

Mathematical Model of Cholera Transmission with Education Campaign and Treatment Through Quarantine

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Authors' contributions

This work was carried out in collaboration between both authors. Author DMM designed the study and wrote the first draft of the manuscript. All authors managed literature searches. Author HON performed the Mathematical analysis of the study and the simulations. All authors read and approved the final manuscript.

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Abstract

Cholera, a water-borne disease characterized by intense watery diarrhea, affects people in the regions with poor hygiene and untreated drinking water. This disease remains a menace to public health globally and it indicates inequity and lack of community development. In this research, SIQR-B mathematical model based on a system of ordinary differential equations is formulated to study the dynamics of cholera transmission with health education campaign and treatment through quarantine as controls against epidemic in Kenya. The effective basic reproduction number is computed using the next generation matrix method. The equilibrium points of the model are determined and their stability is analysed. Results of stability analysis show that the disease free equilibrium is both locally and globally asymptotically stable $R_0 < 1$ while the endemic equilibrium is both locally and globally asymptotically stable $R_0 > 1$. Numerical simulation carried out using MATLAB software shows that when health education campaign is efficient, the number of cholera infected individuals decreases faster, implying that health education campaign is vital in controlling the spread of cholera disease.

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1 Introduction

Cholera infection is caused by ingestion of water and food contaminated with the bacterium *V ibrio cholerae*. Its dynamics depend on the interaction between human, the bacterium *V ibrio cholerae* and the environment, hence the disease is transmitted through human-to-human and environmentto-human, Nelson [1].

Cholera cases mostly are experienced in Sub-Saharan Africa, Asia and some parts of South America where accessibility of clean water and basic sanitation infrastructure cannot be guaranteed. According to WHO [2], in 2016, 132,121 cholera cases with 2420 associated deaths were reported to WHO by 38 countries. Whil[st](#page-10-0) in Kenya, from 1*st* January to 29*th* November 2017, 596 cholera cases with 76 associated deaths were reported to WHO by the Ministry of Health.

Mathematical models are very important tools for understanding the dynamics of infectious diseases, Ande[rs](#page-10-1)on [3]. Modelling of cholera with simple deterministic model was started by Capasso [4], this was to research 1973 outbreak of cholera in the Mediterranean. Since then, several mathematical models have been developed and analyzed: e.g [5, 6, 7, 8, 9, 10].

Accoding to CDC [11], cholera is a quarantinable disease. However, to our knowledge, there are few models of [ch](#page-10-2)olera with quarantine. Nirwani [12] proposed SIQR model for cholera transmiss[ion](#page-10-3) which was analysed and found that disease free equilibrium and endemic equilibrium are locally asymptotically stable if a quarantine reproducti[on](#page-10-4) [n](#page-10-5)[um](#page-10-6)[be](#page-10-7)r *[R](#page-10-8)^q [<](#page-10-9)* 1 and *R^q >* 1 respectively. In this study, we extend the work of Nirwani [12] by investigating the effects of education campaign and treatment through [qua](#page-10-10)rantine in cholera transm[issi](#page-11-0)on.

2 Description and Fo[rm](#page-11-0)ulation of the Model

We formulate a mathematical model with *V ibrio cholerae* population $N_B(t)$ which is denoted $B(t)$ and human population $N_H(t)$. Human population is divided into four compartments; $S(t)$ -Susceptible, I(t)- Infected, $Q(t)$ - Quarantined and R(t)- Recovered with natural death rate μ in all compartments and δ the rate of death from cholera infection, in the infected and quarantined compartments. The model assumed that human population is recruited to susceptible compartment at the rate Λ and become infected with cholera through human-to-human transmission at the rate *βhIS* or through environment-to-human transmission at the rate *^βeBS ^κ*+*^B* where *β^h* and *β^e* are the rate of human-to-human interaction and the rate of Vibrios ingetion from the environment respectively, $\omega\beta_h(0<\omega<1)$ is the reduced rate of human-to-human interaction due to education campaign and treatment and $\omega \beta_e (0 \lt \omega \lt 1)$ is the reduced rate of Vibrios ingetion from the environment due to education campaign and treatment, where *ω* is a measure of education campaign and treatment efficacy, *κ* is the concentration of the bacterium *V ibrio cholerae* that bear 50% chance of contracting cholera. Furthermore, the rate of quarantine of infected individuals is *ε* and quarantined individual will recover through treatment at η rate. Finally, the rate of contribution of infected human to *V ibrio cholerae* concentration in the environment is *α* and on the other hand, decay rate of *V ibrio cholerae* from the environment is *σ*.

