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Modeling Hepatitis C Transmission to Inform Public Health Strategies and Long-Term Control

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Abstract: The majority of people today would suffer and pass away from the hepatitis C virus (HCV); there is little knowledge of the illness in the world. Although many individuals have HCV characteristics and are affected, several individuals do not genuinely believe that this is a major issue. They are simply visiting the hospital to get short-term relief from symptoms like fatigue, nausea, jaundice; in long-term situations, they may experience fluid accumulation in the belly and easily bruise. Later on, it will develop into a chronic illness that causes liver cancer, liver failure, and scarring of the liver (cirrhosis). Since HCV continues to be a major cause of death among the populations, we established a compartmental framework for the nationwide outbreak in the current research, classifying those infected into two sections with the most effective control. To get the fundamental reproduction number, first, we employed the Next-Generation Matrix method to identify the model's endemic and disease-free equilibrium point. Using infected and disease-free equilibrium points with reproduction number coordination, as well as MATLAB software to simulate the model's numerical equations, the local and global stability were analysed.

Keywords: Hepatitis C Virus; Mathematical Modeling; Stability; Equilibrium Points; Reproduction Number

1. Introduction

The hepatitis C virus (HCV) was identified by the Chiron group (Choo, Kuo, Houghton) in the 1980s. Blood transfusions were a major factor in spreading HCV. A watery acute hepatitis outbreak in Kashmir, India, raised the first suspicion of hepatitis E. Hepatitis C is a virus that mainly affects the liver and can cause both short-term and long-term health problems. The HCV causes it and is a major health issue worldwide because serious issues, including liver cancer, liver failure, and scaring of the liver (cirrhosis), can result from it, so preventing it and detecting it early are very important for controlling its spread. Effective vaccines for hepatitis A and B are available, significantly reducing the incidence of these diseases. Advances in treatment for hepatitis C have led to high cure rates, making it a potentially curable chronic disease. Research continues to focus on understanding the natural history of hepatitis B and C, developing new therapies, and preventing transmission. The majority of hepatitis A patients recover without medical assistance because it is an acute condition. Hepatitis B or hepatitis C infections that are persistent and long-lasting can cause chronic liver damage.

It's transmitted from mother to child after childbirth or by intimate relations, sharing of needles, or contact with contaminated blood or body fluids. Elaiw et al. [1] noted that liver inflammation is a key feature of hepatitis and is primarily caused by the hepatitis B virus (HBV) and hepatitis C virus (HCV), which mainly target liver cells. To improve treatment strategies for these co-infections, researchers have developed mathematical models to understand better the interaction between the two viruses and the immune response, particularly the role of CTL's. Enhancing CTL activation rates increases the number of healthy hepatocytes while reducing the levels of both HCV and HBV, ultimately improving liver function. Abiodun et al. [2] stated that by reducing the reproduction number of co-infection, treating HCV first can lower the risk of liver cancer in individuals with both HBV and HCV. Simulation results suggest that HCV treatment may also significantly reduce the negative impact of HCV on the HIV/AIDS epidemic.

Churkin et al. [3] mentioned that clinical research patient data and *in-silico* patient data were integrated to create a precise machine learning method for predicting the time to cure for hepatitis C. Carvalho et al. [4] examined the prevalence of HIV viremia and the effectiveness of therapy in relation to the severity of HIV/HCV co-infection patterns. It has also been discovered that therapy effectiveness influences the natural progression of HCV in HIV/HCV co-infection. Artenie et al. [5] and Pitcher et al. [6] emphasized the importance of economic evaluations to determine the budgetary impact, cost-effectiveness, and optimal impact of HCV eradication initiatives to improve the global public health response are necessary in conjunction with specific expenditures to increase HCV prevention and treatment among drug injectors, given the findings indicating limited coverage.

Hepatitis C can be hard to identify in its early stages since it frequently goes years without showing any symptoms [7]. HCV is eliminated, but a new steady state for infectious virions is obtained that is lower than the prior steady state value for effectiveness below this threshold value [7]. As a result, many individuals do not realise they have the infection until significant liver damage has already occurred. In addition to public health promotion, symptoms such as nausea, jaundice (yellowing of the skin and eyes), stomach discomfort, and fatigue indicate the need to raise awareness among high-risk groups and healthcare professionals and significantly increase prevention, screening, and treatment [8]. Mayanja et al. [9] stated that the finding of reproduction and performing sensitivity analysis to ascertain the relative significance of the various factors affecting the dynamics of HIV-HCV co-infection indicates a significant proportion of people will eventually be co-infected with HIV and latent HCV; thus, we must prevent this and begin therapy as soon as possible. The illness caused by the disease might range from a minor one that lasts a few weeks to a major one that lasts a lifetime; sufficient conditions have been determined in real-life data with a handled model [10].

Our numerical analysis of the fractional order model [11] ensures that it is more informative and has the same behaviour as the classical model. In the fractional case, it should yield accurate findings, as seen in Guedj et al. [12]. Considering the early degradation of HCV RNA following the start of IFN-based antiviral treatment, important aspects of the *in vivo* viral kinetics have been calculated, including the rate of free virus generation and clearance [13]. The dynamical complexity of the model, including the instability of immune control equilibrium and the existence of a stable periodic solution, has led to changes in the way hepatitis C is treated with the advent of direct-acting antiviral (DAA) drugs. Jothi et al. [14] developed a mathematical model concept for the dynamics of spreading disease, using the stability concept of controlling the infection to prevent the spread of disease. Nakabayashi [15] and Durso-Cain [16] create a mathematical model that depicts the whole HCV replication process in a single infected cell; the effect of dendritic cells (DC) and CTL is important to control the HCV. Since these drugs have a cure rate of over 95%, are usually well tolerated, and have a brief course of therapy, it makes sense that they would be more effective than HCV.

Hakami et al. [17] used the most precise estimates of the physico-chemical characteristics of the pharmaceutical drugs to treat hepatitis, which may be obtained via quantitative Structure-Property Relationships (QSPR) analysis. However, many regions like Italy still face challenges in accessing these treatments due to their high cost and healthcare inequalities [18,19]. We demonstrate the elimination plan of Khader et al. [20], Haggar et al. [21], and Belay et al. [22]. The ability to produce viral persistence in cell-to-cell transmission might be the consequence of two complementary modes of transmission. More and more mathematical model approaches are being used for diagnosis, cost-effectiveness, and HCV drug prediction.

Mannan et al. [23] showed the suggested approach's accuracy, robustness, and convergence while emphasising how it may be applied to other nonlinear epidemiological models. Institution like as the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) have set aggressive goals to eradicate hepatitis C as a danger to the public in order to address the disease's worldwide effect; the aim would show Canada's strong

commitment to the general health and welfare of Canadians with chronic HCV infection in addition to placing the country among the world's most successful nations [24–26]. Mathematical modelling is necessary to comprehend the dynamics of infectious disease transmission in Hepatitis C, where heavy alcohol use can accelerate the illness's progression and complicate therapy. By integrating components such as vaccine research, treatment strategies, and behavioural interventions, models can guide effective disease control initiatives. While cost-effectiveness analysis make sure that suggested measures, such as targeted treatment or immunisation, have the best possible public health impact within financial limits, stability analysis aids in determining if an illness will endure or disappear under certain circumstances, so that the spread of the disease would be most effective in terms of the dynamics of the people [27]. We should determine the infection rate utilising modelling approaches, and we demonstrate the optimal solution to stabilise the disease in the high-infection areas of the world. This includes increasing efforts in screening, early diagnosis, and improving access to treatment [27–34]. Hepatitis B is also a long-term disease; utilising screening and vaccination are two of the best ways to prevent hepatitis, but early diagnosis is also important for this [35,36]. In our model, it has to be verified to be controllable.

In this study, we developed a nonlinear Ordinary differential equation (ODE) model with the compartments. Here is the total population, which represents susceptible people, exposed people, acutely infected people, chronically infected people, and recovered people. The exposed people are affected in two ways. One is an acutely infected population, in which acutely infected persons are those who recover spontaneously or after taking antibiotics, but another type of impact is very complicated from I_a. According to the next session's results, this work is organised as follows: various analytical findings are determined with a viewpoint for the purpose of defining boundedness and positivity of the answer and identifying the model's five stable states for every equilibrium. In numerical analysis, we exhibit both local and global stability analysis of steady states, as well as global stability, locally asymptotic stability, and numerical simulation. The final portion serves as the work's finale.

