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A study about mathematical analysis of Hepatitis B virus using Optimal Control approach

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Abstract

This research focuses on the mathematical modeling of Hepatitis B virus (HBV) dynamics using optimal control theory to enhance understanding of transmission patterns and optimize intervention strategies. An ordinary differential equation (ODE) model is proposed, capturing the dynamics of HBV transmission through distinct compartments: susceptible, exposed, infected, liver cirrhosis, and removed. Advanced liver cirrhosis, a severe stage of chronic liver disease caused by sustained and progressive damage, has emerged as a critical non-communicable health concern. The study employs mathematical simulations to analyze the impact of various control measures in mitigating HBV spread. Through the application of optimal control theory and the Hamiltonian principle, the research identifies effective strategies, such as vaccination, treatment, and awareness campaigns, to manage and limit HBV transmission. The primary objective is to minimize the number of individuals in the infected and cirrhotic stages while reducing associated intervention costs. By targeting HBV, a leading cause of cirrhosis, the study aims to lower the incidence of chronic liver disease. The findings highlight the importance of vaccination, effective treatment protocols, and public awareness in curbing the progression of HBV and reducing its long-term health impacts. This research provides crucial insights for public health policies and the development of targeted strategies to combat HBV and its complications.

Keywords: Hepatitis B Virus; Liver cirrhosis latent; Optimal control theory; Treatment control and awareness campaign control.

1. Introduction

A number of viruses, including A, B, C, D, and E, can cause hepatitis, which is defined as inflammation of the liver. Liver illness is characterized by jaundice. Hepatitis B, a potentially lethal liver infection, is caused by the Hepatitis B Virus (HBV), a serious global health concern. Significant advancements have been made in the last ten years in the use of positive antiviral treatment for chronic HBV infection, which was initially documented three decades ago [1]. But this advancement has also made therapy more challenging. With 350 million chronic cases [2] worldwide and 4,000 new cases in the US in 2006, hepatitis B is a very common disease. According to estimates, between 2,000 and 4,000 people die each year from chronic Hepatitis B (CHB) liver disorders[3]. CHB develops in only 5% of immunocompetent persons with severe infection. The World Health Organization (WHO) estimated that in 2015, [4] 257 million people were living with HBV, resulting in nearly 900,000 deaths. A 2006 Sampling Survey for HBV Epidemiology indicated that the HBsAg prevalence among children under five was less than 1%. [5] Chronic HBsAg carriers have significantly higher rates of hepatocellular carcinoma (HCC), cirrhosis, and mortality compared to those who have never been chronically HBsAg-positive. The annual incidence of HCC is 0.1% in asymptomatic HBsAg carriers, 1% in CHB patients, and 3-10% in those with cirrhosis. CHB patients develop cirrhosis at a rate of 2% per year [6]. Studies have shown significant differences in clinical outcomes among various diagnostic groups, including inactive HBsAg carriers, CHB without cirrhosis, and CHB with cirrhosis. A U.S. cohort study followed 400 HBsAg patients [7] (70% born in Asia) for over seven years. Among 110 inactive carriers, none developed HCC or died from liver-related diseases, and only one died from any cause. In CHB patients without cirrhosis, [8] 6% developed HCC and died from it, and 2% died from non-liver-related causes. In contrast, [9]16% of CHB patients with cirrhosis were diagnosed

with HCC, and [10] 42% died during the follow-up period, all from liver-related causes. Vertical transmission refers to the transmission of HBV from mother to fetus.

One of the primary challenges in studying hepatitis B virus (HBV) infection is devising strategies to control and eradicate the infection across the entire population. Mathematical models are instrumental in optimizing resources and implementing control measures more efficiently and effectively. To illustrate the impact of carriers on HBV transmission, Roy M. Anderson and Robert M. May [11] employed a straightforward mathematical model. [12] Graham F. Medley Azra C. Ghani, R. M. Anderson [13] developed a mathematical model to formulate a strategy for eradicating HBV infection in New Zealand [14]. Zheo et al. [15] proposed an age-structured model to predict HBV transmission and assess the effectiveness of vaccination programs in China. Jingjing Pang, Lanlan Xu, Qiao Liu, Min Yao, Yi Zhang, Jie Ren, Yanjun Kang, Meng Wang, Li Liu, Guofeng Chen, Li Zhang, and Jidong Jia [16] created a model to evaluate the impact of vaccination on a population and the effectiveness of other control measures against HBV infection. Zhang and Zhou [17] provided analysis and applications for this model.

The best possible management of infectious diseases Gul Zaman and Imran Khan [19] and Trimoty J. Wilt and Tatyana [18] suggested a mathematical model to control both acute and chronic HBV transmission. [20] Hepatitis B viral model by Julse L. Diyantag. Tahir Khan's Chronic HBV model [21] and Hussan Alrabiah, Mohammad Safi, Bashir et al.'s optimal control study are also available. Additionally, we observed the HBV treatment in a model by Kar.T.K. Batabyal, A [22]. The potential of pulse vaccination to successfully manage epidemics while maintaining stability in vaccination quantity and pulse intervals has been demonstrated using pulse vaccination epidemic models [23–24]. However, vaccine and treatment-based epidemic control methods can be expensive and not always practical. The HBV model can be drawn to prevent [25–26].

2. Hepatitis B virus and liver cirrhosis severity in whole world

Liver cirrhosis has become a significant global health concern, affecting populations across both developed and developing countries. This condition typically results from the long-term progression of liver diseases such as Hepatitis B and C, fatty liver disease, or other previously undiagnosed liver conditions (as shown in Fig. 1). Cirrhosis is the result of ongoing liver damage caused by these diseases. Among these, Hepatitis B is the leading cause of chronic liver cirrhosis. Each year, Hepatitis B infects millions of people worldwide, contributing to a rise in cases of chronic cirrhosis. Of the four countries analyzed China, India, the United States, and Bangladesh, India exhibits a particularly high incidence of liver cirrhosis. The Indian population experiences a higher frequency of cirrhosis, while Bangladesh has a relatively lower incidence compared to the other countries. In Bangladesh, for instance, 5.3 deaths per 100,000 people are attributed to HBV infection. Hepatitis C, on the other hand, accounts for 30% of cirrhosis cases and 17% of hepatocellular carcinoma cases in the country. As illustrated in Fig. 1, the global distribution of liver diseases linked to Hepatitis B varies significantly, highlighting the widespread impact of HBV across different populations.

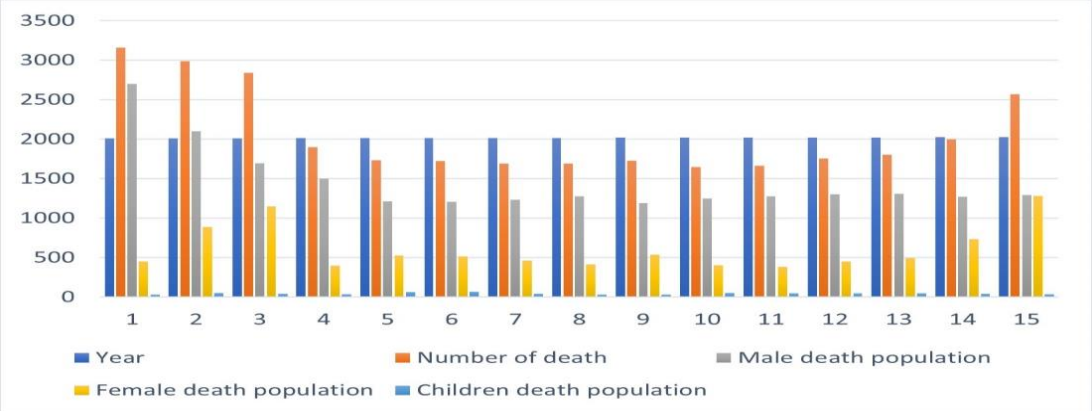

Fig. 1. Hepatitis B infection-associated Death from 2010 to 2024 in the world.

