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

Lawal Jibril 1, Sule Amiru2

1Department of Mathematical Sciences, Federal University Gusau, Zamfara-State, Nigeria
2Department 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 2 hours and 5 days 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, $\frac{\lambda S(t)B(t)}{(k+B(t))}$ 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. Codeço [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] Mukanadavire et al. reported an incidence function in the form $\beta S(t)I(t) + \frac{\lambda S(t)B(t)}{(k+B(t))}$, 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 Kalajdzieva 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, \( \pi \). 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, \( \beta C(t)S(t) \) and, \( \beta I(t)S(t) \), respectively.

3) Transmission occurred through indirect contact of susceptible individuals with pathogen populations at a rate, \( \frac{\beta P(t)S(t)}{1+\alpha P(t)} \).

4) The contact rate is expressed as, \( \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) \). The environment to human transmission rate \( \frac{\beta P(t)S(t)}{1+\alpha P(t)} \) fluctuates randomly as, \( \alpha \) varied to reach a saturation level. The mass action law \( \beta P(t)S(t) \) is obtained as the parameter value, \( \alpha \) 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 assumed 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:

\[
\begin{align*}
\frac{dS(t)}{dt} &= -\pi \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - \mu S(t) \\
\frac{dC(t)}{dt} &= (1-\sigma) \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \epsilon + m) C(t) \\
\frac{dl(t)}{dt} &= \epsilon C(t) + \sigma \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \mu_c + \gamma) I(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t) \\
\frac{dP(t)}{dt} &= d_C C(t) + d_I I(t) - (\mu_c - b) P(t)
\end{align*}
\]

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

\[
\begin{align*}
\frac{dS(t)}{dt} &= -\pi \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - \mu S(t) \\
\frac{dC(t)}{dt} &= (1-\sigma) \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \epsilon + m) C(t) \\
\frac{dl(t)}{dt} &= \epsilon C(t) + \sigma \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \mu_c + \gamma) I(t) \\
\frac{dP(t)}{dt} &= d_C C(t) + d_I I(t) - (\mu_c - b) P(t)
\end{align*}
\]

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.

\[
\begin{align*}
\Gamma &= \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \\
Q_1 &= \mu + \epsilon - m, Q_2 = g \mu + \mu_t + g \gamma, Q_3 = \mu_c - b
\end{align*}
\]

Where, \( \mu \) is the natural death rate, \( \epsilon \) is the death rate due to vibriosis, \( m \) is the birth recruitment rate. The Cholera carrier model equations are given as follows:

\[
\begin{align*}
\frac{dS(t)}{dt} &= \pi - \Gamma S(t) - \mu S(t) \\
\frac{dC(t)}{dt} &= (1-\sigma) \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \epsilon + m) C(t) \\
\frac{dl(t)}{dt} &= \epsilon C(t) + \sigma \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \mu_c + \gamma) I(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t) \\
\frac{dP(t)}{dt} &= d_C C(t) + d_I I(t) - (\mu_c - b) P(t)
\end{align*}
\]

By\( \Delta \)\( \triangleleft \)\( \triangleright \)

\[
\frac{dS(t)}{dt} = \pi - \Gamma S(t) - \mu S(t) \\
\frac{dC(t)}{dt} = (1-\sigma) \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \epsilon + m) C(t) \\
\frac{dl(t)}{dt} = \epsilon C(t) + \sigma \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \mu_c + \gamma) I(t) \\
\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t) \\
\frac{dP(t)}{dt} = d_C C(t) + d_I I(t) - (\mu_c - b) P(t)
\]

