

# Optimization of Cholera Spreading using Sanitation, Quarantine, Education and Chlorination Control

Subchan Subchan<sup>1</sup>, Sentot D. Surjanto<sup>1</sup>, Irma Fitria<sup>2</sup> and Dwita S. Anggraini<sup>1</sup>

<sup>1</sup>*Departement of mathematics, Institut Teknologi Sepuluh Nopember, Surabaya, Indonesia*

<sup>2</sup>*Departement of mathematics, Institut Teknologi Kalimantan, Balikpapan, Indonesia*

**Keywords:** Cholera Model, Optimal Control, Pontryagin Minimum Principle.

**Abstract:** Cholera is a contagious and deadly disease that requires an effective prevention and control actions. In this paper, several efforts are made to prevent the cholera spreading by reconstructing the mathematical model and adding control sanitation, treatment consisted of quarantine and education as well as chlorination on to the bacteria. The Pontryagin Minimum Principle is employed to derive the optimal control solution and solved by Runge-Kutta method. The computational results showed that the control was able to minimize the number of individuals infected by cholera with mild symptoms at the final time as many as 2 individuals and individuals infected by cholera with severe symptoms at the final time as many as 7 individuals as well as minimize the number of bacteria concentrations at the final time as much as 517 cell/ml.

## 1 INTRODUCTION

Cholera is an acute diarrhea infection which is caused by the consumption of food or water contaminated with *Vibrio cholerae* bacteria (Organization, 2008). These bacteria secrete enterotoxins in the intestinal tract which cause diarrhea accompanied with acute and severe vomiting. Therefore, an individual will lose a lot of body fluids only in several days and get dehydration. This condition can cause death if not handled quickly (Johnson and R, 2006). The spreading process of cholera can occur through the mouth, when *Vibrio Cholerae* bacteria successfully entered through the mouth and ingested, then these bacteria will be quickly killed when exposed to stomach acid. However, if *Vibrio Cholerae* bacteria successfully passes the stomach acid, the bacteria will develop in the small intestine (Setiadi, 2014).

About 75% of people infected with *Vibrio cholera* do not experience any symptoms, even though the bacteria are in their feces for 7-14 days after infected (Organization, 2008), but when there is an infection attack then the diarrhea and vomiting suddenly occur with serious condition as acute attack (Sack et al., 2004). Since 1917, cholera has been known as seven pandemics which spread to Europe. The *Vibrio Cholerae* bacteria first appeared in Sulawesi, Indonesia and caused a cholera epidemic. Cholera then spread rapidly to other East Asian countries and

reached Bangladesh in 1963, India in 1964 and the Soviet Union, Iran and Iraq in 1965-1966 (Setiadi, 2014).

Cholera is rapidly spreading in densely populated areas, poor water sanitation and lack of clean water supply. Therefore, cholera is widely identified in poor and developing countries (Subchan et al., 2019). So that it does not rule out the possibility of cholera spreading in Indonesia, for it is important to conduct research on controlling the spread of cholera. Effective precautions of controlling for cholera depend on providing adequate environmental health services, such as increasing access to clean water, sanitation, availability of cholera vaccines, quarantine and treatment (Organization, 2008).

The mathematical model related to cholera spreading with its control had been many conducted in the previous research. The research about cholera disease had been examined by Bakhtiar (Bakhtiar, 2015). He studied optimum control approach of contagious disease with the control variable was the role of education and chlorination. After that, Lemos-Paião et al (Lemos-Paião et al., 2016), concerned on cholera spreading model by giving control in the form of treatment done to the population of quarantined people. The population infected which was given a treatment would be quarantined so that it obtained the quarantined population. In addition, Subchan et al

(Subchan et al., 2019). researched cholera disease spreading model by giving the optimal control in the forms of medication and intervention through the improvement of sanitation, education and quarantine.

