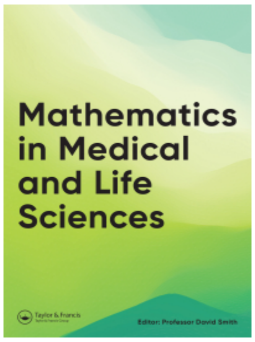

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Modeling the effect of alcohol consumption on the progression dynamics of Hepatitis C infection with treatment

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ABSTRACT

This research introduces a model that integrates a logistic approach to alcohol consumption to delve into the effect of alcohol consumption on the advancement of hepatitis C infection, evaluates the effects of addressing hepatitis C treatment and reducing alcohol consumption to minimal levels in the control of hepatitis C infection. The hepatitis C model was well established and biologically reasonable, indicating that all model solutions were non-negative and associated with non-negative initial conditions. Stability analysis revealed that the disease-free and endemic equilibrium points were locally and globally asymptotically stable whenever the basic reproduction number, $\mathcal{R}_{0H} \leq 1$ and $\mathcal{R}_{0H} > 1$ respectively. Sensitivity and numerical analysis of the model showed that improving treatment for individuals with chronic infection, along with reducing alcohol consumption, led to a notable decrease in the number of people affected by chronic infection.

PLAIN LANGUAGE SUMMARY

Hepatitis C (HCV) remains a major global health challenge. This study uses a mathematical model to explore how alcohol use and medical treatment affect the spread and progression of the disease. The model shows two possible outcomes: either the disease fades away or it continues to spread. Which outcome occurs depends on a key number called the *basic reproduction number*. If this number is less than one, each infected person spreads the virus to fewer than one other person on average, and the disease eventually dies out. But if the number is greater than one, the disease can keep spreading in the population. Computer simulations based on the model suggest that increasing access to treatment for people with chronic HCV and helping them reduce alcohol use can greatly reduce the number of long-term infections. Reducing alcohol consumption not only helps protect the liver but also makes medical treatment more effective.

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Hepatitis C; alcohol consumption; stability analysis; basic reproduction number

1. Introduction

The hepatitis C virus is a highly contagious infection that specifically targets the liver, leading to inflammation [1]. According to Purcell [2], hepatitis C virus (HCV) takes an average of 82.5 days to incubate, with a range of 15 to 150 days. In 2019, approximately 58 million individuals were living with HCV worldwide, 1.5 million newly infected with chronic hepatitis C, 3.2 million cases in children and adolescents and approximately 290, 000 hepatitis C-related deaths annually [3]. HCV is acquired through exposure to infected blood and exposure can occur through sharing needles or syringes during drug use, tattooing, sexual contact, mother-to-child transmission and unsafe medical procedures like organ transplants and transfusions of unscreened blood. A person who contracts HCV may develop a chronic condition or an acute (short-term) infection. Although most people with HCV don't have any symptoms at all, some might feel tired, nauseous, vomit, have dark urine, light-colored stools, lose their appetite, have flu-like symptoms, feel pain or tenderness on the right side of their abdomen, or develop jaundice, abdominal pain, blood in stool or vomit, accumulation of fluids in the abdomen, and itching [4–7]. Approximately 15–45% of individuals with acute HCV often recover on their own in 6 months [8–10]. In the acute phase, the infection does not harm the liver permanently. However, alcohol inhibits certain immune system functions, which makes it possible for HCV infection to persist past the acute phase. According to WHO, 3 million deaths each year are attributable to alcohol [11]. This implies alcohol consumption weakens the immune system and reduces the ability to withstand infectious diseases like viral hepatitis. According to H. Australia [12], drinking 3–4 standard drinks per day accelerates the risk of liver cancer and liver cirrhosis. Therefore, alcohol consumption has been assumed to increase the rate of HCV

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infection after exposure to the virus [13–15] and is thus associated with accelerated progression of liver injury, high frequency of cirrhosis and higher incidence of hepatocellular carcinoma [16–19].

