Dynamical Analysis of Cholera Epidemic Model with Educational Preventive Measures

Mohamad Hasyim Muzaqi, Trisilowati*, Ummu Habibah

Abstract-Cholera is an infectious bacterial disease caused by a bacterium named Vibrio Cholerae. Therefore, this research aims to determine cholera's dynamic analysis due to its rapid spread, using educational preventive measures. The model consists of four human subpopulations, namely susceptible, educated, infected, and recovered, as well as the bacteria population. The infection rate is assumed in the form of a saturated incidence rate found in interactions between the educated and bacteria population and the interactions between the susceptible and bacteria population. Furthermore, the dynamical analysis is performed by determining the equilibrium point, the basic reproduction number, and the stability. We also perform sensitivity analysis of the parameters. The results showed that the model has two equilibrium points, namely disease-free and endemic. The disease-free equilibrium point always exists, while endemic occurs when the basic reproduction number is greater than one. In addition, the disease-free equilibrium point is asymptotically stable when the basic reproduction number is less than one. Meanwhile, the endemic equilibrium point is asymptotically stable under certain conditions. Numerical simulations showed that the results are consistent with the analysis. Furthermore, educational measures have a significant impact on controlling the spread of cholera.

Index Terms—dynamical analysis; cholera; education; saturation.

I. INTRODUCTION

NFECTIOUS diseases are disorders caused by pathogenic microorganisms, such as bacteria, parasites, or viruses and transmitted from one person to another. One of the common types of infectious diseases caused by bacteria is cholera, which is defined as an acute diarrhea disease caused by Vibrio Cholerae bacteria that enters the body through food or drink consumed by the sufferers. When infecting a person, this bacterium produces enterotoxin, which contributes to the provision of diarrhea fluids [1]. In 2013, the World Health Organization (WHO) reported 129,064 cases of cholera by 47 countries globally [2]. Furthermore, from 2016-2018 there were 1,104,683 cases in several countries across the world [3]. These data indicate that cholera is still a significant health problem in several parts of the world. Therefore, based on this reason, a mathematical model capable of describing the dynamics of cholera's spread to obtain optimal solutions and strategies is needed.

Manuscript received October 11, 2020; revised April 30, 2021. This work was supported in part by the Minister of Research and Higher Education Republic of Indonesia under grand number: 037/SP2H/LT/DRPM/2020.

M. H. Muzaqi is a Postgraduate Student of Mathematics Department, Brawijaya University, Jl. Veteran Malang, Indonesia. (e-mail: mohamadhasyimmuzaqi@gmail.com).

Trisilowati is an Associate Professor of Mathematics Department and a member of Research Group of Biomathematics, Brawijaya University, Jl. Veteran Malang, Indonesia. (corresponding author; Phone: 62-341-571142; e-mail: trisilowati@ub.ac.id).

U. Habibah is an Assistant Professor of Mathematics Department and a member of Reserearch Group of Biomathematics, Brawijaya University, Jl. Veteran Malang, Indonesia. (email: ummu_habibah@ub.ac.id).

In recent years, some mathematical models in epidemic have been developed by researcher [4], [5], [6], [7], [8]. In 2015, Edward and Nyerere [6] developed a cholera prevention model by adding educational parameters to susceptible populations. Meanwhile, Lemos-Paiao et al. [7] developed a model used to determine the dynamics of this bacterium's spread using control measures in the form of treatment in quarantined populations. The model was constructed by adding the assumption of the immune loss level in the recovered subpopulation, thereby making it susceptible again. Furthermore, Tian et al. [8] developed a cholera epidemic model consisting of five subpopulations, namely susceptible, vaccinated, infected, recovered, and bacteria, with the saturated transmission rate found in susceptible and vaccinated subpopulations. In general, previous studies on the cholera epidemic model with control measures do not involve educated populations [9], [10], [11].

Therefore, this study modified the model developed by Tian et al. in the early prevention measures of cholera disease, namely education and control water sanitation measures, protecting it from being contaminated. Besides that, modification is also carried out by adding the assumption of the immune loss level in the recovered subpopulation, thereby making it susceptible again.

This paper is organized as follows: The model formulation is provided in Section 2. In Section 3, we discuss the model analysis including the positivity, boundedness, equilibrium points and sensitivity analysis of the parameters. The stability of the model is presented in Section 4 followed by numerical simulation to illustrate the analysis result. Finally, concluding remarks of this paper are given in Section 6.

II. MODEL FORMULATION

This study discusses the cholera epidemic model using educational, preventive measures.

