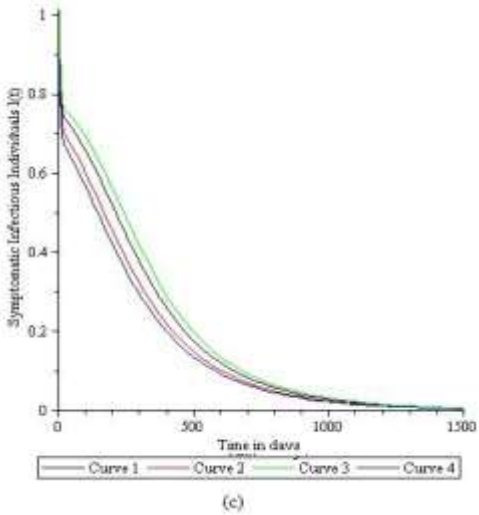
In Figure 1 (a - e), the time plot of model system (1) is presented to exhibit the feasibility of the obtained results when $R_0 < 1$ using $\sigma = 0.8, m = 0.1, \varepsilon = 0.8, d_1 = 0.01$ then, the basic reproduction number, $R_0 = 0.7902 < 1$. By

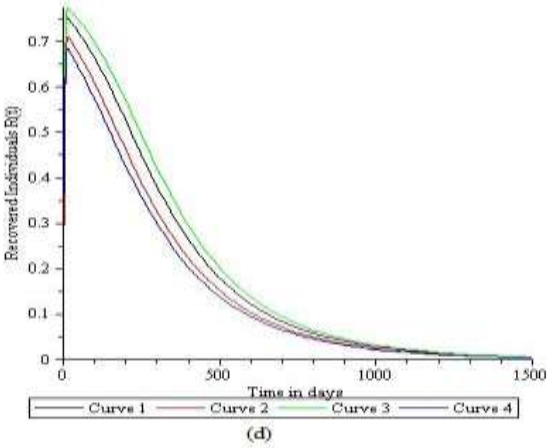
Theorem 1, for any initial values considered and whenever $R_0 < 1$, then the cholera free equilibrium is globally asymptotically stable and eventually cholera disease dies out in the population.

In Figure 2 (a - e), shows the feasibility of the obtained results of the model system (1) when, $R_0 > 1$ using $\sigma = 0, m = 0.1, \varepsilon = 0.8, d_1 = 0.1, \alpha = 0$ then, the basic reproduction number, $R_0 = 2.5592 > 1$. By Theorem 2, for any initial values considered, and whenever $R_0 > 1$ the cholera endemic equilibrium is globally asymptotically stable and it assumed that the cholera disease will invades the population.









