Effect of Educational Campaign on the Transmission Model of Cholera

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ABSTRACT

In this paper, we proposed a mathematical model to describe the transmission model of Cholera, to analyze the stability of the model, and to study the effect of educational campaign on the Cholera transmission. The developed model based on the Cholera model of Mukandavire et al (2012) by taking the educational campaign into account in the model. The standard method was used to analyze the model that was to study the equilibrium points and stability, the basic reproductive number, the analytic solution and the numerical solution. The research findings revealed that the model of Cholera is represented by a system of differential equation consists of four equations, that is, the number of susceptible human, the number of infected human, the number of recovered human and V. cholerae population in the water. There were two equilibrium points: disease free equilibrium point and endemic equilibrium point. The basic reproductive number, $R_0 = \frac{\beta N}{\gamma + \tau + \mu}$ and the stability of each equilibrium point were local asymptotically stable. For the numerical result at the disease free equilibrium, we obtained, $R_0 = 0.109995 < 1$ this mean that no spread of cholera and the numerical result at the endemic equilibrium point, we obtained $R_0 = 1.09995 > 1$, this mean that the cholera will occur. If the effective of educational campaign and water treatment increase then the number of infected human will decrease, this means no outbreaks of disease. The educational campaign and water treatment can be the affective way to control for this disease.

INTRODUCTION

Cholera is an acute, diarrheal illness caused by infection of the intestine with the bacterium Vibrio cholerae. The cholera bacterium is usually found in water or food sources that have been contaminated by feces from a person infected with cholera. Cholera is most likely to be found and spread in places with inadequate water treatment, poor sanitation, and inadequate hygiene. An estimated 3-5 million cases and over 100,000 deaths occur each year around the world. The infection is often mild or without symptoms, but can sometimes be severe. Approximately one in 10 (5-10%) infected persons will have severe disease characterized by profuse watery diarrhea, vomiting, and leg cramps. In these people, rapid loss of body fluids leads to dehydration and shock. Without treatment, death can occur within hours (CDC, 2014). Individuals living in places with inadequate water treatment, poor sanitation, and inadequate hygiene are at a greater risk for cholera. The Ganges River is the main river of life contributes to the spread to other regions. As the world's trade routes by land and by sea. The pandemics in the world several times, From 1918 – 1920, there were a record number of patients with data showing the number of deaths caused major outbreaks in Thailand is number of patients 19,413 person
and die 13,918 person but nowadays in Thailand have not yet found a case report and deceased (IDEP, 2015). Even though early studies of cholera have become exemplars of modern epidemiology predicting and managing cholera outbreaks is still a major challenge in the developing world. Improvements in sanitation and the use of oral rehydration therapy have greatly reduced the burden of disease, but we lack a predictive framework for anticipating outbreaks and planning for interventions. Mathematical modeling is one approach to synthesizing our knowledge of cholera into a quantitative framework. Education, which is a key tool in disease control, is often overlooked. It requires investment in people rather than in biomedical interventions, but it has the potential to lead to enormous benefits for relatively low cost. Conversely, a lack of information can have a severe impact on worsening the spread of the disease. Cholera-specific education includes advising people with symptoms to seek medical care promptly, and improving sanitation and hygienic practices. Failures to provide health education can be traced to barriers at one of six sites: to be effective, messages have to (1) reach the intended audience, (2) gain attention, (3) be correctly understood, (4) be accepted, (5) result in changed behavior and (6) result in improvement in health. During the 1994 cholera epidemic of Guinea-Bissau, health education demonstrated that local preventive rituals, radio and word-of-mouth communication were effective educational tools (Edward and Nyerere, 2015). Mathematical models have been used to study the dynamics of disease outbreaks and predict the effectiveness of potential intervention strategies (Garnett et al., 2011). In this paper, we proposed a mathematical model to describe the Cholera transmission, and to study effects of control measures affecting Cholera transmission. The control measures were the educational campaign.