The model consists of four human subpopulations, namely susceptible (S), educated (E), infected (I), recovered (R), and considered the bacteria population (B). The spread of cholera is illustrated in the flowchart, as shown in Figure 1. Based on the flowchart in Figure 1, the change rate model of the cholera epidemic using educational, preventive measures is as follows,

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta_1 BS}{k+B} + \omega R - \phi S - \mu S, \\ \frac{dE}{dt} &= \phi S - \frac{\beta_2 BE}{k+B} - \mu E, \\ \frac{dI}{dt} &= \frac{\beta_1 BS}{k+B} + \frac{\beta_2 BE}{k+B} - (\gamma + \alpha + \mu)I, \end{aligned}$$
(1)
$$\begin{aligned} \frac{dR}{dt} &= \gamma I - (\omega + \mu)R, \\ \frac{dB}{dt} &= \xi I - \delta B, \end{aligned}$$



Fig. 1. The compartment of cholera epidemic model with educational, preventive measures

where the value $\beta_2 \leq \beta_1$. The description of the parameters that make up the system are given in the Table I. While the parameters values for numerical simulation are provided in Table II.

TABLE I Description of Variables and Parameters

-		
Symbol	Parameters	
S	Susceptible subpopulations	
E	Educated subpopulations	
Ι	Infected subpopulations	
R	Recovered subpopulations	
B	Bacteria populations	
Λ	Human birth rate	
μ	The human natural death rate	
β_1	Interaction rate of susceptible subpopulations with bacteria populations	
β_2	Interaction rate of educated subpopulations with bacteria populations	
k	Constant saturation rate of the bacteria population	
ω	The loss rate of immunity	
ϕ	Educated rate	
γ	The recovery rate of naturally infected populations	
α	Death rate that caused by cholera infection	
ξ	The growth rate of bacteria	
δ	The natural death rate of bacteria	

 TABLE II

 PARAMETERS VALUES FOR NUMERICAL SIMULATION

Symbol	Value	Source
Λ	0.54/ day	Assumed
μ	0.0000548/ day	[8]
β_1	0.59/ day	Assumed
β_2	0.118/ day	Assumed
k	10 ⁶ cells/ml	[8]
ω	0.109589/ day	[7]
ϕ	0.01/ day	Assumed
γ	0.004/ day	[8]
α	0.0029/ day	Assumed
ξ	20 cells/ml	[8]
δ	0.33/ day	[8]

III. THE MODEL ANALYSIS

In the following, we discuss the positivity, boundedness, equilibrium point and sensitivity analysis of the solution of system (1).

A. Positivity and Boundedness

We prove the positivity and boundedness based on Cui et al. [12].

Theorem 1. The solution of S(t), E(t), I(t), R(t), and B(t) of the model (1) are nonnegative for all t > 0 with the non negative initial value.

Proof: System (1) can be written in the form of a matrix as follows,

$$X' = M(X),$$

where $X = (S, E, I, R, B)^T \in \mathbb{R}^5$ and M(X) is given by

$$M(X) = \begin{bmatrix} M_1(X) \\ M_2(X) \\ M_3(X) \\ M_4(X) \\ M_5(X) \end{bmatrix},$$
$$= \begin{bmatrix} \Lambda - \frac{\beta_1 BS}{k+B} + \omega R - \phi S - \mu S \\ \phi S - \frac{\beta_2 BE}{k+B} - \mu E \\ \frac{\beta_1 BS}{k+B} + \frac{\beta_2 BE}{k+B} - (\gamma + \alpha + \mu)I \\ \gamma I - (\omega + \mu)R \\ \varepsilon I - \delta B \end{bmatrix}.$$

We have

$$\begin{split} \left. \frac{dS(t)}{dt} \right|_{S=0} &= \Lambda + \omega R \ge 0, \\ \left. \frac{dE(t)}{dt} \right|_{E=0} &= \phi S \ge 0, \\ \left. \frac{dI(t)}{dt} \right|_{I=0} &= \frac{\beta_1 BS}{k+B} + \frac{\beta_2 BE}{k+B} \ge 0, \\ \left. \frac{dR(t)}{dt} \right|_{R=0} &= \gamma I \ge 0, \\ \left. \frac{dB(t)}{dt} \right|_{B=0} &= \xi I \ge 0. \end{split}$$

Therefore,

$$M_i|_{X_i(t)=0, x_i \in \mathbb{R}^5_+} \ge 0, i = 1, 2, 3, 4, 5.$$

Based on Lemma 2 in [13], any solution of system (1) is such that $X(t) \in \mathbb{R}^5_+$ for all t > 0. This completes the proof of Theorem 1.

Theorem 2. All solution S(t), E(t), I(t), R(t), and B(t) of system (1) are bounded.