2. The Model Description of HPS

In our model, we have five stages. The first one is the susceptible people, normal people with low immunity, who cannot fight back against the virus HPS. They are considered to be in the first stage, and then the second stage is exposed. In that stage of exposure, individuals with a disease expose themselves and being exposed is going to the chronic stage because it is also like the individual having the acute stage of the disease, like having live cancer and cirrhosis. So we consider the acute stage as all are curable with their immunity and medication, if they properly continue for a particular time period, and the chronic stage means having cancer and liver failure. The previous studies focus on early prevention, but our model differs from others because we prove that exposure to chronic conditions when symptoms are negative can put an individual at risk. In this study, we must prevent the infection before that stage using the reproduction number and by checking the stability criteria of the model. In the exposed stage, individuals are exposed to the highly infectious virions of HPS, but because the individuals do not have any symptoms, the disease is not effective, which means they are already in the acute stage of liver disease. After the effectiveness of the infections, they should change to highly infected individuals—chronic stage, as described in our model. To prevent the illness of the individuals, we have to diagnose the infection at an early stage, and find the reproduction number. Our model should help prevent the spread in the early stage. In the early stage, we had the immunotherapy to combat that virus and were highly expecting a higher recovery rate and lower infection rate. If they are having recovery, they will be going through the stage of being exposed to acute to recovery, a key aspect of this model.

3. Mathematical Statement of the Model

The collection of ODEs from the flow chart **Figure 1** is written as Equations (1)–(5).

$$\frac{dS}{dt} = u - (\mu + \alpha)S \tag{1}$$

$$\frac{dE}{dt} = \alpha S - (\mu + \beta + \gamma)E \tag{2}$$

$$\frac{dI_a}{dt} = \gamma E - (\mu + \varepsilon)I_a \tag{3}$$

$$\frac{dI_c}{dt} = \beta E - (\tau + \mu)I_c \quad (4)$$

$$\frac{dR}{dt} = \varepsilon I_a - \mu R \quad (5)$$

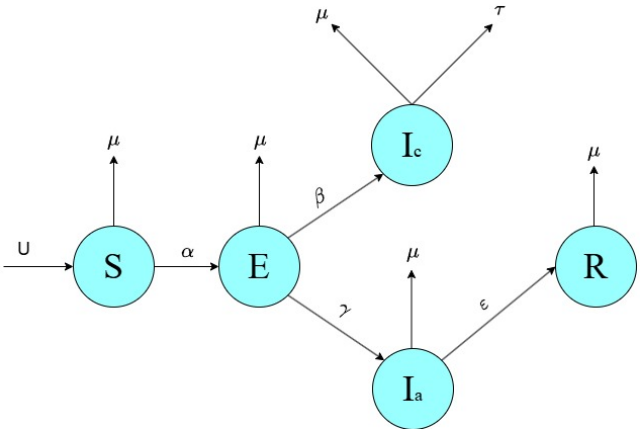

Figure 1. The compartment model of HCV.

Note: u = individual population; S = susceptible individuals; E = exposed people (infected- symptoms not recognized); I_a = acutely infected people (exposing symptoms); I_c = chronically infected people (suddenly highly infected); R = recovered people; α = the rate of exposed people; β = infection rate of chronically infected individuals; γ = infection rate of acutely infected individuals; ε = recovery rate of acutely infected individuals; μ+τ = the mortality rate of people with chronic infections; μ = individual death rate.

4. State of Equilibrium

- A result that remains constant in the absence of a disease state is called equilibrium. This suggests that since the system began at equilibrium, it will continue to be in that condition until the end of time.
- A system that is constantly changing is said to be dynamic. When time is calculated in various periods, we have a discrete dynamical system.
- The sequence described by the recurrence relation $\{x_n\}+1 = f\{x_n\}$, where is a real-valued function, is a discrete dynamical system.
- An iteration from on a function:
- A fixed point is occurs $[x_{n+1} = x_n = x^* = x^* = f(x^*)]$ when the system has same solution, it is the dynamical systems equilibrium.
- A differential equation of the following type describes a continuous dynamical system: $\frac{dx}{dt} = f(x)$
- An equilibrium, also referred to as a steady state point, occurs when: $\frac{dx}{dt} = 0$; This indicates that there is no change over time and the system is in rest.

5. Steady State-Equilibrium Points

In this model, the population is free of the virus as no one is afflicted with it.

Consider now that HCV is a disease-free state.

Now

$$\dot{D} = (\dot{S}, \dot{E}, \dot{I_a}, \dot{I_c}, \dot{R})$$
$$= (\dot{S}, 0, 0, 0, 0)$$

$$\frac{dS}{dt} = 0$$

$$\Rightarrow u - (\mu + \alpha)S = 0$$

$$\dot{S} = \frac{u}{\mu + \alpha}$$

Here is the HCV's disease free equilibrium points [Equation (6)].

$$E_0 = (\dot{S}, \dot{E}, \dot{I_a}, \dot{I_c}, \dot{R}) = (\frac{u}{\mu+\alpha}, 0, 0, 0, 0) \quad (6)$$

6. HCV's Basic Reproduction Number $\{R_0\}$

- After establishing the compartments, we will calculate the fundamental reproduction number (R_0) in our model using the next generation matrix approach.
- The HCV, in the model virus infected states are I_a and I_c. Hence, we find the reproduction number must be determined the $R_0(a)$ and $R_0(c)$ of the HCV model.

Now, the state of HCV's Reproduction number is $[R_0(a)]$
i.e.,

$$\frac{dI_a}{dt} = \gamma E - (\mu+\varepsilon)I_a$$

We have that F=[γ] and v=[μ + ε]
Now the inverse matrix of v is,

$$v^{-1} = \left[\frac{1}{\mu+\varepsilon}\right]$$

Let

$$R_0(a) = Fv^{-1}$$

Then,

$$R_0(a) = \gamma\left[\frac{1}{\mu+\varepsilon}\right],$$
$$R_0(a) = \left[\frac{\gamma}{\mu+\varepsilon}\right].$$

Furthermore, the HCV reproduction number state $[R_0(c)]$,
i.e.,

$$\frac{dI_c}{dt} = \beta E - (\tau+\mu)I_c$$

we have that F=[β] and v=[τ + μ]
for v, the inverse matrix is

$$v^{-1} = \left[\frac{1}{\mu+\tau}\right]$$

Now,

$$R_0(a) = Fv^{-1}$$

Then,

$$R_0(c) = [\beta][\frac{1}{\mu+\tau}],$$
$$R_0(c) = [\frac{\beta}{\mu+\tau}].$$

The represented state conditions are shown below. On average, one person from each of the two infectious classes contributes to a new infection.
thus, $R_0 = R_0(a) + R_0(c)$.

7. Positivity and Boundedness

Theorem 1. *The equation combined with Equation (6), the proposed model's in Equations (1)–(5) solution set is positive for all time* $t > 0$.

Proof of Theorem 1. We evaluate, Equation (1) while taking into consideration non-linear system of equations.

$$\frac{dS}{dt} + (\mu+\alpha)S = u$$

To find the Integrating Factor, which implies that:

$$S(t) = \frac{u}{\alpha + \mu} + ce^{-(\mu+\alpha)t}$$

Now

$$S_0 = S(0)$$

$$S_0 = \frac{u}{\mu + \alpha} + c$$

$$c = S_0 - \frac{u}{\mu + \alpha}$$

Substitute c in S(t)

$$S(t) = u\alpha + \mu + \left(S_0 - \frac{u}{\mu + \alpha}\right)e^{-(\alpha+\mu)t}$$

This goes

$$S(t) \geq 0$$

Let $\Omega=\{S,\ E,\ I_a,\ I_c,\ R\} \in R_5^+, N \leq \frac{U}{\mu}, S_0 \geq 0,\ E_0 \geq 0,\ I_a \geq 0,\ I_c \geq 0, R \geq 0$. The model's responses will always stay positive for the model for all time $t > 0$

8. Uniqueness Solution

Lemma 1. *If all initial conditions, can be satisfied* $S(t) > 0,\ E(t) > 0,\ I_a(t) > 0,\ I_c(t) > 0,\ R(t) > 0$, *then in this model, for all then , will exist in* $\mathbb{R}_5^+$.

Proof of Lemma 1. Therefore, if it is continuously differentiable on $\mathbb{R}_5^+$, we may conclude that f is Lipschitz Locally in $\mathbb{R}_+^5$

$$x=\begin{pmatrix} S \\ E \\ I_a \\ I_c \\ R \end{pmatrix} and f(t) = \begin{pmatrix} U - (\mu + \alpha)S \\ \alpha S - (\mu + \beta + \gamma)E \\ \gamma E - (\mu + \varepsilon)I_a \\ \beta E - (\mu + \tau)I_c \\ \varepsilon I_a - \mu R \end{pmatrix}$$

Theorem 2 (Uniqueness and Existence theorem). *f:*$\mathbb{R}^n \to \mathbb{R}^n$ *is the function we assume that it is continuously differentiable and thus, the differential equation* $\frac{dy}{dt}$ *=f(y) with the interval* τ *is have a answer, it's y(t) if for* $\forall t \in \tau$ *then y(t) is derive on the interval* τ *and y(t) is in* $\mathbb{R}^5$ *and* $\frac{dy}{dt}$ *=f(y), given* $y_0 \in \mathbb{R}^n$ *and, the initial valued Problem had a solution ,that is y(t).*

$$\frac{dy}{dt} = f(y)$$

$$y(t_0) = y_0$$

Based on the distinctness, we may presume that the ODE's model Equation (1) has positive, unique, and bounded outcomes. Existence and uniqueness of the preceding theorem, which we recently stated without verification, as well as the proven lemmas of the solutions are positive and bounded.