HCV mathematical models have been developed to study its transmission, prevalence and control. In the study by Tresna, S.T, [20], treatment of infected population and educational programs on uneducated drug injectors would suppress the number of infected population and uneducated drug injectors. Ayobami *et al.* [21] suggested that proper education, adequate awareness, and proper intensive treatment given at the initial phase of the disease outbreak would reduce HCV transmission. Kondili *et al.* [22] emphasized that efforts to increase new diagnoses should prioritize screening people who inject drugs, individuals with tattoos, and young people at risk of sexually transmitted infections.

While most studies on HCV transmission have explored treatment and educational programs as control strategies, the impact of alcohol consumption on the progression of HCV infection in the context of treatment has been largely overlooked. This study addresses this gap by incorporating the effects of alcohol use into the dynamics of HCV proliferation. Specifically, it evaluates the role of treatment and reduction of alcohol intake to minimal levels in mitigating the burden of HCV infection.

2. Model formulation

2.1. *Variables and parameters of Hepatitis C virus model*

The whole population is partitioned into five compartments namely, susceptible individuals to HCV $S(t)$, exposed individuals to HCV $E(t)$, acutely infected individuals $I_A(t)$, the chronically infected individuals $I_C(t)$ and the recovered individuals $R(t)$ such that the total population at time t, denoted by $N(t)$, is given by $N(t) = S(t) + E(t) + I_A(t) + I_C(t) + R(t)$ and $H(t)$ is the amount of alcohol consumed per day in grams.

In model formulation for the progression dynamics of HCV, it is assumed that all susceptible individuals have an equal likelihood of contracting HCV. Susceptible, exposed, acutely and undiagnosed chronically infected individuals consume alcohol daily, with the rate of progression from exposure to infection varying according to their level of alcohol consumption. Recovery from infection does not confer lifelong immunity, indicating the possibility of reinfection. Furthermore, there is no natural recovery for individuals with chronic HCV infection. The model also assumes that treatment of acutely infected individuals is negligible, as they are typically asymptomatic and unlikely to seek medical attention at this stage. Additionally, chronically infected individuals are assumed to abstain from alcohol upon diagnosis to enhance treatment effectiveness.

The hepatitis C virus model parameters are described in Table 1.

Table 1. Parameter description.

Parameter	Description
Λ	Rate of recruitment into the susceptible population
$\omega(H)$	Rate of HCV acquisition
ρ	Relative infectiousness rate of chronic over acute infection
θ	Rate of loss of immunity of recovered individuals
μ	Natural mortality rate
$\delta(H)$	Rate at which the exposed progress to infected class
γ_c	HCV induced mortality rate
$\alpha(H)$	Rate at which acutely infected individuals progress to chronic stage
ϕ	Recovery rate of acutely infected individuals
ψ	Rate of treatment of chronically infected individuals
ϵ	Alcohol consumption growth rate
$H_{\min}$	Lower threshold of alcohol consumption in grams
$H_{\max}$	Upper threshold of alcohol consumption in grams

2.2. *Equations of hepatitis C virus model*

From the parameters described in Table 1, assumptions, and compartmental diagram in Figure 1, the following equations are derived:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\omega(H)(I_A + \rho I_C)S}{N} + \theta R - \mu S,\\
\frac{dE}{dt} &= \frac{\omega(H)(I_A + \rho I_C)S}{N} - (\delta(H) + \mu)E,\\
\frac{dI_A}{dt} &= \delta(H)E - (\mu + \alpha(H) + \phi)I_A,\\
\frac{dI_C}{dt} &= \alpha(H)I_A - (\gamma_c + \mu + \psi)I_C,\\
\frac{dR}{dt} &= \psi I_C + \phi I_A - (\mu + \theta)R,\\
\frac{dH}{dt} &= \epsilon(H(t) - H_{\min})\left(1 - \frac{H(t)}{H_{\max}}\right),
\end{aligned} \tag{1}$$

where

$$\omega(H) = \omega_0 + \tau\left(\frac{H(t) - H_{\min}}{H_{\max}}\right),$$

and ω_0 is the average rate of transmission of hepatitis C infection and τ is the average progression rate as a result of alcohol consumption.

$$\delta(H) = \delta_0 + \tau\left(\frac{H(t) - H_{\min}}{H_{\max}}\right),$$

and δ_0 is the mean progression rate to acute infection at minimum alcohol consumption and τ is the average