Proof: System (1) is divided by two part, the human population i.e. S(t), E(t), I(t), and R(t) and bacteria population i.e. B(t). From the first four equation of system (1),

Volume 29, Issue 3: September 2021

we have

$$\begin{aligned} \frac{d(S+E+I+R)}{dt} &= \Lambda - \mu S - \mu E - (\alpha + \mu)I - \mu R, \\ &= \Lambda - \mu (S+E+I+R) - \alpha I, \\ &\leq \Lambda - \mu (S+E+I+R), \end{aligned}$$

then it follows that $\limsup_{t\to\infty} (S+E+I+R) \leq \frac{\Lambda}{\mu}$. From the first equation, we get

$$\begin{split} \frac{dS(t)}{dt} &\leq \Lambda + \omega R - \phi S - \mu S, \\ &\leq \Lambda + \frac{\omega \Lambda}{\mu} - (\phi + \mu) S. \end{split}$$

Thus $\frac{dS(t)}{dt} \leq 0$, as $S(t) \geq \frac{\Lambda(\mu + \omega)}{\mu(\phi + \mu)}$.

From the second equation, we obtain

$$\begin{aligned} \frac{dE(t)}{dt}\phi S - \mu E, \\ &\leq \phi \left(\frac{\Lambda}{\mu} - E\right) - \mu E \\ &\leq \frac{\Lambda \phi}{\mu} - (\phi + \mu)E. \end{aligned}$$

Thus $\frac{dE(t)}{dt} \leq 0$, as $E(t) \geq \frac{\Lambda\phi}{\mu(\phi+\mu)}$. Similarly, from the fourth equation, we have

$$\begin{split} \frac{dR(t)}{dt} &= \gamma I - (\omega + \mu) R, \\ &\leq \gamma \left(\frac{\Lambda}{\mu} - R\right) - (\omega + \mu) R, \\ &\leq \frac{\gamma \Lambda}{\mu(\gamma + \omega + \mu)} - R. \end{split}$$

Thus $\frac{dR(t)}{dt} \leq 0$, as $R(t) \geq \frac{\gamma \Lambda}{\mu(\gamma + \omega + \mu)}$. Finally, from the last equation, we can get

$$\frac{dB(t)}{dt} = \xi I - \delta B,$$

$$\leq \xi \frac{\Lambda}{\mu} - \delta B.$$

Hence, $\frac{dB(t)}{dt} \leq 0$, as $B \geq \frac{\xi \Lambda}{\mu \delta}$. Therefore, all solutions S(t), E(t), I(t), R(t), and B(t) of system (1) are bounded.

Thus, the feasible region of the human population of system (1) is

$$\Omega_{H} = \left\{ (S, E, I, R) \mid S + E + I + R \leq \frac{\Lambda}{\mu}, \\ 0 \leq S \leq \frac{\Lambda(\mu + \omega)}{\mu(\phi + \mu)}, 0 \leq E \leq \frac{\Lambda\phi}{\mu(\phi + \mu)}, I \geq 0, \\ 0 \leq R \leq \frac{\gamma\Lambda}{\mu(\gamma + \omega + \mu)} \right\},$$

and the feasible region of bacteria population for system (1) is

$$\Omega_B = \left\{ B \; \middle| \; 0 \le B \le \frac{\xi \Lambda}{\mu \delta} \right\}.$$

B. Equilibrium Point and Reproduction Number

The equilibrium point of system (1) is obtained when $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dB}{dt} = 0$. System (1) has two equilibrium points, namely disease-free (T^0) and endemic (T^*) . The disease-free equilibrium point (T^0) is obtained when I = 0 in the following form.

$$T^{0} = (S^{0}, E^{0}, I^{0}, R^{0}, B^{0}) = \left(\frac{\Lambda}{\phi + \mu}, \frac{\phi \Lambda}{\mu(\phi + \mu)}, 0, 0, 0\right).$$

The epidemic model consists of a unique number, called the basic reproduction number (R_0) , which determines the possible occurrence of a disease's outbreak [14]. The nextgeneration matrix method [15] is used to determine the basic reproduction number (R_0) .

Let $X = (x_1, x_2)^T$ is the number of compartments containing new infections, then we have

$$x_i' = \mathcal{F}_i - \mathcal{V}_i, i = 1, 2.$$

where $\mathcal{F} = \left(\frac{\beta_1 B\xi}{k+B} + \frac{\beta_2 BE}{k+B}, 0\right)^T$ and $\mathcal{V} = \left((\gamma + \alpha + \mu)I, \delta B - \xi I\right)^T$. The Jacobian matrix of \mathcal{F} and \mathcal{V} with respect

to I and B at point T^0 are

$$DF(T^{0}) = \begin{bmatrix} 0 & \frac{\beta_{1}s^{0}}{k} + \frac{\beta_{2}s^{0}}{k} \\ 0 & 0 \end{bmatrix}$$

and

$$DV(T^0) = \begin{bmatrix} \gamma + \alpha + \mu & 0 \\ -\xi & \delta \end{bmatrix}$$

Thus, R_0 , the spectral radius of $(DF)(DV)^{-1}$ is $R_0 =$ $\Lambda\xi(\mu\beta_1+\phi\beta_2)$