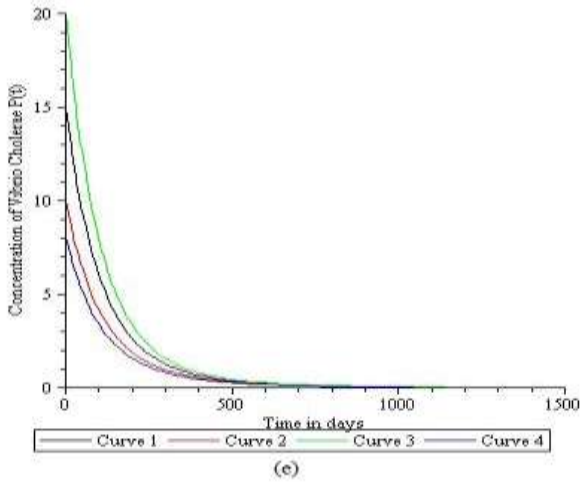
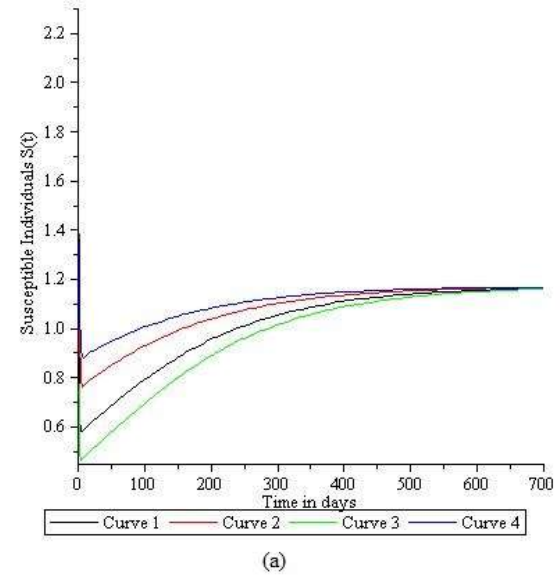
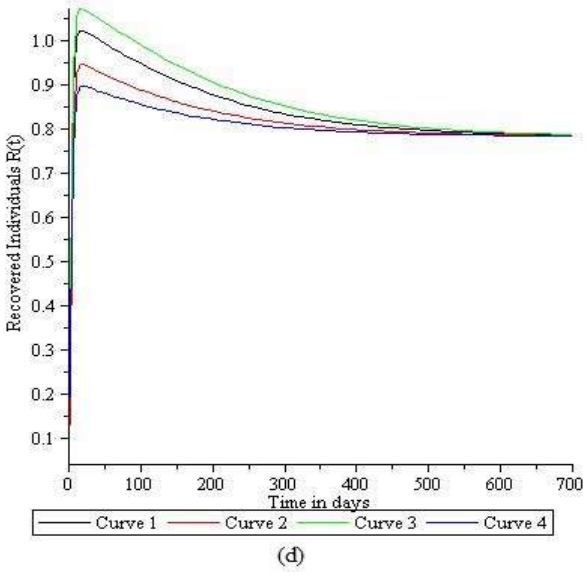
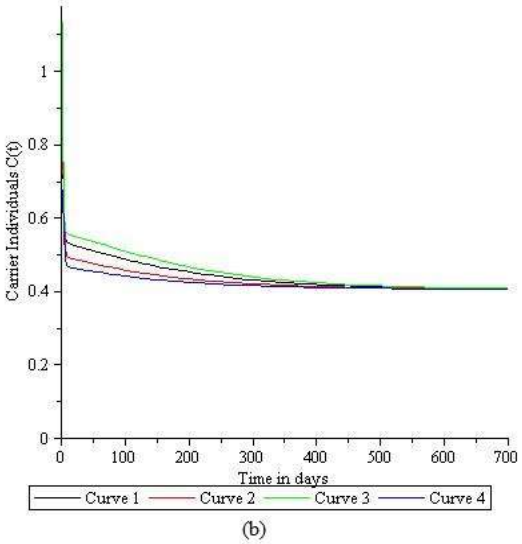
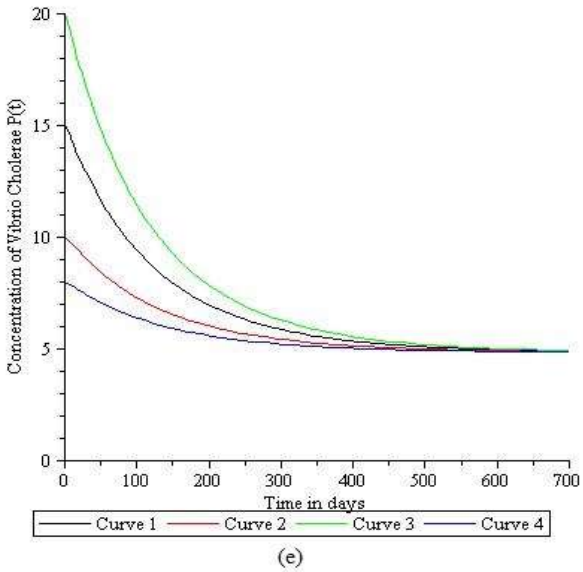


Figure 1 (a-e): Simulations results depicting time plots of (a) Susceptible population (b) Carrier population (c) Symptomatic population (d) Recovered population (e) Pathogen population, converges to the Cholera free states whenever $R_0 < 1$ thus the disease will die out.









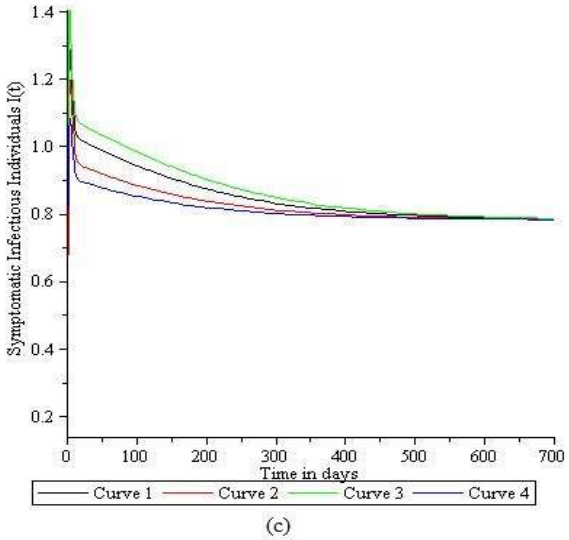


Figure 2 (a-e): Simulation results depicting time plots of (a) Susceptible population (b) Carrier population (c) Symptomatic population (d) Recovered population (e) Pathogen population approaches their unique endemic steady state whenever $R_0 > 1$, thus the disease will invades the population.

5. Conclusion

A cholera carrier epidemic model with both mass action incidence and saturated incidence rates using deterministic model were formulated and analyzed. The major findings of this study are:

1) Model system (1) is tested for invariant region and was found to be epidemiologically and mathematically well-posed in the region of study.
2) Using Lyapunov functional approach it is establish that the cholera free equilibrium was GAS whenever the associated basic reproduction number is less than unity. Thus, the Cholera dies out in the community.
3) Using Lyapunov functional approach (a special case) it is establish that the model has a unique endemic equilibrium which is GAS whenever the associated basic

reproduction number exceed unity. Thus, the Cholera will persist in the population.

