JOURNAL OF MECHANICS OF CONTINUA AND MATHEMATICAL SCIENCES
www.journalimcms.org

ISSN (Online) : 2454 -7190 , Special Issue, No.-12, August (2025) pp 34-48 ISSN (Print) 0973-8975

TRANSMISSION DYNAMICS OF HEPATITIS B MATHEMATICAL MODEL USING A FIXED POINT APPROACH

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https://doi.org/10.26782/jmcms.spl.12/2025.08.00004

(Received: May 14, 2025; Revised: July 22, 2025; Accepted: August 05, 2025)

Abstract

Hepatitis B remains a serious global health concern, affecting approximately one-third of the world's population and causing nearly one million deaths annually. The SEI_CI_AR model that distinguishes between acutely and chronically infected individuals becomes a significant addition to public health research about Hepatitis B virus transmission. This study provides rigorous insights using fixed-point theory with generalised Hyers-Ulam stability criteria to produce thorough results about solution existence, uniqueness, and stability. The model demonstrates through visualisation using the RK-5 method that proper population control measures, such as vaccination systems, transmission rate, lead populations toward the eradication of disease states. This research both enhances mathematical epidemiology and supports worldwide hepatitis B elimination programs.

Keywords: Hepatitis B Virus, Banach Contraction Principle, Fixed Point Approach, Picard Theorem, Generalised Hyers-Ulam stability.

I. Introduction:

Hepatitis B Virus (HBV) is a highly contagious infection that infects over one-third of the global population and kills nearly a million people per year. In 2024, the World Health Organisation (WHO) reports that more than 14 million individuals in the European Region are affected by chronic hepatitis B. Each year, European Centre for Disease Prevention and Control (2024), the disease leads to approximately 43,000 fatalities, primarily due to severe complications like cirrhosis and liver cancer. HBV is an infection that leads to the inflammation of the liver, and when the virus is competent to come into contact with the bloodstream and affects the liver, then it causes the infection. In the Hepatitis B infection, the virus multiplies rapidly in the liver and discharges enormous amounts of virus into the bloodstream. The Acute Hepatitis B and Chronic Hepatitis B are the two classes of HBV. Dienstag (2008), McMahon (2009), and Seto et al. (2018) focused on the Chronic Hepatitis B virus

because it often leads to serious liver disease, such as fibrosis, cirrhosis, and hepatocellular cancer. Cirrhosis or primary liver cancer are complications of chronic HBV infection.

Seto et al. (2018) and Garg and Chauhan (2024) provide papers that show that the HBV can be spread in various ways, such as it can be detected in specific body fluids or blood, and it is transferred when infected specific body fluid or blood enters the body of an uninfected individual. Sexual contact with a diseased individual, blood-to-blood products, and sometimes sharing their things like razors and brushes are all examples of how this might happen. Sometime during pregnancy or breastfeeding, mothers pass the infection to their children and many other ways.

Eliminating HBV as a public health threat requires universal screening, timely diagnosis, effective treatment, and an understanding of the virus's transmission dynamics. Mathematical modeling is crucial in this effort. Moneim and Khalil (2015) investigated the model in which infectious latent infection is present in HBV disease. The scientists also used an $\mathcal{SEIR}$, a model with a static vaccination rate, to investigate the worldwide behaviour of the disease's spread. The infectivity throughout the incubation stage is a secondary mode of transmission. There are major flaws in that study, i.e., the acute infected persons and chronic infected persons are clubbed into one compartment, which may result in the treatment not being taken into account. Then Desta and Koya (2019) introduced the modified model of $\mathcal{SEIR}$, which is entitled as $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model in which created the individual sections house for both acute and chronically sick people. In the $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model vaccine and treatment are both considered to prevent the overspread of the Hepatitis B virus.

In this research paper, the fixed point approach is used to provide the theoretical result, which is used to solve complex $\mathcal{SEIR}$ models. Fixed point approach is used to analyse the existence and uniqueness of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model. Furthermore, the generalised Hyers-Ulam stability criteria is carried out to determine the stability condition for $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model. Also, for the first time, simulation of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model is conducted using the Runge-Kutta (RK-5) method to determine the system's dynamic behaviour of every compartment of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model and displayed graphically. The simulated data show that the point of convergence is the disease-free equilibrium point (fixed point).

The structure of this paper is as follows: in section two, fundamental definitions are stated; in section three, the identified system's governing equations and some parameter values are described; in section four, the main result is presented, in which we evaluate the "existence and uniqueness" of solution with the help of fixed point approach; in section five, model's stability results is given; in section six, numerical simulation is presented and in section seven, the results are briefly summarised.

II. Preliminaries:

Here we are giving some fundamental definitions that will be used in the sequel.

Ansari (2010), Kutbi and Sintunavarat (2014), Phiangsungnoen et al. (2014), Singh and Aggarwal (2016), Khan(2019) provide the fundamental definition of metric space,

Banach contraction principle, Picard theorem, and generalised Hayer-Ulam stability can be found in many papers.