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

<table>
<thead>
<tr>
<th>Variables</th>
<th>Description of Variables</th>
<th>Value</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>( S(t) )</td>
<td>Susceptible Population</td>
<td>0.6</td>
<td>Estimate</td>
</tr>
<tr>
<td>( C(t) )</td>
<td>Carrier Population</td>
<td>0.1</td>
<td>Varied</td>
</tr>
<tr>
<td>( I(t) )</td>
<td>Symptomatic Population</td>
<td>0.2</td>
<td>Estimate</td>
</tr>
<tr>
<td>( R(t) )</td>
<td>Removed Population</td>
<td>0.05</td>
<td>[10]</td>
</tr>
<tr>
<td>( P(t) )</td>
<td>Pathogen Population</td>
<td>0.05</td>
<td>[11]</td>
</tr>
<tr>
<td>( \pi )</td>
<td>Birth rate</td>
<td>1.5 ( \beta )</td>
<td>[11]</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>Cholera carrier symptomatic</td>
<td>0.05</td>
<td>[11]</td>
</tr>
<tr>
<td>( \mu )</td>
<td>Natural death</td>
<td>0.05</td>
<td>[11]</td>
</tr>
<tr>
<td>( \mu_c )</td>
<td>Immigrants with carrier</td>
<td>0.01</td>
<td>[10]</td>
</tr>
<tr>
<td>( \mu_t )</td>
<td>Death due to V. Cholera</td>
<td>0.015</td>
<td>[24]</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>Death of Vibriosis</td>
<td>0.02</td>
<td>Estimate</td>
</tr>
<tr>
<td>( \beta )</td>
<td>The Growth of V. cholera</td>
<td>0.01</td>
<td>[24]</td>
</tr>
<tr>
<td>( b )</td>
<td>The Growth of V. cholera</td>
<td>0.01</td>
<td>[24]</td>
</tr>
<tr>
<td>( \gamma )</td>
<td>Clearnace rate due to treatment</td>
<td>0.2</td>
<td>[9]</td>
</tr>
<tr>
<td>( d_c )</td>
<td>V. cholera Contribution</td>
<td>0.1</td>
<td>Varied</td>
</tr>
<tr>
<td>( d_I )</td>
<td>V. cholera Contribution</td>
<td>0.01</td>
<td>[13]</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>Saturation constant</td>
<td>0.02</td>
<td>[11]</td>
</tr>
</tbody>
</table>

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

\[
\Phi = \left\{ (S, C, I, R) \in \mathbb{R}_+^4 : S + C + I + R \leq \frac{\pi}{\mu} ; P \leq \frac{\pi}{\mu} (\mu_c - b) \right\}
\]

is positively-invariant and attracting with respect to the model system (1).

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

\[
\frac{dS(t)}{dt} = \pi - \Gamma S(t) - \mu S(t) \\
\frac{dC(t)}{dt} = (1-\sigma) \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \epsilon + m) C(t) \\
\frac{dl(t)}{dt} = \epsilon C(t) + \sigma \left( \beta C(t) + \beta I(t) + \frac{\beta P(t)}{1+\alpha P(t)} \right) S(t) - (\mu + \mu_c + \gamma) I(t) \\
\frac{dR(t)}{dt} = \gamma I(t) - \mu R(t) \\
\frac{dP(t)}{dt} = d_C C(t) + d_I I(t) - (\mu_c - b) P(t)
\]
The basic reproduction number of the model system (1) is given by

\[ R_0 = \rho(FV^{-1}) \]

Where \( \rho \) 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, \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

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

\[ S^* = \frac{\pi}{\mu + \mu}, \quad C^* = \frac{\Gamma \pi L_0}{Q_0 Q_0 (T + \mu)}, \quad I^* = \frac{\Gamma \pi L_0}{Q_0 Q_0 (T + \mu)}, \quad P^* = \frac{\Gamma \pi L_0}{Q_0 Q_0 (T + \mu)} \]

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

\[ S^* = \frac{\pi}{\mu R_0}, \quad C^* = \frac{\mu R_0 (1 - \sigma)}{\mu R_0}, \quad I^* = \frac{\mu R_0 (1 - \sigma)}{\mu R_0}, \quad P^* = \frac{\mu R_0 (1 - \sigma)}{\mu R_0} \]

With

\[ L_1 = d_1 Q_1 (1 - \sigma) + d_2 (1 - \sigma) + \sigma Q_1, \quad L_2 = \sigma - \sigma Q_1, \quad L_3 = (1 - \sigma) \]

\[ \Gamma = \beta C + \beta I + \frac{\beta P}{1 + \alpha P}, \quad Q_3 = \mu + \mu + \mu + \mu + \mu \]

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

**Lemma 3.** If \( R_0 < 1 \), then the point \( E^* \) does not exist and there exists a unique endemic equilibrium if and only if \( R_0 > 1 \).

### 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, \( R_0 \leq 1 \).

**Proof.** Consider the Lyapunov function

\[ L_1 = \left( \frac{\beta Q_1 Q_1 + \beta d_1 Q_1 + \beta (\beta Q_1 + \beta d_1)}{Q_1 \Omega} \right) C + \left( \frac{\beta Q_2 + \beta d_2}{Q_2} \right) I + (\beta)^P \]

With the Lyapunov derivative given by

\[ L_i = \left( \frac{\beta Q_1 Q_1 + \beta d_1 Q_1 + \beta (\beta Q_1 + \beta d_1)}{Q_1 \Omega} \right) C + \left( \frac{\beta Q_2 + \beta d_2}{Q_2} \right) I + (\beta)^P \]