In this research, the problem was reconstructed by the mathematical model of the spread of cholera with the control variable in the form of chlorination in bacteria, improved sanitation, education and quarantine. Control was given to reduce the number of individuals infected with cholera with mild and severe symptoms and reduce proliferation of the *Vibrio Cholerae* bacteria.

## 2 MATHEMATICAL MODEL

In this research, the type of mathematical model of cholera spreading used was the type of SEIQR which was reconstructed by adding chlorine to the bacteria ( $u_4$ ). Other optimal controls were based on the research (Subchan et al., 2019), which are improvement of sanitation ( $u_1$ ), control treatment in the form of medication during quarantine for infected individuals ( $u_2$ ) and education for vulnerable individuals ( $u_3$ ).

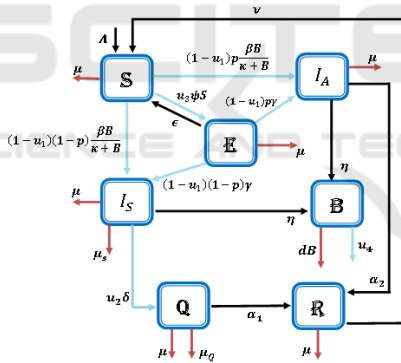


Figure 1: SEIQR Compartment Diagram the Spread of Cholera.

The spread of water-based diseases, especially cholera, can be reduced by the use of chlorine which is believed to be effective in reducing bacteria. In addition, sanitation improvements are carried out to reduce the level of absorption of bacteria caused by infected individuals. Control treatment is also given to people with cholera through quarantine to accelerate healing of infected individuals and prevent spread to vulnerable individuals. In addition, education is also provided to individuals who are vulnerable to cholera as an effort to prevent the outbreak of the disease.

The interpretation of the mathematical model of cholera spreading by giving optimal control to the compartment diagram as shown in the Fig. 1. The

mathematical models of the spread of cholera are as follows.

$$\frac{ds}{dt} = \Lambda + vR + \epsilon E - \mu S - u_a \psi - (1 - u_1) \frac{\beta B}{\kappa + B} S \quad (1)$$

$$\frac{dE}{dt} = u_a \psi S - \epsilon E - \mu E - (1 - u_1) \gamma E \quad (2)$$

$$\frac{dI_A}{dt} = (1 - u_1) p \frac{\beta B}{\kappa + B} S + (1 - u_1) p \gamma E - \mu I_A - \alpha_2 I_A \quad (3)$$

$$\frac{dI_S}{dt} = \{(1 - u_1)(1 - p) \frac{\beta B}{\kappa + B} S + (1 - u_1) (1 - p) \gamma E - \mu I_S - \mu_S I_S = u_2 \delta I_S\} \quad (4)$$

$$\frac{dQ}{dt} = u_2 \delta I_S - \mu Q - \mu_Q Q - \alpha_1 Q \quad (5)$$

$$\frac{dR}{dt} = \alpha_1 Q + \alpha_2 I_A - \mu R - vR \quad (6)$$

$$\frac{dB}{dt} = \eta I_A + \eta I_S - dB - U_4 B \quad (7)$$

with the variables and parameters that formed up the system can be seen in Figure 2. It is assumed that  $S, E, I_A, I_S, Q, R \geq 0$  and all parameters are positive, which are taken from (Subchan et al., 2019)

## 3 OPTIMAL CONTROL PROBLEMS

The purpose of this research is to obtain control by minimizing the number of infected human populations, bacterial populations and minimizing the costs incurred for controls by considering equations (1)-(7). The objective function can be defined as follows

$$J(x, u) = \left\{ \frac{1}{2} \int_{t_f}^{t_0} [C_1 I_S^2(t)] + C_2 I_A^2(t) + C_3 B^2(t) + C_4 u_1^2(t) + C_5 u_2^2(t) + C_6 u_3^2(t) + C_7 u_4^2(t) dt \right\} \quad (8)$$

with  $t_0$  as initial time and  $t_f$  is final time, and  $C_i$  was the parameter weight or price coefficient issued at each control, where  $C_i > 0$  for each  $i = 1, 2, 3, 4, 5, 6, 7$ .