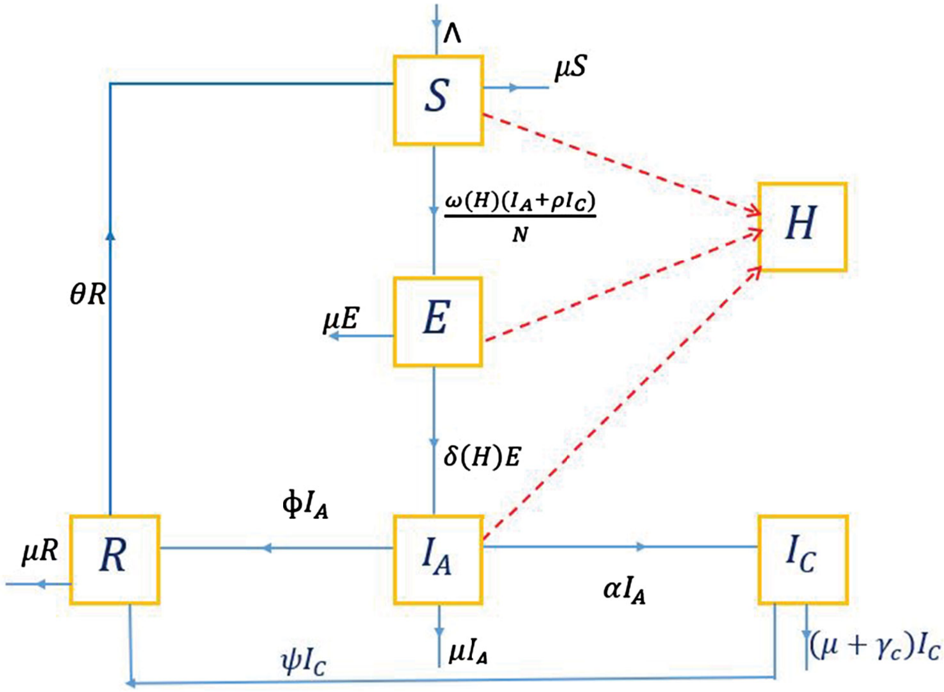


Figure 1. Compartmental diagram for the model.

progression rate as a result of alcohol consumption.

$$\alpha(H) = \alpha_0 + \tau\left(\frac{H(t) - H_{\min}}{H_{\max}}\right),$$

and α_0 is the average rate at which the acutely infected individuals become chronically infected at minimum alcohol consumption and τ is the average progression rate as a result of alcohol consumption.

Let initial data of the system (1) be $R(0) = R_0 \geq 0, H(0) = H_{\min} > 0$.

3. Analysis of Hepatitis C virus model

3.1. Basic properties of the model

In this section, the basic properties of the model system (1) useful in the proofs of stability of the system are established.

Theorem 3.1: *The solution of the system* (1) $\{(S(t), E(t), I_A(t), I_C(t), R(t), H(t))\}$ *uniquely exists and is non-negative for all* $t \geq 0$ *with the initial data of the system.*

Proof: To show the positivity of the states $(S(t), E(t), I_A(t), I_C(t), R(t), H(t))$, we shall first obtain the solution of the first equation of the system (1).

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \frac{\omega(H)(I_A + \rho I_C)S}{N} + \theta R - \mu S,\\ &\geq -\frac{\omega(H)(I_A + \rho I_C)S}{N} - \mu S.\end{aligned} \tag{2}$$

By using the integrating factor technique, it follows from (2) that

$$\frac{dS}{dt} + \left(\frac{\omega(H)(I_A + \rho I_C)}{N} + \mu\right) S \geq 0. \tag{3}$$

Integrating inequality (3) with initial condition $S(0) = S_0$ leads to

$$S(t) > S_0\left[e^{-\left(\mu t + \int_0^t \frac{\omega(H)(I_A(x) + \rho I_C(x))}{N}\,dx\right)}\right] > 0. \tag{4}$$