Definition 2.1: Metric space (Singh and Aggarwal (2016), Khan(2019)**)** A function $d : A^2 \to \mathbb{R}^+ \cup \{0\}$ (where $A \neq \{\emptyset\}$) is called a distance function (or metric) on A, if it meets the subsequent requirements:

1) $d(ד"_1, ד"_2) \geq 0$ if $ד"_1 \neq ד"_2$ and $ד"_1 = ד"_2 \Leftrightarrow d(ד"_1, ד"_2) = 0$,
2) $d(ד"_1, ד"_2) = d(ד"_2, ד"_1)$,
3) $d(ד"_1, ד"_3) + d(ד"_3, ד"_2) \geq d(ד"_1, ד"_2), \forall\ ד"_1, ד"_2, ד"_3 \in A.$

So the function "d" composed with a set "A" is titled as a metric space, which is denoted by (A, d).

Definition 2.2: Contraction mapping (Ansari (2010)) Let $(ב, d)$ be a metric space and $\Phi: ב \to ב$ is said to be a contraction mapping, if $\exists\ \tau \in ב$ such that

$$d\big(\Phi(ב_1), \Phi(ב_2)\big) \leq \tau d(ב_1, ב_2)\ \forall ב_1, ב_2 \in ב, \text{ where } \tau \in (0,1)$$

Theorem 2.1: "Banach Contraction Principle (B.C.P.)" (Ansari (2010)) Let $\Phi: ב \to ב$ be a contraction mapping where $(ב, d)$ be a complete metric space. Then there is a fixed point in Φ that is unique.

Theorem 2.2: Picard Theorem (Ansari (2010)) Let $\Phi(ב_1, ב_2)$ be a continuous function of two variables defined on a rectangle $Å = \{(ב_1, ב_2): \alpha \leq ב_1 \leq \beta, \gamma \leq ב_2 \leq \delta\}$ and fulfill the subsequent Lipschitz condition in the second variable:

$$|\Phi(ב_1, ב_2) - \Phi(ב_1', ב_2')| \leq \rho|ב_1 - ב_2|, \forall\ ב_2, ב_2' \in [\gamma, \delta].$$

Further, let $(ב_\alpha, ב_\beta)$ be an interior point of Å. Then the differential equation $\frac{dב_2}{dב_1} = \Phi(ב_1, ב_2)$ with the given initial condition $ב_2(ב_\alpha) = ב_\beta$ has a unique solution.

Definition 2.3: Hayers-Ulam stability (Phiangsungnoen et al. (2014)**)** Suppose $(ב, d)$ be a metric space and $\Phi: ב \to ב$ have a fixed solution $\rho = \Phi(\rho)$, if $\exists\ c > 0$ such that $\forall\ \epsilon > 0$ and $\omega' \in \Phi$ which is an ϵ-solution of fixed point, i.e., satisfy the inequality

$$d(\omega', \Phi(\omega')) \leq \epsilon$$

$\exists\ \varpi' \in \Phi$ satisfying $\rho = \Phi(\rho)$,

and

$$d(\omega', \varpi') \leq c\epsilon$$

Then $\mathcal{H}$ is called generalised Hayers-Ulam stable.

Definition 2.4: Generalised Hayers-Ulam stability(Kutbi and Sintunavarat (2014)) Suppose $(ב, d)$ be a metric space and $\Phi: ב \to ב$ has a fixed solution $\rho = \Phi(\rho)$ if $\exists$ an increasing function $\varrho: \mathbb{R}^+ \longrightarrow \mathbb{R}^+$also continuous at 0 and $\varrho(0) = 0$ such that $\forall\ \epsilon > 0$ and $\omega' \in \Phi$, which is an ϵ-solution of a fixed point, i.e., satisfying the inequality

$$d(\omega', \Phi(\omega')) \leq \epsilon$$

$\exists\ \varpi' \in \Phi$ satisfying $\rho = \Phi(\rho)$,

and

$$d(\omega', \varpi') \leq \varrho(\epsilon)$$

Then Φ is called generalised Hayers-Ulam stable.

III. Identified Mathematical Model and Parameter Values:

Desta and Koya (2019) proposed the $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model, in which the total population is distributed into five compartments, such as $\mathcal{S}$: the susceptible population, $\mathcal{E}$: the exposed population, $\mathcal{I}_{\mathcal{C}}$: the chronic infectious population, $\mathcal{I}_{\mathcal{A}}$: the acute infective population and $\mathcal{R}$: the recovered population.

The likelihood of becoming infected is unaffected by age, gender, social standing, or race. Only by coming into contact with contagious people, the susceptible person gets infected. Individuals who have been exposed are not contagious, i.e., they are incapable of transmitting the virus.