The first step to solve the optimal control problem using the Pontryagin Minimum Principle is to define (Subchan and Zbikowski, 2007) as follows

$$\begin{aligned}
 H = & \left\{ \frac{1}{2}(C_1 I_S^2(t) + C_2 I_A^2(t) \right. \\
 & + C_3 B^2(t) + C_4 u_1^2(t) + C_5 u_2^2(t) \\
 & + C_6 u_3^2(t) + C_7 u_4^2(t)) + \\
 & \lambda_S(\Lambda + \nu R + \varepsilon E - \mu S - u_3 \Psi S - (1 - u_1) \\
 & \frac{\beta B}{(\kappa + B)} S) + \lambda_E(u_3 \Psi S - \varepsilon E - \\
 & \mu E - (1 - u_1) \gamma E) + \lambda_{I_A}((1 - u_1) p \\
 & \frac{\beta B}{(\kappa + B)} S + (1 - u_1) p \gamma E - \\
 & \mu I_A - \alpha_2 I_A) + \lambda_{I_S}((1 - u_1)(1 - p) \\
 & \frac{\beta B}{(\kappa + B)} S + (1 - u_1)(1 - p) \gamma E \\
 & - \mu I_S - \mu_S I_S - u_2 \delta I_S) + \lambda_Q \\
 & (u_2 \delta I_S - \mu Q - \mu_Q Q - \alpha_1 Q) \\
 & + \lambda_R(\alpha_1 Q + \alpha_2 I_A - \mu R - \nu R) \\
 & \left. + \lambda_B(\eta I_A + \eta I_S - dB - u_4 B) \right\}
 \end{aligned} \tag{9}$$

where  $\lambda_i$  for each  $i = S, E, I_A, I_S, Q, R, B$  was the costate vector or Lagrange multiplier that depended on the state. Next, the optimal control value  $u_1^*, u_2^*, u_3^*$  and  $u_4^*$  was found as follows

$$u_1^* = \left\{ \frac{1}{C_4} \left( \frac{\beta B}{\kappa + B} S (P \lambda_{I_A} + \lambda_{I_S} (1 - p) - \lambda_S) \right. \right. \tag{10}$$

$$\left. + \gamma E (\lambda_{I_A} P + \lambda_{I_S} (1 - p) - \lambda_E) \right\}$$

$$u_2^* = \frac{\delta I_S (\lambda_{I_S} - \lambda_Q)}{C_5} \tag{11}$$

$$u_3^* = \frac{\Psi S (\lambda_S - \lambda_E)}{C_6} \tag{12}$$

$$u_4^* = \frac{\lambda_B B}{C_7} \tag{13}$$

The optimal control  $u^*$  was obtained from  $\frac{\partial H}{\partial u}$  and had the following characteristics

