Implementation of an optimal control for reducing individuals infected by hepatitis B virus

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Abstract. Hepatitis B is an inflammatory liver disease caused by hepatitis B virus, whose spread can be contagious and becomes a serious world health problem. The spread of hepatitis B can be modeled using the Susceptible, Infected, and Recovered (SIR) model. This paper aims is to reduce individuals infected with hepatitis B virus by applying optimal control strategies. The SIR model is given optimal control with three control variables, namely health promotion, treatment, and vaccination. Optimal control form is determined by the Pontryagin Maximum Principle (PMP) method to minimize the number of infected individuals and the associated costs. The step of PMP method i.e.Identify the Lagrange multipliers, and then minimize Hamiltonian function, further obtain stationary conditions, and finally find an optimal control. The results of numerical simulations completed by the Runge-Kutta Order 4 show effectiveness in minimizing infected individuals. In addition, it can also maximize individual recovery by giving the proposed three control variables.

1. Introduction
Hepatitis B is a liver disease resulting from infection with the hepatitis B virus (HBV) [1]. The hepatitis B infected people has high risk of death from liver cirrhosis and cancer [2]. Hepatitis B is a contagious disease that is a serious health problem in the world. This is evidenced by hepatitis B which is the main cause of morbidity and mortality in the continents of Africa and Asia [3]. According to 2015 WHO data, it shows that there have been 887,000 deaths per year due to complications of hepatitis B (HBV) including cirrhosis of the liver and liver cancer [4]. Indonesia is one of the countries in Asia that has a high endemicity of hepatitis B, the second largest after Myanmar from a member country of the South East Asian Region (SEAR). Based on 2013 data from the Indonesian Ministry of Health, hepatitis B sufferers reached 649,875 (21.8%) of the total population in Indonesia who suffer from hepatitis [5].

Referring to cases of hepatitis B which are easily contagious and can cause death, indicates hepatitis B should receive special treatment. Another reason for studying hepatitis B virus (HBV) infection is to improve control and ultimately stop infection from the population. Mathematical models can be a useful tool in this approach which helps to optimize the use of limited resources or only for the purpose of more impressive (incidence of infection) control measures.

Kamyad et al [6] use the SEICR mathematical model to analyze and apply optimal controls to determine the likely impact of vaccination susceptible individuals and treatment for infected individuals. Khatun and Biswas [7] also used control in the form of vaccinations and treatment in the SEILCR mathematical model, which is a model that divides 6 classes, namely susceptible, exposed, infected, liver cirrhosis, and recovered classes. Fitrinani et al [8] analyze the effect of vaccination and disinfection
in the dynamical population of the spread of cholera. The population divided into two groups, namely the human population (N) with SIR models and V. Cholera population (B). Then, this model considers the vaccination and disinfection to control the spread of cholera. The SEIR model that was constructed by Rosyada et al [9] describes the transmission of the influenza virus by paying attention to the disease resistance in humans. So that individuals who recover but have low immunity can return to being susceptible individuals. And in the spread of Tuberculosis disease can also use a mathematical model as constructed by Mahardika et al [10], they divide human into five subpopulations, namely susceptible class (S), latent class (E), infectious treated at home class (I1), contagious treated at hospital class (I2), and recovery class (R).

In this paper, we present a mathematical model to clarify the dynamics of hepatitis B disease transmission, which can be controlled by health promotion, treatment, and vaccination. In this study, we analyzed a mathematical model of SIR that was constructed by Khan et al [11] and also applied optimal control theory by considering three control variables for disease prevention and minimization. Finally, we performed numerical simulations to interpret the results and also to demonstrate the effectiveness of health promotion and vaccination in preventing hepatitis B infection and appropriate treatment to control hepatitis B disease. In the numerical analysis section the efficiency index was also carried out to determine which controls were more effective than others. The main objective of this work is to minimize individual hepatitis B infection by using health promotion, treatment, and vaccination as controls as well as the associated costs of implementing the three control measures.