Based on the above description we have the following assumptions and flow chart;

Assumptions:

- i. Human birth and natural death takes place at different rates.
- ii. Quarantined individuals do not shed *V ibrio cholerae* into the aquatic environment.
- iii. There is lifetime immunity on recovery.
- iv. All identified individuals with cholera infection are quarantined.

Fig. 1. Flow chart

From the flow chart, Figure 1, we obtain the following differential equations of the model with *S*(0) *>* 0, *I*(0) *≥* 0, Q (0) *≥* 0, R (0) *≥* 0 and *B*(0) *≥* 0, non-negative initial conditions.

$$
\frac{dS}{dt} = \Lambda - (1 - \omega) \left[\frac{\beta_e B}{\kappa + B} + \beta_h I \right] S - \mu S
$$
\n
$$
\frac{dI}{dt} = (1 - \omega) \left[\frac{\beta_e B}{\kappa + B} + \beta_h I \right] S - (\varepsilon + \delta + \mu) I
$$
\n
$$
\frac{dQ}{dt} = \varepsilon I - (\eta + \delta + \mu) Q
$$
\n
$$
\frac{dR}{dt} = \eta Q - \mu R
$$
\n
$$
\frac{dB}{dt} = (1 - \omega) \alpha I - \sigma B
$$
\n(2.1)

3 Model Analysis

Since the system (2.1) describes human population and *V ibrio cholerae* population, all the solutions of state variable with non-negative initial conditions are non-negative *∀ t >* 0 and they are bounded $\text{in the feasible region } \Gamma = \{ (S, I, Q, R) \in \mathbb{R}^4_+; B \in \mathbb{R}_+; S > 0; I, Q, R, B \geq 0; N_H \leq \frac{\Lambda}{\mu}; N_B \leq \frac{\Lambda}{\mu} \}$ $\frac{\Lambda(1-\omega)\alpha}{\mu\sigma}$ }

Since the variable $R(t)$ does not appear in the first three and last equations of the model (2.1), it

suffices to consider the following model:

$$
\frac{dS}{dt} = \Lambda - (1 - \omega) \left[\frac{\beta_e B}{\kappa + B} + \beta_h I \right] S - \mu S
$$
\n
$$
\frac{dI}{dt} = (1 - \omega) \left[\frac{\beta_e B}{\kappa + B} + \beta_h I \right] S - (\varepsilon + \delta + \mu) I
$$
\n
$$
\frac{dQ}{dt} = \varepsilon I - (\eta + \delta + \mu) Q
$$
\n
$$
\frac{dB}{dt} = (1 - \omega)\alpha I - \sigma B
$$
\n(3.1)

3.1 Disease-free Equilibrium(DFE) Point

The disease free equilibrium point denoted by E^0 is the steady state solution of the model in the absence of disease. To obtain the DFE of the system (3.1), we equate the right hand side of the system (3.1) to zero and let $S = S^0$ $I = I^0 = 0$, $Q = Q^0 = 0$, and $B = B^0 = 0$. By doing so, we remain with one equation;

$$
\Lambda - \mu S^0 = 0
$$

From w[hich](#page-3-0) we have $E^0 = (S^0, 0, 0, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$