$$u_1^* = \min(u_{1min}, \max(\hat{u}_1^*, u_{1max}))$$

$$u_2^* = \min(u_{2min}, \max(\hat{u}_2^*, u_{2max}))$$

$$u_3^* = \min(u_{3min}, \max(\hat{u}_3^*, u_{3max}))$$

$$u_4^* = \min(u_{4min}, \max(\hat{u}_4^*, u_{4max}))$$

Variables and Parameters	Description
$S(t)$	Number of healthy and susceptible individuals infected with cholera at the $t$ time
$E(t)$	Number of healthy and susceptible individuals infected with cholera at the $t$ time
$I_A(t)$	Number of individuals infected with cholera with mild symptoms at the $t$ time
$I_S(t)$	Number of individuals infected with cholera with severe symptoms at the $t$ time
$Q(t)$	Number of individuals who are on treatment through quarantine at the $t$ time
$R(t)$	The number of individuals who have recovered from cholera and are assumed to be resistant to disease at the $t$ time
$B(t)$	Number of Vibrio Cholera bacteria at the $t$ time
$\Lambda$	Rate the addition of individuals into vulnerable subpopulation
$\mu$	Rate natural death
$\beta$	Rate the consumption of bacteria through contaminated sources
$\kappa$	Half constant saturation from the bacterial subpopulation
$\beta B(t)$	Rate transmission from vulnerable subpopulations to subpopulations infected with cholera
$\kappa + B(t)$	
$\delta$	Rate subpopulations of educated vulnerable individuals
$\mu_S$	Rate subpopulations of educated individuals stop taking precautions
$\gamma$	Rate an educated subpopulation (very small) suffering from cholera
$p$	The proportion of subpopulations of individuals infected with mild symptoms
$1 - p$	The proportion of subpopulations of individuals infected with severe symptoms
$\mu_A$	Rate healing of subpopulations of individuals infected with severe symptoms
$\mu_S$	Rate healing of subpopulations of individuals infected with mild symptoms
$\delta$	Rate time of individual quarantined
$\mu_Q$	Rate the death of individuals infected with severe symptoms
$\mu_Q$	Rate the death of quarantined individuals
$\alpha_1$	Rate the loss of immunity of individuals who have recovered to become vulnerable again
$\alpha_2$	Rate the increase of bacterial concentration due to contributions from infected individuals
$d$	Rate bacterial death

Figure 2: Variables and Parameters on Mathematical Models of the Spread of Cholera.

Equations (10)-(13) were substituted to Equation (9) so that it had the optimal Hamiltonian function  $H^*$ . The next step was to determine the state equation (Subchan and Zbikowski, 2007) as follows

$$\dot{S}^* = \{ \Lambda + \nu R + \varepsilon E - \mu S - u_3^* \Psi S - (1 - u_1^*) \frac{\beta B}{(\kappa + B)} S \} \tag{14}$$

$$\dot{E}^* = u_3^* \Psi S - \varepsilon E - \mu E - (1 - u_1^*) \gamma E \tag{15}$$

$$\dot{I}_A^* = p \frac{\beta B}{(\kappa + B)} S + (1 - u_1^*) p \gamma E - \mu I_A - \alpha_2 I_A \tag{16}$$

$$\dot{I}_S^* = \{ (1 - u_1^*) (1 - p) \frac{\beta B}{(\kappa + B)} S + (1 - u_1^*) (1 - p) \gamma E - \mu I_S - \mu_S I_S - u_2^* \delta I_S \} \tag{17}$$

$$\dot{Q}^* = u_2^* \delta I_S - \mu Q - \mu_Q Q - \alpha_1 Q \tag{18}$$

$$\dot{R}^* = \alpha_1 Q + \alpha_2 I_A - \mu R - \nu R \tag{19}$$

$$\dot{B}^* = \eta I_A + \eta I_S - dB - u_4^* B \tag{20}$$

And costate equations can be derived as follows

$$\begin{aligned}
 \dot{\lambda}_S^* = & -(-\mu \lambda_S - u_3^* \Psi \lambda_S - (1 - u_1^*) \\
 & \frac{\beta B}{\kappa + B} \lambda_S + u_3^* \Psi \lambda_E + (1 - u_1^*) p \\
 & \frac{\beta B}{\kappa + B} \lambda_{I_A} + (1 - u_1^*) (1 - p) \\
 & \frac{\beta B}{\kappa + B} \lambda_{I_S})
 \end{aligned} \tag{21}$$

$$\begin{aligned}
 \dot{\lambda}_E^* = & -(\varepsilon \lambda_S - \varepsilon \lambda_E - \mu \lambda_E - (1 - u_1^*) \\
 & \gamma \lambda_E + (1 - u_1^*) p \gamma \lambda_{I_A} + (1 - u_1^*) \\
 & (1 - p) \gamma \lambda_{I_S})
 \end{aligned} \tag{22}$$