 $\overline{\frac{\mu k \delta(\phi + \mu)(\gamma + \alpha + \mu)}{\text{Next, the endemic equilibrium point } (T^*)}}$ is obtained when $I \neq 0$, of the form

$$T^* = (S^*, E^*, I^*, R^*, B^*)$$

= $\left(\frac{\Lambda A_3 A_4}{F}, \frac{\phi \Lambda A_3 A_4}{A_2 F}, \frac{\Lambda A_4 G L}{F}, \frac{\gamma \Lambda G L}{F}, \frac{\xi \Lambda A_4 G L}{F \delta}\right),$

where

$$\begin{split} A_{1} &= \phi + \mu, A_{2} = \beta_{2}L + \mu, A_{3} = \gamma + \alpha + \mu, A_{4} = \omega + \mu \\ G &= \beta_{1} + \frac{\phi\beta_{2}}{A_{2}}, \\ F &= \frac{A_{2}A_{3}A_{4}(\beta_{1}L + A_{1}) - A_{2}\omega\gamma L\beta_{1} - \omega\gamma L\phi B_{2}}{A_{2}} > 0, \\ L &= \frac{-b \pm \sqrt{b^{2} - 4ac}}{2a}, \\ a &= k\delta\beta_{1}\beta_{2}A_{3}A_{4} + \Lambda\xi\beta_{1}\beta_{2}A_{3}A_{4} - \omega\gamma k\delta\beta_{1}\beta_{2} > 0 \\ b &= \mu k\delta A_{3}A_{4} + k\delta\beta_{2}A_{1}A_{3}A_{4} + \mu\Lambda\delta\beta_{1}A_{4} + \Lambda\beta_{2}\xi\phi A_{4} \\ &- \mu k\delta\omega\gamma\beta_{1} - k\delta\omega\gamma\beta_{2}\phi - \Lambda\beta_{1}\beta_{2}\delta A_{4} > 0 \\ c &= \mu k\delta A_{1}A_{2}A_{3}(1 - R_{0}). \end{split}$$

The existence of an endemic equilibrium point (T^*) depends on R_0 . If $R_0 < 1$, then the endemic equilibrium point does not exist because c > 0, it is obtained L < 0, therefore the value of $I^*, R^*, B^* < 0$. If $R_0 = 1$, then the equilibrium point $T^* = T^0$ that results in the endemic equilibrium point (T^*) do not exist. If $R_0 > 1$, then c < 0, resulting in L > 0, therefore all values of S^*, E^*, I^*, R^*, B^* are positive and the unique endemic equilibrium point exists.

Volume 29, Issue 3: September 2021

C. Sensitivity Analysis

The sensitivity analysis of the basic reproduction number (R_0) is done to determine which parameter that influences the disease transmission. The most sensitive parameter can be used as the key factor in controlling the disease.

Definition 1. ([16], [17]) The normalized forward sensitivity index of R_0 that depends differentiably on a parameter ρ is defined by

$$\Psi_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho} \frac{\rho}{R_0}.$$

Some example for the normalized forward sensitivity index of R_0 with respect to the given parameter are obtained as

$$\begin{split} \Psi_{\beta_1}^{R_0} &= \frac{\beta_1 \mu}{\beta_1 \mu + \beta_2 \phi} > 0, \\ \Psi_{\beta_2}^{R_0} &= \frac{\beta_2 \phi}{\beta_1 \mu + \beta_2 \phi} > 0, \\ \Psi_{\gamma}^{R_0} &= -\frac{\gamma}{\gamma + \alpha + \mu} < 0, \\ \Psi_{\alpha}^{R_0} &= -\frac{\alpha}{\gamma + \alpha + \mu} < 0, \\ \Psi_{\phi}^{R_0} &= -\frac{\phi(\beta_1 - \beta_2)\mu}{(\beta_1 \mu + \beta_2 \phi)(\phi + \mu)}. \end{split}$$

Based on parameter values as in Table II, we obtain the sensitivity index of R_0 which is given in Table III. The parameters are ordered from the most sensitive to the least sensitive. The sensitivity index of parameters Λ, ξ, β_1 , and β_2 are positive. This result indicates that increasing the interaction rate of susceptible subpopulations with bacteria population (β_1) increases the reproduction number. On the other hand, the sensitivity index of the parameter $k, \delta, \mu, \gamma, \alpha$, and ϕ are negative. This result indicates that increasing the educated rate (ϕ) reduces the reproduction number. The most sensitive parameters are Λ, ξ, k , and δ , which means that a small change in these parameters can cause a major effect on the disease transmission. The least sensitive parameters are β_1 and ϕ . An increase in the interaction rate of susceptible subpopulations with bacteria population (β_1), for instance, does not have a significant effect on the disease transmission.