9. HCV Dynamic Model

Given an all-out population and the model of Equations (1)–(5), we distinguish according to time and procedures. We get,

$$\frac{d\hat{N}(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI_a(t)}{dt} + \frac{dI_c(t)}{dt} + \frac{dR(t)}{dt}$$

When we combine Equations (1) through (5), we obtain Equation (7).

$$= u - (\mu + \alpha)S + \alpha S - (\mu + \beta + \gamma)E + \gamma E - (\mu + \varepsilon)I_a + \beta E - (\tau + \mu)I_c + \varepsilon I_a - \mu R$$

$$= u - \mu(S + E + I_a + I_c + R) - \tau I_c$$

$$\frac{d\hat{N}(t)}{dt} = u - \mu\hat{N}(t) - \tau I_c \tag{7}$$

Hence, the population's dynamics of variances may, there will be in the Equation (7).

Theorem 3. *Considering that Equation (7) includes a model solution in manner of Equations (1)–(5) beginning circumstances, in what else is there in the set of compact (π) as t → ∞. Next, a positive continuous set of the model, the feasible Answer, is provided by*

$$\pi = \left\{ (S(t) + E(t) + I_a(t) + I_c(t) + R(t)) \in R_+^5 \geq 0 : \hat{N}(t) \leq \frac{u}{\mu} \right\}$$

Proof of Theorem 3. *In Equation (7), variation in N cause changes in every variable in the population.*
i.e., $\hat{N} = (S(t) + E(t) + I_a(t) + I_c(t) + R(t))$, and we obtain Equation (8).

$$\frac{d\hat{N}(t)}{dt} = u - \mu\hat{N}(t) - \tau I_c \tag{8}$$

Assumed that at the beginning stage, there is no disease, say next, Equation (8) provides Equation (9).

$$\frac{d\,\hat{N}(t)}{dt} = u - \tau I_c \tag{9}$$

From Equation (9), As we see this if so we obtain Equation (10).

$$\frac{d\hat{N}(t)}{dt} \leq u - \tau I_c \tag{10}$$

Modifying Equation (10), we obtain:

$$\frac{d\hat{N}(t)}{dt} + \tau I_c \leq u$$

Using an integrating factor that is linear, I.f = $e^{\int \mu dt} = e^{\mu t}$.
This refers to the common answer of Equation (10) that is found.

$$\hat{N}(t).(I.f) \leq \int (I.f)dt + c$$

Equivalently,

$$\begin{aligned} \hat{N}(t).e^{\mu t} &\leq \int u e^{\mu t} dt + c, \\ \hat{N}(t).e^{\mu t} &\leq u\, e^{\mu t} + c \\ \hat{N}(t) &\leq \frac{u}{\mu} + c\, e^{-\mu t} \end{aligned} \tag{11}$$

Using $\hat{N}(t = 0) = N_0$, we have

$$\begin{aligned} \hat{N}(0) &= \frac{u}{\mu} + c, \\ \left[\hat{N}(0) - \frac{u}{\mu}\right] &= c \end{aligned} \tag{12}$$

From Equation (11) and (12), we have Equation (13).

$$\hat{N}(t) \leq \frac{u}{\mu} + \left[\hat{N}(0) - \frac{u}{\mu}\right] e^{-\mu t} \tag{13}$$

Where in Equation (13), the overall population reduces $\hat{N}(t) \leq \frac{u}{\mu}$, it implies at that point, $0 \leq \hat{N}(t) \leq \frac{u}{\mu}$, there are restricted regulations for models of Equations (1) to (5) in the area (π). This completes a statement.

Thus, ∀ t >0, the region suggested by:

$$\pi = \left\{ \begin{array}{c} (S(t),\ E(t),\ I_a(t),\ I_c(t),\ R(t)) \in R_+^5 \geq 0, \\ S(t) + E(t) + I_a(t) + I_c(t) + R(t) \leq \hat{N}(t),\ \ \hat{N}(t) \leq \frac{u}{\mu} \end{array} \right\},$$

stands for a model's region of feasible.

Based on this, it is positively invariant. As a result, both mathematically and epidemiologically, the model in Equations (1)–(5) is given quite well. As a result, it is sufficient to focus on the model's dynamics in the region π.

10. Dimensionless Transformation

Using the state variables, we perform dimensionless adjustments to enhance the analysis of model S(t), E(t), $I_a(t)$, $I_c(t)$, R(t). The consistent set of models becomes into Equations (14)–(18).

$$S'(t) = u - (\mu + \alpha)S \tag{14}$$

$$E'(t) = \alpha S - (\mu + \beta + \gamma)E \tag{15}$$

$$I_a'(t) = \gamma E - (\mu + \varepsilon)I_a \tag{16}$$

$$I_c'(t) = \beta E - (\tau + \mu)I_c \tag{17}$$

$$R'(t) = \varepsilon I_a - \mu R \tag{18}$$

Adding Equations (14)–(18) yield Equation (19).

$$\begin{aligned} &S'(t) + E'(t) + I_a'(t) + I_c'(t) + R'(t) = u - \mu(S + E + I_a + I_c + R) - \tau I_c \\ &= u - (\mu + \tau I_c) \end{aligned} \tag{19}$$

Where $(S + E + I_a + I_c + R) = 1$.

11. Region of Feasible

- All of the state variables in the models are consistently positive as the population being studied is the human population. Thus, in the area π, the system Equations (14)–(18) model equations are limited to an a positive state.
- $\pi = \{(S, E, I_a, I_c, R) : S > 0, E > 0, I_a > 0, I_c > 0, R > 0\} \in R_+^5$,
- Our model Equations (14)–(18) have biological value except in cases where the feasible area is positively invariant.

12. Positivity Solution

- Since, they deal with the human population, we demonstrate in this section that each condition variable is not negative.

13. HCV's Endemic Equilibrium Points($\hat{E}_*$)

Consider $\hat{E}_* = (S^*, E^*, I_a^*, I_c^*, R^*) \in \pi$ are the organization's points of equilibrium, as shown by the configuration of Equations (1)–(5). Setting the condition yields the states of equilibrium points are:

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI_a}{dt} = \frac{dI_c}{dt} = \frac{dR}{dt} = 0$$

$$\frac{dS}{dt} = u - (\mu + \alpha)S = 0,$$

$$S^* = \frac{u}{\mu + \alpha},$$

$$\frac{dE}{dt} = \alpha S - (\mu + \beta + \gamma)E = 0$$

$$E^* = \frac{\alpha u}{(\mu + \beta + \gamma)(\mu + \alpha)},$$

$$\frac{dI_a}{dt} = \gamma E - (\mu + \varepsilon)I_a = 0$$

$$I_a{}^* = \frac{\gamma\alpha u}{(\alpha + \mu)(\varepsilon + \mu)(\beta + \gamma + \mu)},$$

$$\frac{dI_c}{dt} = \beta E - (\tau + \mu)I_c = 0$$

$$I_c^* = \frac{\beta\gamma\alpha u}{(\tau+\mu)(\varepsilon+\mu)(\mu+\beta+\gamma)\,(\alpha+\mu)},$$

$$\frac{dR}{dt} = \varepsilon I_a - \mu R = 0$$

$$R^* = \frac{\varepsilon\beta\gamma\alpha u}{(\mu)(\tau+\mu)(\varepsilon+\mu)\,(\alpha+\mu)(\beta+\gamma+\mu)}$$

Accordingly the given a equilibrium points is:

$$\hat{E_*} = (S^*,\, E^*,\, I_a^*,\, I_c^*,\, R^*) = E^*$$
$$= \left(\frac{u}{\alpha+\mu},\ \frac{\alpha u}{(\mu+\beta+\gamma)\,(\mu+\alpha)},\ \frac{\gamma\alpha u}{(\mu+\varepsilon)(\mu+\beta+\gamma)\,(\mu+\alpha)},\ \frac{\beta\gamma\alpha u}{(\tau+\mu)(\mu+\varepsilon)(\mu+\beta+\gamma)\,(\mu+\alpha)},\ \frac{\varepsilon\beta\gamma\alpha u}{(\mu)(\tau+\mu)(\varepsilon+\mu)\,(\alpha+\mu)(\beta+\gamma+\mu)} \right)$$

Theorem 4. *In the even that $R_0 > 1$, then the area π is the Global Asymptotic Stability (GAS) point of equilibrium for the endemic.*

Proof of Theorem 4. Assuming that $R_0 > 1$, we are able to establish the global stability by defining and deriving the Lyapunov P function as Equation (20).

$$P(y_1, y_2, \ldots,\, y_n) = \sum_{1}^{n} \frac{1}{2}\left[y_i - y_i^*\right]^2 \tag{20}$$