2. Model Formulation:

In our model, we assume that the human is constant. We formulate the model of cholera transmission by using basic ideas taken from epidemiology control measures, the educational campaign. The model is obtained by assuming:

The total human population \( N \) is divided into four compartments: Susceptible human denote by \( S \), Infected human denoted by \( I \) and Recover human denoted by \( R \). The concentration of \( V. \) cholerae in the water denoted by \( B \). The dynamics of the disease is depicted in the flow chart shown in Fig. 1.

\[ \frac{dS}{dt} = \mu N - (1 - u) \frac{\beta_e B}{k + B} S - \frac{\beta}{h} SI - \mu S \]  
\[ \frac{dI}{dt} = (1 - u) \frac{\beta_e B}{k + B} S + \frac{\beta}{h} SI - (\gamma + \tau + \mu) I \]  
\[ \frac{dR}{dt} = (\gamma + \tau) I - \mu R \]  
\[ \frac{dB}{dt} = x(1 - v) I - \delta B \]

with \( N = S + I + R \)

where

- \( S \) is the susceptible human,
- \( I \) is the infected human,
- \( R \) is the recovered human,
- \( B \) is the concentration of \( V. \) cholerae in the water
- \( N \) is the total human population,
- \( \mu \) is the natural birth or death rate,
- \( \beta_e \) is the rate of infection from water contaminated with \( V. \) cholerae.

Fig. 1: Flow chart of the dynamics of cholera.
$\beta_h$ is the *V. cholerae* infection rate from human to human,

is the concentration of bacteria that causes cholera,

$\gamma$ is the rate of cure of the infection,

$\delta$ is the death rate induced disease,

$s$ is the rate of infected human with cholera-infected discharges into the water,

$\alpha$ is the rate of infection with *V. cholerae*,

$\mu$ is the effectiveness of the educational campaign,

$v$ is the rate of water treatment,

$\tau$ is the retention rate

3. Analysis of the Model:

**Equilibrium Points:**
The system has two equilibrium points; a disease free equilibrium point and an endemic equilibrium point. We obtained these by setting the right hand sides of equations. (1) - (4) to zero. Doing this, we obtained

**Disease Free Equilibrium Point** ($E_0$):
In the absence of the disease, i.e., $I = 0$, equation (1), (3), (4) becomes:

$$
\frac{dS}{dt} = \mu N - (1-u) \frac{\beta e BS}{k+B} - \beta_h S I - \mu S,
$$

$$
\frac{dR}{dt} = (\gamma + \tau) I - \mu R \quad \text{and} \quad \frac{dB}{dt} = x(1-v) I - \delta B.
$$

The solution to this equation is $S = N$, $R = 0$ and $B = 0$. The disease free state is $E_0 = (N, 0, 0, 0)$

**Endemic Equilibrium Point** ($E_1$):
In the case where the disease is presented, by setting $I \neq 0$. This gives:

$$
S^* = \frac{\mu N(k \delta + x(1-v) I^*)}{(1-u) \beta e x(1-v) I^* + (k \delta + x(1-v) I^*)(\beta_h I^* + \mu)},
$$

$$
R^* = \frac{\mu}{\mu},
$$

$$
B^* = \frac{x(1-v) I^*}{\delta},
$$

Where

$$
A_1 = \frac{\beta e B^*}{k + B} + \beta_h I^* + \mu, A_2 = \frac{(1-u) \beta e B^*}{k + B} + \beta_h I^*.
$$

$$
A_3 = -\beta_h S^*,
$$

$$
A_4 = \gamma + \tau + \mu - \beta_h S^*.
$$

$$
A_5 = \gamma + \tau.
$$

$$
A_6 = \frac{-((k + B^*)(1-u)(\beta e S^*) - \beta e (1-u)B^* S^*)}{(k + B^*)^2},
$$

Thus
Basic Reproductive Number:

The basic reproductive number is obtained by the next generation matrix. In the notation of Van den Driessche and Watmough (2002), we start with

\[
\frac{dx}{dt} = F(x) - V(x) \tag{5}
\]

where \( F(x) \) is the matrix of new infectious and \( V(x) \) is the matrix of the transfers between the compartments in the infective equations. We obtained

\[
F(x) = \begin{bmatrix}
0 & \frac{(1-u)\beta_{p}BS}{k+B} + \beta_{H}SI \\
0 & 0 \\
-\mu N + \frac{(1-u)\beta_{p}BS}{k+B} + \beta_{H}SI + \mu S \\
(\gamma + \tau + \mu)I \\
-\gamma - \sigma + \mu R \\
-x(1-v)I + \delta B
\end{bmatrix}
\]