TABLE III Sensitivity Index

Parameters	Sensitivity Index
Λ	1
ξ	1
k	-1
δ	-1
μ	-0.9867
β_2	0.9733
γ	-0.5751
α	-0.4170
β_1	0.0267
ϕ	-0.0212

IV. STABILITY ANALYSIS

In this section, we discuss the local stability of disease-free and endemic equilibrium. **Theorem 3.** The disease-free equilibrium point T^0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian matrix at T^0 is

$$J(T^{0}) = \begin{bmatrix} -A_{1} & 0 & 0 & \omega & -\frac{\beta_{1}S_{0}}{k} \\ \phi & -\mu & 0 & 0 & -\frac{\beta_{2}E_{0}}{k} \\ 0 & 0 & -A_{3} & 0 & \frac{\beta_{1}S_{0}}{k} + \frac{\beta_{2}E_{0}}{k} \\ 0 & 0 & \gamma & -A_{4} & 0 \\ 0 & 0 & \xi & 0 & -\delta \end{bmatrix}.$$

Based on calculation with $|J(T^0) - rI| = 0$, the following was obtained $r_1 = -A_1$, $r_2 = -\mu$, $r_4 = -A_4$, and r_3, r_5 that meets

$$\begin{vmatrix} -A_3 - r_3 & \frac{\beta_1 \Lambda}{A_1 k} + \frac{\beta_2 \Lambda \phi}{A_1 \mu k} \\ \xi & -\delta - A_5 \end{vmatrix} = 0.$$

Then, using the trace and determinant methods [18], r_3 , r_5 are negative assuming $\frac{\Lambda\xi(\mu\beta_1+\beta_2\phi)}{\mu k\delta A_1A_3} < 1$ or $R_0 < 1$. Consequently, the disease-free equilibrium point (T^0) is locally asymptotically stable when $R_0 < 1$ and otherwise unstable.

Theorem 4. The endemic equilibrium point T^* is locally asymtotically stable if and only if

 $\begin{array}{ll} 1) & b_1b_2 > b_3, \\ 2) & b_1b_2b_3 + b_1b_5 > b_3^2 + b_1^2b_4, \\ 3) & b_1b_2b_3b_4 + 2b_1b_4b_5 + b_2b_3b_5 > b_1^2b_4^2 + b_1b_2^2b_5 + b_3^2b_4 + b_5^2, \\ 4) & b_5 > 0. \end{array}$

Proof: The stability of endemic equilibrium point (T^*) is obtained by determining the eigenvalues of the Jacobian matrix system (1) at T^* . The Jacobian matrix at T^* is of the form

$$I(T^*) = \begin{bmatrix} -\beta_1 L - A_1 & 0 & 0 & \omega & -J_1 \\ \phi & -\beta_2 L - \mu & 0 & 0 & -J_2 \\ \beta_1 L & \beta_2 L & -A_3 & 0 & J_1 + J_2 \\ 0 & 0 & \gamma & -A_4 & 0 \\ 0 & 0 & \xi & 0 & -\delta \end{bmatrix}.$$

 $\frac{1}{2}$,

where

$$J_1 = \frac{\beta_1 k \Lambda A_3 A_4}{F\left(k + \frac{\Lambda \xi A_4 G}{F\delta}\right)}$$

and

$$J_2 = \frac{\beta_2 k \phi \Lambda A_3 A_4}{A_2 F \left(k + \frac{\Lambda \xi A_4 G}{F \delta}\right)^2}.$$

The characteristic equation of a matrix $J(T^*)$ is obtained by solving $|J(T^*) - rI| = 0$, that is

$$\begin{vmatrix} A & 0 & 0 \\ \phi & -\beta_2 L - \mu - r & 0 & \omega & -J_1 \\ \beta_1 L & \beta_2 L & -A_3 - r & 0 & -J_2 \\ 0 & 0 & \gamma & -A_4 - r & 0 \\ 0 & 0 & \xi & 0 & -\delta - r \end{vmatrix} = 0,$$

where
$$A = -\beta_1 L - A_1 - r$$
.

$$b^{5} + b_{1}r^{4} + b_{2}r^{3} + b_{3}r^{2} + b_{4}r + b_{5} = 0,$$
 (2)