3.2 The Basic Reproduction Number (R_0)

*R*⁰ refers to the number of secondary infections generated by a single infective individual in a completely susceptible population. We use next generation matrix, the approach by [13] to determine R_0 . Using this method the basic reproduction number is given by $\rho(F_0V_0^{-1})$ (the dominant eigenvalue of $F_0 V_0^{-1}$) where F_0 is the Jacobian of f_i at E^0 , where f_i is the rate at which new infections appear in compartment *i* and V_0 is the Jacobian of v_i at E^0 , where v_i is the rate of transfer of individuals into and out of compartment *i*. The infected population is captured [in](#page-11-1) the following system of equations.

$$
\begin{array}{rcl}\n\frac{dI}{dt} & = & (1 - \omega) \left[\frac{\beta_e B}{\kappa + B} + \beta_h I \right] S - (\varepsilon + \delta + \mu) I \\
\frac{dQ}{dt} & = & \varepsilon I - (\eta + \delta + \mu) Q \\
\frac{dB}{dt} & = & (1 - \omega)\alpha I - \sigma B\n\end{array}\n\tag{3.2}
$$

From system (3.2), we have

$$
f_i(I, Q, B) = \begin{bmatrix} (1 - \omega)(\frac{\beta_e B S}{\kappa + B} + \beta_h I S) \\ 0 \\ 0 \end{bmatrix}
$$
 and $v_i(I, Q, B) = \begin{bmatrix} (\varepsilon + \delta + \mu)I \\ (\eta + \delta + \mu)Q - \varepsilon I \\ \sigma B - (1 - \omega)\alpha I \end{bmatrix}$

It follows that

$$
F_0 = \begin{bmatrix} (1-\omega)\frac{\beta_h \Lambda}{\mu} & 0 & (1-\omega)\frac{\beta_e \Lambda}{\mu \kappa} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V_0 = \begin{bmatrix} (\varepsilon + \delta + \mu) & 0 & 0 \\ -\varepsilon & (\eta + \delta + \mu) & 0 \\ -(1-\omega)\alpha & 0 & \sigma \end{bmatrix}
$$

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$$
F_0 V_0^{-1} = \begin{bmatrix} \frac{(1-\omega)\beta_h \Lambda}{\mu(\varepsilon+\delta+\mu)} + \frac{(1-\omega)^2 \alpha \beta_e \Lambda}{\mu\kappa(\varepsilon+\delta+\mu)\sigma} & 0 & \frac{(1-\omega)\beta_e \Lambda}{\sigma\mu\kappa} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}
$$

Thus

$$
R_0 = \rho(F_0 V_0^{-1}) = \frac{(1 - \omega)\beta_h \Lambda}{\mu(\varepsilon + \delta + \mu)} + \frac{(1 - \omega)^2 \alpha \beta_e \Lambda}{\mu \kappa(\varepsilon + \delta + \mu)\sigma}
$$
(3.3)

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3.3 Endemic Equilibrium Point

This refers to a spreading point of disease in the population. Let $E^* = (S^*, I^*, Q^*, B^*)$ be the endemic equilibrium point, where $S^*, I^*, Q^*, B^* > 0$.

Theorem 1. *A unique endemic equilibrium point of system (3.1) exists if* $R_0 > 1$ *.*

Proof. Equating the right hand side of the system (3.1) to zero and replacing (S,I,Q,B) with (S^*, I^*, Q^*, B^*) , the third equation and fourth equation of the system gives

$$
Q^* = \frac{\varepsilon I^*}{\eta + \delta + \mu} \tag{3.4}
$$

and

$$
B^* = \frac{(1 - \omega)\alpha I^*}{\sigma} \tag{3.5}
$$

respectively, and adding first and second equations of the same system, we have

$$
S^* = \frac{\Lambda - (\varepsilon + \delta + \mu)I^*}{\mu} \tag{3.6}
$$

In view of equation (3.5) and (3.6), the second equation of system (3.1) at endemic equilibrium becomes;

$$
(1 - \omega) \left[\frac{\beta_e (1 - \omega) \alpha}{\sigma \kappa + (1 - \omega) \alpha I^*} + \beta_h \right] \left[\frac{\Lambda - (\varepsilon + \delta + \mu) I^*}{\mu} \right] I^* - (\varepsilon + \delta + \mu) I^* = 0 \tag{3.7}
$$