$$\begin{aligned}
 \dot{\lambda}_{I_A}^* = & -(C_2 I_A - \mu \lambda_{I_A} - \alpha_2 \lambda_{I_A} \\
 & + \alpha_2 \lambda_R + \eta \lambda_B)
 \end{aligned} \tag{23}$$

$$\lambda_{I_S}^* = - (C_1 I_S - \mu \lambda_{I_S} - \mu_S \lambda_{I_S} - u_2^* \delta \lambda_{I_S} + u_2^* \delta \lambda_Q + \eta \lambda_B) \quad (24)$$

$$\lambda_Q^* = - (-\mu \lambda_Q - \mu_Q \lambda_Q - \alpha_1 \lambda_Q + \alpha_1 \lambda_R) \quad (25)$$

$$\lambda_R^* = - (v \lambda_S - \mu \lambda_R - v \lambda_R) \quad (26)$$

$$\begin{aligned} \lambda_R^* = & - (C_3 B + \lambda_S (1 - u_1^*)) \frac{\beta S \kappa}{(\kappa + B)^2} \\ & + \lambda_{I_A} (1 - u_1^*) \frac{p \beta S \kappa}{(\kappa + B)^2} \\ & + \lambda_{I_S} (1 - u_1^*) \end{aligned} \quad (27)$$

The optimal state and costate then can be determined by considering the boundary condition  $x(0) = x_0$  and  $\lambda(t_f) = 0$ .

#### 4 COMPUTATIONAL RESULT

The parameter values are taken from (Subchan et al., 2019) and used for numerical simulation. The simulation is solved by using Forward-Backward Sweep Runge-Kutta Order 4 method (Lenhart and Workman, 2007; Burden et al., 2016; Lindfield and Penny, 1995). The purpose of numerical simulation was to determine the effectiveness of optimal control in each population. The simulation result on every population can be seen on Fig. 3 and the control can be seen on Fig. 4.

Based on Fig. 2, it was known that infected asymptomatic individual decreased. The number of infected asymptomatic individuals with the final time without optimal control was 133 individuals while with control the number of individuals at the end of time was 2 individuals. This was due to the large influence of  $\beta$  parameter, which was the level of consumption of bacteria through contaminated sources. If the value of  $\beta$  was getting bigger, then the number of individuals without optimal control would be even less. It caused the individuals with mild symptoms to change into severe symptoms if individuals were not aware of the symptoms because of lacking the knowledge or neglected the individual education. So, in this case the effectiveness of giving control in the infected asymptomatic subpopulation had an effect of 98.50%. So, the objective to minimize the number of individuals with mild symptoms had been reached.

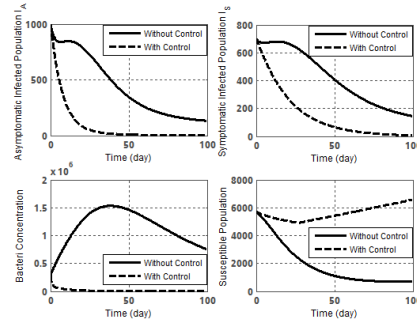


Figure 3: The Change of Rate on the Number of Infected Asymptomatic ( $I_A$ ) population, Infected Symptomatic ( $I_S$ ), Bacteria and Susceptible concentration.

Furthermore, the infected symptomatic individual also decreased when control is given. The number of infected symptomatic individuals with the final time without control was 145 individuals while with control, the number of individuals at the end was 7 individuals. The number of infected symptomatic individuals without control increased 680 individuals at  $t = 15$ , while with control, the number of infected symptomatic individuals were decreased at the beginning until the end. It means that the level of control effectiveness had an effect of 95.17%. The concentration of bacteria decreased with control. The amount of bacterial concentration at the end without control was  $7.5 \times 10^5$  while with control the number of bacterial concentrations at the end was 517. In this case, control had an effect of 99.93%. Based on Fig. 3, it was shown that the level of control of individuals and bacteria was on maximum value 1 and sanitation control was at value 0.4.