where

$$\begin{split} b_1 &= \delta + \beta_2 L + \mu + \beta_1 L + A_3 + A_4 + A_1, \\ b_2 &= \delta A_4 + \delta A_3 + \delta \beta_1 L + \delta A_1 + \delta \beta_2 L + \delta \mu \\ &+ A_3 A_4 + A_4 \beta_1 L + A_1 A_4 + A_4 \beta_2 L + A_4 \mu \\ &+ A_3 \beta_1 L + A_1 A_3 + A_3 \beta_2 L + A_3 \mu + \beta_1 \beta_2 L^2 \\ &+ \beta_1 L \mu + A_1 \beta_2 L + A_1 \mu - \xi (J_1 + J_2), \\ b_3 &= \delta A_3 A_4 + \delta A_4 \beta_1 L + \delta A_1 A_4 + \delta A_4 \beta_2 L + \delta A_4 \mu \\ &+ \delta A_3 \beta_1 L + \delta A_1 A_3 + \delta A_3 \beta_2 L + \delta A_3 \mu + \delta \beta_1 \beta_2 L^2 \\ &+ \delta \beta_1 L \mu + \delta A_1 \beta_2 L + \delta A_1 \mu + A_3 A_4 \beta_1 L + A_1 A_3 A_4 \\ &+ A_3 A_4 \beta_2 L + A_3 A_4 \mu + A_4 \beta_1 \beta_2 L^2 + A_4 \beta_1 L \mu \\ &+ A_1 A_4 \beta_2 L + A_1 A_4 \mu + A_3 \beta_1 \beta_2 L^2 + A_3 \beta_1 L \mu \\ &+ A_1 A_3 \beta_2 L + A_1 A_3 \mu - \xi L (\beta_1 J_1 + \beta_2 J_2) - \omega \gamma \beta_1 L \\ &- \xi (J_1 + J_2) (\beta_2 L + A_4 + \mu + \beta_1 L + A_1), \\ b_4 &= A_3 A_4 \beta_1 \beta_2 L^2 + \delta A_3 \beta_1 \beta_2 L^2 + \delta A_4 \beta_1 \beta_2 L^2 \\ &+ A_1 A_3 A_4 \beta_2 L + \delta A_1 A_3 \beta_2 L + \delta A_1 A_4 \beta_2 L \\ &+ \delta A_3 A_4 \beta_1 L + A_3 A_4 \beta_1 L \mu + \delta A_3 A_4 \beta_2 L \\ &+ \delta A_3 \beta_1 L \mu + \delta A_4 \beta_1 L \mu + \xi \phi_2 L J_1 + \delta A_1 A_3 A_4 \\ &+ \mu A_1 A_3 A_4 + \delta \mu A_1 A_3 + \delta \mu A_1 A_4 + \delta \mu A_3 A_4 \\ &- L^2 \beta_1 \beta_2 \omega \gamma - A_1 J_1 \xi \beta_2 L - A_4 J_1 \xi \beta_2 L - A_4 J_2 \xi \beta_1 I \\ &- \mu J_2 \xi \beta_1 L - \beta_1 L \delta \gamma \omega - \beta_1 L \mu \gamma \omega - \gamma \omega \phi \beta_2 L \\ &- A_4 \mu J_1 \xi - A_4 \mu J_2 \xi, \\ b_5 &= \delta A_3 A_4 \beta_1 \beta_2 L^2 + A_4 J_1 \xi \phi \beta_2 L + \delta A_1 A_3 A_4 \beta_2 L \\ &+ \delta A_1 A_3 A_4 \mu + \delta A_3 A_4 \beta_1 L \mu - \delta \gamma \omega \beta_1 \beta_2 L^2 \\ &- A_1 A_4 J_1 \xi \beta_2 L - \mu A_4 J_2 \xi \beta_1 L - \beta_1 L \delta \gamma \omega \mu \\ &- \beta_2 L \gamma \omega \phi \delta - A_1 A_4 J_1 \xi \mu - A_1 A_4 J_2 \xi \mu. \end{split}$$

The stability of the endemic equilibrium point (T^*) is obtained using the Routh-Hurwitz criteria [19]. Based on equation (2), the endemic equilibrium point T^* is asymptotically stable, if only if

1)
$$b_1b_2 - b_3 > 0$$
,
2) $b_1b_2b_3 + b_1b_5 - b_3^2 - b_1^2b_4 > 0$,
3) $b_1b_2b_3b_4 + 2b_1b_4b_5 + b_2b_3b_5 - b_1^2b_4^2 - b_1b_2^2b_5 - b_3^2b_4 - b_5^2 > 0$,
4) $b_5 > 0$.

Thus, the proof is complete.

V. NUMERICAL SIMULATION

In this section, numerical simulation is used to illustrate the main result in Sections III. These were conducted using the Runge-Kutta 4th order method to show the cholera epidemic model dynamics with preventive measures through education and the parameter values in the following Table II.

The stability simulation of the disease-free equilibrium point is shown in Figure 2-6. The parameter values used are presented in Table II, where $\beta_1 = 0.0943$, $\beta_2 = 0.03$, and $\xi = 5$ obtained $R_0 = 0.6516 < 1$. Based on these parameter values, the disease-free equilibrium point is

 $T^0 = (53.7, 9800.3, 0, 0, 0)$. Figure 2-6 shows the solution when $R_0 < 1$ with three different initial values, namely

$$NA_1 = (100, 6000, 80, 40, 125000),$$

 $NA_2 = (50, 3000, 200, 15, 150000),$
 $NA_3 = (15, 500, 120, 80, 75000).$

The results of this simulation show that with some initial values, the solution leads to the disease-free equilibrium point (T^0) , this means that after a long time, no infected individual is found. The numerical simulation results obtained support the analysis results in Section III, which states that when $R_0 < 1$ disease-free equilibrium (T^0) point is locally asymptotically stable.