2.1 Model formulation

In this paper, we can express the communication forces at work and HBV resistor by using ordinary differential equations. The below model is given by five ordinary differential equations (ODEs) to demonstrate the active performance of hepatitis B virus. The collective human population at any instantaneous of time $\boldsymbol{t}$ represented by $\boldsymbol{N(t)}$ has five different classes, namely $\boldsymbol{S(t)}$ the susceptible class, $\boldsymbol{E(t)}$ the exposed class i.e. are not infected at time $\boldsymbol{t}$, $\boldsymbol{I(t)}$ the infective class who are affected by infections and can convey at any time, $\boldsymbol{L_c}$ the liver cirrhotic latent class who are affected by liver cirrhosis of the liver,

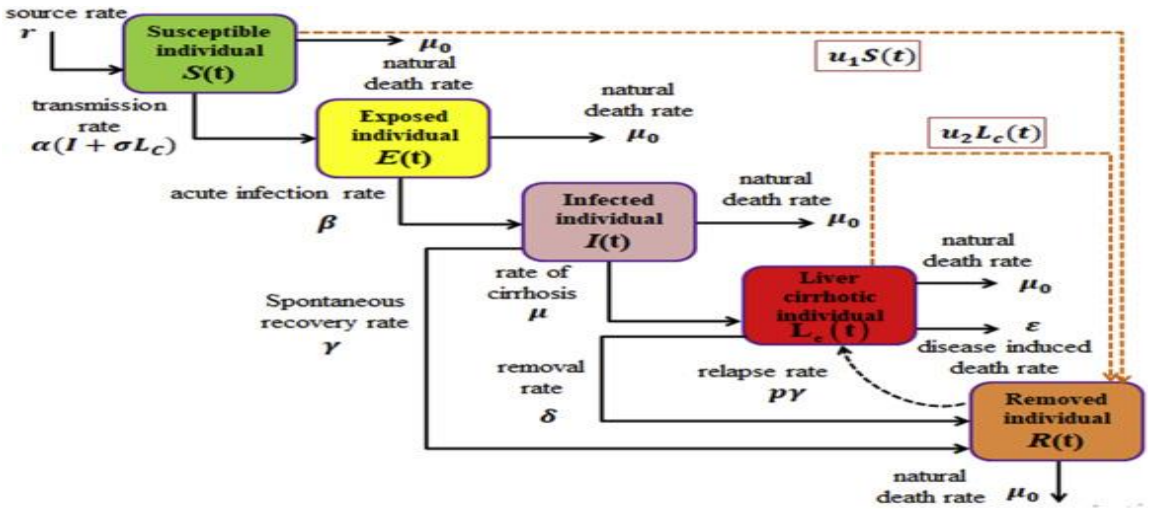

Fig. 2. Flow diagram of the compartmentalized model.

$\boldsymbol{R(t)}$ is the removed class who are removed for recovery, death, and any other means. Fig.2.[25] characterizes the flow chart of the model which embodies the segments of all the variable quantity taken from the population. We style a statement that the quantity of newborn transporters is overall lower than the sum of the shippers that have died and the population is fluctuating from hauler public to a constant immune public.
Therefore

$$\boldsymbol{N(t) = S(t) + I(t) + L_c(t) + R(t).}$$

In Fig. 2, we have presumed that a steady population with birth rate of per heads $\boldsymbol{r}$ and per heads death rate $\boldsymbol{\mu_0}$. $\boldsymbol{\sigma}$ is the infectiousness of haulers qualified to critical infection, $\boldsymbol{\beta}$ is the serious infection rate of exposed class, $\boldsymbol{\alpha}$ is the transmission rate and $\boldsymbol{\gamma}$ is the natural recovery rate of infective class. Here, the parameter $\boldsymbol{\mu}$ is infected class cirrhotic getting number, $\boldsymbol{\delta}$ is the retrieval rate of cirrhotic class, and $\boldsymbol{\epsilon}$ is the disease-persuaded death rate. We assumed two control parameters of vaccination rate control $\boldsymbol{u_1}$ and treatment rate control $\boldsymbol{u_2}$, and awareness campaign rate control $\boldsymbol{u_3}$. Also, $\boldsymbol{p\gamma}$ is the rate of infections cirrhosis declining in the liver cirrhotic class after being recovered. At the identical rate all epidemiological classes occur in ordinary death. Thus, the following equation describes the dynamics of the susceptible:

$$\boldsymbol{\frac{dS}{dt} = r - \alpha(I + \sigma L)S - (\mu_0 + u_1)S}$$

The population of buried compartment demonstrates the susceptible class infection. This population cuts by the transmission rate $\boldsymbol{\sigma}$ to the serious (infection) class and the rate of normal death per capita death rate $\boldsymbol{\mu_o}$.

Now, we have the following equation:

$$\boldsymbol{\frac{dE}{dt} = \alpha(I + \sigma L_c)S - (\mu_0 + \beta)E}$$

The population of exposed class demonstrates the susceptible class for infection. This population reductions by the transfer rate $\boldsymbol{\sigma}$. And the number of serious populations decreases by the natural death rate μ_0, and this class is abridged by the class moving to the continuing class at the rate $\boldsymbol{p\gamma}$.
Thus, for the infected individual class, we obtain the following equation:

$$\frac{dI}{dt} = \boldsymbol{\beta E} - (\mu + \boldsymbol{\gamma} + \mu_0)\boldsymbol{I}$$

The hepatitis B virus is intensely diseased class grow the lingering period and then change to the lingering class at the rate $\boldsymbol{p\gamma}$ and the newborn broods instinctive to diseased mas are un-immunized and treatment for this liver cirrhosis latent applicable in this situation, this class is amplified at the rate of $\boldsymbol{u_2}$, and the class for education awareness campaign of people about the epidemiology about liver cirrhosis is amplified at the rate of $\boldsymbol{u_3}$. We can see it is decreased by the natural death at the rate μ_0.
So, to define the dynamics of the liver cirrhotic latent class, we express the following equation:

$$\frac{dL_c}{dt} = (\mu + \boldsymbol{p\gamma})\boldsymbol{I} - (\mu_0 + \boldsymbol{\delta} + \boldsymbol{\varepsilon} - \boldsymbol{u_2} + \boldsymbol{u_3})\boldsymbol{L_c}$$

Lastly, the recovered class is summary by the same natural death rate and the following differential equation express by:

$$\frac{dR}{dt} = (\boldsymbol{\delta} + \boldsymbol{u_2} + \boldsymbol{u_3})\boldsymbol{L_c} - \mu_0\boldsymbol{R} + (\boldsymbol{\gamma} - \boldsymbol{p\gamma})\boldsymbol{I} + \boldsymbol{u_1 S}$$

Therefore, combining all the differential equations, we finally obtain the following system of HBV called model (1):

$$\begin{aligned}
&\frac{dS}{dt} = \boldsymbol{r} - \boldsymbol{\alpha}(\boldsymbol{I} + \boldsymbol{\sigma L_c})\boldsymbol{S} - (\boldsymbol{\mu_0} + \boldsymbol{u_1})\boldsymbol{S}, \qquad (1)\\
&\frac{dE}{dt} = \boldsymbol{\alpha}(\boldsymbol{I} + \boldsymbol{\sigma L_c})\boldsymbol{S} - (\boldsymbol{\mu_0} + \boldsymbol{\beta})\boldsymbol{E},\\
&\frac{dI}{dt} = \boldsymbol{\beta E} - (\boldsymbol{\mu_0} + \boldsymbol{\mu} + \boldsymbol{\gamma})\boldsymbol{I},\\
&\frac{dL_c}{dt} = (\boldsymbol{\mu} + \boldsymbol{p\gamma})\boldsymbol{I} - (\boldsymbol{\mu_0} + \boldsymbol{\delta} + \boldsymbol{\varepsilon} + \boldsymbol{u_2} + \boldsymbol{u_3})\boldsymbol{L_c},\\
&\frac{dR}{dt} = (\boldsymbol{\delta} + \boldsymbol{u_2} + \boldsymbol{u_3})\boldsymbol{L_c} - \mu_0\boldsymbol{R} + (\boldsymbol{\gamma} - \boldsymbol{p\gamma})\boldsymbol{I} + \boldsymbol{u_1 S},
\end{aligned}$$

And, initial conditions $\boldsymbol{S > 0, E \geq 0, I \geq 0, L_c \geq 0, R \geq 0}$.

Now, we have three control variable quantity $(\boldsymbol{u_1, u_2, u_3})$:

(i) Vaccination against hepatitis B prior to infection to prevent the disease.
(ii) Post-infection treatment targeting the underlying causes that have led to the development of liver cirrhosis.
(iii) Raising awareness campaign and implementing control measures for hepatitis B virus (HBV).

So, $\boldsymbol{u_1(t)}$, $\boldsymbol{u_2(t)}$ and $\boldsymbol{u_3(t)}$ denotes the vaccination control, treatment control and awareness campaign control individually. We get, the system is an optimal control model.
i.e set of control variables for this system $(\boldsymbol{u_1(t), u_2(t), u_3(t)}) \in \boldsymbol{U}$.Then for Labesgue measurable we can get,

$$\boldsymbol{U} = \{(\boldsymbol{u_1(t), u_2(t), u_3(t)}): \boldsymbol{0 \leq a_i \leq u_i(t) \leq b_i \leq c_i \leq 1, i = 1, 2, 3}\};$$

$\forall \boldsymbol{t} \in [\boldsymbol{0, T}]$.