2. Methods
The method used to solve the problem of optimal control of the spread of hepatitis B is the Pontryagin’s Maximum Principle (PMP) method [12]. The step of PMP method i.e.

a. Identify the Lagrange multipliers.

\[ L = J + \int_{t_0}^{t_f} \lambda (g(x(t),u(t),t) - \dot{x}(t)) dt \]

with

\[ J[u(t)] = \int_{t_0}^{t_f} f(x(t),u(t),t) dt \] (Functionally Objective)

\[ \dot{x}(t) = g(x(t),u(t),t) \] (Constraint Function)

b. Minimize a Hamiltonian function.

\[ H(x(t),u(t),t) = f(x(t),u(t),t) + \lambda(g(x(t),u(t),t)) \]

c. Obtain stationary conditions

\[ \dot{x} = \frac{\partial H}{\partial \lambda} \] (State equations)

\[ \dot{\lambda} = -\frac{\partial H}{\partial x} \] (Co-state equations)

\[ \frac{\partial H}{\partial u_i} = 0 \] (Stationary Conditions)

d. Find an optimal control

\[ \frac{\partial H}{\partial u_1} = \frac{\partial H}{\partial u_2} = \frac{\partial H}{\partial u_3} = 0 \]

3. Mathematical model without control
The SIR model for the spread of hepatitis B consists of three variables, namely susceptible individuals (S), infected individuals (I), and recovered or immune individuals (R). While the parameters used in the
SIR model of the spread of hepatitis B are birth rate (\(\lambda\)), hepatitis B virus spread rate (\(\alpha\)), human population (\(N\)), natural mortality rate (\(\mu_0\)), death rate due to disease (\(\mu_1\)), rate of increase. (\(\beta\)), vaccination (\(\nu\)), and saturation incidence ratio (\(\gamma\)).

The assumptions used in the process of modelling the spread of hepatitis B are as follows, all new births will go to the susceptible class only, the incidence rate is taken to be the saturated incidence rate, the recovered population has permanent immunity, and the successfully vaccinated population will go to the recovered class.

The SIR model transmission scheme for the spread of hepatitis B can be presented [11]

\[
\begin{align*}
\frac{dS}{dt} &= \lambda - \frac{\alpha S I}{1 + \gamma I} - (\mu_0 + \nu) S \\
\frac{dI}{dt} &= \frac{\alpha S I}{1 + \gamma I} - (\mu_0 + \mu_1 + \beta) I \\
\frac{dR}{dt} &= \beta I + \nu S - \mu_0 R
\end{align*}
\]

with

\[
S(0) > 0, \quad I(0) > 0, \quad R(0) > 0
\]

The total individuals are \(N(t)\), where \(N(t) = S(t) + I(t) + R(t)\). So, we have

\[
\frac{dN}{dt} = \lambda - \mu_0 N(t)
\]

Thus, we have \(N(t) \leq \frac{\lambda}{\mu_0} + N(0)e^{-\mu_0 t}\). It showsthat \(N(t) \leq \frac{\lambda}{\mu_0}\), if \(N(t) \leq \frac{\lambda}{\mu_0}\). The disease free equilibrium is given by, \(SFE = \left\{ \frac{\lambda}{(\mu_0 + \nu)}, 0, \frac{\nu \lambda}{\mu_0 (\mu_0 + \nu)} \right\} \). The basic reproduction ratio is defined by

\[
\mathcal{R}_0 = \frac{\alpha \lambda}{(\mu_0 + \nu)(\mu_0 + \mu_1 + \beta)}
\]

It represents the number of individuals who are susceptible to infected. Next, we provide the existence and uniqueness of endemic equilibrium of the model in the following theorem

**Theorem 1.** [11] If \(\mathcal{R}_0 > 1\), then the disease endemic equilibrium point \(E^*\) of the proposed model (1) is locally asymptotically stable and unstable, when \(R_0 < 1\).

**Proof:**

Taking the Jacobian matrix of the proposed model (1) at DEE (\(E^*\)), we get

\[
J(E^*) = \begin{bmatrix}
\frac{-\alpha I^*}{1 + \gamma I^*} - (\mu_0 + \nu) & \frac{-\alpha S}{1 + \gamma I^*} & 0 \\
\frac{\alpha I^*}{1 + \gamma I^*} & \frac{\alpha S}{(1 + \gamma I^*)^2} - (\mu_0 + \mu_1 + \beta) & 0 \\
\nu & \beta & -\mu_0
\end{bmatrix}
\]