Using the Equations (1)–(5), where y_i=human population classes and y_i^*= endemic equilibrium point of individual people $\hat{E_*}$, Equation (20) becomes Equation (21).

$$P\,(S, E, I_a,\, I_c,\, R) = \frac{1}{2}\left[(S-S^*) + (E-E^*) + (I_a - I_a^*) + (I_c - I_c^*) + (R-R^*)\right]^2. \tag{21}$$

Equation (21) differentiate it with respect time t, relating to the Equations (1)–(5), we get Equation (22):

$$\begin{gathered}\frac{dP}{dt}\,(S, E, I_a,\, I_c,\, R) = \left[(S-S) + (E-E^*) + (I_a - I_a^*) + (I_c - I_c^*) + (R-R^*)\right]. \\ \frac{d}{dt}\left[S + E + I_a + I_c + R\right], \\ \frac{dP}{dt}\,(S, E, I_a,\, I_c,\, R) = \left[(S + E + I_a + I_c + R) - \left(S^* + E^* + I_a^* + I_c^* + R^*\right)\right]. \\ \frac{d}{dt}\left[S + E + I_a + I_c + R\right],\end{gathered} \tag{22}$$

but, in Equation (7) from, we getting Equation (23).

$$\frac{d\hat{N}(t)}{dt} = \frac{d}{dt}\left[S + E + I_a + I_c + R\right] \tag{23}$$

Thus, substitute Equation (8), and then Equation (23) yields Equation (24):

$$\frac{d\hat{N}(t)}{dt} = u - \mu\hat{N}(t), \tag{24}$$

but , from Equation (13), we getting Equation (25):

$$\left(S^* + E^* + I_a^* + I_c^* + R^*\right) = \frac{u}{\mu}, \tag{25}$$

in Equation (22) ,we substitute Equations (23)–(25), we getting Equation (26) and (27).

$$\frac{dP}{dt} = \left[\hat{N}(t) - \frac{u}{\mu}\right]\left[u - \mu\hat{N}(t)\right] \tag{26}$$

$$\frac{dP}{dt} = -\frac{1}{\mu}\left[\mu\hat{N}(t) + u\right]\left[u - \mu\hat{N}(t)\right]$$
$$\frac{dP}{dt} = -\frac{1}{\mu}\left[u - \mu\hat{N}(t)\right]^2 \quad (27)$$

Subsequently, from Equation (27), which follows $\frac{dP}{dt} < 0$, makes it obvious that is a function of Lyapunov strictly, indicating that GAS, which is made up of the area π, is the endemic point of equilibrium. According to this, For a very long period, hepatitis (HPS) will remain physiologically stable in the human population.

Once again in Equation (27), and subsequently, in the region π, converges positively as $t \to 0$

Thus, it is becomes closer to the proof.

Theorem 5. *The Disease-free equilibrium of our model Equations (1)–(5), Locally asymptotically stable (LAS) models have a reproduction number of* $R_0 < 1$*; otherwise, they are unstable.*

Proof of Theorem 5. At the disease-free equilibrium point, we are now use the Jacobian matrix to determine the local stability of Equations (1)–(5) in the HPS disease free equilibrium.

Applying Equations (1)–(5) of the system as follows:

$$j(\hat{E_0}) = \begin{bmatrix} -(\mu+\alpha) & 0 & 0 & 0 & 0 \\ \alpha & -(\beta+\gamma+\mu) & 0 & 0 & 0 \\ 0 & \gamma & -(\varepsilon+\mu) & 0 & 0 \\ 0 & \beta & 0 & -(\tau+\mu) & 0 \\ 0 & 0 & \varepsilon & 0 & -(\mu) \end{bmatrix} \quad (28)$$

we now handle matrix **Equation (28)**, the LAS will depend on the eigenvalues' outcome of the matrix.

Now here and the unit matrix is $I = 5 \times 5$; are eigenvalues.

Then,

$$\left[\begin{pmatrix} -(\mu+\alpha) & 0 & 0 & 0 & 0 \\ \alpha & -(\mu+\beta+\gamma) & 0 & 0 & 0 \\ 0 & \gamma & -(\mu+\varepsilon) & 0 & 0 \\ 0 & \beta & 0 & -(\mu+\tau) & 0 \\ 0 & 0 & \varepsilon & 0 & -(\mu) \end{pmatrix} - \begin{pmatrix} -(\lambda) & 0 & 0 & 0 & 0 \\ 0 & -(\lambda) & 0 & 0 & 0 \\ 0 & 0 & -(\lambda) & 0 & 0 \\ 0 & 0 & 0 & -(\lambda) & 0 \\ 0 & 0 & 0 & 0 & -(\lambda) \end{pmatrix}\right]$$
$$= \begin{vmatrix} -(\mu+\alpha)-\lambda & 0 & 0 & 0 & 0 \\ \alpha & -(\mu+\beta+\gamma)-\lambda & 0 & 0 & 0 \\ 0 & \gamma & -(\mu+\varepsilon)-\lambda & 0 & 0 \\ 0 & \beta & 0 & -(\mu+\tau)-\lambda & 0 \\ 0 & 0 & \varepsilon & 0 & -(\mu)-\lambda \end{vmatrix} = 0$$

when the above mentioned equation is evaluated,

$$\lambda_1 = -(\mu+\alpha)\ , \lambda_2 = -(\mu+\beta+\gamma), \lambda_3 = -(\mu+\varepsilon)\ , \lambda_4 = -(\mu+\tau)\ ,\ \lambda_5 = -(\mu)$$

Since all the eigenvalues of $j(\hat{E_0}) < 1$, that is, $\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 < 1$

Hence, $R_0 < 1$, the proof of this statement is indicates that, disease-free equilibrium point is LAS.

Theorem 6. *The global asymptotically stable (GAS) equilibrium of the endemic point on π will exist if* $R_0 < 1$.

Proof of Theorem 6. Now we consider the Lyapunov function [Equation (29)].

$$L_f(S, E, I_a, I_c, R) = S - S^* \ln S + k_1(E - E^* \ln E) + k_2(I_a - I_a^* \ln I_a) + k_3(I_c - I_c^* \ln I_c) + k_4(R - R^* \ln R) \quad (29)$$

Characterized and continuous and fulfills

$$\frac{dL_f}{dt} = \frac{\partial L_f}{\partial S}\frac{dS}{dt} + \cdots + \frac{\partial L_f}{\partial R}\frac{dL_f}{dt}$$

Which turns on

$$\dot{L_f}(S, E, I_a, I_c, R) = \dot{S} - S^* \frac{1}{S} \dot{S} + k_1(\dot{E} - E^* \frac{1}{E} \dot{E}) + k_2 (\dot{I_a} - I_a^* \frac{1}{I_a} \dot{I_a})$$
$$+k_3 (\dot{I_c} - {I_c}^* \frac{1}{I_c} \dot{I_c}) + k_4(\dot{R} - R^* \frac{1}{R} \dot{R})$$
$$\dot{L_f}(S, E, I_a, I_c, R) = \left(1 - S^* \frac{1}{S}\right) \dot{S} + k_1 \left(1 - E^* \frac{1}{E}\right) \dot{E} + k_2 \left(1 - I_a^* \frac{1}{I_a}\right) \dot{I_a}$$
$$+k_3 \left(1 - {I_c}^* \frac{1}{I_c}\right) \dot{I_c} + k_4 \left(1 - R^* \frac{1}{R}\right) \dot{R}$$

After simplification we obtain the Equation (30).

$$\dot{L_f}(S, E, I_a, I_c, R) = \left(1 - S^* \frac{1}{S}\right) (u - (\mu + \alpha)S) + k_1 \left(1 - E^* \frac{1}{E}\right) (\alpha S - (\mu + \beta + \gamma)E)$$
$$+k_2 \left(1 - I_a^* \frac{1}{I_a}\right) (\gamma E - (\mu + \varepsilon)I_a) + k_3 \left(1 - {I_c}^* \frac{1}{I_c}\right) (\beta E - (\tau + \mu)I_c) \quad (30)$$
$$+k_4 \left(1 - R^* \frac{1}{R}\right) (\varepsilon I_a - \mu R)$$