Form, these three control variables we can get now the model,

$$\boldsymbol{min J(u_1, u_2, u_3)} = \int_{\boldsymbol{0}}^{\boldsymbol{T}} \left(\boldsymbol{I(t)} + \boldsymbol{L_c(t)} + \frac{\boldsymbol{\mathcal{A}}}{\boldsymbol{2}}\boldsymbol{u_1^2} + \frac{\boldsymbol{\mathcal{B}}}{\boldsymbol{2}}\boldsymbol{u_2^2} + \frac{\boldsymbol{\mathcal{C}}}{\boldsymbol{2}}\boldsymbol{u_3^2}\right)\boldsymbol{dt}$$

We can redevelop the model as,

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$$(P_c) = \begin{cases} \min J(x,u) = \int_0^T L\big(t, x(t), u(t)\big)dt \\ \text{subject to} \\ \dot{x}(t) = f\big(x(t)\big) + g\big(x(t)\big) + h\big(x(t)\big), u(t), \forall t \in [0,T] \\ u(t) \in U(t), \forall t \in [0,T] \\ x(0) = x_0 \end{cases}$$

here, $x(t) = \begin{pmatrix} S(t) \\ E)t) \\ I(t) \\ L_c(t) \\ R(t) \end{pmatrix}$, $g(x) = \begin{pmatrix} -S & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & -L_c \\ S & L_c \end{pmatrix}$, $h(x) = \begin{pmatrix} -S & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & -L_c \\ S & L_c \end{pmatrix}$,

$$f(x) = \begin{pmatrix} r - \alpha(I + \sigma L_c)S - \mu_0 S \\ \alpha(I + \sigma L_c)S - (\mu_0 + \beta)E \\ \beta E - (\mu_0 + \mu + \gamma)I \\ (\mu + p\gamma)I - (\mu_0 + \delta + \varepsilon)L_c \\ \delta L_c + (1-p)\gamma I - \mu_0 R \end{pmatrix}$$

$u(t) = \begin{pmatrix} u_1(t) \\ u_2(t) \\ u_3(t) \end{pmatrix}$ and the integrand of the presentation index is,

$$L(x,u) = I(t) + L_c(t) + \frac{\mathcal{A}}{2}u_1^2 + \frac{\mathcal{B}}{2}u_2^2 + \frac{\mathcal{C}}{2}u_3^2.$$

3. Methods and materials

This aspect is vital for explaining the positivity and boundedness of model (1), as these terms represent the population. Positivity and boundedness in population reviews can be seen as a common constraint due to the acceptance of limited resources. In this context, we present key findings on the existence of equilibrium for model (1), positive invariance, and the boundedness of solutions.

3.1 System invariance of positivity

Reorganize model (1) in relations explained as,

$\emptyset(t) = F\big(\emptyset(t)\big)$,

Here, $\emptyset(t) = (\emptyset_1, \emptyset_2, \emptyset_3, \emptyset_4, \emptyset_5)^T = (S, E, I, L_c, R)^T$,

$\emptyset(0) = (\mathbf{S(0), E(0), I(0), L_c(0), R(0)})^{\mathbf{T}} \in \mathbf{R_+^5}$

$$F(\emptyset) = \begin{pmatrix} F_1(\emptyset) \\ F_2(\emptyset) \\ F_3(\emptyset) \\ F_4(\emptyset) \\ F_5(\emptyset) \end{pmatrix} = \begin{pmatrix} r - \alpha(I + \sigma L_c)S - (\mu_0 + u_1)S \\ \alpha(I + \sigma L_c)S - (\mu_0 + \beta)E \\ \beta E - (\mu_0 + \mu + \gamma)I \\ (\mu + p\gamma)I - (\mu_0 + \delta + \varepsilon + u_2 + u_3)L_c \\ (\delta + u_2 + u_3)L_c - \mu_0 R + (\gamma - p\gamma)I + u_1 S, \end{pmatrix}$$

The condition is stress-free because of the shape $F_i(\emptyset)|_{\emptyset_i} \geq 0, i = 1, \ldots, 5$. As said by the famous result of Nagumo, model (1) solution with an initial point $\emptyset_0 \in R_+^5$, say $\emptyset(t) = \emptyset(t; \emptyset_0)$, is such that $\emptyset(t) \in R_+^5 \; \forall \, t > 0$.

3.2 Boundedness

Theorem 1:
There exists a positive $\emptyset$ for nonzero for all solutions meet $\emptyset > \big(\boldsymbol{S(t), E(t), I(t), L_c(t), R(t)}\big)$ for the extended time t.

Proof:

Completely model (1) solutions are greater than zero, In first compartment of (1) as

$$\frac{\boldsymbol{dS(t)}}{\boldsymbol{dt}} = \boldsymbol{r - \alpha(I + \sigma L_c)S - (\mu_0 + u_1)S \leq r - (\mu_0 + u_1)S}.$$

Now, $\frac{\boldsymbol{dS(t)}}{\boldsymbol{dt}} < \boldsymbol{1 + \frac{r}{\mu_0 + u_1}}$ for extreme time $\boldsymbol{t}$,
Let, $\boldsymbol{t > t_0}$.

Then we get, $\boldsymbol{R_1(t) = \big(S(t), E(t), I(t), L_c(t)\big)}$.

Now differentiating $\boldsymbol{R_1}$ with respect to the model (1) solutions we get,

$$\begin{aligned}\frac{\boldsymbol{dR_1(t)}}{\boldsymbol{dt}} &= \boldsymbol{-(\mu_0 + u_1)S - \mu_0 E - (\mu_0 + \mu + \gamma)I - (\mu_0 + \delta + \varepsilon)L_c + r} \\ &\leq \boldsymbol{hR_1(t) + r},\end{aligned}$$

Here, $\boldsymbol{h = min\,((\mu_0 + u_1), \mu_0, (\mu_0 + \mu + \gamma), (\mu_0 + \delta + \varepsilon))}$.

Then we get,
$\boldsymbol{S(t) \leq 1 + \frac{r}{\mu_0 + u_1}}$ for $\boldsymbol{t > t_0}$.
$\boldsymbol{\emptyset_1}$ exists, depends on the model (1), So we can say $\boldsymbol{R_1(t) \leq \emptyset_1}$, finally $\boldsymbol{t \geq t_0, E(t), I(t)}$ and $\boldsymbol{L_c(t)}$ are bounded above.
Afterward the 3rd & 4th equations of model (1) and $\boldsymbol{R(t)}$ are ultimately bounded above, where $\emptyset$ is maximum.
Fig. 5 previously states that retain an appreciation on now & proven. This displays that model (1) is critical.

$$\mathrm{C} = \{\boldsymbol{(S, E, I, L_c, R)} | \boldsymbol{1 + \frac{r}{\mu_0 + u_1} \geq S \geq 0, I \geq 0, \emptyset \geq R}\}.$$

Clearly, C is convex.

4. Optimal control presence

To establish the existence of the optimal control, it is imperative to demonstrate the presence of both the state and the objective functional.

4.1 Presence of the state variable

The initial condition, along with state equation (1), can be expressed in the following manner,

$$\begin{aligned}
\boldsymbol{S'(t)} &= \boldsymbol{r - \alpha(I + \sigma L_c - \mu_0)S + (0)E(t) + (0)I(t) + (0)L_c(t) + (0)R(t)} \\
\boldsymbol{E'(t)} &= \boldsymbol{\alpha(I + \sigma L_c)S - (\mu_0 + \beta)E + (0)I(t) + (0)L_c(t) + (0)R(t)} \\
\boldsymbol{I'(t)} &= \boldsymbol{(0)S(t) + \beta E + (0)I(t) + (0)L_c(t) + (0)R(t)} \\
\boldsymbol{L_c'(t)} &= \boldsymbol{(0)S(t) + (0)E(t) + (\mu + p\gamma)I - (\mu_0 + \delta + \epsilon)L_c + (0)R(t)} \\
\boldsymbol{R'(t)} &= \boldsymbol{(0)S(t) + (0)E(t) + (1 - p)\gamma I + \delta L_c - \mu_0 R}
\end{aligned} \tag{2}$$

We know that for total population size $\boldsymbol{N(t)}$ we can get for this system,

$$\boldsymbol{N(t) = S(t) + E(t) + I(t) + L_c(t) + R(t)}$$

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Differentiating this system with respect to t we get,

$$\boldsymbol{N'(t) = S'(t) + E'(t) + I'(t) + L_c'(t) + R'(t)} \quad (3)$$

Substituting the right-hand side of equation (2) into equation (3), we obtain

$$\boldsymbol{N'(t) = r - \varepsilon L_c - \mu_0 N(t)}$$
$$\boldsymbol{\Rightarrow N'(t) + \varepsilon L_c = r - \mu_0 N(t)}$$
$$\boldsymbol{i.e\ N'(t) \leq r - \mu_0 N(t)}$$

From this we get,

$$\boldsymbol{N(t) \leq \frac{r}{\mu_0} + \left(N_0 - \frac{r}{\mu_0}\right) e^{-\mu_0 t} = K_1 \in R_+} \text{ and}$$
$$\boldsymbol{\lim_{t\to\infty} sup\, N(t) \leq K_1}$$

This leads to the following conclusion,

$$\boldsymbol{S(t), E(t), I(t), L_c(t), R(t) \leq K_1} \text{ as } \boldsymbol{t \to \infty}.$$