4) The simulations of the model system (1) are conducted to support the analytical results obtained and the displayed results established in Theorem 1 and Theorem 2.

References

[1] A. S. Azman, K. E. Rudolph, D. A. T. Cummings, J. Lessler, The Incubation Period of Cholera: A Systematic Review, Journal of Infection 66 (2013) 432-438.

[2] Assessment Capacities Projects, ACAPS, Cholera Epidemic: the Central African Republic, Available at http://www.acaps.org (2016) Accessed 15th March 2017.

[3] V. Capasso and S. L. Paveri-Fontana, A Mathematical Model for the 1973 Cholera Epidemic in the European Mediterranean region, Revenue Depidemoligie Et de Sante Publique 27(2) (1979) 121-132.

[4] C. T. Codeco, Endemic and Epidemic Dynamics of Cholera: The Role of the Aquatic Reservoir, Biomed Central: Infectious Disease 1(1) (2001).

[5] J. Cui, Z. Wu, X. Zhou, Mathematical Analysis of a Cholera Model with Vaccination, J. of Appl. Math Volume 2014 Article ID 324767 http://dx.doi.org/10.1155/2014/324767.

[6] Y. Emvudu, and E. Kokomo, Stability analysis of cholera epidemic model of a closed population. J. of Applied Maths and Bioinformatics 2(1) (2012) 69-97.

[7] N. H. Gazi, K. Das, Z. Mukandavire, C. Chiyaka, and P. Das, A Study of Cholera Model with Environmental Fluctuations, Int. J. of Mathe. Mod. Meth. App. Sci. 3(4) (2010) 150-156.

[8] D. M. Hartley, J. G. Morris, D. L. Smith, Hyperinfectivity: a critical element in the ability of V. cholerae to cause epidemics? PLoS Med. 3(1) e7 (2006).

[9] A. O. Isere, J. E. Osemwenkhae, D. Okuonghae, Optimal control model for the outbreak of cholera in Nigeria, Afri. J. Math. Operat. Res. 7(2) (2014) 24 -30.

[10] D. Kalajdzievska, and M. Y. Li, Modeling the effects of carriers on Transmission Dynamics of Infectious Diseases. Mathe. Biosc. Eng. 8(3): (2011) 711 -727.

[11] M. A. Khan, A. Ali, L. C. C. Dennis, S. Islam, I. Khan, M. Ullah, T. Gul, the Dynamical behavior of Cholera epidemic model with nonlinear incidence rate. App. Mathe. Sci. 9(20) (2015) 989 - 1002.

[12] Z. Mukandavire, S. Liao, J. Wang, H. Gaff, D. L. Smith, Estimating the Basic Reproduction Number for the 2008-2009 Cholera Outbreak in Zimbabwe, proceeding of the Natio. Aca. Sci. U.S.A. 108(21) (2011) 8767-8772.

[13] Z. Mukandavire, D. L. Smith, J. G. Morris, Cholera in Haiti: Reproductive numbers and Vaccination Coverage Estimates. Scientific Reports 3 1997 (2013) DOI: 10:1038/srep 00997.

[14] C. C. McCluskey, Global Stability of an SIR Epidemic Model with Delay and General Nonlinear Incidence, Math. Biosc. Eng. 7(4) (2010) 837-850.

[15] F. Muthuramalingan, V. Ananthaswamy, R. Lakshmanan, Nonlinear Analysis of Cholera Epidemics, Applied Mathematics 4(1): (2014) 12-21.

[16] R. L. M. Neilan, E. Schaefer, H. Gaff, K. R. Fister, S. Lenhart S, Modeling Optimal Intervention Strategies for Cholera, Bull. Math. Biol. 72 (2010) 2004-2018.

[17] J. M. Ochoche, Modeling HIV in the Presence of Infected Immigrants and Vertical Transmission: the Role of Incidence Function, Int. J. Sci. Tech. Res. 2(11) (2013).

[18] K. O. Okosun and O. D. Makinde, A Co-infection Model of Malaria and Cholera Disease with Optimal Control. Mathematical Biosciences 000: (2014) 1-14.

[19] D. Okuonghae, A. B. Gumel, M. A. Safi, Dynamics of a Two Strain Vaccination Model for Polio, Nonlinear Analysis: Real World Applications 25 (2015) 157-189.

[20] M. A. Safi, A. B. Gumel, The Effect of Incidence Functions on the Dynamics of a Quarantine/ Isolation Model with Delay, Nonlinear Analysis: Real World Applications 12 (2011) 215-235.

[21] O. Sharomi and A. B. Gumel, Mathematical Study of a Risk Structured Two Group Model for Chlamydia Transmission Dynamics, App. Math. Modeling 35 (2011) 3653-3673.

[22] J. P. Tian, J. Wang, Global Stability for Cholera Epidemic Models, Mathematical Biosciences 232 (2011) 31-41.

[23] World Health Organization (2012), Cholera: epidemic disease. Available at http://www.who.int/gho/epidemicdisease/cholera/en/index (Accessed February, 2016).