In the Susceptible population $(S(t))$, likely to get infected individuals take place (i.e., an individual is not yet infected but has a chance to get infected in the future). In the exposed population $(E(t))$, the non-infectious infected individuals take place (i.e., infected individuals who are incapable of transferring the virus). The rate of HBV transmission is determined by the rate of contact between the susceptible and the infective. So, Nana-Kyere (2017) introduced the incidence rate $\left[\frac{(\beta_1 \mathcal{I}_{\mathcal{C}} + \beta_2 \mathcal{I}_{\mathcal{A}})}{\mathcal{N}}\right]$ is used in the growth of the exposed population. The infectious individuals are classified as chronic population $(C(t))$ and acute population $(A(t))$. The acute infections are not treated with antiviral drugs, but they may be cured in some cases due to natural immunity. On the other hand, chronically infected people can transmit the disease. However, a chronic group of people requires medical treatment. The Recovered population $\left(R(t)\right)$is recovered from the infection.

The symbolizations and characterisation of the variables/ constants utilised in the $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model are listed in Table 1.

Table 1: The symbolizations and characterisation of the variables/ constants utilised in the $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model equations

Variables/ Constants	Characterization
$\mathcal{N}$(t)	Global population size
β_1	Rate of transmission from $\mathcal{S}$ to $\mathcal{I}_{\mathcal{C}}$
β_2	Rate of transmission from $\mathcal{S}$ to $\mathcal{I}_{\mathcal{A}}$
ς	Rate of recruitment
θ	Death rate in $\mathcal{I}_{\mathcal{C}}$
γ_1	Treatment rate in $\mathcal{I}_{\mathcal{C}}$
γ_2	Recovered rate of $\mathcal{I}_{\mathcal{A}}$
c_1	Transfer rate from $\mathcal{E}$ to $\mathcal{I}_{\mathcal{C}}$
c_2	Transfer rate from $\mathcal{E}$ to $\mathcal{I}_{\mathcal{A}}$
η	Rate of natural death
α	Death rate in $\mathcal{I}_{\mathcal{A}}$
ζ	Rate of vaccination

In the flow chart, all the compartments and flow direction of their input and output parameters are indicated, where parameters include recruitment rate, treatment, vaccination, transfer, and death rate.

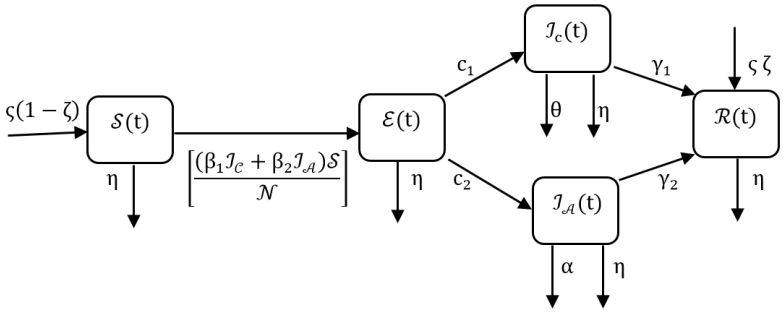

Fig. 1. Flow chart of $\mathcal{SEI_C I_A R}$ model.

The $\mathcal{SEI_C I_A R}$ mathematical model as follows:

$$\frac{d\mathcal{S}}{dt} = \varsigma(1-\zeta) - \left[\frac{(\beta_1\mathcal{I}_\mathcal{C}+\beta_2\mathcal{I}_\mathcal{A})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S} \quad (1)$$

$$\frac{d\mathcal{E}}{dt} = \left[\frac{(\beta_1\mathcal{I}_\mathcal{C}+\beta_2\mathcal{I}_\mathcal{A})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{E} - c_1\mathcal{E} - c_2\mathcal{E} \quad (2)$$

$$\frac{d\mathcal{I}_\mathcal{C}}{dt} = c_1\mathcal{E} - \theta\mathcal{I}_\mathcal{C} - \eta\mathcal{I}_\mathcal{C} - \gamma_1\mathcal{I}_\mathcal{C} \quad (3)$$

$$\frac{d\mathcal{I}_\mathcal{A}}{dt} = c_1\mathcal{E} - \alpha\mathcal{I}_\mathcal{A} - \eta\mathcal{I}_\mathcal{A} - \gamma_2\mathcal{I}_\mathcal{A} \quad (4)$$

$$\frac{d\mathcal{R}}{dt} = \gamma_2\mathcal{I}_\mathcal{A} + \gamma_1\mathcal{I}_\mathcal{C} + \varsigma\zeta - \eta\mathcal{R} \quad (5)$$

In this model, the global population size is represented by $\mathcal{N}$. Where, $\mathcal{N} = \mathcal{S} + \mathcal{E} + \mathcal{I}_\mathcal{C} + \mathcal{I}_\mathcal{A} + \mathcal{R}$.

For demonstrating the modified $\mathcal{SEI_C I_A R}$ model is significant and well defined; all the variables need to be non-negative to establish the necessary fact. This fact is presented in this theorem.

Theorem 3.1: Positivity of the solution (Desta and Koya (2019)**)** The size of the population at any time (t) is non-negative if the model's initial population sizes are non-negative. In other words, if $\mathcal{S}(0), \mathcal{E}(0), \mathcal{I}_\mathcal{C}(0), \mathcal{I}_\mathcal{A}(0), \mathcal{R}(0) \geq 0$ then the solutions of $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}_\mathcal{C}(t), \mathcal{I}_\mathcal{A}(t), \mathcal{R}(t)$ are non-negative for all $t > 0$.