Upon expansion we h[ave](#page-4-0) $I^* = 0$ [or](#page-4-1)

$$
AI^{*2} + BI^* + C = 0 \tag{3.8}
$$

Where

$$
A = -(1 - \omega)^2 (\varepsilon + \delta + \mu) \alpha \beta_h
$$

\n
$$
B = (1 - \omega)^2 \Lambda \alpha \beta_h - ((1 - \omega)^2 \alpha \beta_e + (1 - \omega) \sigma \beta_h \kappa) (\varepsilon + \delta + \mu) - (1 - \omega) (\varepsilon + \delta + \mu) \alpha \mu
$$

\n
$$
C = (1 - \omega)^2 \Lambda \alpha \beta_e + (1 - \omega) \Lambda \sigma \beta_h \kappa - (\varepsilon + \delta + \mu) \sigma \kappa \mu
$$

There exists endemic equilibrium of the system if equation (3.8) has real positive roots. Using Descartes' rule of signs as in [14], we determine if there is real positive roots. Since the sign of A is negative and that of C is positive when $R_0 > 1$, there is at least one real positive root hence the endemic equilibrium exists. \Box

3.4 Local Stability of the Disease Free Equilibrium Point

Now, we investigate local satability of DFE of system (3.1)

Theorem 2. Disease Free Equilibrium Point E^0 of the system (3.1) is locally asymptotically stable *if* $R_0 < 1$ *and unstable if* $R_0 > 1$ *.*

Proof. Evaluating the jacobian matrix (J) system (3.1) at E^0 , we have

$$
J(E^{0}) = \begin{bmatrix} -\mu & \frac{-(1-\omega)\beta_{h}\Lambda}{\mu} & 0 & \frac{-(1-\omega)\Lambda\beta_{e}}{\mu\kappa} \\ 0 & \frac{(1-\omega)\beta_{h}\Lambda}{\mu} - (\varepsilon + \delta + \mu) & 0 & \frac{(1-\omega)\Lambda\beta_{e}}{\mu\kappa} \\ 0 & \varepsilon & -(\eta + \delta + \mu) & 0 \\ 0 & (1-\omega)\alpha & 0 & -\sigma \end{bmatrix}
$$

Clearly $-\mu$ and $-(\eta + \delta + \mu)$ are the eigenvalues, the remaining eigenvalues are given by reducing matrix $(J(E^0))$ into 2×2 matrix as shown below;

$$
D(E^{0}) = \begin{bmatrix} \frac{(1-\omega)\beta_{h}\Lambda}{\mu} - (\varepsilon + \delta + \mu) & \frac{(1-\omega)\Lambda\beta_{e}}{\mu\kappa} \\ (1-\omega)\alpha & -\sigma \end{bmatrix}
$$

The characteristics equation of matrix $D(E^0)$ is given by

$$
\lambda^2 + B\lambda + C = 0\tag{3.9}
$$

Where

$$
B = \left[(\varepsilon + \delta + \mu) + \sigma - \frac{(1 - \omega)\beta_h \Lambda}{\mu} \right] \text{ and } C = -\left[\frac{\sigma (1 - \omega)\beta_h \Lambda}{\mu} - \sigma (\varepsilon + \delta + \mu) + \frac{(1 - \omega)^2 \Lambda \alpha \beta_e}{\mu \kappa} \right]
$$

By use of Routh-Hurwitz criterion, all the eigenvalues of $D(E^0)$ (roots of equation (3.9)) have negative real part when $B > 0$, $C > 0$ and $BC > 0$.