Substituting model system (1) into (5)

\[ L_i = \left( \frac{\beta Q_1 Q_1 + \beta d_1 Q_1 + \beta (\beta Q_1 + \beta d_1)}{Q_1 \Omega} \right) C + \left( \frac{\beta Q_2 + \beta d_2}{Q_2} \right) I + (\beta)^P \]

\[ + \left( \beta d_1 \frac{\beta + \beta d_1}{Q_1} \right) \left( \beta - 1 \right) C + \left( \beta d_1 \frac{\beta + \beta d_1}{Q_1} \right) I + (\beta)^P \]

\[ \left( \beta d_1 \frac{\beta + \beta d_1}{Q_1} \right) \left( \beta - 1 \right) C + \left( \beta d_1 \frac{\beta + \beta d_1}{Q_1} \right) I + (\beta)^P \]
Using \( \frac{S}{1+\alpha P} \leq S \leq \frac{\pi}{\mu} \) with further simplification to obtain
\[
\dot{S} \leq Q_1 (\beta C + \beta_2 I + \beta_3 P)(R_0 - 1)
\]
Thus, \( \dot{S} = 0 \) only when, \( C = I = P = 0 \). On substituting \( C = 0 \), \( I = 0 \) and \( P = 0 \) into the model system (1) shows that \( S \rightarrow \frac{\pi}{\mu} \) as \( t \rightarrow \infty \). Obviously, the largest compact invariant set in \( \{(S, C, I, P) \in \Omega : \dot{S} \leq 0\} \) is the singleton set \( \{\epsilon_0\} \). Thus, the global stability of \( \{\epsilon_0\} \) when \( R_0 \leq 1 \) 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, \( t \rightarrow \infty \).

**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
\[
L_0 = \left[ S - S \ln \frac{S}{S^*} - (1 - \frac{1}{C}) \left( \frac{(\beta C_I + \beta_2 I + \beta_3 P)S - Q_1}{Q_1} \right) \right] + \frac{\beta S}{Q_1} \left( P - P^* - \frac{P}{P^*} P \right)
\]
With Lyapunov derivatives
\[
\dot{L}_0 = \left[ 1 - \frac{S}{S^*} (1 - \frac{1}{C}) \right] \dot{S} + \frac{\beta S}{Q_1} \left( P - P^* - \frac{P}{P^*} P \right)
\]
By substitution model system (1) into equation (6)
\[
\dot{L}_0 = \left[ 1 - \frac{S}{S^*} (1 - \frac{1}{C}) \right] S + \frac{\beta S}{Q_1} \left( P - P^* - \frac{P}{P^*} P \right)
\]
Using
\[
\pi = \left( \beta C + \beta_2 I + \beta_3 P \right)S^* + \mu S^* \quad \text{and} \quad \dot{Q}_1 = \epsilon Q_1^* \quad \dot{P}^* = \frac{d C}{Q_1^*} + \frac{d I}{Q_1^*} - \frac{d Q}{Q_1^*}
\]
With simplifications
\[
-\mu S \left[ \frac{S}{S^*} \left( \beta C + \beta_2 I + \beta_3 P \right)S^* - \frac{\beta C + \beta_2 I + \beta_3 P}{S^*} \right] \left( \beta C + \beta_2 I + \beta_3 P \right) \mu S^*
\]
Further simplification yields
\[
-\mu S \left[ \frac{S}{S^*} \left( \beta C + \beta_2 I + \beta_3 P \right)S^* - \frac{\beta C + \beta_2 I + \beta_3 P}{S^*} \right] \left( \beta C + \beta_2 I + \beta_3 P \right) \mu S^*
\]
Obviously, the arithmetic mean exceeds the geometric mean, and then it follows that
\[
2 \left( \frac{S}{S^*} \right) \leq 2 \leq 0.7902 \leq 1 \quad \text{and} \quad P = P^* \quad \text{Obviously, the largest}
\]
compact invariant set in \( \{(S, C, I, P) \in \Omega : \dot{S} \leq 0\} \) is the endemic equilibrium point as \( t \rightarrow \infty \) for \( R_0 > 1 \). Hence, the EEP is globally asymptotically stable (GAS) in \( \Omega \).

**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
\[
N(t) = S(t) + C(t) + I(t) + R(t) = 3
\]
In Figure 1 (a - c), the time plot of model system (1) is presented to exhibit the feasibility of the obtained results when \( R_0 < 1 \) using \( \sigma = 0.8, m = 0.1, \epsilon = 0.8, d_1 = 0.01 \) then, the basic reproduction number, \( R_0 = 0.7902 < 1 \).
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 $\sigma = 0, m = 0.1, \epsilon = 0.8, d_1 = 0.1, \alpha = 0$ 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.
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 basic reproduction number is greater than unity.
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