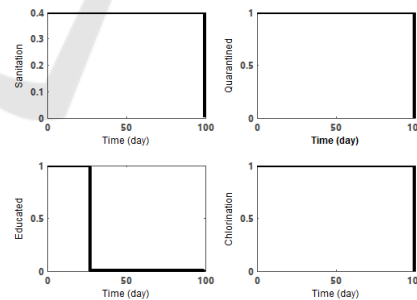


Figure 4: Sanitation ( $u_1^*$ ), Quarantine ( $u_2^*$ ), Education ( $u_3^*$ ) and Chlorination ( $u_4^*$ ).

Based on Fig. 5, it can be seen that the level of susceptible population decreased since the beginning. This was caused by the number of susceptible population interacted with cholera bacterial-contaminated environment so the population were infected. Furthermore, the population increased at about  $t = 27$ . The educated population was increased from the very first time and it was proportional to susceptible population level. The quarantine population increased sharply at

the beginning till day 15, then it decreased until the end. This is caused by infected symptomatic population decreased.

In this case, the recovered population increased from the beginning then it stay in the certain value until the end. The asymptomatic and symptomatic infected population decreased because bacteria concentration level kept decrease until the end.

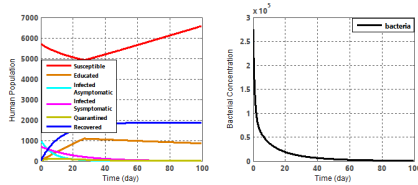


Figure 5: The Change of Subpopulation Rate and Bacteria Concentration with Control.

## 5 CONCLUSION

In this paper, system of differential equations were given as the dynamics model of cholera spreading that was divided into human population and bacteria population classes. The optimal control in the form of sanitation, chlorination, education, and quarantine were given as the attempt to control cholera spreading.

The simulation result showed the effect of the given control. Based on the computational result, controls affected the number of infected population and bacteria experienced decline so that cholera endemic was not quite big problem. This showed that the optimum control strategy in the form of sanitation, chlorination, education, and quarantine gave significant positive effect to minimize the spread of cholera.

## ACKNOWLEDGEMENTS

The authors wish to thank Department of Mathematics, Institut Teknologi Sepuluh Nopember and Institut Teknologi Kalimantan for their support and funding.

## REFERENCES

- Bakhtiar, T. (2015). Peran edukasi dan klorinasi dalam pengendalian penyakit menular: Sebuah pendekatan kontrol optimum. *Semirata*, 1.
- Burden, R., Faires, J., and Burden, A. (2016). *Numerical Analysis*. Cengage Learning, Boston USA.
- Johnson and R, L. (2006). *Mathematical modeling of Cholera: from bacterial life histories to human epidemics*. s.l.:University of California, Santa Cruz.
- Lemos-Paião, S., a., A., Torres, C., and FM, D. (2016). An epidemic model for cholera with optimal control treatment. *Journal of Computational and Applied Mathematics*.
- Lenhart, S. and Workman, J. T. (2007). *Optimal control applied to biological models*. CRC Press, Taylor and Francis Group, London.
- Lindfield, G. and Penny, J. (1995). *Numerical Method Using MATLAB*. MPG Book Ltd, Botmin Cornwall.
- Organization, W. H. (2008). *Prevention and control of cholera outbreaks: WHO policy and recommendations*. s.n, s.l.
- Sack, David, A., Sack, R., Nair, G., and Siddique, A. (2004). Cholera. *Lancet*, 363:223–33.
- Setiadi, S. (2014). *Ilmu Penyakit Dalam*. Interna Publishing, Jakarta.
- Subchan, S., A.M., a. I., and F. (2019). *An epidemic cholera model with control treatment and intervention*. IOP Publishing, s.l.
- Subchan, S. and Zbikowski, R. (2007). Computational optimal control of the terminal bunt manoeuvre—part 1: Minimum altitude case. *Optimal Control Applications and Methods*, 28(5):311–325.