By assuming Equations (1)–(5):

$$u = (\mu + \alpha)S^*, \alpha S^* = (\mu + \beta + \gamma)E^*, \gamma E^* = (\mu + \varepsilon)I_a^*, \beta E^* = (\tau + \mu)I_c^*, \varepsilon I_a^* = \mu R^*$$

$$\dot{L_f}(S, E, I_a, I_c, R) = \left(1 - S^* \frac{1}{S}\right) ((\mu + \alpha)S^* - (\mu + \alpha)S)$$
$$+k_1 \left(1 - E^* \frac{1}{E}\right) \left(\frac{(\beta + \mu + \gamma)E^*}{S^*}.S - (\mu + \beta + \gamma)E\right)$$
$$+k_2 \left(1 - I_a^* \frac{1}{I_a}\right) \left(\frac{(\mu + \varepsilon)I_a^*}{E^*}.E - (\mu + \varepsilon)I_a\right)$$
$$+k_3 \left(1 - {I_c}^* \frac{1}{I_c}\right) \left(\frac{(\tau + \mu)I_c^*}{E^*}E - (\tau + \mu)I_c\right) + k_4 \left(1 - R^* \frac{1}{R}\right) \left(\frac{\mu R^*}{I_a^*}I_a - \mu R\right)$$
$$\dot{L_f}(S, E, I_a, I_c, R) = \left(\frac{S - S^*}{S}\right) (S^* - S)(\mu + \alpha)$$
$$+k_1 \left(\frac{E - E^*}{E}\right) \left(\frac{E^*.S - E}{S^*}\right) (\mu + \gamma + \beta)$$
$$+k_2 \left(\frac{I_a - I_a^*}{I_a}\right) \left(\frac{I_a^*.E - I_a}{E^*}\right) (\mu + \varepsilon)$$
$$+k_3 \left(\frac{I_c - {I_c}^*}{I_c}\right) \left(\frac{I_c^*.E - I_c}{E^*}\right) (\tau + \mu) + k_4 \left(\frac{R - R^*}{R}\right) \left(\frac{R^*.I_a - R}{I_a^*}\right) (\mu)$$

After simplification, we have

$$\dot{L_f}(S, E, I_a, I_c, R) = -\left(\frac{(\mu + \alpha)}{S}\right) (S - S^*)^2$$
$$-k_1 \left(\frac{\mu + \beta + \gamma}{E\,S^*}\right) (E.S^* - E^*.S)(E - E^*)$$
$$-k_2 \left(\frac{\varepsilon + \mu}{I_a\,E^*}\right) (I_a.E^* - I_a^*.E)(I_a - I_a^*)$$
$$-k_3 \left(\frac{\tau + \mu}{I_c\,E^*}\right) (I_c.E^* - I_c^*.E)(I_c - {I_c}^*)$$
$$-k_4 \left(\frac{\mu}{R\,I_a^*}\right) (R.I_a^* - R^*.I_a)(R - R^*)$$

$$\dot{L_f} = A(S - S^*)^2 + B(E - E^*) + C(I_a - I_a^*) + D(I_c - {I_c}^*) + E(R - R^*).$$

Therefore, for Note that It follows, the most collection of compact invariant in is such that is the singular $\hat{E}_*$, where is the endemic equilibrium. At this point, Lasalle's invariant principle implies that is GAS in the interior of π.

Table 1. The value of parameters used in the numerical simulation.

Parameter	Parameters Value	Source
u	2,25,000	WHO
α	58,500	WHO
β	8,000	Assumed
γ	22,000	Assumed
ε	8,775	Assumed
τ	4,000	Assumed
μ	42,000	WHO

Table 1 contains the parameters of our model as well as some values derived from our illness model's real-world data. The World Health Organization has approved these values for our model of Hepatitis C infection; thus, we are using them together with the numbers from the numerical simulation. In addition to predicting the future epidemiology of disease control, having these real-world data allows us to verify stability and control, which should be helpful when making decisions in the future.

14. Numerical Simulations

Here, the model contains five individual compartments. Each compartment has to be real and finite, so we showed the model of different dimensional dynamical pictures of stability control using some real-world values.

In the 2D plots of **Figure 2**, the HCV is infection-populations over time are with some world reality values of the South-East Asia region, as follows; the values we applied in **Figure 2**: u = 225000, μ = 0.42000, γ = 0.172711, α = 0.58500, β = 0.172711, τ = 0.107000, and ε = 0.84000.

In **Figure 3**, we applied the real world values from the region of the Eastern Mediterranean Region, u = 183000, μ = 0.42000, γ = 0.172711, α = 0.58500, β = 0.172711, τ = 0.107000, and ε = 0.84000.

In **Figure 4**, we applied the real world values from the region of the European Region, u = 126000, μ = 0.42000, γ = 0.172711, α = 0.58500, β = 0.172711, τ = 0.107000, and ε = 0.84000.

In **Figure 5**, we applied the real world values from the region of the Americas, u = 176000, μ = 0.42000, γ = 0.172711, α = 0.58500, β = 0.172711, τ = 0.107000, and ε = 0.84000. The total population of all the compartments are approaching control and stability states. In **Figures 2–5**, all the values are based on real-world phenomena of the region, and we also assume additional values to match our prediction based on our spreading rate and infection rate per year.

In **Figure 6**, the 2D plot represents individual stability rates of the differential equation of this model of HCV dynamics.

In **Figures 7–9**, the 3D plot shows the dynamic perspective simulation values, based on the region of the epidemic states, such as susceptible vs. Exposed vs. Recovered and Susceptible vs. Acute vs. Chronic Infection and Exposed vs. Acute vs. Recovered. These state trajectories illustrate the stability of this dynamical model.

In **Figure 10**, we can visualise the 3D coloured plot, which illustrates the population changing over time in the real-world region's susceptible-exposed-recovery stage. The hepatitis C transition from susceptible to exposed, acute, chronic, and recovered phases is shown in the 3D simulation figures. With one graphic displaying colour progression dependent on time, they depict how people move through various compartments over time.

In **Figure 11**, the mesh-based 3D plot is given, based on the SEIR dynamical structure of the stages, with real-world values and some random values for all stages.

Figure 12 is the 3D surface plot of the population. As the population goes through the susceptible and exposed stages, we see the stability controlled of the overall population of this HCV dynamic epidemiology. We show all the plots with the random values and the stability control of the dynamic population. All things considered, the figures concisely convey important aspects of infection, persistence, and recovery.

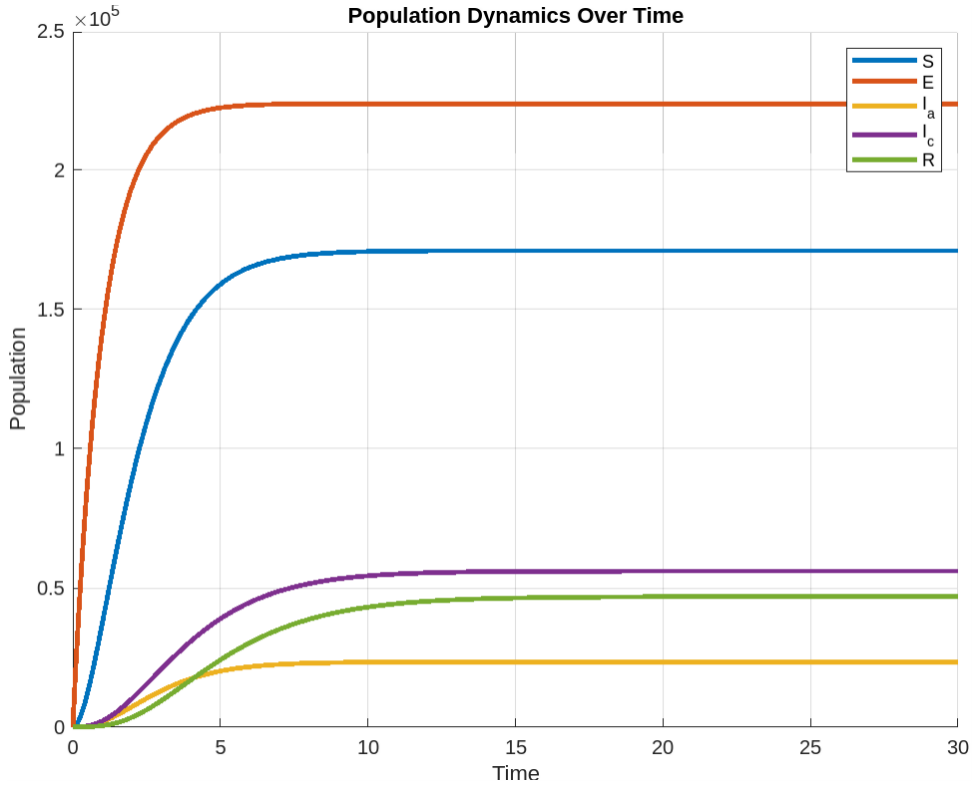

Figure 2. Population stability over time when U = 225000.