Subsequently, we can rewrite equation (2) in the following manner:

$$\boldsymbol{\omega_t = T\omega + E(\omega)} \quad (4)$$

Where, $\boldsymbol{\omega} = \begin{pmatrix} S(t) \\ E(t) \\ I(t) \\ L_c(t) \\ R(t) \end{pmatrix}$, $\boldsymbol{\omega_t} = \begin{pmatrix} S'(t) \\ E'(t) \\ I'(t) \\ L_c'(t) \\ R'(t) \end{pmatrix}$, $\boldsymbol{E(\omega)} = \begin{pmatrix} -\alpha(I+\sigma L_c)S \\ \alpha(I+\sigma L_c)S \\ 0 \\ 0 \\ 0 \end{pmatrix}$ and

$$\boldsymbol{T} = \begin{bmatrix} -\mu_0 & 0 & 0 & 0 & 0 \\ 0 & -(\mu_0+\beta) & 0 & 0 & 0 \\ 0 & \beta & -(\mu_0+\mu+\gamma) & 0 & 0 \\ 0 & 0 & (\mu+p\gamma) & -(\mu_0+\delta+\varepsilon) & 0 \\ 0 & -\mu_0 & 0 & (1-p)\gamma & \delta \end{bmatrix}$$

$$\boldsymbol{E(\omega_1) - E(\omega_2)} = \begin{pmatrix} -\alpha\left(I_1+\sigma L_{c_1}\right)S_1 \\ \alpha\left(I_1+\sigma L_{c_1}\right)S_1 \\ 0 \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} -\alpha\left(I_2+\sigma L_{c_2}\right)S_2 \\ \alpha\left(I_2+\sigma L_{c_2}\right)S_2 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Equation (4) represents a nonlinear system with coefficients that are bounded, so we get

$$\boldsymbol{Q(\omega) = \omega_t = T\omega + E(\omega)}.$$

To establish the existence of optimal control and the optimality system, it is necessary to ensure that the solution of the system remains bounded for a finite time. We, adopt that for $\boldsymbol{u \in U}$, there present a bounded solution.

$$\boldsymbol{|E(\omega_1) - E(\omega_2)| = \left|-\alpha\left(I_1+\sigma L_{c_1}\right)S_1 + \alpha\left(I_2+\sigma L_{c_2}\right)S_2\right| + \left|\ \alpha\left(I_1+\sigma L_{c_1}\right)S_1 - \ \alpha\left(I_2+\sigma L_{c_2}\right)S_2\right|}$$
$$\boldsymbol{\leq 2\alpha(|S_1||I_1 - I_2| + |S_1 - S_2|\left|I_2+\sigma L_{c_1}\right| + |\sigma S_2|\left|L_{c_1} - L_{c_2}\right|)}$$
$$\boldsymbol{\leq M|\omega_1 - \omega_2|}$$

here $\boldsymbol{M = 2gK_1}$.
So, we get $\boldsymbol{|Q(\omega_1) - Q(\omega_2)| \leq \|T\||\omega_1 - \omega_2| + M|\omega_1 - \omega_2| \leq K|\omega_1 - \omega_2|}$,
Where $\boldsymbol{K = max(M, \|T\|)} < \infty$.

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Thus, it surveys that the function $\boldsymbol{Q}$ is uniformly Lipschutz continuous. Given the definition of the control $\boldsymbol{U(t)}$ and the constraints on $\boldsymbol{(S, E, I, L_c)}$; $\boldsymbol{R \geq 0}$ we can infer that a solution to the system (4) exists.

4.2 Presence of objective function

To prove the existence of the objective functional, we can apply the following theorem.

Theorem 2:

Let us consider the following, $\bar{\mathfrak{X}}(t) = \begin{bmatrix} \mathfrak{X}_1(t) \\ \vdots \\ \mathfrak{X}_n(t) \end{bmatrix}$

Where $\boldsymbol{n}$ is the system of state variables, and let us consider $\boldsymbol{u(t)}$ be a control variable by a set of acceptable controls $\boldsymbol{U}$, that satisfy the following differential equation $\mathfrak{X}_i'(t) = g\big(t, \mathfrak{X}_i(t), u(t)\big)$ for $i = 1, 2, \ldots\ldots n$ with the related index.

$$J(u) = \int f(t, \bar{\mathfrak{X}}(t), u(t))\, dt$$

There, presence an optimal control which minimizes $\bar{J}(u)$ if following conditions are satisfied:

(i) $\mathcal{F}$ is a non-empty set.
(ii) The control set $\boldsymbol{U}$ is closed.
(iii) And, the control set $\boldsymbol{U}$ is convex.
(iv) The right-hand side of the state system is continuous, bounded above by a linear amalgamation of the control and state variables, and can be expressed as a linear function with coefficients dependent on time and state.
(v) The cohesive of the objective functional is a convex set on $\boldsymbol{U}$. It is bounded underneath by $-D_2 + D_1(u)^{\eta}$; $D_1 > 0$ & $\eta > 0$.

Now, defining $\mathcal{F}$ as a class of $(S_0, E_0, I_0, L_{c_0}, R_0, u)$.

So, $\boldsymbol{u}$ is a piecewise function on $[0, T]$ with the control set in $\boldsymbol{U}$.

Proof (i):

Let us consider,

$$\dot{S} = \mathcal{F}_1(t, S, E, I, L_c, R), \tag{5}$$

$$\dot{E} = \mathcal{F}_2(t, S, E, I, L_c, R),$$

$$\dot{I} = \mathcal{F}_3(t, S, E, I, L_c, R),$$

$$\dot{L_c} = \mathcal{F}_4(t, S, E, I, L_c, R),$$

$$\dot{R} = \mathcal{F}_5(t, S, E, I, L_c, R),$$

Where $\mathcal{F}_1, \mathcal{F}_2, \mathcal{F}_3, \mathcal{F}_4$ and $\mathcal{F}_5$ build up the righthand side of system below,

$$\frac{dS}{dt} = r - \alpha(I + \sigma L_c)S - (\mu_0 + u_1)S$$

$$\frac{dE}{dt} = \alpha(I + \sigma L_c)S - (\mu_0 + \beta)E$$

$$\frac{dI}{dt} = \beta E - (\mu_0 + \mu + \gamma)I$$

$$\frac{dL_c}{dt} = (\mu + p\gamma)I - (\mu_0 + \delta + \varepsilon + u_2 + u_3)L_c$$

$$\frac{dR}{dt} = (\delta + u_2 + u_3)L_c - \mu_0 R + (\gamma - p\gamma)I + u_1 S$$

Let us consider $\boldsymbol{u(t) = D}$ for some constant $\boldsymbol{D}$.

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Now, $\mathcal{F}_1, \mathcal{F}_2, \mathcal{F}_3, \mathcal{F}_4$ and $\mathcal{F}_5$ functions must adhere to linearity and exhibit continuity throughout. Additionally, their partial derivatives must be accounted for $\mathcal{F}_1, \mathcal{F}_2, \mathcal{F}_3, \mathcal{F}_4$ and $\mathcal{F}_5$ with respect to altogether all states are constants. they are also continuous universally.

According to the aforementioned theorem, there is a sole solution that exists. Now,

$S = \Omega_1(t), E = \Omega_2(t), I = \Omega_3(t), L_c = \Omega_4(t)$ and $R = \Omega_5(t)$

According to the aforementioned theorem, there is a sole solution that exists.

The solution that satisfies the initial conditions is thus established. Consequently, the collection of controls and corresponding state variables is not void. Therefore, condition (i) is fulfilled.

Proof (ii):

From definition, U is closed.

Now we yield three controls,

$(u_1, u_2, u_3) \in U$ and $\lambda \in [0, 1]$ such that $0 \leq \lambda u_1 + 2\lambda u_2 + (1 - 3\lambda)u_3$.

Now we see that $\lambda u_1 \leq \lambda$, $2\lambda u_2 \leq \lambda$, and $(1 - 3\lambda)u_3 \leq (1 - 3\lambda)$.

So, $\lambda u_1 + 2\lambda u_2 + (1 - 3\lambda)u_3 \leq \lambda + 2\lambda + (1 - 3\lambda) = 1$.

Hence, we get,

$0 \leq \lambda u_1 + 2\lambda u_2 + (1 - 3\lambda)u_3 \leq 1$

$\forall (u_1, u_2, u_3) \in U$ and $\lambda \in [0, 1]$.

So, condition (ii) is fulfilled.

Proof (iii):

Let,

$\mathcal{F}_1 \leq r - u_1 S$

$\mathcal{F}_2 \leq \mathcal{K}_1 E$

$\mathcal{F}_3 \leq \beta E - \mathcal{K}_2 I$

$\mathcal{F}_4 \leq \mathcal{K}_3 I - P_1 L_c - u_2 L_c - u_3 L_c$

$\mathcal{F}_5 \leq \delta L_c + P_2 I + u_1 S + u_2 L_c + u_3 L_c$

Then, form system (5) we get,

$$\bar{\mathcal{F}}(t, \bar{\mathfrak{X}}, u) \leq \bar{m}\left(t, \begin{bmatrix} S \\ E \\ I \\ L_c \\ R \end{bmatrix}\right)\bar{\mathfrak{X}} + \bar{n}\left(t, \begin{bmatrix} S \\ E \\ I \\ L_c \\ R \end{bmatrix}\right)u(t),$$

Where $\bar{m}\left(t, \begin{bmatrix} S \\ E \\ I \\ L_c \\ R \end{bmatrix}\right) = \begin{bmatrix} -S & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & -L_c \\ S & L_c \end{bmatrix}$

This results in a linear function of the control u, defined in terms of time and state variables. Next, we can determine the bound of the right-hand side. Notably, all parameters are constant and non-negative.