Fig. 2. Dynamic behavior of the disease free-equilibrium point for susceptible subpopulation



Fig. 3. Dynamic behavior of the disease free-equilibrium point for educated subpopulation

The result of stability simulation of the endemic equilibrium point is provided in Figure 7-11, while the parameter values used are shown in Table II, with $R_0 = 10.3536 > 1$. Based on these parameter values, the disease-free equilibrium point $T^0 = (53.7, 9800.3, 0, 0, 0)$ and the endemic equilibrium point $T^* = (76, 603, 170, 6, 10306)$. Section III is

.



Fig. 4. Dynamic behavior of the disease free-equilibrium point for infected subpopulation



Dynamic behavior of the disease free-equilibrium point for Fig. 5. recovered subpopulation



Fig. 6. Dynamic behavior of the disease free-equilibrium point for bacteria population

explained that the roots of the characteristic equation (2) are negative value when they meet the Routh-Hurwitz criteria. Based on the parameter values in Table II, the Routh-Hurwitz criteria values are as follows:

1)
$$b_1b_2 - b_3 = 0.0201 > 0$$
,
2) $b_1b_2b_3 + b_1b_5 - b_3^2 - b_1^2b_4 = 1.2722 \times 10^{-5} > 0$,
3) $b_1b_2b_3b_4 + 2b_1b_4b_5 + b_2b_3b_5 - b_1^2b_4^2 - b_1b_2^2b_5 - b_3^2b_4 - b_5^2 = 7.3109 \times 10^{-12} > 0$,
4) $b_5 = 2.0593 \times 10^{-9} > 0$,
where

$$b_1 = 0.4632,$$

 $b_2 = 0.04473,$
 $b_3 = 6.4024 \times 10^{-4},$
 $b_4 = 7.2023 \times 10^{-7},$

This shows that the Routh-Hurwitz criterion is fulfilled, therefore, the endemic equilibrium point is locally asymptotically stable.



Fig. 7. Dynamic behavior of the endemic equilibrium point for susceptible subpopulation



Fig. 8. Dynamic behavior of the endemic equilibrium point for educated subpopulation



Fig. 9. Dynamic behavior of the endemic equilibrium point for infected subpopulation



Fig. 10. Dynamic behavior of the endemic equilibrium point for recovered subpopulation



Fig. 11. Dynamic behavior of the endemic equilibrium point for bacteria population

Figure 7-11 show the solution of cholera epidemic model, using three different initial values, namely

$$NA_1 = (250, 15, 80, 40, 275000),$$

 $NA_2 = (400, 20, 160, 15, 150000),$
 $NA_3 = (25, 5, 120, 80, 75000).$

This simulation shows that with some initial values, the solution leads to an endemic equilibrium point (T^*) , which means cholera is spreading in the populations. The numerical simulation results obtained support the analysis results in Section III, therefore, the endemic equilibrium point (T^*) is asymptotically stable when it meets the Routh-Hurwitz criteria.



Fig. 12. Simulation of sensitivity analysis by varying δ , other parameters are taken from Table II



Fig. 13. Simulation of sensitivity analysis by varying ϕ , other parameters are taken from Table II

Some simulations representing the sensitivity analysis of the parameters are illustrated in Figure 12 and Figure 13. Here, we take two examples: δ as the most sensitive parameter and ϕ as the less sensitive parameter. The parameter values are taken from Table II by varying δ or ϕ . Increasing the parameter values δ from 0.33 to 0.3993 decreases the reproduction number, R_0 , from 10.3536 to 8.5567. Furthermore, increasing the parameter values ϕ from 0.01 to 0.0121 decreases the reproduction number, R_0 , from 10.3536 to 10.3155. Figure 12 shows varying the parameter δ causes a significant effect in the disease transmission. However, varying the parameter ϕ does not have a major change in the transmission of the disease (see Figure 13). Although the rate of educational, ϕ , is less sensitive, it still affects the disease dynamic as described in Figure 14.

Figure 14 shows that when the value $\phi = 0$, it means no educational action is taken, the number of infected subpopulations is 161 individuals at t = 200. However, when the value $\phi = 0.01$, it means that education is carried out at a rate of 0.01. The number of infected subpopulations experiences a decline of 120 individuals at t = 200. Therefore, the value $\phi = 0.1$, which means education is carried out at a rate of 0.1. The number of infected subpopulations experiences a decline of 86 individuals at t = 200. This simulation shows that educational, preventive measures significantly decrease the number of infected subpopulations.