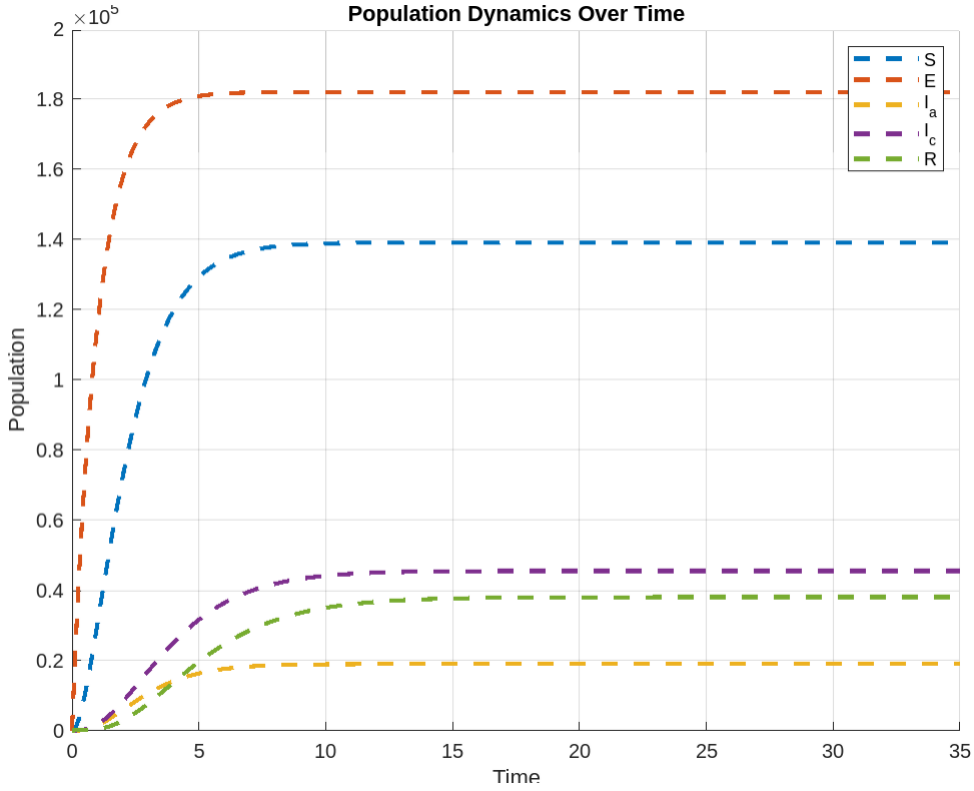

Figure 3. Population stability over time when U = 183000.

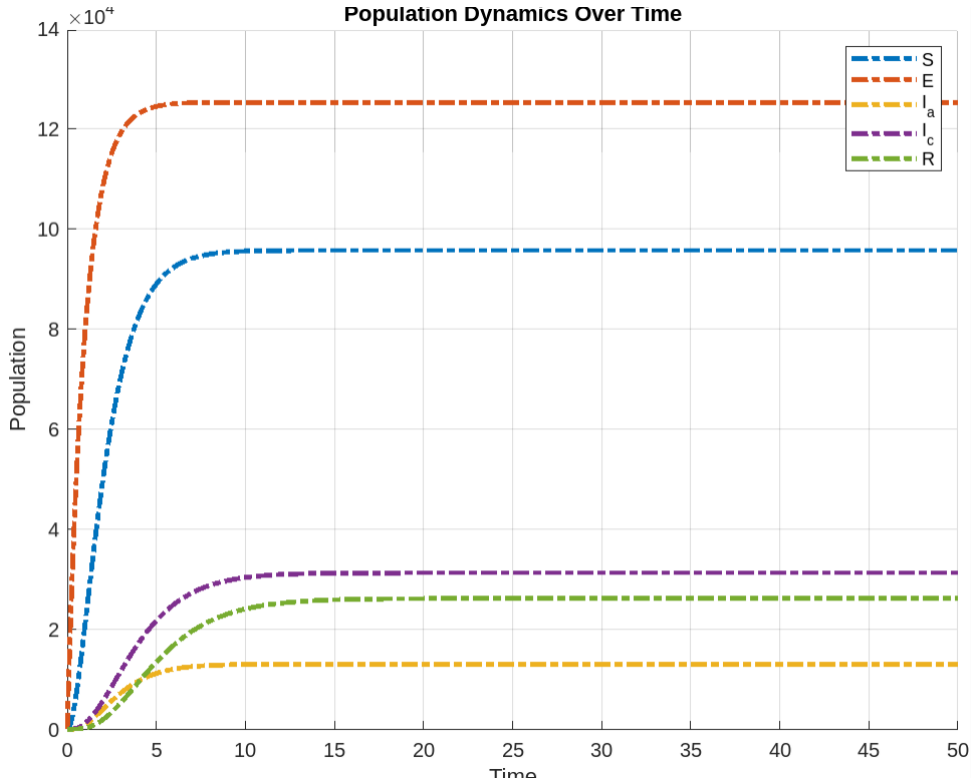

Figure 4. Population stability over time when U = 126000.

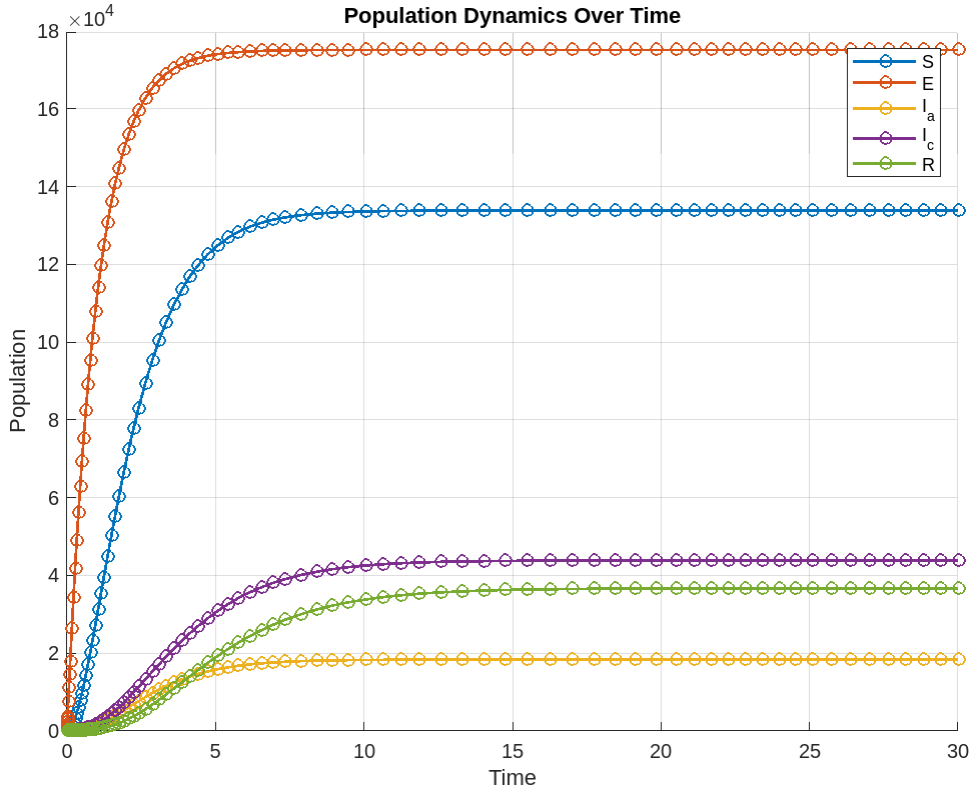

Figure 5. Population stability over time when U = 176000.

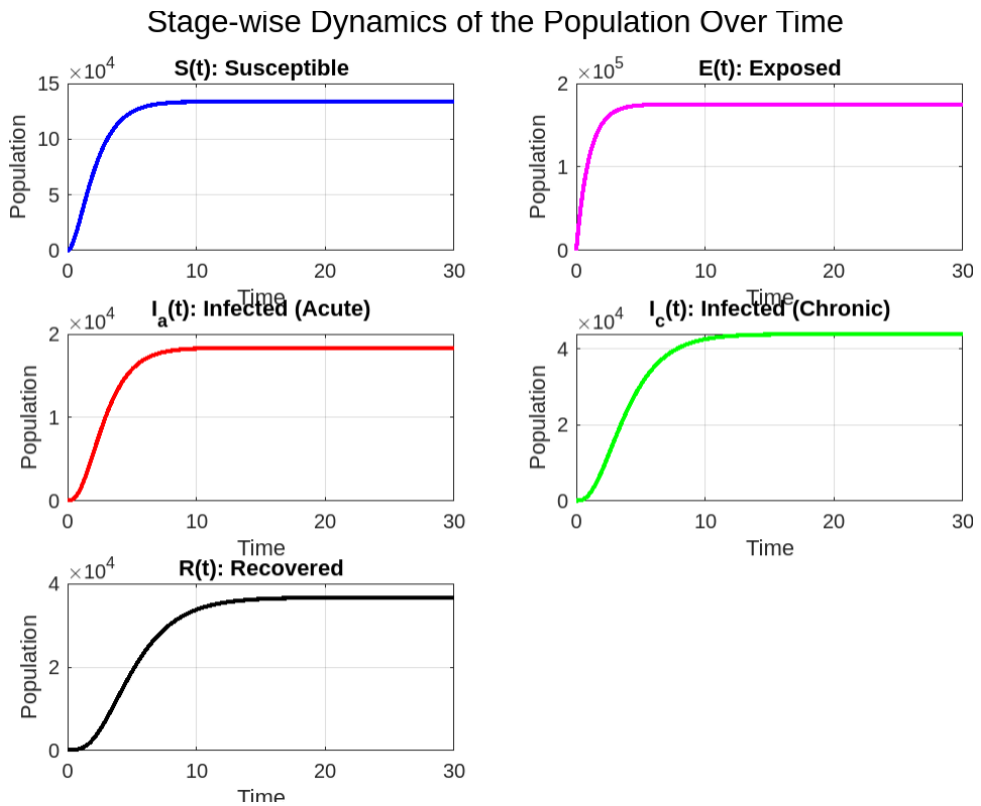

Figure 6. Time-based changes in all stages.