Given that, $\boldsymbol{S}$ and $\boldsymbol{L_c}$ are restricted, and $\boldsymbol{q}$ comprises the upper bound of the constant matrix, it follows that the right-hand side is bounded by a sum of the state and control variables. Therefore, condition (iii) is satisfied.

Proof (iv):

Let,

$\boldsymbol{L(u) = I(t) + L_c(t) + u^2}$, where $\frac{\mathcal{A}}{2}\boldsymbol{u_1^2} + \frac{\mathcal{B}}{2}\boldsymbol{u_2^2} + \frac{\mathcal{C}}{2}\boldsymbol{u_3^2} = \boldsymbol{u^2}$.

Where the three controls variables are $\boldsymbol{(u_1, u_2, u_3) \in U}$ and $\boldsymbol{0 < \varphi < 1}$.

Then we can get,

$$\boldsymbol{u_1^2 - 2u_1u_2 - 2u_2u_3 - 2u_3u_1 + u_2^2 + u_3^2 = (u_1 - u_2 - u_3)^2 \geq 0}$$

$$\boldsymbol{\Rightarrow u_1^2 + u_2^2 + u_3^2 \geq 2u_1u_2 + 2u_2u_3 + 2u_3u_1}$$

$$\boldsymbol{\Rightarrow \varphi(1-\varphi)u_1^2 + \varphi(1-\varphi)u_2^2 + \varphi(1-\varphi)u_3^2}$$

$$\boldsymbol{\geq 2\varphi(1-\varphi)u_1u_2 + 2\varphi(1-\varphi)u_2u_3 + 2\varphi(1-\varphi)u_3u_1}$$

$$\boldsymbol{i.e.\ \varphi\, L(u_1) + (1-\varphi)\, L(u_2) + \varphi(1-\varphi)L(u_3) \geq L(\varphi + (1-\varphi)u_2 + \varphi(1-\varphi)u_3}.$$

So, this satisfy, $\boldsymbol{L(u)}$ is convex on $\boldsymbol{U}$.

Now, we will prove,

$\boldsymbol{J(u) \geq -D_2 + D_1(u)^{\eta}}$ with initial conditions i.e. $\boldsymbol{\eta > 0, C_1 \geq 0}$

Here,

$$\boldsymbol{J(u) = I(t) + L_c(t) + \frac{\mathcal{A}}{2}u_1^2 + \frac{\mathcal{B}}{2}u_2^2 + \frac{\mathcal{C}}{2}u_3^2,}$$

$$\boldsymbol{J(u) = I(t) + L_c(t) + u^2}$$

[where $\boldsymbol{\frac{\mathcal{A}}{2}u_1^2 + \frac{\mathcal{B}}{2}u_2^2 + \frac{\mathcal{C}}{2}u_3^2 = u^2}$]

Now we get,

$$\boldsymbol{J(u) \geq -[I(t) + L_c(t)] + u^2}$$

$$\boldsymbol{= -D_2 + D_1u^2}$$

With initial condition $\boldsymbol{D_2 > 0}$ which rest on upper bounds of $\boldsymbol{I(t)}$ & $\boldsymbol{L_c(t)}$.
We also get, $\boldsymbol{\eta = 2 > 1, D_1 > 0}$.
Hence, the requirement (iv) is also fulfilled. So, the existence of objective functional has been recognized.

4.3. Optimal strategy with control variable

In this valuable chapter, optimal control philosophy of the Hepatitis B Virus model submission is conversed. Optimal theory for control variable that qualifies us to enterprise a strategy for the control of many kinds of infectious diseases of many kinds. Now the optimal control theory is functional to the infection model of Hepatitis B Virus. For controlling the spread of this virus in the population, we know that, there exists three control variable they are $\boldsymbol{u_1(t)}$ is vaccination control variable, $\boldsymbol{u_2(t)}$ is treatment control variable and $\boldsymbol{u_3(t)}$ is awareness campaign control rate. For these three optimal control variables $\boldsymbol{u_1(t), u_2(t)}$ and $\boldsymbol{u_3(t)}$ is complete to aid the determination. These three control variables represent three techniques for controlling the disease in the population, $\boldsymbol{u_1(t)}$ the requirement is to maintain continuity within a region, representing the administration of the drug to prevent the generation of new infected cells, $\boldsymbol{u_2(t)}$ the requirement that means treatment is helping to reduction of the construction rate of the viruses in the infected class, and lastly, $\boldsymbol{u_3(t)}$ makes the awareness campaign control rate that stated infection causing in public can get control by using education about these topic. Our main area of concentration here is to minimize the infection of Hepatitis B virus in total populations N(t) through a correct way that verves through susceptible class to infective class $\boldsymbol{S(t)}$, exposed class $\boldsymbol{E(t)}$, liver

cirrhotic latent class $\boldsymbol{L_c(t)}$, critical infection class $\boldsymbol{I(t)}$ complete the way foremost to a protecting resistant population $\boldsymbol{R(t)}$.

$$\frac{dS}{dt} = r - \alpha(I + \sigma L_c)S - (\mu_0 + u_1)S - (1 - u_1)S - (1 - u_3)S, \quad (6)$$
$$\frac{dE}{dt} = \alpha(I + \sigma L_c)S - (\mu_0 + \beta)E - (1 - u_1)E,$$
$$\frac{dI}{dt} = \beta E - (\mu_0 + \mu + \gamma)I - (1 - u_2)I,$$
$$\frac{dL_c}{dt} = (\mu + p\gamma)I - (\mu_0 + \delta + \varepsilon + u_2 + u_3)L_c - (1 - u_2)L_c - (1 - u_3)L_c,$$
$$\frac{dR}{dt} = (\delta + u_2 + u_3)L_c - \mu_0 R + (\gamma - p\gamma)I + u_1 S + (1 - u_1)S + (1 - u_3)S + (1 - u_1)E + (1 - u_2)I + (1 - u_2)L_c + (1 - u_3)L_c$$

When,

$$S(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0,\ L_c(0) \geq 0, R(0) \geq 0 \quad (7)$$

To characterize the weight constants, we take $\mathfrak{A}_1^*, \mathfrak{A}_2^*, \mathfrak{A}_3^*, \mathfrak{A}_4^*, \mathfrak{A}_5^*, \mathfrak{A}_6^*, \mathfrak{A}_7^*$ and $\mathfrak{A}_8^*$. Let for these objective function maximizations,

$$J\big(u_1(t), u_2(t), u_3(t)\big) = \int_0^t \Big\{\mathfrak{A}_1^* S(t) + \mathfrak{A}_2^* E(t) + \mathfrak{A}_3^* I(t) + \mathfrak{A}_4^* L_c(t), + \mathfrak{A}_5^* R(t) + \frac{1}{2}\mathfrak{A}_6^* u_1^2(t) + \frac{1}{2}\mathfrak{A}_7^* u_2^2(t) + \frac{1}{2}\mathfrak{A}_8^* u_3^2(t)\Big\} dt \quad (8)$$

Also, to minimize the objective functions, we have to discover the optimal control variables $\boldsymbol{u_1^*(t)}$, $\boldsymbol{u_2^*(t)}$, and $\boldsymbol{u_3^*(t)}$,
i.e.

$$J\{u_1^*(t), u_2^*(t), u_3^*(t)\} = min\{J(u_1(t), u_2(t), u_3(t)), u_1(t), u_2(t), u_3(t) \in U\}, \quad (9)$$

Conditional on the system (6).
Where the control set,
$\boldsymbol{U = \{u_1(t), u_2(t), u_3(t) \backslash u_i(t)\}}$

This set is Lebesgue measurable on $[0, 1]$ region , $0 \leq u_i(t) \leq 1, i = \{1, 2, 3\}$.