Clearly, one eigenvalues, \(\lambda_1 = -\mu_0\) among the eigenvalues of the \(J(E^*)\) have negative real part. For the remaining, we take the following matrix

\[
A = \begin{bmatrix}
\frac{-\alpha I^*}{1 + \gamma I^*} - (\mu_0 + \nu) & \frac{-\alpha S}{1 + \gamma I^*} \\
\frac{\alpha I^*}{1 + \gamma I^*} & \frac{\alpha S}{(1 + \gamma I^*)^2} - (\mu_0 + \mu_1 + \beta)
\end{bmatrix}
\]

The eigenvalues of the above matrix \(A\) are negative, if the Hurwitz (\(H_1\)): \(\text{trace}(A) < 0\) and \(\det(A) < 0\) criterion satisfied. Therefore \(\text{trace}(A) < 0\) of matrix \(A\) becomes
\[ trace(A) = -\frac{\alpha l^*}{1 + y l^*} - (\mu_0 + v) + \frac{a S}{(1 + y l^*)^2} - (\mu_0 + \mu_1 + \beta) \]

\[ trace(A) = -\alpha l^* (1 + y l^*) - (\mu_0 + v) (1 + y l^*)^2 - (\mu_0 + \mu_1 + \beta) y l^* (2 + y l^*) \]

\[ trace(A) = -\frac{a (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) (1 + \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1)) - (\mu_0 + v) \left( 1 + \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \right)^2 - \]

\[ (\mu_0 + \mu_1 + \beta) \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \left( 2 + \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \right) \]

\[ trace(A) < 0 \text{ is met if } R_0 > 1. \]

Similarly determinant of matrix \( A \) becomes

\[ det(A) = \left( -\frac{\alpha l^*}{1 + y l^*} - (\mu_0 + v) \right) \left( \frac{a S}{(1 + y l^*)^2} - (\mu_0 + \mu_1 + \beta) \right) + \left( \frac{\alpha l^*}{1 + y l^*} \frac{a S^*}{1 + y l^*} \right) \]

\[ det(A) = \alpha v (\mu_0 + \mu_1 + \beta) (1 + y l^*) (1 + 2 y l^*) + (\mu_0 + \mu_1 + \beta) (\mu_0 + v) (1 + y l^*)^2 (y l^*) \]

\[ det(A) = \alpha \frac{\mu_0 + v}{\mu_0 + \mu_1 + \beta} (R_0 - 1) (\mu_0 + \mu_1 + \beta) \left( 1 + \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \right) \left( 1 + 2 \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \right) + \]

\[ (\mu_0 + \mu_1 + \beta) (\mu_0 + v) \left( 1 + \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \right)^2 \frac{y (\mu_0 + v)}{\mu_0 + \mu_1 + \beta} (R_0 - 1) \]

\[ det(A) > 0 \text{ is met if } R_0 > 1. \]

Based on Hurwitz (\( H_1 \)) criteria [13], \( E^l \) locally asymptotically stable if \( trace(A) < 0 \) and \( det(A) > 0 \), these conditions are met if \( R_0 > 1. \)

4. Results and discussion

The problem that occurs in a population where the disease is endemic is the level of disease spread. The spread of disease that occurs can be reduced through health promotion, administration of treatment for infected individuals, and vaccination for susceptible individuals. The application of this optimal control is to determine the possible impact of health promotion (\( u_1 \)) in susceptible and infected individuals, treatment (\( u_2 \)) in infected individuals, and vaccination (\( u_3 \)) in susceptible individuals. In this case \( u_1, u_2, \) and \( u_3 \) are time dependent control variables.

The number of susceptible individuals (\( S \)), infected individuals (\( I \)), and individuals recovered (\( R \)) affects the magnitude of the level of health promotion, treatment, and vaccination in an effort to minimize the spread of hepatitis B. It is assumed that individuals who recover or are immune after treatment and vaccination will be included in the recovered or immune (\( R \)) class.