Clearly, $B > 0$, $C > 0$ and $BC > 0$ are satisfied when $R_0 < 1$. Hence disease-fee equilibrium point E^0 is locally asymptotically stable. □

3.5 Global Stability of the Disease Free Equilibrium Point

Considering the approach by [15] Castillo-Chavez theorem, the system (3.1) can be expressed as ;

$$
\frac{\frac{dX}{dt} = \mathbf{F}(X, Z)}{\frac{dZ}{dt} = \mathbf{G}(X, Z), \mathbf{G}(X, 0) = 0
$$

Where $X \in \mathbb{R} = (S)$ $X \in \mathbb{R} = (S)$ $X \in \mathbb{R} = (S)$, the nu[mbe](#page-11-2)r of non-infected individuals and $Z \in \mathbb{R}^3 = (I, Q, B)$, the infected compartments.

The following conditions are for global stability of disease-free equilibrium point $E^0 = (S^0, 0, 0, 0)$ $\left(\frac{\Lambda}{\mu}, 0, 0, 0\right) = (X^0, 0), \text{ for } X^0 = \frac{\Lambda}{\mu}$

- 1. $\frac{dX}{dt} = \mathbf{F}(X,0)$, X^0 is globally asymptotically stable.
- 2. **G** $(X, Z) = WZ \hat{G}(X, Z), \hat{G}(X, Z) \ge 0$ for $(X, Z) \in \Omega$

where $W = D_Z \mathbf{G}(X^0, 0)$ is an M-matrix (in that the off diagonal elements of W are positive) and Γ is the region where the equations of the model makes epidemiological sense. If conditions 1 and 2 are satisfied by system (3.1), the following theorem holds.

Theorem 3. *Provided that R*⁰ *<* 1 *and the conditions 1 and 2 are satisfied, the disease free equilibrium point* $E^0 = (X^0, 0)$ $E^0 = (X^0, 0)$ $E^0 = (X^0, 0)$ *of the system* (3.1) *is globally asymptotically stable.*

Proof. Since X=(S) and Z=(I,Q,B), $\frac{dX}{dt}$ = **F**(*X*,0) (condition 1) can be written as;

 $\frac{dS}{dt} = \Lambda - \mu S$ which gives

 $\Lambda - \mu S(t) = (\Lambda - \mu S(0))e^{-\mu t}$ \Rightarrow $S(t) = \frac{\Lambda - (\Lambda - \mu S(0))e^{-\mu t}}{\mu}$ $\Rightarrow S(t) \to \frac{\Lambda}{\mu} \text{ as } t \to \infty$

hence E^0 is globally asymptotically stable.

In view of $\mathbf{G}(X, Z) = WZ - \widehat{\mathbf{G}}(X, Z)$ (condition 2), we have

$$
\begin{aligned}\n\mathbf{G}(X,Z) &= WZ - \mathbf{G}(X,Z) \\
\mathbf{G}(X,Z) &= \begin{bmatrix}\n(1-\omega)\left[\frac{\beta_e B}{\kappa + B} + \beta_h I\right] S - (\varepsilon + \delta + \mu)I \\
\varepsilon I - (\eta + \delta + \mu)Q \\
(1-\omega)\alpha I - \sigma B\n\end{bmatrix} \\
W &= D_Z \mathbf{G}(X^0,0) = \begin{bmatrix}\n(1-\omega)\beta_h S^0 - (\varepsilon + \delta + \mu) & 0 \\
\varepsilon & -(\eta + \delta + \mu) & 0 \\
(1-\omega)\alpha & 0 & -\sigma\n\end{bmatrix} \\
\text{WZ} &= \begin{bmatrix}\n(1-\omega)\beta_h I S^0 - (\varepsilon + \delta + \mu)I + \frac{(1-\omega)\beta_e B S^0}{\kappa} \\
\varepsilon I - (\eta + \delta + \mu)Q \\
(1-\omega)\alpha I - \sigma B\n\end{bmatrix}\n\end{aligned}
$$

Therefore

$$
\widehat{\mathbf{G}}(X,Z) = \left[\begin{array}{c} (1-\omega)\beta_h(S^0-S) + \frac{(1-\omega)\beta_e B(S^0 B + \kappa(S^0-S))}{\kappa(\kappa+B)} \\ 0 \\ 0 \end{array} \right]
$$

Since all off diagonal entries of matrix W are positive, it implies that W is an M-matrix.