4.3.1 Presence of optimal control problem

With the aim of display the existence of the optimal control problem, we start the reference [36]. For this reason, we prove the presence of the optimal control problems, we take into thought that the control system consuming all the conditions at initial stage at time $t = 0$. Positivity invariance bounded solution to the state system and with positive initial conditions seized the existence of bounded Lebesguemeasurable control [37-39]. For this determination we take the optimal control problems in system (6) and (9). So, for these optimal control problems (6) and (9), survey of the Lagrangian and Hamiltonian is essential. The Lagrangian optimal control problem is the following equation given below:

$$Ł\{S(t), E(t), I(t), L_c(t), R(t), u_1(t), u_2(t), u_3(t)\} = \mathfrak{A}_1^* S(t) + \mathfrak{A}_2^* E(t) + \mathfrak{A}_3^* I(t) + \mathfrak{A}_4^* L_c(t), + \mathfrak{A}_5^* R(t) + \frac{1}{2}\mathfrak{A}_6^* u_1^2(t) + \frac{1}{2}\mathfrak{A}_7^* u_2^2(t) + \frac{1}{2}\mathfrak{A}_8^* u_3^2(t). \quad (10)$$

For optimize the minimal value of the Lagrangian equation, we can take Hamiltonian $\boldsymbol{H}$ then the optimal control will represent as:

$$H = Ł\big(S(t), E(t), I(t), L_c(t), R(t), u_1(t), u_2(t), u_3(t)\big) + \mathfrak{F}_1 \frac{dS(t)}{dt} + \mathfrak{F}_2 \frac{dE(t)}{dt} + \mathfrak{F}_3 \frac{dI(t)}{dt} + \mathfrak{F}_4 \frac{dL_c(t)}{dt} + \mathfrak{F}_5 \frac{dR(t)}{dt}. \quad (11)$$
…

For this equation we can get some adjoint variables $\mathfrak{F}_1(t), \mathfrak{F}_2(t), \mathfrak{F}_3(t)(t), \mathfrak{F}_4(t)$, and $\mathfrak{F}_5(t)$ with respect to the optimal control variables $u_1(t), u_2(t)$, and $u_3(t)$,

$$\mathfrak{F}_1'(t) = -\{\mathfrak{A}_1^* - \mathfrak{F}_1(\mu_0 + \alpha I + \alpha\sigma L_c + u_1 - u_1 - u_3 + 2) + \mathfrak{F}_2(\alpha I + \alpha\sigma L_c) + \mathfrak{F}_5(2 - u_1 - u_3)\}$$

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$$\mathfrak{F}_2'(t) = -\{\mathfrak{U}_2^* - \mathfrak{F}_2(\mu_0 + \varepsilon + \beta - 1) + \mathfrak{F}_3\varepsilon + \mathfrak{F}_5(1 - u_1)\},$$

$$\mathfrak{F}_3'(t) = -\{\mathfrak{U}_3^* - \alpha S(\mathfrak{F}_1 - \mathfrak{F}_2) - \mathfrak{F}_3(\mu_0 + \gamma + u_2 - 1) + \mathfrak{F}_4 p\gamma + \mathfrak{F}_5(\gamma - p\gamma - u_2 + 1)\},$$

$$\mathfrak{F}_4'(t) = -\{\mathfrak{U}_4^* - \mathfrak{F}_1(r + \sigma\alpha S) + \mathfrak{F}_2\sigma\alpha S + \mathfrak{F}_4(r - \mu_0 - \epsilon - \delta + u_2 + u_3 - 2) + \mathfrak{F}_5(\delta - u_2 + u_3)\},$$

$$\mathfrak{F}_5'(t) = -\{\mathfrak{U}_4^* - \mathfrak{F}_5\mu_0\},$$

$$u_1(t) = \left\{\frac{-\mathfrak{F}_1 S - \mathfrak{F}_2 E + \mathfrak{F}_5(E+S)}{\mathfrak{U}_6^*}\right\},$$

$$u_2(t) = \left\{\frac{-\mathfrak{F}_3 I - \mathfrak{F}_4 L_c + \mathfrak{F}_5(I+L_c)}{\mathfrak{U}_7^*}\right\},$$

$$u_3(t) = \left\{\frac{-\mathfrak{F}_1 S - \mathfrak{F}_4 L_c + \mathfrak{F}_5(S-L_c)}{\mathfrak{U}_8^*}\right\}.$$

Theorem 3:

We can get from the control system (6), the three optimal controls,
$u^*(t) = (u_1^*(t), u_2^*(t), u_3^*(t)) \in U.$

Then we get,

$$\min_{(u_1(t),u_2(t),u_3(t))\in U} J(u_1(t), u_2(t), u_3(t)) = J(u_1^*(t), u_2^*(t), u_3^*(t)).$$

Proof:

For, proving this, we can use many several techniques shown in [38]. Now all states variables are positive. So, we can say that this process will reducing the problem, And the obligatory convexness of these objective functional is explained as $u_1(t), u_2(t)$ and $u_3(t)$ is satisfied. The control variables set $u_1, u_2, u_3 \in U$ is convex and closed.

Furthermore, the integrand of the objective functional is given below

$$\mathfrak{U}_1^* S(t) + \mathfrak{U}_2^* E(t) + \mathfrak{U}_3^* I(t) + \mathfrak{U}_4^* L_c(t), + \mathfrak{U}_5^* R(t) + \frac{1}{2}\mathfrak{U}_6^* u_1^2(t) + \frac{1}{2}\mathfrak{U}_7^* u_2^2(t) + \frac{1}{2}\mathfrak{U}_8^* u_3^2(t)$$

This objective functional is convex proceeding the control set U, which implies the proof. Now for our planned control problem, we excavated an optimal solution. We can also use the Pontryagin maximum principle [39] for finding solution to our control problem. By using this principle, we can get the Hamiltonian,

$$H = Ł\big(S(t), E(t), I(t), L_c(t), R(t), u_1(t), u_2(t), u_3(t)\big) + \mathfrak{F}_1\frac{dS(t)}{dt} + \mathfrak{F}_2\frac{dE(t)}{dt} + \mathfrak{F}_3\frac{dI(t)}{dt} + \mathfrak{F}_4\frac{dL_c(t)}{dt} + \mathfrak{F}_5\frac{dR(t)}{dt}.$$

Now we get the nontrivial vector function $\mathfrak{F}(t) = (\mathfrak{F}_1(t), \mathfrak{F}_2(t), \dots, \mathfrak{F}_n(t))$ that exists.
For considering (y^*, u^*) as an optimal solution we can get the solutions,

$$\frac{dy}{dt} = \frac{\partial H(t, y, u, \mathfrak{F})}{\partial u},$$
$$0 = \frac{\partial H(t, y, u, \mathfrak{F})}{\partial u},$$
$$\mathfrak{F}'(t) = -\frac{\partial H(t, y, u, \mathfrak{F})}{\partial u}.$$

For the necessary conditions of the Hamiltonian, we can get the above results.

5. Numerical Simulation

In this section, we solve the proposed $\mathbf{SEIL_cR}$ model numerically. The model contains ten parameters, where some of which are obtained from literature and some are assumed. Graphical results are displayed using the initial values $S = 0.99, E = 0.01, I = 0, L_c = 0, R = 0$ and all the parameters showed in Table 1. The simulations are

performed with time 160 days. Dynamics of SEIL_cR model we can get firstly in Fig. 3. Then the optimal control model is simulated. For the simulations of the optimal control model (3), we first solve the optimality systems when no treatment is employed. So that we take the control variable $\boldsymbol{u_1 \neq 0}$(i.e treatment control,$\boldsymbol{u_2 = 0}$). The simulation results in the absence of treatment are shown in Figs.4-10. Also, we run the program of the optimal control model (3) when no vaccination strategy is employed. Hence, we take the control variable $\boldsymbol{u_2 \neq 0}$ (i.e vaccination control, $\boldsymbol{u_1 = 0}$) and the simulations are presented in Figs.7-10.

Table 1. Parameter specifications of model (3).

Descriptions	**Parameters**	**Values**	**Ref.**
Transmission rate	$\boldsymbol{\alpha}$	0.4	Assumed
Rate of exposed to infected	$\boldsymbol{\beta}$	0.2	Assumed
Recovery rate of infected	$\boldsymbol{\gamma}$	0.06	Assumed
Rate of infection to liver cirrhosis	$\boldsymbol{\mu}$	0.03	Assumed
Recovery rate of liver cirrhosis latent	$\boldsymbol{\delta}$	0.02	Assumed
Per heads birth rate	$\boldsymbol{r}$	0.0121	[26]
Per heads death rate	$\boldsymbol{\mu_0}$	0.95	[26]
the infectiousness of haulers qualified to critical infection	$\boldsymbol{\sigma}$	0.00693	[26]
Disease infected death rate	$\boldsymbol{\varepsilon}$	0.02	[26]
Rate of moving from convalesce to liver cirrhosis transmits	$\boldsymbol{p}$	0.25	[26]
Vaccination rate control	$\boldsymbol{u_1}$	0.1	Assumed
Treatment rate control	$\boldsymbol{u_2}$	0.1	Assumed

The dynamics of the **SEI$\boldsymbol{L_cR}$** model is presented in Fig. 3. This figure shows the behavior of susceptible, exposed, infected populations of HBV, liver cirrhosis populations and recovered individuals. This indicates the individuals of all the compartment will tend to zero except the susceptible class. This figure also showed that the susceptible class decreases with time and about 45 days it stops decreasing as the recovered individuals become re susceptible due to loss of immunity and join the class S. It is also shown that the exposed population with time. Similar results also observe in the case of infected individuals.