Theorem 3.2: Boundedness of the solution (Desta and Koya (2019)**)** All the solutions $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}_\mathcal{C}(t), \mathcal{I}_\mathcal{A}(t), \mathcal{R}(t)$ of modified $\mathcal{SEI_C I_A R}$ model equations (1) to (5) are bounded.

Now, any solution of modified $\mathcal{SEI_C I_A R}$ model is bounded when the global population size $(\mathcal{N}(t))$ is between $\left[0, \frac{\varsigma}{\eta}\right]$. (i.e, $0 \leq \mathcal{N}(t) \leq \frac{\varsigma}{\eta}$)

IV. Main Result: Existence and Uniqueness of the Solution of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model using Fixed Point Approach:

The main result is presented in this section, i.e., the "existence and uniqueness" of the equilibrium point of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model is presented in this section.

Theorem 4.1: Let $\mathfrak{I}'(t) = K\big(t, \mathfrak{I}(t)\big)$ with $\mathfrak{I}(0) = \mathfrak{I}_0; t > 0$ is a linear differential equation where $K\big(t, \mathfrak{I}(t)\big)$ is a function depending on t and $\mathfrak{I}(t)$, where t is the time variable and $\mathfrak{I}(t)$ is another function of various variables depending on $\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}_{\mathcal{C}}(t), \mathcal{I}_{\mathcal{A}}(t), \mathcal{R}(t)$.

The following suppositions are true for the existence of the solution.

(P1) $K(t, \mathfrak{I})$ is a continuous function on a compact subset $A \subseteq \mathbb{R}^2$. So $\exists$ constant $k > 0$ s.t.,

$$|K(t, \mathfrak{I})| \leq k;\ \forall\ (t, \mathfrak{I}) \in \left[0, \frac{\varsigma}{\eta}\right] = A$$

(P2) There exists constant $\alpha > 0$ such that $\forall\ \mathfrak{I}_1(t), \mathfrak{I}_2(t)$ satisfy the Lipschitz condition, i.e.,

$$\big|K\big(t, \mathfrak{I}_1(t)\big) - K\big(t, \mathfrak{I}_2(t)\big)\big| \leq \alpha|\mathfrak{I}_1(t) - \mathfrak{I}_2(t)|$$

Further, under the assumption (P1) and (P2), the differential equation has a unique solution.

Proof: For the existence of the solution of $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model using fixed point approach, we convert equation (1) to (5) into the following integral equation using the initial conditions, we get

$$\int_0^t \mathcal{S}'ds = \int_0^t (\varsigma(1-\zeta) - \left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S})ds$$

$$\mathcal{S}(t) - \mathcal{S}(0) = \int_0^t (\varsigma(1-\zeta) - \left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S})ds$$

$$\mathcal{S}(t) = \mathcal{S}(0) + \int_0^t (\varsigma(1-\zeta) - \left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S})ds$$

$$\mathcal{S}(t) = \mathcal{S}(0) + \int_0^t K_1(s, \mathcal{S})\, ds \tag{6}$$

Where,

$$K_1(s, \mathcal{S}) = \varsigma(1-\zeta) - \left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S} \tag{7}$$

Similarly,

$$\int_0^t \mathcal{E}'ds = \int_0^t (\left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{E} - c_1\mathcal{E} - c_2\mathcal{E})ds$$

$$\mathcal{E}(t) = \mathcal{E}(0) + \int_0^t (\left[\frac{(\beta_1\mathcal{I}_{\mathcal{C}} + \beta_2\mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{E} - c_1\mathcal{E} - c_2\mathcal{E})ds$$

$$\mathcal{E}(t) = \mathcal{E}(0) + \int_0^t K_2(s, \mathcal{E})\, ds \tag{8}$$

Where,

$$K_2(s, \mathcal{E}) = \left[\frac{(\beta_1 \mathcal{I}_\mathcal{C} + \beta_2 \mathcal{I}_\mathcal{A})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{E} - c_1\mathcal{E} - c_2\mathcal{E} \tag{9}$$

Similarly,

$$\int_0^t \mathcal{I}_\mathcal{C}' ds = \int_0^t (c_1\mathcal{E} - \theta\mathcal{I}_\mathcal{C} - \eta\mathcal{I}_\mathcal{C} - \gamma_1\mathcal{I}_\mathcal{C})ds$$

$$\mathcal{I}_\mathcal{C}(t) = \mathcal{I}_\mathcal{C}(0) + \int_0^t (c_1\mathcal{E} - \theta\mathcal{I}_\mathcal{C} - \eta\mathcal{I}_\mathcal{C} - \gamma_1\mathcal{I}_\mathcal{C})ds$$

$$\mathcal{I}_\mathcal{C}(t) = \mathcal{I}_\mathcal{C}(0) + \int_0^t K_3(s, \mathcal{I}_\mathcal{C})\, ds \tag{10}$$