Similarly, from the second and third equations of system (1), with $E(0) = E_0$ and $I_A(0) = I_{A0}$ respectively, we obtain

$$E(t) = E_0\left[e^{-\left(\mu t + \int_0^t \delta(H)\,dx\right)}\right] \geq 0. \tag{5}$$

$$I_A(t) = I_{A0}\left[e^{-\left((\mu+\phi)t + \int_0^t \alpha(H)\,dx\right)}\right] \geq 0. \tag{6}$$

We also obtain an inequality from the fourth equation of (1) as

$$\frac{dI_C}{dt} \geq -(\gamma_c + \mu + \psi)I_C. \tag{7}$$

Integrating (7) with $I_C(0) = I_{C0}$ yields

$$I_C(t) = I_{C0}e^{-(\mu+\gamma_c+\psi)t} \geq 0. \tag{8}$$

Similarly, from the fifth equation of system (1), with $R(0) = R_0$, we obtain

$$R(t) = R_0e^{-(\mu+\theta)t} \geq 0. \tag{9}$$

From the sixth equation of system (1), we obtain

$$\frac{dH}{dt} \geq -\epsilon H_{\min}\left(1+\frac{H^2}{H_{\min}H_{\max}}\right) \tag{10}$$

Letting $k = \frac{1}{H_{\min}H_{\max}}$, then (10) becomes

$$\frac{dH}{dt} \geq -\epsilon H_{\min}\left(1+k(H(t))^2\right) \tag{11}$$

Separation of variables and integrating inequality (11) yields

$$\int \frac{dH}{\frac{1}{k}+(H(t))^2} \geq -\int \epsilon H_{\min}k\,dt. \tag{12}$$

Applying tan substitution to (12), and letting

$$H(t) = \frac{1}{k^{\frac{1}{2}}}\tan\theta \implies H(t)k^{\frac{1}{2}} = \tan\theta \tag{13}$$

Integration leads to

$$\theta \geq -\epsilon H_{\min}k^{\frac{1}{2}}t + C, \tag{14}$$

where C is a constant of integration. From Equation (13), it follows that $\theta = \tan^{-1}(H(t)k^{\frac{1}{2}})$. Substituting for θ in inequality (14) yields

$$\tan^{-1}(H(t)k^{\frac{1}{2}}) \geq -\epsilon H_{\min}k^{\frac{1}{2}}t + C. \tag{15}$$

Applying the initial condition $H(0) = H_{\min}$, then $\tan^{-1}(H_{\min}k^{\frac{1}{2}}) \geq C$ which leads to

$$H(t) \geq k^{-\frac{1}{2}}\tan\left(\tan^{-1}(H_{\min}k^{\frac{1}{2}}) - \epsilon H_{\min}k^{\frac{1}{2}}t\right) \geq 0. \tag{16}$$

Thus the solutions of the model are all nonnegative for $t \geq 0$. ■

Theorem 3.2: *The closed set*

$$\Omega = \left\{(S,E,I_A,I_C,R) \in \mathbb{R}^5_+ : N \leq \frac{\Lambda}{\mu}\right\} \tag{17}$$

is a biologically feasible region of the initial value problem (1).

Proof: Adding the first five equations of system (1) gives

$$\frac{dN}{dt} = \Lambda - \mu N - \gamma_C I_C. \tag{18}$$

Equation (18) leads to the inequality

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{19}$$

With initial condition $N(0) = N_0$, integration leads to

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t}. \tag{20}$$

As $t \to \infty$, inequality (20) becomes

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty}\left[\frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t}\right] = \frac{\Lambda}{\mu}. \tag{21}$$

Thus

$$N(t) \leq \frac{\Lambda}{\mu}. \tag{22}$$

Hence, for all $t \geq 0$, each solution of the initial value problem of (1) remains in Ω. Thus Ω is a well-posed and biologically meaningful region since $0 \leq N(t) \leq \frac{\Lambda}{\mu}$. ■