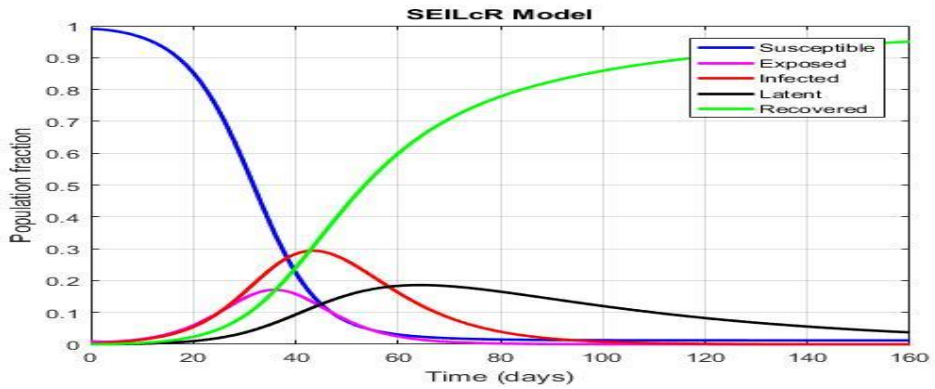

Fig.3. Dynamics of **SEI$\boldsymbol{L_cR}$** model.

Now we can get the (Fig. 3-4) for the dynamics of susceptible, exposed, Infective, liver cirrhosis latent and recovered individuals when only vaccination control ($\boldsymbol{u_1}$) is employed as optimal control by using optimal control theory.

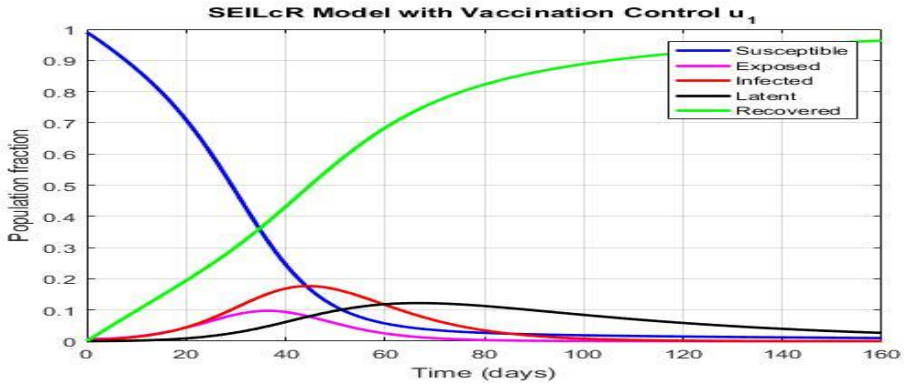

Fig. 4. Dynamics of $\mathbf{SEI}\boldsymbol{L_cR}$ model when only vaccination control ($\boldsymbol{u_1}$) is employed as optimal control.

In Fig. 4 this model provides insights into the effects of vaccination control ($\boldsymbol{u_1}$) on the spread and dynamics of a disease within the populations of susceptible, exposed, infected, liver cirrhosis latent and recovered. This model showing us that the susceptible population reduces and the recovered population recovered with vaccination control by using optimal control theory. Now when we take $\boldsymbol{u_1 \neq 0}$(i.e treatment control,$\boldsymbol{u_2 = 0}$). Then we get the Fig. 8 dynamics of susceptible and recovered populations in the presence of vaccination, providing insight into how influences the population fractions over time.

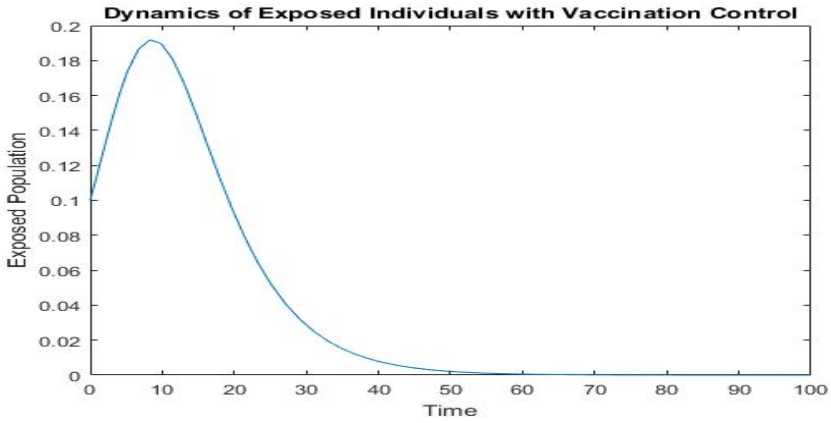

Fig. 5. Dynamics of exposed individuals when only vaccination control ($\boldsymbol{u_1}$) is employed as optimal control.

In Fig. 5 provides a more comprehensive understanding of the impact of vaccination control (i.e $\boldsymbol{u_1 \neq 0}$ and treatment control, $\boldsymbol{u_2 = 0}$) on exposed individuals within the population. It includes the effect of vaccination on reducing the exposed population and increasing the recovered population.

This Fig. 10 provides a comprehensive view of how vaccination of infected individuals affects the spread of disease and the dynamics of different population compartments.

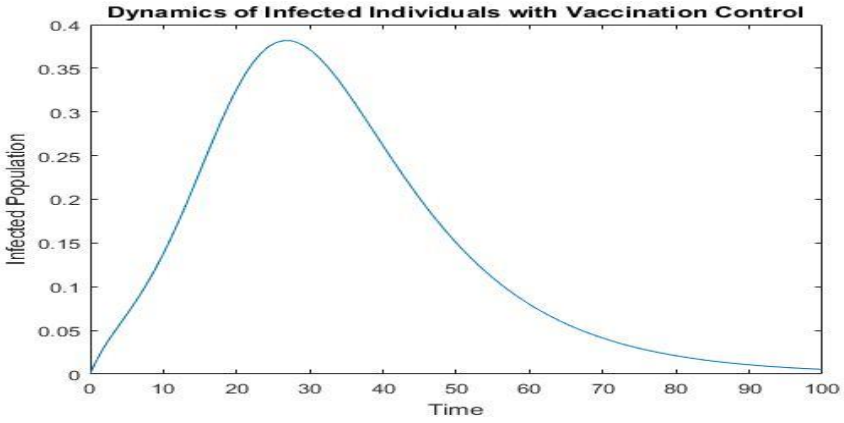

Fig. 6. Dynamics of infected individuals when only vaccination control ($\boldsymbol{u_1}$) is employed as optimal control.

This Fig. 6 provides a clear representation of the progression and control of liver cirrhosis within a population, including the impact of vaccination on thepopulation dynamics. vaccination control (i.e $\boldsymbol{u_1 \neq 0}$ and treatment control, $\boldsymbol{u_2 = 0}$) gives the liver cirrohosis control with time. The plot shows the imact of vaccination on individuals with cirrhosis.This Fig. 7 simplifies the dynamics but effectively illustrates the balance between recovery and vaccination in the context of a recovered population . This plot demonstrate the effects of constant new recoveries and reduction due to vaccination.Similarly, we can get the Fig. 8-9 for the dynamics of susceptible, exposed, Infective, liver cirrhosis latent and recovered individuals when only treatment control ($\boldsymbol{u_2}$) is employed as optimal control by using optimal control theory. Now when we take treatment control $\boldsymbol{u_2 \neq 0}$(i.e vaccination control,$\boldsymbol{u_1 = 0}$), then we can get the figures below, and this figures discuss how the disease spreads and envolves in a population considering the treatment effect on infected individuals. All the plot below discuss visual insightsinto the dynamics of each population compartment over time.

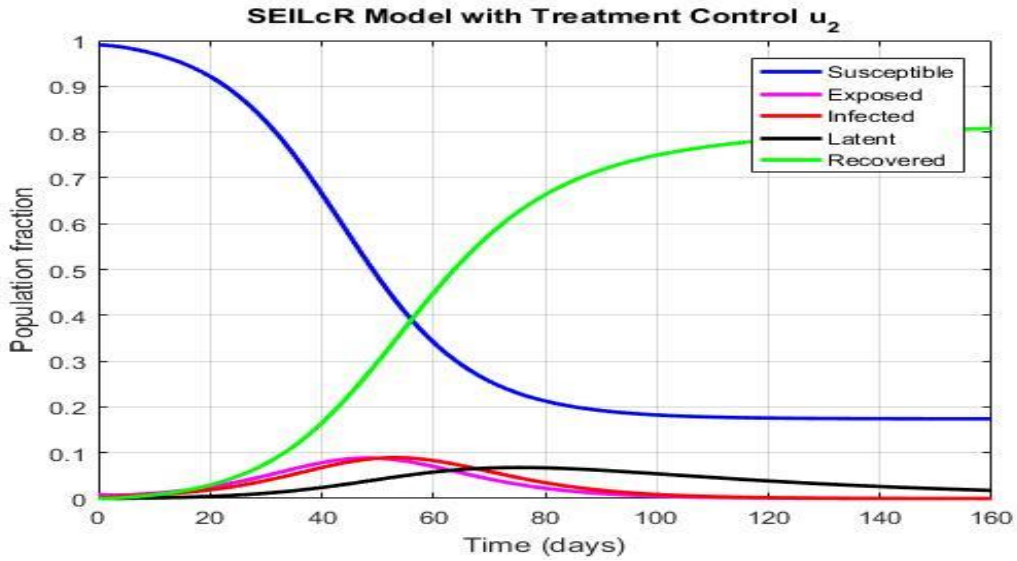

Fig. 7. Dynamics of $\mathbf{SEIL_cR}$ model when only treatment control ($\boldsymbol{u_2}$) is employed as optimal control.