Many susceptible individuals (\( S \)) are the number of individuals who are healthy but susceptible to hepatitis B transmission. Changes in the number of individuals susceptible to hepatitis B will increase due to a natural birth rate of \( \lambda \), then it will decrease due to interactions between susceptible individuals and infected individuals. The spread rate of hepatitis B virus is \( \alpha \) with saturation incidence ratio \( \gamma \) and the number of susceptible individuals will also decrease due to the natural death rate \( \mu_0 \), vaccination in susceptible individuals, and control giving in the form of vaccination (\( u_3 \)). The growth rate of susceptibles population is

\[ \frac{dS}{dt} = \lambda - \frac{a S I}{1 + y l^*} (1 - u_1(t)) - (\mu_0 + u_3(t)) S \]  \( \text{(4)} \)

The number of infected individuals (\( I \)) is the number of individuals infected with hepatitis B. The change in the number of individuals infected with hepatitis B will increase due to the interaction between susceptible individuals and infected individuals, the rate of spread of the hepatitis B virus is \( \alpha \) with the saturation incidence ratio \( \gamma \). And the number of infected individuals will decrease due to the natural death rate of \( \mu_0 \), the death rate due to disease of \( \mu_1 \), recovery in infected individuals at the \( \beta \) rate, and control of health promotion (\( u_1 \)) and treatment (\( u_2 \)) of infected individuals. Thus the growth rate of infected class is,
\[ \frac{dI}{dt} = \frac{asI}{1+\gamma I} (1 - u_1(t)) - (\mu_0 + \mu_1 + \beta + u_2(t))I \]

(5)

Many individuals who recover or are immune (R) are the number of individuals who have recovered or become immune to hepatitis B. The change in the number of recovered or immune to hepatitis B will increase due to the rate of recovery for infected individuals by \( \beta \) and provision of control in the form of treatment(\( u_2 \)) for individuals infected and vaccination against susceptible individuals. And the number of recovered or immune individuals will decrease because of the natural death rate of \( \mu_0 \). So that the growth rate of recovered class is,

\[ \frac{dR}{dt} = (\beta + u_2(t))I + u_3(t)S - \mu_0 R \]

(6)

So that the constraint function of mathematical model with control is formed as follows:

\[ \begin{align*}
\frac{dS}{dt} &= \lambda - \frac{asI}{1+\gamma I} (1 - u_1(t)) - (\mu_0 + u_3(t))S \\
\frac{dI}{dt} &= \frac{asI}{1+\gamma I} (1 - u_1(t)) - (\mu_0 + \mu_1 + \beta + u_2(t))I \\
\frac{dR}{dt} &= (\beta + u_2(t))I + u_3(t)S - \mu_0 R
\end{align*} \]

(7)

with

\[ S(0) > 0, I(0) > 0, R(0) > 0 \]

The objective functional of optimal control can be expressed in the following equation:

\[ J(u_1, u_2, u_3) = \int_0^T \left[ w_1 I + \frac{1}{2} \left( w_2 u_1^2(t) + w_3 u_2^2(t) + w_4 u_3^2(t) \right) \right] dt \]

where \( w_1, w_2, w_3, w_4 \) are the relative weights of infected individuals, health promotion, treatment, and vaccination. Terms \( w_2 u_1^2(t) \), \( w_3 u_2^2(t) \) and \( w_4 u_3^2(t) \) represent costs related to education/media campaign, treatment, and vaccination respectively.

Hamiltonian of the system can be determine by

\[ H(x, u, \lambda) = w_1 I + \frac{1}{2} \left( w_2 u_1^2(t) + w_3 u_2^2(t) + w_4 u_3^2(t) \right) + \lambda_1 \left( \frac{dS}{dt} \right) + \lambda_2 \left( \frac{dI}{dt} \right) + \lambda_3 \left( \frac{dR}{dt} \right) \]

(9)

next, we prove Theorem 2 below

**Theorem 2.** Consider optimal control of variable \( u_1^*, u_2^*, u_3^* \) and solutions \( S^*, I^*, R^* \) of the system (7) for minimizing \( J(u_1, u_2, u_3) \) over \( U \). Then there exists an adjoint variables \( \lambda_1, \lambda_2, \lambda_3 \) satisfying

\[ \begin{align*}
\frac{d\lambda_1}{dt} &= \lambda_1 \left( \frac{aI}{1+\gamma I} (1 - u_1(t)) + (\mu_0 + u_3(t)) \right) - \lambda_2 \left( \frac{aI}{1+\gamma I} (1 - u_1(t)) \right) + \lambda_3 u_3 \\
\frac{d\lambda_2}{dt} &= -w_1 + \lambda_1 \left( \frac{asI}{1+\gamma I} (1 - u_1(t)) \right) - \lambda_2 \left( \frac{asI}{1+\gamma I} (1 - u_1(t)) - (\mu_0 + \mu_1 + \beta + u_2(t)) \right) - \lambda_3 (\beta + u_2(t)) \\
\frac{d\lambda_3}{dt} &= \lambda_3 \mu_0 \end{align*} \]