3.2. *Disease-free equilibrium point of Hepatitis C virus model, DFE*

The DFE $\mathcal{E}_0$ is the steady state at which HCV is being controlled within a population to the level of complete elimination. The DFE can be found by setting the right-hand side of system (1) equal to zero to obtain; $S(t) = \Lambda/\mu, E(t) = 0, I_A(t) = 0, I_C(t) = 0, R(t) = 0$, and $H_{\max}$. The DFE $\mathcal{E}_0(S,E,I_A,I_C,R)$ is given as either

$$\mathcal{E}_{01} = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, H_{\min}\right) \quad \text{or}$$
$$\mathcal{E}_{02} = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, H_{\max}\right) \tag{23}$$

Thus the DFE given by $\mathcal{E}_0(S,E,I_A,I_C,R) = \mathcal{E}_{01}$ or $\mathcal{E}_{02}$. This result suggests that, regardless of the level of alcohol consumption, Hepatitis C will not persist within the population.

3.3. *Computation of reproduction number, $\mathcal{R}_{0H}$ of hepatitis C virus model*

According to Diekmann *et al.* [23], we re-define, $\mathcal{R}_{0H}$ as the number of secondary hepatitis C infections due to a single hepatitis C infectious individual during his or her entire infectious period in a completely susceptible population. To obtain $\mathcal{R}_{0H}$, we use the DFE and the next generation matrix as applied in [24, 25]. The infected compartments are re-arranged as follows:

$$\begin{aligned}
\frac{dE}{dt} &= \frac{\omega(H)(I_A+\rho I_C)S}{N} - x_1E,\\
\frac{dI_A}{dt} &= \delta(H)E - x_2I_A,\\
\frac{dI_C}{dt} &= \alpha(H)I_A - x_3I_C,
\end{aligned} \tag{24}$$

where $x_1 = \delta(H)+\mu, x_2 = \mu+\alpha(H)+\phi, x_3 = \gamma_C+\mu+\psi$. Let F_i be the matrix of infection terms and V_i be the matrix of transfer terms of system (1). Then we have

$$F_i = \begin{pmatrix} \frac{\omega(H)(I_A+\rho I_C)S}{N} \\ 0 \\ 0 \end{pmatrix} \tag{25}$$

and

$$V_i = \begin{pmatrix} x_1 E \\ x_2 I_A - \delta E \\ x_3 I_C - \alpha I_A \end{pmatrix}. \tag{26}$$

We note that at the beginning of the epidemic, $\frac{S}{N} \approx 1$, thus, (25) becomes

$$F_i = \begin{pmatrix} \omega(H)(I_A + \rho I_C) \\ 0 \\ 0 \end{pmatrix}. \tag{27}$$

The Jacobian matrices $\mathcal{F}$ and $\mathcal{V}$ of F_i and V_i respectively are evaluated at DFE to obtain

$$\mathcal{F} = \begin{pmatrix} 0 & \omega(H) & \omega(H)\rho \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \tag{28}$$

$$\mathcal{V} = \begin{pmatrix} x_1 & 0 & 0 \\ -\delta(H) & x_2 & 0 \\ 0 & -\alpha(H) & x_3 \end{pmatrix}. \tag{29}$$

Thus, from Equations (28) and (29), the next generation matrix is computed as follows:

$$\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \left(\frac{\omega(H)\delta(H)}{x_1x_2} + \frac{\omega(H)\rho\delta(H)\alpha(H)}{x_1x_2x_3}\right) & \left(\frac{\omega(H)}{x_2} + \frac{\omega(H)\rho\alpha(H)}{x_2x_3}\right) & \left(\frac{\omega(H)\rho}{x_3}\right) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{30}$$

From the next generation matrix (30), we compute the characteristic polynomial $\mathcal{P}(\lambda) = |FV^{-1} - \lambda I| = 0$. Thus

$$\mathcal{P}(\lambda) = \begin{vmatrix} \left(\frac{\omega(H)\delta(H)}{x_1x_2} + \frac{\omega(H)\rho\delta(H)\alpha(H)}{x_1x_2x_3}\right) - \lambda & \left(\frac{\omega(H)}{x_2} + \frac{\omega(H)\rho\alpha(H)}{x_2x_3}\right) & \left(\frac{\omega(H)\rho}{x_3}\right) \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix}. \tag{31}$$