Also since $0 < \omega < 1$ and $S^0 \ge S \ \forall \ (X, Z) \in \Gamma$, $\widehat{G}(X, Z) \ge 0$.

Therefore, condition 2 can be expressed as

$$
\frac{dZ}{dt} \le WZ
$$

Since $S^o = \frac{\Lambda}{\mu}$, the characteristic equation of W is given by

$$
\{-(\eta + \delta + \mu) - \lambda\}(\lambda^2 + B\lambda + C) = 0
$$

or $\lambda = -(\eta + \delta + \mu)$ and

$$
\lambda^2 + B\lambda + C = 0\tag{3.10}
$$

Where

$$
B=[(\varepsilon+\delta+\mu)+\sigma-\tfrac{(1-\omega)\beta_h\Lambda}{\mu}]\text{ and }C=-[\tfrac{\sigma(1-\omega)\beta_h\Lambda}{\mu}-\sigma(\varepsilon+\delta+\mu)+\tfrac{(1-\omega)^2\Lambda\alpha\beta_e}{\mu\kappa}]
$$

Clearly equation (3.10) is the same as equation (3.9). Using Routh-Hurwitz criterion as in section $(3.4), B > 0, C > 0$ and $BC > 0$ are satisfied when $R_0 < 1$. Since the conditions 1 and 2 have been met and $R_0 < 1$, the proof is complete. \Box

3.6 Local Stability of the Endemic Equilibrium Point

Theorem 4. *The endemic equilibrium point* (*E ∗*) *of system (3.1) is locally asymptotically stable when* $R_0 > 1$

Proof. The jacobian matrix (J) evaluated at the endemic equilibrium point is given by Ĭ.

> $\overline{1}$ \mathbf{I} Ť

$$
J(E^*) = \begin{bmatrix} -X - \mu & -Y & 0 & -Z \\ X & Y - L & 0 & Z \\ 0 & \varepsilon & -M & 0 \\ 0 & N & 0 & -\sigma \end{bmatrix}
$$

Where

$$
X = (1 - \omega) \begin{bmatrix} \frac{\beta_e B^*}{\kappa + B^*} + \beta_h I^* \end{bmatrix}
$$

$$
Y = (1 - \omega) \beta_h S^*
$$

$$
Z = \frac{(1 - \omega) \beta_e S^*}{(\kappa + B^*)^2}
$$

$$
L = (\varepsilon + \delta + \mu)
$$

$$
M = (\eta + \delta + \mu)
$$
and

The characteristic equation is given by

$$
\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0 \tag{3.11}
$$

where

 $N = (1 - \omega)\alpha$

 \sim

 $a_1 = [L + X - Y + \mu + \sigma] = -Y + L + \sigma + X + \mu$ $a_2 = [Lμ + LX + μσ + Lσ + Xσ - Yσ - Yμ - ZN]$ $a_3 = XYN - XZN + LX\sigma + L\mu\sigma - Y\mu\sigma - ZN\mu$

Applying Routh Hurwitz criterion, all roots of equation (3.11) are negative when $a_1 > 0$, $a_2 > 0$, *a*³ *>* 0 and *a*1*a*² *− a*³ *>* 0.