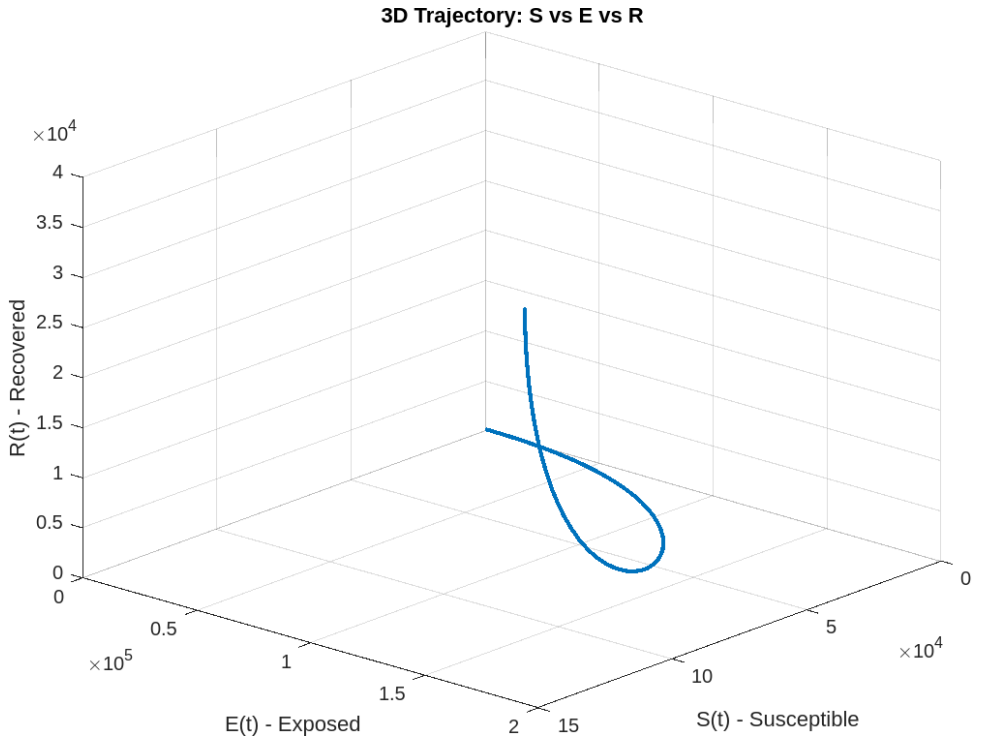

Figure 7. Evolution of epidemic states: susceptible vs. exposed vs. recovered.

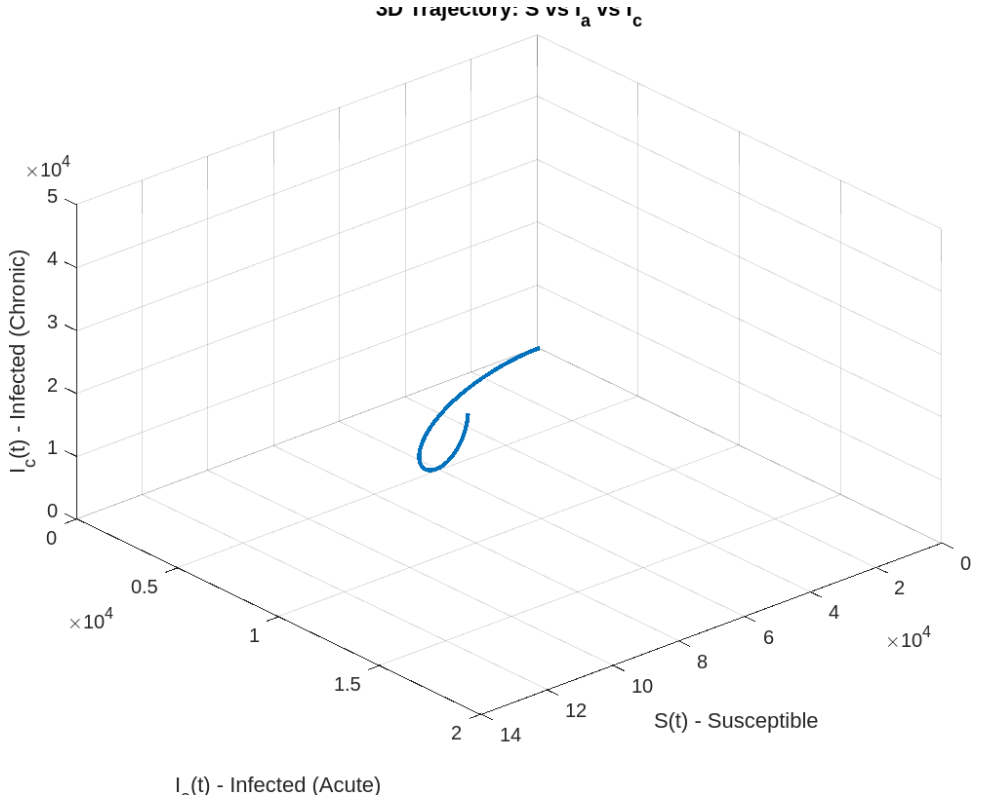

Figure 8. Evolution of epidemic states: susceptible vs. acute vs. chronic infection.

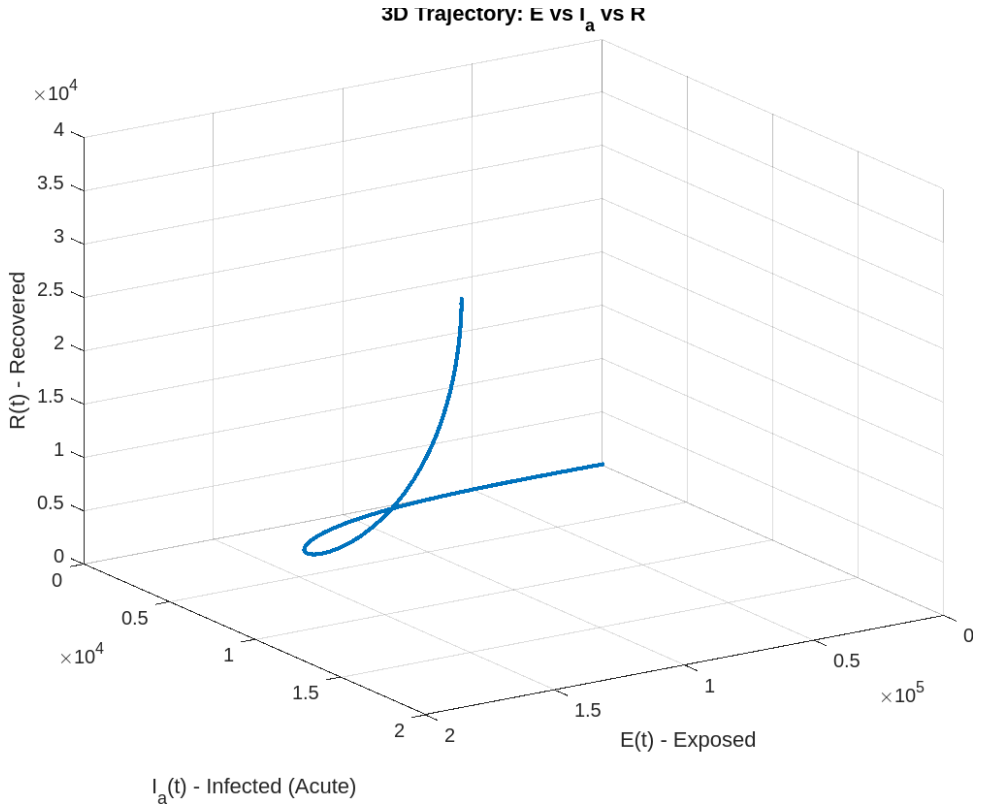

Figure 9. Evolution of epidemic states: exposed vs. acute vs. recovered.