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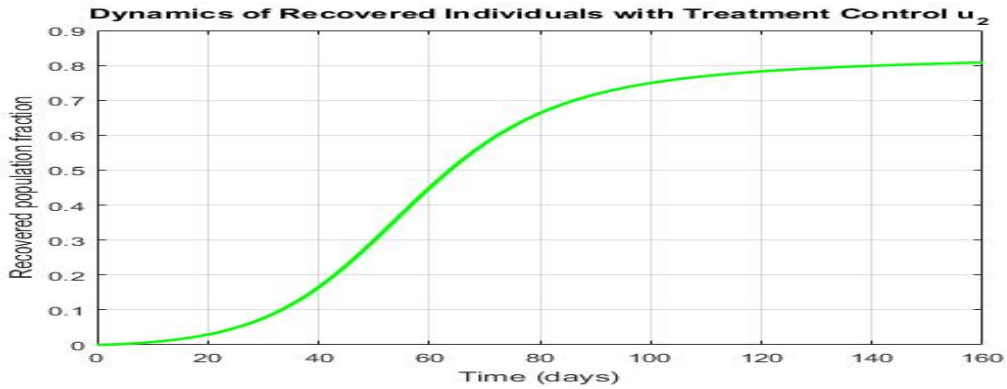

Fig. 8. Dynamics of recovered individuals when only treatment control ($\boldsymbol{u_2}$) is employed as optimal control.

Again, numerical simulations of the optimal model (2) are performed considering both the two controls: vaccination control (i.e. $\boldsymbol{u_1}$), and treatment control (i.e. $\boldsymbol{u_2}$) and also the results are shown in Fig. 8-9. Considering the vaccination and treatment controls at a great extent,$(\boldsymbol{i.e.\, u_1 = 0.1, u_2 = 0.1})$.

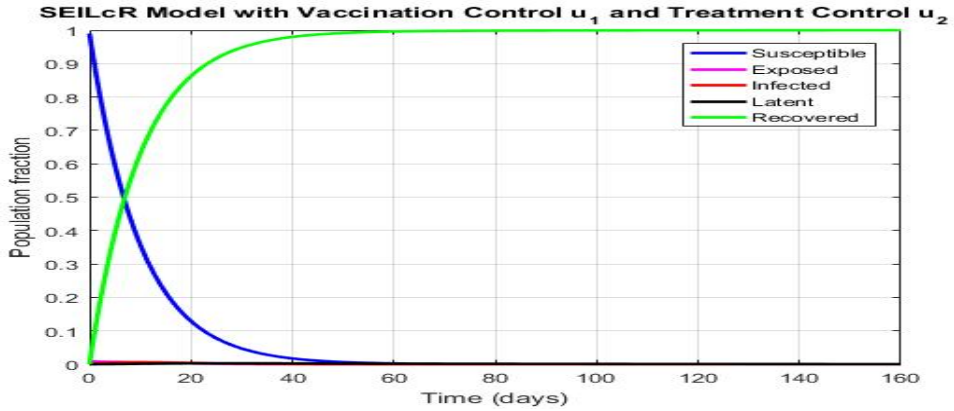

Fig. 9. Dynamics of $\mathbf{SEI}\boldsymbol{L_cR}$ model when both vaccination control ($\boldsymbol{u_1}$) and treatment control ($\boldsymbol{u_2}$) is employed as optimal control.

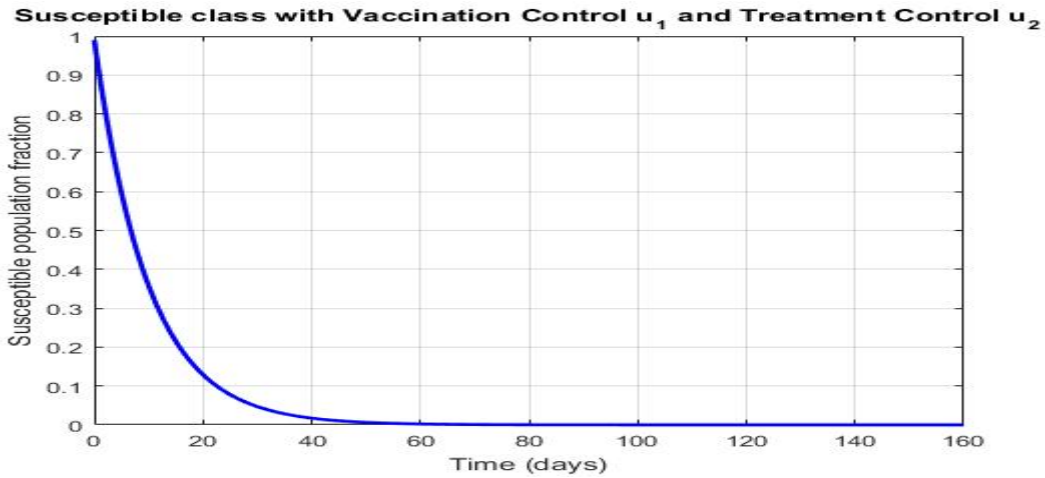

Fig. 10. Dynamics of susceptible individuals when both vaccination control ($\boldsymbol{u_1}$) and treatment control ($\boldsymbol{u_2}$) is employed as optimal control.

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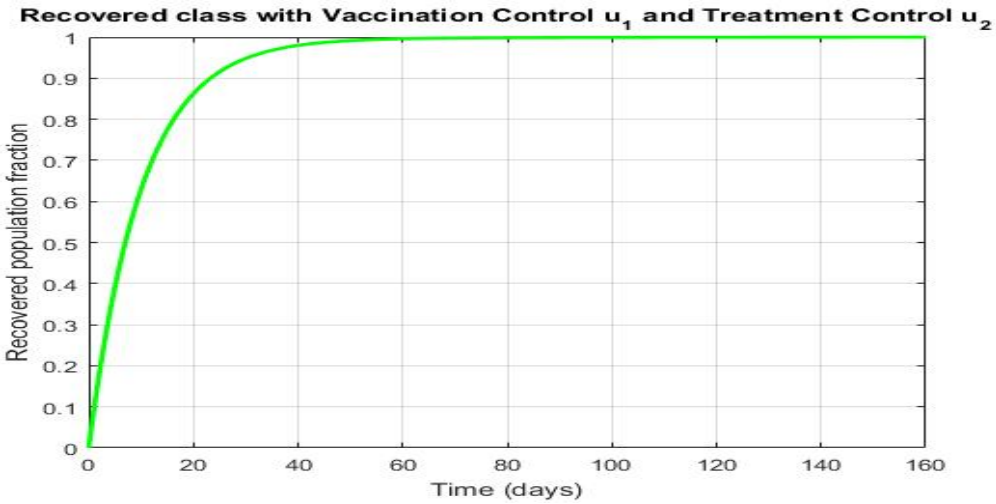

Fig. 11. Dynamics of recovered individuals when both vaccination control ($\boldsymbol{u_1}$) and treatment control ($\boldsymbol{u_2}$) is employed as optimal control.

In Figs. 7-11 represent the effects of vaccination as a control measure on the susceptible, exposed, infected, liver cirrhotic and recovered individuals for 100 days timeline from 160 days observation. It has been noticed that the control measure slightly influences the susceptible individuals, but significantly controls the exposed, infected, liver cirrhotic and recovered individuals. As expected, both the infected and liver cirrhotic individuals have increased in the absence of vaccination than the individuals with having the control measure. Here, both the infected and liver cirrhotic individuals have decreased noticeably for the presence of treatment control than the individuals without having the control measure. It has been observed that the control measure slightly influences the susceptible population, but significantly controls the exposed, infected, liver cirrhotic and recovered individuals.

5. Conclusions

This paper presents an optimal control model incorporating two control variables, developed using Pontryagin's maximum principle. Numerical simulations were conducted to validate the analytical findings. The results demonstrate that implementing optimal vaccination and treatment strategies significantly reduces the number of exposed, infected, and liver cirrhotic individuals while maximizing the number of recovered individuals and minimizing the costs associated with these interventions. Given the availability of vaccination strategies for hepatitis B a condition that often progresses to chronic liver cirrhosis—the simulations confirm that an optimal combination of vaccination and treatment is highly effective in controlling disease progression. To curb infections, it is crucial to initiate hepatitis B vaccination immediately after birth. Liver cirrhosis, a leading global cause of morbidity and mortality, impacts millions of individuals regardless of age, sex, region, or race. Addressing this life-threatening disease is an urgent global priority.

Data availability

Not applicable.

Conflict of interest: The authors declare that they have no known conflicting financial interests or personal relationships that could have influenced the work disclosed in this publication.

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