To show that $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 - a_3 > 0$, we substitute equation (3.5) in the second equation of system (3.1) at endemic equilibrium point to get

$$
(1 - \omega) \left[\frac{\beta_e (1 - \omega) \alpha}{\sigma \kappa + (1 - \omega) \alpha I^*} + \beta_h \right] S^* - (\varepsilon + \delta + \mu) = 0 \text{ or}
$$

$$
\frac{\beta_e (1 - \omega)^2 \alpha S^*}{\sigma \kappa + (1 - \omega) \alpha I^*} - L = -Y
$$
 (3.12)

Substituting equation (3.5) and (3.12) in a_1 , a_2 and a_3 appropriately, we obtain

$$
a_1 = \frac{\beta_e (1 - \omega)^2 \alpha S^*}{\sigma \kappa + (1 - \omega)\alpha I^*} + \sigma + X + \mu > 0
$$

\n
$$
a_2 = LX + \mu \sigma + X\sigma + \frac{\beta_e (1 - \omega)^2 \alpha \mu S^*}{\sigma \kappa + (1 - \omega)\alpha I^*} + \frac{\beta_e (1 - \omega)^2 \alpha \sigma S^* B^*}{\sigma (\kappa + B^*)^2} > 0
$$

\n
$$
a_3 = XYN + \frac{\beta_e (1 - \omega)^2 \alpha \mu \sigma S^* B^*}{\sigma (\kappa + B^*)^2} + LX\sigma - XNZ > 0
$$
 and
\n
$$
a_1 a_2 - a_3 > 0
$$

Since $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1a_2 - a_3 > 0$, the endemic equilibrium is locally asymptotically stable. \Box

3.7 Global Stability of the Endemic Equilibrium Point

Theorem 5. The Endemic Equilibrium Point E^* of the system (3.1) is globally asymptotically *stable if* $R_0 > 1$ *.*

Proof. To prove global stability of E^* , we apply LaSalle [16] approach by constructing the following Lyapunov function

 $V(S, I, Q, B) = (S - S^* ln \frac{S}{S^*}) + (I - I^* ln \frac{I}{I^*}) + (Q - Q^* ln \frac{Q}{Q^*}) + (B - B^* ln \frac{B}{B^*})$ Differentiating V , we get $\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{Q^*}{Q}\right)\frac{dQ}{dt} + \left(1 - \frac{B^*}{B}\right)\frac{dB}{dt}$ Substituting $\frac{dS}{dt}$, $\frac{dI}{dt}$, $\frac{dQ}{dt}$ and $\frac{dB}{dt}$ from system (3.1), we obtain

$$
\frac{dV}{dt} = (1 - \frac{S^*}{S})\{\Lambda - (1 - \omega)\left[\frac{\beta_e B}{\kappa + B} + \beta_h I\right]S - \mu S\} + (1 - \frac{I^*}{I})\{(1 - \omega)\left[\frac{\beta_e B}{\kappa + B} + \beta_h I\right]S - (\varepsilon + \delta + \mu)I\} + (1 - \frac{Q^*}{Q})\{\varepsilon I - (\eta + \delta + \mu)Q\} + (1 - \frac{B^*}{B})\{(1 - \omega)\alpha I - \sigma B\}
$$
\n(3.13)

Rearranging system (3.1) at endemic equilibrium point, we have

$$
\Lambda = (1 - \omega) \left[\frac{\beta_e B^*}{\kappa + B^*} + \beta_h I^* \right] S + \mu S^*
$$

$$
(\varepsilon + \delta + \mu) = \frac{(1 - \omega)}{I^*} \left[\frac{\beta_e B^*}{\kappa + B^*} + \beta_h I^* \right] S^*
$$

$$
(\eta + \delta + \mu) = \frac{\varepsilon I^*}{Q^*}
$$

$$
\sigma = \frac{(1 - \omega)\alpha I^*}{B^*}
$$
(3.14)