Where,

$$K_3(s, \mathcal{I}_\mathcal{C}) = c_1\mathcal{E} - \theta\mathcal{I}_\mathcal{C} - \eta\mathcal{I}_\mathcal{C} - \gamma_1\mathcal{I}_\mathcal{C} \tag{11}$$

Similarly,

$$\int_0^t \mathcal{I}_\mathcal{A}' ds = \int_0^t (c_1\mathcal{E} - \alpha\mathcal{I}_\mathcal{A} - \eta\mathcal{I}_\mathcal{A} - \gamma_2\mathcal{I}_\mathcal{A})ds$$

$$\mathcal{I}_\mathcal{A}(t) = \mathcal{I}_\mathcal{A}(0) + \int_0^t (c_1\mathcal{E} - \alpha\mathcal{I}_\mathcal{A} - \eta\mathcal{I}_\mathcal{A} - \gamma_2\mathcal{I}_\mathcal{A})ds$$

$$\mathcal{I}_\mathcal{A}(t) = \mathcal{I}_\mathcal{A}(0) + \int_0^t K_4(s, \mathcal{I}_\mathcal{A})\ ds \tag{12}$$

Where,

$$K_4(s, \mathcal{I}_\mathcal{A}) = c_1\mathcal{E} - \alpha\mathcal{I}_\mathcal{A} - \eta\mathcal{I}_\mathcal{A} - \gamma_2\mathcal{I}_\mathcal{A} \tag{13}$$

Similarly,

$$\int_0^t \mathcal{R}' ds = \int_0^t (\gamma_2\mathcal{I}_\mathcal{A} + \gamma_1\mathcal{I}_\mathcal{C} + \varsigma\zeta - \eta\mathcal{R})ds$$

$$\mathcal{R}(t) = \mathcal{R}(0) + \int_0^t (\gamma_2\mathcal{I}_\mathcal{A} + \gamma_1\mathcal{I}_\mathcal{C} + \varsigma\zeta - \eta\mathcal{R})ds$$

$$\mathcal{R}(t) = \mathcal{R}(0) + \int_0^t K_5(s, \mathcal{R})\, ds \tag{14}$$

Where,

$$K_5(s, \mathcal{R}) = \gamma_2\mathcal{I}_\mathcal{A} + \gamma_1\mathcal{I}_\mathcal{C} + \varsigma\zeta - \eta\mathcal{R} \tag{15}$$

Using equations (6)-(15), we can express the model in the following way[15].

$$\mathfrak{I}'(t) = K\big(t, \mathfrak{I}(t)\big) \tag{16}$$

with $\mathfrak{I}(0) = \mathfrak{I}_0; t > 0$

where,

$$\Im(t) = (\mathcal{S}(t), \mathcal{E}(t), \mathcal{I}_{\mathcal{C}}(t),\ \mathcal{I}_{\mathcal{A}}(t), \mathcal{R}(t)\ ),$$

$$\Im(0) = \Im_0 = (\mathcal{S}(0), \mathcal{E}(0), \mathcal{I}_{\mathcal{C}}(0),\ \mathcal{I}_{\mathcal{A}}(0), \mathcal{R}(0)\ )$$

and

$$K(t, \Im(t)) = \begin{pmatrix} K_1 \\ K_2 \\ K_3 \\ K_4 \\ K_5 \end{pmatrix} = \begin{pmatrix} \varsigma(1-\zeta) - \left[\frac{(\beta_1 \mathcal{I}_{\mathcal{C}} + \beta_2 \mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{S} \\ \left[\frac{(\beta_1 \mathcal{I}_{\mathcal{C}} + \beta_2 \mathcal{I}_{\mathcal{A}})\mathcal{S}}{\mathcal{N}}\right] - \eta\mathcal{E} - c_1\mathcal{E} - c_2\mathcal{E} \\ c_1\mathcal{E} - \theta\mathcal{I}_{\mathcal{C}} - \eta\mathcal{I}_{\mathcal{C}} - \gamma_1\mathcal{I}_{\mathcal{C}} \\ c_1\mathcal{E} - \alpha\mathcal{I}_{\mathcal{A}} - \eta\mathcal{I}_{\mathcal{A}} - \gamma_2\mathcal{I}_{\mathcal{A}} \\ \gamma_2\mathcal{I}_{\mathcal{A}} + \gamma_1\mathcal{I}_{\mathcal{C}} + \varsigma\zeta - \eta\mathcal{R} \end{pmatrix}$$

The solution to differential equation (16) is the same as the solution of the integral equation. Now, convert the differential equation (16) into an integral equation. we get,

$$\Im(t) - \Im(0) = \int_0^t K(s, \Im(s))\, ds$$

$$\Im(t) = \Im_0 + \int_0^t K(s, \Im(s))\, ds \qquad (17)$$

Further, we define transformation $\Phi: \mathcal{L} \to \mathcal{L}$ defined by

$$\Phi(\Im(t)) = \Im_0 + \int_0^t K(s, \Im(s)) ds$$

Now, for the uniqueness, the following steps should be taken.

Step 1: First of all, show that Φ is well-defined.