\[
V(x) = \begin{bmatrix}
\frac{\partial F_i(E_0)}{\partial x_j} \\
\frac{\partial V_i(E_0)}{\partial x_j}
\end{bmatrix}
\]

for all \( i, j = 1, 2, 3, 4 \). This are the Jacobian matrix of \( F(x) \) and \( V(x) \) at \( E_0 \). The basic reproductive number, \( R_0 \), is the threshold for indicating the degree of epidemiology of the disease. It can be determined by noting that

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

For our model, the Jacobian matrices are

\[
F = \begin{bmatrix}
0 & 0 & 0 & 0 \\
\frac{(1-u)\beta_{p}BS}{k+B} + \beta_{H}SI & 0 & \frac{(k+B)(1-u)\beta_{p}S - (1-u)\beta_{p}SB}{k+B} \\
0 & 0 & 0 & 0 \\
\frac{(1-u)\beta_{p}BS}{k+B} + \beta_{H}SI + \mu S & (\gamma + \tau + \mu)I & -\gamma - \sigma + \mu R & -x(1-v)I + \delta B
\end{bmatrix}
\]

and

\[
V = \begin{bmatrix}
\frac{(1-u)\beta_{p}BS}{k+B} + \beta_{H}SI + \mu S & \beta_{H}S & \frac{(k+B)\beta_{p}S - (1-u)\beta_{p}SB}{k+B} \\
0 & 0 & 0 & 0 \\
0 & -\gamma - \tau & 0 & 0 \\
0 & -x(1-v) & 0 & \mu
\end{bmatrix}
\]

The inverse of \( V \) is

\[
V^{-1} = \begin{bmatrix}
\frac{1}{\mu} & \frac{\beta_{p}(1-v)(1-w)N}{k\mu(\gamma + \tau + \mu)} & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & \frac{\gamma + \tau}{\mu(\gamma + \tau + \mu)} & \frac{1}{\mu} & 0 \\
0 & \frac{x(1-v)}{\delta(\gamma + \tau)} & 0 & \frac{1}{\delta}
\end{bmatrix}
\]

This leads to
Local Asymptotically Stability:

The local stability of an equilibrium point is determined from the Jacobian matrix of the ordinary differential equation (1) – (4) evaluated at \( E_0 \). The Jacobian at \( E_0 \) is

\[
J_0 = \begin{bmatrix}
-\mu & -\beta_N & 0 & 0 \\
0 & -\beta_N & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\delta
\end{bmatrix}
\]

The eigenvalues of the \( J_0 \) are obtained by solving \( \text{det}(J_0 - \lambda I) = 0 \). From this, we obtain the characteristic equation,

\[
(\lambda + \mu)^2 [\lambda^2 + C\lambda + D] = 0
\]

where

\[
a_1 = -\beta_N \\
a_2 = \gamma + \tau + \mu - \beta_N \\
a_3 = \frac{(1-u)\beta_N}{k} \\
C = \delta + a_2 \\
D = a_2 \delta - a_3 \mu(1-v)
\]

From the characteristic equation, we see that two eigenvalues are \( \lambda_{1,2} = -\mu < 0 \). The other two are the solutions of the characteristic equation. The roots of this equation will be negative if two coefficients satisfied with the Routh-Hurwitz criteria (Allen,2006).

1) \( C > 0 \)
2) \( D > 0 \).

Disease Endemic Equilibrium Point:

To determine the stability of the endemic equilibrium point. We examine the eigenvalues of Jacobian matrix at \( E_1 \), which is

\[
J_1 = \begin{bmatrix}
\frac{(1-u)\beta}{k+B} & -\beta_N & 0 & 0 \\
\beta_N & -\beta_N & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\delta
\end{bmatrix}
\]