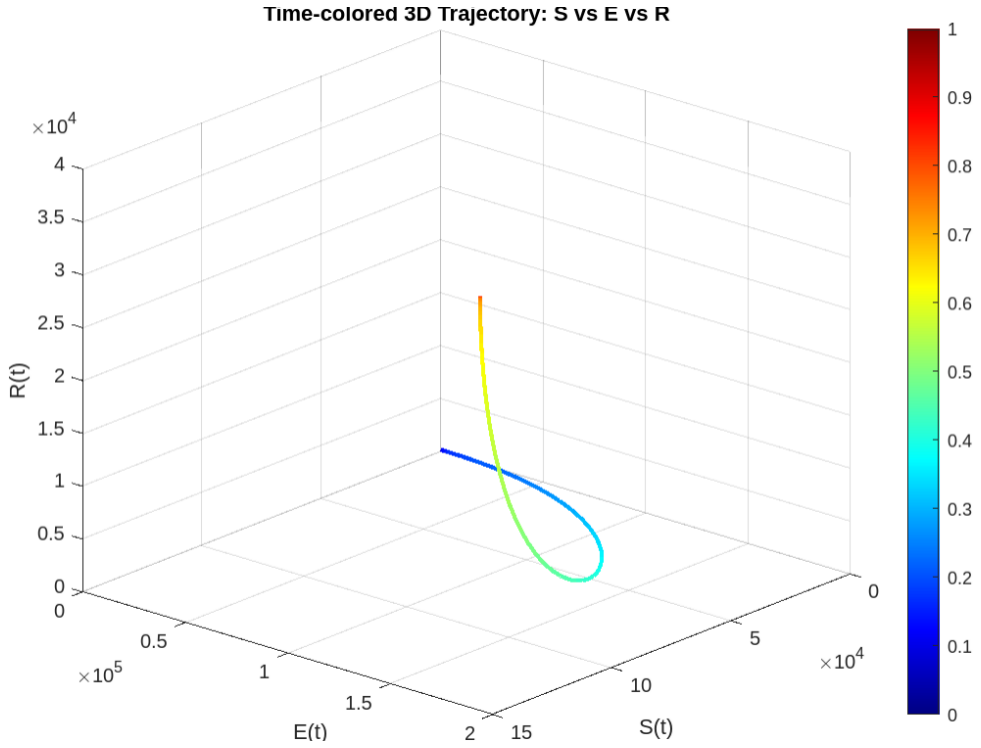

Figure 10. Dynamic visualization of susceptible-recovered stages.

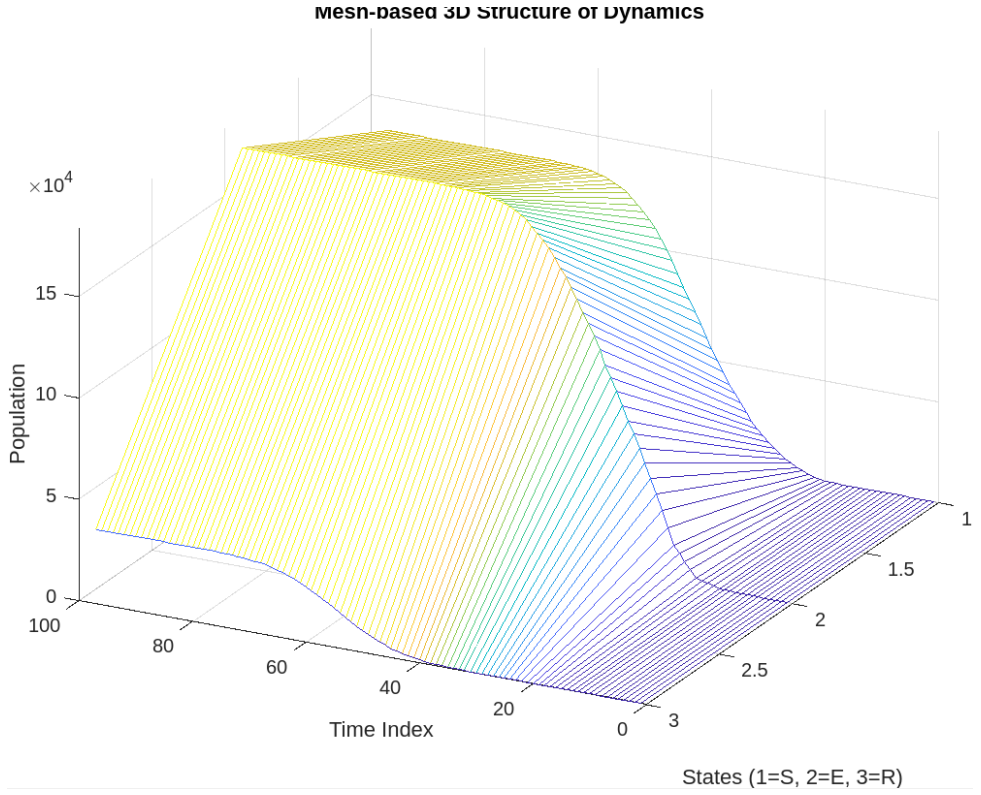

Figure 11. 3D temporal evolution of exposed-SEIR model states.

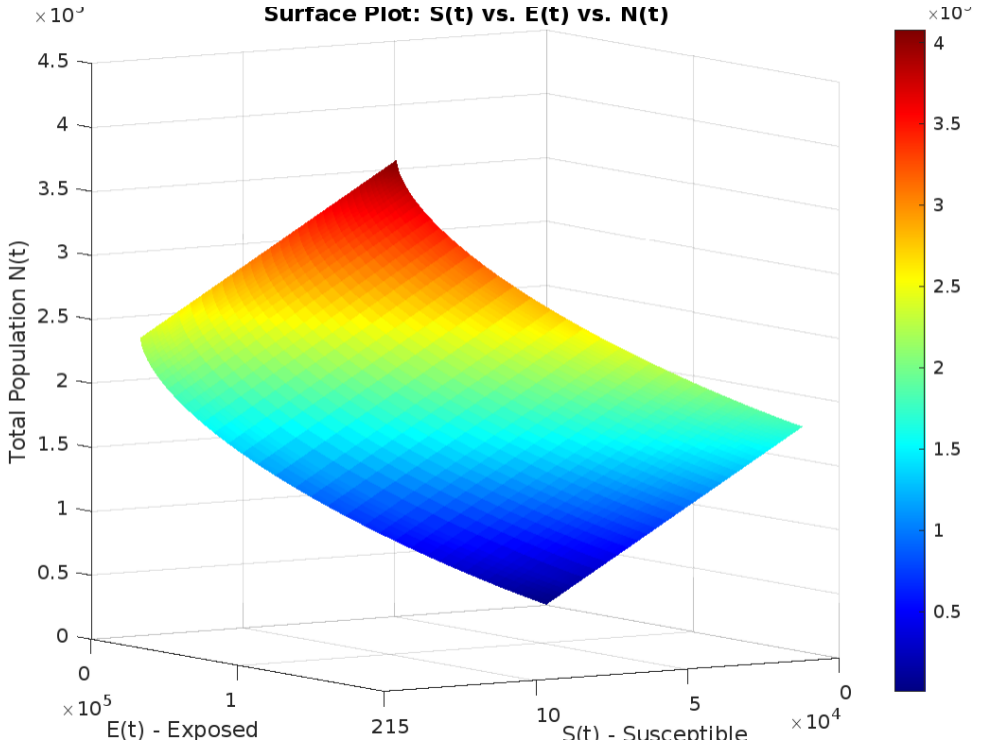

Figure 12. 3D surface visualization of N(t) as a function of S(t) and E(t).

15. Conclusion

In this work, we discussed the acute and chronic stages of HCV, as the majority of individuals worldwide live with these stages without being aware that they are infected. The population's ignorance of the HCV's symptoms is the primary issue. We used five compartments in the compute model. It was the most appropriate cause of death in the world. According to this paradigm, we stop the illness before it develops into a chronic, long-term condition. We would decrease the duration of illness and enhance the rate of recovery if we were to detect the early impact of this disease. This model must be stabilised in order to identify an equilibrium point devoid of sickness. To ascertain if the conditions remain stable, we evaluate R_0. We determine that the infectiousness condition is stable based on the R_0. Having these results, we prevent the infection at the stage of arrival and take the immunity to clear the virus, even if it has emerged in the individual's body. Lastly, we provide a few reliable figures, based on global data for this condition, using MATLAB. Early illness detection will be uncovered in the future, which will help reach more individuals and stop the sickness.

Author Contributions

Conceptualization, N.K.J. and J.M.; methodology, N.K.J. and J.M.; software, N.K.J., J.M., S.D., and C.M.; validation, N.K.J., J.M., S.D., and V.V.; formal analysis, N.K.J. and J.M.; investigation, N.K.J., J.M., and C.M.; resources, N.K.J., J.M., S.D., V.V., and C.M.; data curation, N.K.J. and J.M.; writing—original draft preparation, N.K.J. and J.M.; writing—review and editing, N.K.J., J.M., and V.V.; visualization, N.K.J., J.M., S.D., and V.V.; supervision, N.K.J., J.M., S.D., V.V., and C.M.; project administration, N.K.J., J.M., and C.M.; funding acquisition, N.K.J., J.M., S.D., and C.M. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement

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Conflicts of Interest

The authors declare no conflict of interest.

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