Substituting (3.14) in (3.13) , we get

$$
\begin{split} &\frac{dV}{dt}=\big(1-\tfrac{S^*}{S}\big)\left[(1-\omega)\{\left[\tfrac{\beta_e S B^*}{\kappa+B^*}+\beta_h S I^*\right]-\left[\tfrac{\beta_e S B}{\kappa+B}+\beta_h S I\right]\}+\mu (S^*-S)\right]+ \big(1-\tfrac{I^*}{I}\big)(1-\omega)\\ &\{\left[\tfrac{\beta_e S B}{\kappa+B}+\beta_h S I\right]-\tfrac{I}{I^*}\left[\tfrac{\beta_e S^* B^*}{\kappa+B^*}+\beta_h S^* I^*\right]\}+\big(1-\tfrac{Q^*}{Q}\big)\varepsilon\big(I-\tfrac{Q I^*}{Q^*}\big) +\big(1-\tfrac{B^*}{B}\big)(1-\omega)\alpha\left[I-\tfrac{B I^*}{B^*}\right] \end{split}
$$

When $S = S^*$, $I = I^*$, $Q = Q^*$ and $B = B^*$, we obtain $\frac{dV}{dt} = 0$. Hence by LaSalle's invariance principle, every solution of the system (3.1) with initial conditions in $\Gamma = \{(S, I, Q, R) \in \mathbb{R}^4_+; B \in$ \mathbb{R}_+ ; $S > 0$; $I, Q, R, B \geq 0$; $N_H \leq \frac{\Lambda}{\mu}$; $N_B \leq \frac{\Lambda(1-\omega)\alpha}{\mu\sigma}$ tends to the endemic equilibrium point E^* . It follows that E^* is globally asymptotically stable.

4 Numerical Simulation

Using MATLAB, we simulated the system (3.1) to investigate the role of education campaign and treatment through quarantine. This is achieved by using parameter values in Table 1. Results of the simulation are presented in the figures below.

Figures $2(a)$, $2(b)$ and $2(c)$ show how education campaign and treatment can reduce the infected individuals, quarantined individuals and *V i[brio](#page-3-0) cholerae* bacterium respectively. As the education campaign and treatment efficacy increases, the infected individuals, quarantined individuals and *V ibrio cholerae* bacterium reduce. This implies that people need to be educated about cholera infection and how it can be prevented especially those in slums, refugee camps and institutions as well as treating the quarantined individuals. Education campaign should target both environmentto-human and human-to-human transmissions. This can be achieved through posters, radio, social media, television and word-of-mouth communication.

Fig. 2. The impact of education campaign and treatment on (a) infected individuals, (b) quarantined individuals and (c) Vibrio cholerae

Parameters	Values	Reference
л	$9.6274 \times 10^{-5}/\text{day}$	[17]
μ	$\sqrt{2.537 \times 10^{-5} / \text{day}}$	$[17]$
β_e	0.75/day	Estimate
β_h	0.0005/day	Estimate
κ	10^6 cells/ml	$\lceil 5 \rceil$
η	0.3 /day	Estimate
δ	$4.0 \times 10^{-1}/day$	[18]
α	10 cells/ml-day	[8]
σ	0.23 /day	[18]
ϵ	0.3 /day	Estimate
ω	$0 < \omega < 1$	Assumed

Table 1: Parameter values of the model

5 Discussion and Conclusion

In this paper, we formulated a mathematical model of cholera transmission with education campaign and treatment through quarantine. We studied the stability of the disease free and endemic equilibrium. The results of the disease free equilibrium showed that the model is both locally and globally asymptotically stable when $R_0 < 1$. This implies that when R_0 is below unity, the spread of cholera disease reduces. Next we studied the endemic equilibrium which we found to be both locally and globally asymptotically stable when *R*⁰ *>* 1. Numerical simulation indicates that when effective health education campaign and treatment are in place as control strategies of cholera, they lead to a faster reduction of the disease and eventually the disease decreases to zero. While ineffective health education campaign and treatment leads to increase of infectious individuals and *V ibrio cholerae* in the population, which is unfavourable for the elimination of cholera. Since we have not carried out persistence analysis of the model, we hereby recommend it to be explored for further studies of SIQR-B model of cholera.

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Competing Interest

We declare that no competing interests exist.

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