Fig. 14. Dynamic behavior of the infected subpopulation by varying ϕ , other parameters are taken from Table II

VI. CONCLUSION

The cholera epidemic model with educational, preventive measures has two equilibrium points: disease-free and endemic. Therefore, based on the analysis results, a diseasefree equilibrium point always exists, while endemic occurs when the basic reproduction number is greater than one, i.e. $R_0 > 1$. The disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and the endemic equilibrium point is locally asymptotically stable under certain conditions. From analysis sensitivity, we concluded that increasing the value of the parameters Λ, ξ, β_1 , or β_2 , increases the basic reproduction number, while increasing the value of the parameters k, $\delta, \mu, \gamma, \alpha$ or ϕ , decreases the basic reproduction number. Numerical simulations performed show that the results are consistent with the analysis. Furthermore, educational measures have a significant impact in controlling the spread of cholera.

REFERENCES

- N. F. Pierce, W. B. Greenough, and C. C. J. Carpenter. "Vibrio Cholerae Enterotoxin and Its Mode of Action". *Bacteriological Re*views, vol. 35, pp. 1-13, 1971.
- [2] World Health Organization. "Cholera 2013". Weekly Epidemiological Record, vol. 89, pp. 345356, 2014.
- [3] A. Camacho, M. Bouhenia, R. Alyusfi, et al. "Cholera Epidemic in Yemen, 2016-2018 an Analysis of Surveillance Data". *Lancet Global Health*, vol. 6, pp. e680-e690, 2018.
- [4] S. Olaniyi, M. A. Lawal, and O. S. Obabiyi. "Stability and Sensitivity Analysis of a Deterministic Epidemiological Model with Pseudorecovery". *IAENG International Journal of Applied Mathematics*, vol. 46, no. 2, pp. 160-167, 2016.
- [5] W. Du, S. Qin, J. Zhang and J. Yu. "Dynamical Behavior and Bifurcation Analysis of SEIR Epidemic Model and its Discretization", *IAENG International Journal of Applied Mathematics*, vol. 47, no. 1, pp. 1-8, 2017.
- [6] S. Edward and N. Nyerere. "A Mathematical Model for the Dynamics of Cholera with Control Measures". *Applied and Computational Mathematics*, vol. 4, pp. 53-63, 2015.
- [7] A. P. Lemos-Paiao, C. J. Silva, and D. F. M. Torres. "An Epidemic Model for Cholera with Optimal Control Treatment". *Journal of Computational and Applied Mathematics*, vol. 318, pp. 168-180, 2017.
- [8] X. Tian, R. Xu, and J. Lin. "Mathematical Analysis of a Cholera Infection Model with Vaccination Strategy". *Applied Mathematics and Computation*, vol. 361, pp. 517-535, 2019.
- [9] A. K. Misra, A. Gupta, and E. Venturino. "Cholera Dynamics with Bacteriophage Infection a Mathematical Study". *Nonlinear Science Nonequilibrium and Complex Phenomena*, vol. 91, pp. 610-621, 2016.
- [10] G. G. Kolaye, S. Bowong, R. Houe, M. A. Aziz-Alaoui, and M. Cadivel. "Mathematical Assessment of the Role of Environmental Factors on the Dynamical Transmission of Cholera". *Communications in Nonlinear Science and Numerical Simulation*, vol. 67, pp. 203-222, 2018.
- [11] G. Q. Sun, J. H. Xie, S. H. Huang, Z. Jin, and M. T. Li. "Transmission Dynamics of Cholera Mathematical Modelling and Control Strategies". *Communications in Nonlinear Science and Numerical Simulation*, vol. 45, pp. 235-244, 2016.
- [12] J. Cui, Z. Wu, and X. Zhou. "Mathematical Analysis of a Cholera Model with Vaccination". *Journal of Applied Mathematic*, vol. 2014, Article ID 3324767, 16 pages, 2014.
- [13] X. Yang, L. Chen, and J. Chen. "Permanence and Positive Periodic Solution for The Single-Species Nonautonomous Delay Diffusive Models". *Computers and Mathematics with Applications*, vol. 32, no. 4, pp. 109-116, 1996.
- [14] J. M. Heffernan, R. J. Smith, and L. M. Wahl. "Perspectives on the Basic Reproductive Ratio". *Journal of the Royal Society Interface*, vol. 2, pp. 281-293, 2005.
- [15] F. Brauer and C. C. Chavez. "Mathematical Models in Population Biology and Epidemiology", Second Edition, Springer-Verlag, New York, 2010.
- [16] N. Chitnis, J. M. Hyman, and J. M. Cushing. "Determining Important Parameters in the Spread of Malaria Through the Sensitivity Analysis of a Mathematical Model". *Bulletin of Mathematical Biology*. vol. 70, no. 5, pp. 1272-1296, 2016.
- [17] C. J. Silva and D. F. M. Torres. "A SICA Compartmental Model in Epidemiology with Application to HIV/AIDS in Cape Verde". *Ecological Complexity*. vol. 30, pp. 70-75, 2017.
- [18] A. Panvilov. "Qualitative Analysis of Differential Equations", Utrecht University, Utrecht, 2004.
- [19] J. D. Murray. "Mathematical Biology I. An Introduction", Third Edition, Springer-Verlag, Berlin Heidelberg, 2002.