Take constant $\wp$ (positive) so that $\wp\,\alpha < 1$, and also take a rectangle B which is contained in A, where $B = \{(t, \Im): -\wp + 0 \le t \le \wp + 0, -\wp k + \Im_0 \le \Im \le \wp k + \Im_0\}$.

Let $\mathcal{L} \in \mathbb{C}(\left[0, \frac{\varsigma}{\eta}\right], \mathbb{R})$ defined on $[-\wp + 0, \wp + 0]$ such that

$$d(\Im(t), \Im_0) \le \wp k.$$

The set $\mathcal{L}$ is a closed subset of the metric space $\mathbb{C}[-\wp + 0, \wp + 0]$ with the sup metric. Since $\mathbb{C}[-\wp + 0, \wp + 0]$ is complete, then $\mathcal{L}$ is complete.

$$d(\Phi(\Im(t)), \Im_0) = \sup \left| \Im_0 + \int_0^t K(s, \Im(s)) ds - \Im_0 \right|$$

$$= \sup \left| \int_0^t K(s, \Im(s)) ds \right|$$

$$\le \int_0^t \sup |K(s, \Im(s))|\, ds$$

$$\le k(t - 0) = k\,\wp$$

$\Rightarrow \Phi(\Im(t)) \in \mathcal{L}$, so Φ is well defined.

Step 2: We have to show that Φ satisfies the self-contraction condition:

$$d\big(\Phi(\Im_1(\mathrm{t})),\Phi(\Im_2(\mathrm{t}))=\sup\left|\Im_0+\int_0^{\mathrm{t}}\mathrm{K}\big(\mathrm{s},\Im_1(\mathrm{s})\big)\mathrm{ds}\ -\varphi_0-\int_0^{\mathrm{t}}\mathrm{K}\big(\mathrm{s},\Im_2(\mathrm{s})\big)\mathrm{ds}\right|$$

$$=\sup\left|\int_0^{\mathrm{t}}\mathrm{K}\big(s,\Im_1(\mathrm{s})\big)-\mathrm{K}\big(\mathrm{s},\Im_2(s)\big)\mathrm{ds}\right|$$

$$\leq\int_0^{\mathrm{t}}\sup\left|\mathrm{K}\big(\mathrm{s},\Im_1(\mathrm{s})\big)-\mathrm{K}\big(\mathrm{s},\Im_2(s)\big)\right|\mathrm{ds}$$

$$\leq\alpha\int_0^{\mathrm{t}}|\Im_1(\mathrm{s})-\Im_2(s)|\,\mathrm{ds}$$

$$\leq\wp\,\alpha\,\mathrm{d}\big(\Im_1(\mathrm{t}),\Im_2(\mathrm{t})\big)$$

$$\Longrightarrow\ d\big(\Phi(\Im_1(\mathrm{t})),\Phi(\Im_2(\mathrm{t}))\leq\ \mathrm{m}.d\big(\Im_1(\mathrm{t}),\Im_2(\mathrm{t})\big)$$

Where, $0\leq \mathrm{m}=\wp\,\alpha<1$.

Hence, Φ is a self-contraction mapping on $\mathcal{L}$, and using Banach contraction theorem (Singh and Aggarwal (2016)), we demonstrate that Φ has a fixed point that's unique, hence $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model has a unique solution.

V. Stability Result

In this section, the stability conditions are explored, and for this, we proceed as follows.

Let transformation $\Phi:\mathcal{L}\to\mathcal{L}$ have a fixed solution

$$\Phi(\Im)=\Im,\ (\text{where},\Im\in\Phi) \tag{18}$$

Remark (1). If there exists $\epsilon_*^*(\mathrm{t})\in C\left(\left[0,\frac{\varsigma}{\eta}\right],R\right)$, then $\Im_\kappa\in\Phi$ satisfies (19) if,

i) $|\epsilon_*^*(\mathrm{t})|\leq\omega_*^*,\ \forall t>0,$
ii) $\Phi(\Im_{\mathrm{k}}(t))=\Im_{\mathrm{k}}(t)+\epsilon_*^*(\mathrm{t}),\ \forall t>0,$

For further analysis, consider the perturbed equation for the Perturbed problem (16), we have

$$\Im'(\mathrm{t})=\mathrm{K}\big(\mathrm{t},\Im(\mathrm{t})\big)+\epsilon_*^*(\mathrm{t}) \tag{23}$$

with $$\Im(0)=\Im_0;\mathrm{t}>0$$

Lemma (1): The result mentioned below holds for equation (23),

$$|\Im(\mathrm{t})-\Phi(\Im(t))|\leq\wp*\omega_*^*,\ \text{where}\ \wp=1/\alpha$$

Proof: From the remark (1) and equation (16), this result is easily followed.