(10)

with conditions

\[ \lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = 0 \]

(11)

and the control \( u_1^*, u_2^*, u_3^* \) satisfy the optimality condition

\[ \begin{align*}
u_1^* &= \frac{1}{w_2} \left( \frac{as^* I}{1+\gamma I} (\lambda_1 - \lambda_2) \right) \\
u_2^* &= \frac{1}{w_3} \left( I^* (\lambda_2 - \lambda_3) \right) \\
u_3^* &= \frac{1}{w_4} \left( S^* (\lambda_1 - \lambda_3) \right)
\end{align*} \]

(12)

**Proof.** Differentiating the Hamiltonian in (9) with respect to each state variable, we obtain the differential equation for corresponding adjoint variables. Also, because \( S(t), I(t), \) and \( R(t) \), the values of
the adjoint state at the final time are zero. We obtain the differential equation system corresponding adjoint variables,
\[
\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial s} = \lambda_1 \left( \frac{a_1}{1+y_l} (1-u_1(t)) + (\mu_0 + u_3(t)) \right) - \lambda_2 \left( \frac{a_1}{1+y_l} (1-u_1(t)) \right) + \lambda_3 u_3
\]
\[
\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial l} = -w_1 + \lambda_1 \left( \frac{a_S}{1+y_l} (1-u_1(t)) \right) - \lambda_2 \left( \frac{a_S}{1+y_l} (1-u_1(t)) \right) - (\mu_0 + \mu_1 + \beta + u_2(t))
\]
\[
\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial R} = \lambda_3 \mu_0
\]
with conditions
\[
\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = 0
\]
Differentiating Hamiltonian function (9) with respect to each \(u_1^*, u_2^*, u_3^*\) and evaluating at optimal control variables, we have
\[
0 = \frac{\partial H}{\partial u_1} = w_2 u_1^* + \frac{a_S d_1^*}{1+y_l} (\lambda_1 - \lambda_2)
\]
\[
0 = \frac{\partial H}{\partial u_2} = w_3 u_2^* - I^* (\lambda_2 - \lambda_3)
\]
\[
0 = \frac{\partial H}{\partial u_3} = w_4 u_3^* - S^* (\lambda_1 - \lambda_3)
\]
Thus, we obtain
\[
u_1^* = \frac{1}{w_2} \left( -\frac{a_S d_1^*}{1+y_l} (\lambda_1 - \lambda_2) \right)
\]
\[
u_2^* = \frac{1}{w_3} (I^* (\lambda_2 - \lambda_3))
\]
\[
u_3^* = \frac{1}{w_4} (S^* (\lambda_1 - \lambda_3))
\]
If \(\frac{\partial H}{\partial u_i} < 0\), at \(t\), then \(u_i^*(t) = 0\), for \(i = 1, 2, 3\), conversely, if \(\frac{\partial H}{\partial u_i} > 0\), at \(t\), we take \(u_i^*(t) = 1\).

Therefore, optimal control variables of \(u_1^*, u_2^*, u_3^*\) are characterized by
\[
u_1^*(t) = \min\{u_{1\text{max}}, \max\left(0, \frac{1}{w_2} \left( -\frac{a_S d_1^*}{1+y_l} (\lambda_1 - \lambda_2) \right) \right)\}
\]
\[
u_2^*(t) = \min\{u_{2\text{max}}, \max\left(0, \frac{1}{w_3} (I (\lambda_2 - \lambda_3)) \right)\}
\]
\[
u_3^*(t) = \min\{u_{3\text{max}}, \max\left(0, \frac{1}{w_4} (S (\lambda_1 - \lambda_3)) \right)\}
\]
It exhibits the uniqueness of the optimal control of the system (9), (13), and (14) with characterization (17). Based on Theorem 2, the optimal control value is obtained from the three control variables used. So the problem of optimum control of the spread of the hepatitis B virus through health promotion, treatment, and vaccination is the problem of minimizing the number of individuals infected with the hepatitis B virus. With the dynamic system constraints (7), the adjoint system (13) and \(u_1^*, u_2^*, u_3^*\) which fulfill the initial conditions \(S(0) = S_0, I(0) = I_0, R(0) = R_0\) and the final condition \(\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = 0\).