This gives the characteristic equation as

$$\lambda^2 \left[\left(\frac{\omega(H)\delta(H)}{x_1x_2} + \frac{\omega(H)\rho\delta(H)\alpha(H)}{x_1x_2x_3}\right) - \lambda\right] = 0.$$

Thus the eigenvalues are as follows:

$$\lambda_1 = \left(\frac{\omega(H)\delta(H)}{x_1x_2} + \frac{\omega(H)\rho\delta(H)\alpha(H)}{x_1x_2x_3}\right), \lambda_2 = 0, \lambda_3 = 0.$$

Since the basic reproduction number, $\mathcal{R}_{0H}$ is the dominant eigenvalue of the next generation matrix (30), then it is given by

$$\mathcal{R}_{0H} = \left(\frac{\omega(H)\delta(H)}{x_1x_2} + \frac{\omega(H)\rho\delta(H)\alpha(H)}{x_1x_2x_3}\right) = \frac{\omega(H)\delta(H)(x_3 + \rho\alpha(H))}{x_1x_2x_3}. \tag{32}$$

From Equation (32), we have two options for $\mathcal{R}_{0H}$ depending on alcohol consumption levels. The first expression for reproduction number $\mathcal{R}_{01}$ is obtained when a chronically hepatitis C infected individual drinks minimum quantity of alcohol (that is $H(t) = H_{\min}$).

$$\mathcal{R}_{01} = \frac{\omega_0\delta_0(x_3 + \rho\alpha_0)}{x_1^* x_2^* x_3}, \tag{33}$$

where $x_1^* = \delta_0 + \mu, x_2^* = \mu + \alpha_0 + \phi$, and $x_3 = \mu + \gamma_C + \psi$.

The second expression $\mathcal{R}_{02}$ is obtained when a chronically hepatitis C infected individual drinks maximum quantity of alcohol (that is $H(t) = H_{\max}$) and is given as

$$\mathcal{R}_{02} = \frac{\omega(H_{\max})\delta(H_{\max})(x_3 + \rho\alpha(H_{\max}))}{x_1^{**} x_2^{**} x_3} \tag{34}$$

where $\omega(H_{\max}) = \omega_0 + B^*\tau, \delta(H_{\max}) = \delta_0 + B^*\tau, \alpha(H_{\max}) = \alpha_0 + B^*\tau, B^* = (\frac{H_{\max}-H_{\min}}{H_{\max}}), x_1^{**} = \delta(H_{\max}) + \mu, x_2^{**} = \mu + \alpha(H_{\max}) + \phi$, and $x_3 = \mu + \gamma_C + \psi$. From Equations (33) and (34), we conclude that $\mathcal{R}_{0H} = \mathcal{R}_{01}$ *or* $\mathcal{R}_{02}$.

The relationship between basic reproduction numbers $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ is given by

$$\mathcal{R}_{02} = \frac{\omega(H_{\max})\delta(H_{\max})(x_3 + \rho\alpha(H_{\max}))x_1^* x_2^* \mathcal{R}_{01}}{x_1^{**} x_2^{**} \omega_0\delta_0(x_3 + \rho\alpha_0)}. \tag{35}$$

This expression quantifies how the basic reproduction number $\mathcal{R}_{02}$ for maximum alcohol consumption is related to $\mathcal{R}_{01}$ for baseline conditions, that is minimal alcohol intake. This relationship shows that alcohol consumption can increase the disease reproduction number by enhancing transmission ω, progression rates δ and α, and by altering the steady-state levels of individuals, potentially making $\mathcal{R}_{02}$ greater than $\mathcal{R}_{01}$.