Where are given by equations (5) . The characteristic equation of Jacobian matrix at \( E_1 \) given by equations (1) – (4), becomes, \( (\lambda + \mu)[\lambda^2 + B_1\lambda^2 + B_2\lambda + B_3] = 0 \)

where

\[
A_1 = \frac{\beta \cdot B}{k+B} + \beta_N \cdot I + \mu, \quad A_2 = \frac{(1-u)\beta \cdot B}{k+B} + \beta_N \cdot I, \\
A_3 = -\beta_N \cdot S, \quad A_4 = \gamma + \tau + \mu - \beta_N \cdot S, \quad A_4 = \gamma + \tau
\]
The three eigenvalues of $A = \begin{pmatrix} 0 & 1 & 0 \\ -k & 0 & 1 \\ 0 & -1 & 0 \end{pmatrix}$ will have negative real part if they satisfy the Routh-Hurwitz criteria (Allen, 2006), that is $B_1 > B_3$. The value of parameters used in the numerical simulation are given in Table 1.

Table 1: Parameter values used in numerical simulations at disease free state.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\mu$</td>
<td>0.000045</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$\beta_g$</td>
<td>0.99999</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>0.00011</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$k$</td>
<td>1,000,000</td>
<td>cells ml day$^{-1}$</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>0.5</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$\delta$</td>
<td>0.3</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$\chi$</td>
<td>35</td>
<td>cell person ml day$^{-1}$</td>
</tr>
<tr>
<td>$\tau$</td>
<td>0.5</td>
<td>day$^{-1}$</td>
</tr>
<tr>
<td>$u$</td>
<td>0.95</td>
<td>-</td>
</tr>
<tr>
<td>$v$</td>
<td>0.05</td>
<td>-</td>
</tr>
<tr>
<td>$N$</td>
<td>10,000</td>
<td>person</td>
</tr>
</tbody>
</table>

Stability of the disease free state: Using the values of parameters listed in Table 1. We find the eigenvalues and basic reproductive number to be: $\lambda_1 = -0.9169$, $\lambda_2 = -0.273$, $\lambda_3 = -0.000045$, $\lambda_4 = -0.000045$ and $R_0 = 0.109995 < 1$. Since all of the eigenvalues are negative and the basic reproductive number is less than one, the equilibrium state will be the disease free state $E_0$, as seen in Fig. 2.
Stability of the endemic state: Using the values of parameter listed in Table 1, except the value of \( u \) we set to be equal 0.01. This values represented the value of the effectiveness of an educational campaign.

![Image](image.png)

**Fig. 3:** The time series of (a) the susceptible human, (b) the infected human, (c) the recovered human and (d) the concentration of bacteria in the water. Only the value of \( u \) has been changed to \( u = 0.01 \). All the state variables approach to endemic state \( E_1 = (4553.2, 0.2452, 0.000045, 0.000045, 0.2452) \).

The eigenvalues and basic reproductive number become greater than one and the outcome is quite different:

\[
\lambda_1 = -0.49909, \quad \lambda_2 = -0.300103, \quad \lambda_3 = -0.000099, \quad \lambda_4 = -0.000045 \quad \text{and} \quad R_0 = 1.09995 > 1
\]

Since all of the eigenvalues are to be negative and the basic reproductive number is greater than one, the equilibrium state will be the endemic state, \( E_1 \) as demonstrated in **Fig. 3**.

### 5 Discussion and Conclusion:

In this study, we proposed a transmission model of cholera by take into account the effectiveness of the educational campaign. The simulation results we can see that from Fig. 2, when \( u = 0.95 \) the basic reproductive number \( R_0 = 0.10999 \) which less than one in this case the disease will not occur. But when we change the value of \( u = 0.01 \) the basic reproductive number \( R_0 = 1.09995 \) which is greater than one in this case the disease will persist as the study of Suksawat and Naowarat (2014). Then the disease will persist in the community as shown in Fig. 3.

In this paper, we proposed effects of an educational campaign on the model of cholera transmission. Mathematical model consist of a system of four nonlinear differential equations. We found that there were two equilibrium points; disease free and endemic equilibrium points. The qualitative results are depended on a basic reproductive numbers. It concluded that if the effectiveness of the educational campaign is low, the number of cholera infection will be increase.

**ACKNOWLEDGMENTS**

Surapol Naowarat would like to thank Department of Mathematics, Faculty of Science and Technology, Surathani Rajabhat University for equipment support and the anonymous reviewers for their kind helpful comments.

**REFERENCES**