Further, we proposed simulations Results. Parameters used for numerical simulation based on the parameter from Tahir Khan, et al [11], Khatun, et al [7] and Setiawan [14] are as follows, the birth rate
\[ (\lambda) = \frac{417}{365} \text{ / day, transmission rate of hepatitis B } (\alpha) = 0.005 \text{ / day, the natural death rate } (\mu_0) = \frac{0.95}{365} \text{ / day, the disease death rate } (\mu_1) = \frac{0.02}{365} \text{ / day, recovery rate } (\beta) = 0.2 \text{ / day, the vaccination rate } (v) = 0.02 \text{ / day, and saturation rate } (\gamma) = 0.900/\text{day.}\]

Based on calculation results, the basic reproductive number \((R_0)\) is obtained as follows:

\[ R_0 = \frac{\alpha \lambda}{(\mu_0 + v)(\mu_0 + \mu_1 + \beta)} = 1.1249 > 1 \]

Because \(R_0 > 1\) hence the system will be asymptotically stable at the endemic equilibrium point, means that there is spread of hepatitis B. Therefore, to prevent the spread of hepatitis B, three controllers are given, namely health promotion for susceptible and infected individuals, vaccination for susceptible individuals, and treatment for individuals infected with the hepatitis B virus. The simulation process is carried out by observing the number of populations of susceptible individuals, infected individuals, and recovered or immune individuals, both before control and after control. By using the MATLAB R2015a application the following results are obtained:

![Figure 2](image1.png) **Figure 2.** Individual populations susceptible with and without controls

The population dynamics of individuals who are susceptible to hepatitis B virus infection in Figure 2 shows the effect of control provision in the form of health promotion \((u_1)\) and vaccination \((u_3)\) on susceptible individuals. The population of susceptible individuals has decreased after the provision of health promotion controls and vaccinations.

![Figure 3](image2.png) **Figure 3.** Individual populations infected with and without controls

Figure 3 shows that the population of infected individuals without control with the initial condition of the population decreases until day 30 and moves constantly towards the equilibrium point. While the population of infected individuals with control in the form of health promotion \((u_1)\) and treatment \((u_2)\) decreased more rapidly than without control.
From figure 4 shows that there is an increase in the population of individuals who recover or are immune after giving control to the system compared to without control.

Figure 4. Individual populations recovered with and without controls

Figure 5 shows a single comparison of control giving in the form of health promotion ($u_1$), medication ($u_2$), vaccination ($u_3$), and the combination of the three controls. In this case the overall efficiency is defined as $\epsilon = 1 - (1-u_1) (1-u_2) (1-u_3) = 0.5$. This shows that the control provision in the form of vaccination is more effective than the health promotion of treatment, and the combination of the three controls.

From all the results obtained, broadly speaking the provision of control in the form of health promotion, treatment, and vaccination in the system can reduce the spread of the hepatitis B virus according to their respective roles. In this case the effectiveness of the controller at approximately 0 to 100 days can reduce the individual population infected with the hepatitis B virus so that the spread of hepatitis B can be suppressed and can maximize the individual population to recover or be immune and be able to minimize the costs required in providing control. The results of this numerical simulation were obtained using the MATLAB R2015a application.

5. Conclusion

The model used in controlling the spread of the hepatitis B virus in humans is SIR (Susceptible, Infected, Recovered). There are three controls used in this model, namely health promotion, treatment, and vaccination. Health promotion is given to susceptible and infected individuals, treatment is given to infected individuals, and vaccination is given to susceptible individuals. The numerical simulation results show that the provision of control in the form of health promotion, treatment, and vaccination can reduce the individual population infected with the hepatitis B virus, so that the spread of hepatitis B can be suppressed and can maximize the individual population to recover or be immune. From the three control
variables, it was found that the provision of control in the form of vaccination was more effective than medical health promotion, and the combination of the three controls.

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