Theorem (2): Under Lemma (1), the solution of the equation (16) is stable, provided

$$\frac{\wp}{(1-\wp\alpha)}<1(\text{where},\wp<1)$$

Proof: Let $\Im_\iota(t) \in \Phi$ be the unique solution of (16) and $\exists\, \Im_\kappa \in \Phi$ satisfying $\Phi(\Im_k) = \Im_k$, then,

$$d\big(\Im_\iota(t), \Im_\kappa(t)\big) = \sup|\Im_\iota(t) - \Im_\kappa(t)|$$

$$\leq \sup|\Im_\iota - \Phi(\Im_\iota)| + \sup|\Phi(\Im_\iota) - \Phi(\Im_k)|$$

$$d(\Im_\iota(t), \Im_\kappa(t)) \leq \wp + \wp\, \alpha\, d\big(\Im_l(t), \Im_k(t)\big)$$

$$d(\Im_\iota(t), \Im_\kappa(t)) - \wp\, \alpha\, d\big(\Im_l(t), \Im_k(t)\big) \leq \wp$$

$$(1 - \wp\alpha) d\big(\Im_l(t), \Im_k(t)\big) \leq \wp$$

$$\Rightarrow \quad d\big(\Im_l(t), \Im_k(t)\big) \leq \frac{\wp}{(1-\wp\alpha)}$$

Showing that the problem (16) is UH stable, it is also GUH stable, by defining

$$\Rightarrow \quad \varrho(\epsilon_*^*) = \frac{\wp}{(1-\wp\alpha)} \text{ with } \varrho(0) = 0.$$

Then, Φ is called Generalised Hayers-Ulam stable and Hayers-Ulam stable.

VI. Validation of Theoretical Approach using Numerical Simulation:

For the qualitative characteristics of parameters, legitimate values that are biologically plausible are assigned for simulation purposes. The numerical methodology is developed with the help of the Runge-Kutta (RK-5) method (Hussain 2020, Minggi et al. 2021) to solve the $\mathcal{SEI}_\mathcal{C}\mathcal{I}_\mathcal{A}\mathcal{R}$ model. For analysing the qualitative behaviour of the model, a list of all the desired parameters is given in Table 2. In addition, the size of the initial population is considered $\mathcal{S}(0) = 5000, \mathcal{E}(0) = 4000, \mathcal{I}_\mathcal{C}(0) = 3000,\ \mathcal{I}_\mathcal{A}(0) = 2000, \mathcal{R}(0) = 1000$. The source for the parameters is Desta and Koya (2019) proposed $\mathcal{SEI}_\mathcal{C}\mathcal{I}_\mathcal{A}\mathcal{R}$ model.

Table 2: List of desired parameters for $\mathcal{SEI}_\mathcal{C}\mathcal{I}_\mathcal{A}\mathcal{R}$ model's simulation.

Parameters	Values
β_1	0.017
β_2	0.017
ς	2000
θ	0.025
γ_1	0.025
γ_2	0.024
c_1	0.3
c_2	0.2
η	0.03
α	0.015
ζ	0.4

Consequently, we get some graphs from the given data that are shown in figures 2-6, where the behaviour of $\mathcal{S}(\mathrm{t}), \mathcal{E}(\mathrm{t}), \mathcal{I}_{\mathcal{C}}(\mathrm{t}),\ \mathcal{I}_{\mathcal{A}}(\mathrm{t})$ and $\mathcal{R}(\mathrm{t})$ are given. We consider the time interval [0,200]. The result of the simulation is described along with the stated objective of controlling the spread of the virus, which is to reduce the size of the exposed, chronic, and acute populations and increase the size of the recovered population.

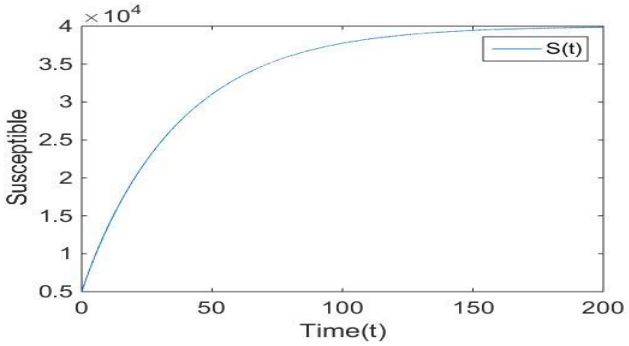

Fig. 2. The solution behaviour of the susceptible population in $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model.

Based on Figure 2, it can be seen that the size of the susceptible population is constantly increasing up to $t = 200$.

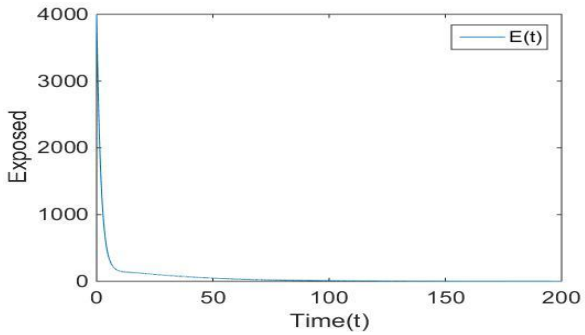

Fig. 3. The solution behaviour of the exposed population in $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model.

Based on Figure 3, it can be seen that the size of the exposed population dropped drastically till $t = 60$, then after $t = 60$ the size of the exposed population in an equilibrium state is 0, which means that after $t = 60$ the model reaches a disease eradication stage.

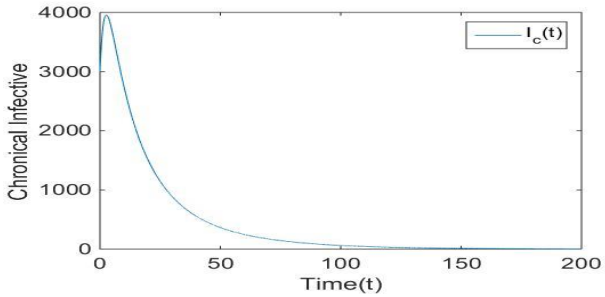

Fig. 4. The solution behaviour of the chronically infected population in $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model.

Based on Figure 4, it can be seen that the size of the chronically infected population dropped drastically till $t = 120$, then after $t = 120$ the size of the chronically infected population is in an equilibrium state is 0, which means after $t = 120$ the chronic class of the model reaches a disease eradication stage.

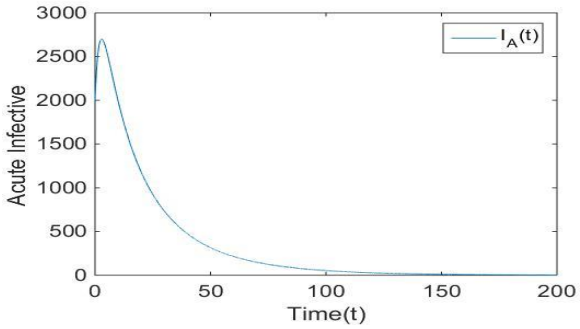

Fig. 5. The solution behaviour of the acute infected population in $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model.

Based on Figure 5, it can be seen that the size of the acute infected population dropped drastically till $t = 125$, then after $t = 125$ the size of the acute infected population iapproaches an equilibrium state is 0, which means after $t = 125$ the class of acute infectives reaches the disease-free state.

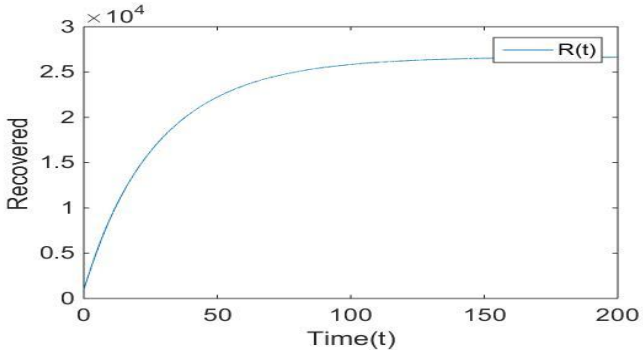

Fig. 6. The solution behaviour of the recovered population in $\mathcal{SEI}_{\mathcal{C}}\mathcal{I}_{\mathcal{A}}\mathcal{R}$ model.

Based on Figure 6, it can be seen that the size of the recovered population constantly increases until $t = 80$, and then it begins to balance with the number of recovered individuals.

VII. Discussion and Conclusion

This study enhances HBV dynamics comprehension by deploying a fixed-point solution in the advanced SEI_CI_AR population model for acute and chronic population representation. The implementation of fixed-point methodology provides researchers with better tools to prove the existence and uniqueness of solutions.

The Theoretical results Theorem 4.1 shows that if the linear combination of all parameters in each compartment lies between $0 \leq \wp\alpha < 1$, then all compartments approach their equilibrium state. All the parameters are considered according to the theoretical conditions to visualize that under the considered condition, all compartments are attaining their equilibrium state. The mathematical model evaluation runs smoothly through this approach because it solves both the challenges of disease transmission non-linearities and the simplification of mathematical calculations.

Research utilises generalised Hyers-Ulam stability criteria to ensure model solutions preserve behavior during minor disturbances because this stability aspect significantly impacts long-term systems analysis. The stability framework proves that both theoretical and real-world conditions support the existence of a disease-free equilibrium, making the model reliable for predictions. The RK-5 method serves as the base for simulating dynamic patterns from the proposed model. The study generates visual outputs from MATLAB numerical simulations showing how each population segment (susceptible, exposed, and infected acute, infected chronic, and recovered) changes temporally during the simulation period.

The simulation outcomes demonstrate how the population moves toward becoming disease-free, which indicates that particular assumptions with proper control methods can eliminate the disease or reduce it to minimal levels. The population compartments converge to this fixed point to verify that properly designed interventions like static vaccination can deliver sustained disease management results.

Researchers who evaluate infectious disease models can use well-established mathematical concepts that merge fixed-point analyses with stability measures because of their powerful RK-5 computational technique. These dual benefits result from the research because it expands the mathematical epidemiological knowledge base and enhances Hepatitis B transmission control outcomes in public health systems. Public health delivers optimal solutions through research findings only when supported by actual epidemiological data, combined with theoretical modeling along computational simulations.

VIII. Acknowledgement

The authors sincerely thank DST-Inspire and Chandigarh University for providing support and the necessary facilities for this study.

Conflicts of Interest

This work does not have any conflicts of interest.

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