3.4. *Endemic equilibrium point of Hepatitis C virus model, EE*

The EE of system (1) is the steady state at which HCV infection persists within the population for a given period. Equating the right-hand side of system (1) to zero and solving for $S^*, E^*, I_A^*, I_C^*, R^*$ yields the first endemic equilibrium point, $\mathcal{E}^*{}_1 = \{(S^*(t), E^*(t), I_A^*(t), I_C^*(t), R^*(t), H^*(t))\}$

which depends on $\mathcal{R}_{01}, H^*(t) = H_{\min}$ and is given as

$$
\begin{aligned}
S^* &= \frac{N}{\mathcal{R}_{01}},\\
E^* &= \frac{(\Lambda\mathcal{R}_{01} - \mu N)x_2^* x_3 x_4}{\delta_0\left[\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0)\right]},\\
I_A^* &= \frac{(\Lambda\mathcal{R}_{01} - \mu N)x_3 x_4}{\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0)},\\
I_C^* &= \frac{(\Lambda\mathcal{R}_{01} - \mu N)\alpha_0 x_4}{\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0)},\\
R^* &= \frac{(\Lambda\mathcal{R}_{01} - \mu N)(x_3\phi + \psi\alpha_0)}{\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0)},\\
N &= \frac{\Lambda}{\mu}\left[\frac{\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0) - \mathcal{R}_{01}\gamma_C\alpha_0 x_4}{\omega_0 x_4(x_3 + \rho\alpha_0) - \mathcal{R}_{01}\theta(x_3\phi + \psi\alpha_0) - \gamma_C\alpha_0 x_4}\right],
\end{aligned} \tag{36}
$$

where $x_2^* = \mu + \alpha_0 + \phi, x_3 = \mu + \gamma_C + \psi$, and $x_4 = \mu + \theta$. This implies that if $\mathcal{R}_{01} > 1$, then there exists at least one positive disease endemic equilibrium point.

The second endemic equilibrium point, $\mathcal{E}_2^*$ depends on $\mathcal{R}_{02}, H^*(t) = H_{\max}$ and it is given as

$$
\begin{aligned}
S^* &= \frac{N}{\mathcal{R}_{02}},\\
E^* &= \frac{(\Lambda\mathcal{R}_{02} - \mu N)x_2^{**} x_3 x_4}{\delta(H_{\max})\left[\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max})) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max}))\right]},\\
I_A^* &= \frac{(\Lambda\mathcal{R}_{02} - \mu N)x_3 x_4}{\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max}) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max}))},\\
I_C^* &= \frac{(\Lambda\mathcal{R}_{02} - \mu N)\alpha(H_{\max})x_4}{\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max})) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max}))},\\
R^* &= \frac{(\Lambda\mathcal{R}_{02} - \mu N)(x_3\phi + \psi\alpha(H_{\max}))}{\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max})) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max}))}.\\
N &= \frac{\Lambda}{\mu}\left[\frac{\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max})) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max})) - \mathcal{R}_{02}\gamma_C\alpha(H_{\max})x_4}{\omega(H_{\max})x_4(x_3 + \rho\alpha(H_{\max})) - \mathcal{R}_{02}\theta(x_3\phi + \psi\alpha(H_{\max})) - \gamma_C\alpha(H_{\max})x_4}\right],
\end{aligned} \tag{37}
$$

where $x_2^{**} = \mu + \alpha(H_{\max}) + \phi$, $\omega(H_{\max}) = \omega_0 + \tau(1 - \frac{(H_{\min})}{H_{\max}})$, $\alpha(H_{\max}) = \alpha_0 + \tau(1 - \frac{(H_{\min})}{H_{\max}})$, and $\delta(H_{\max}) - \delta_0 + \tau(1 - \frac{(H_{\min})}{H_{\max}})$. Thus the endemic equilibrium of system (1) exists and is given by $\mathcal{E}^* = \mathcal{E}_1^*$ or $\mathcal{E}_2^*$.

Theorem 3.3: *If $\mathcal{R}_{0H} > 1$, then the model (1) admits a unique endemic equilibrium.*

Proof: If HCV is endemic in the community, then $\frac{dI_A}{dt} > 0$ and $\frac{dI_C}{dt} > 0$ that is

$$\delta(H^*)E^* - x_2 I_A^* > 0, \tag{38}$$

$$\alpha(H^*)I_A^* - x_3 I_C^* > 0. \tag{39}$$

From the second equation of system (1), since HCV is endemic then $\frac{S}{N} < 1$ implying that

$$E < \frac{\omega(H)(I_A + \rho I_C)}{x_1}.$$

It follows that

$$I_A < \frac{\omega(H)\delta(H)(I_A + \rho I_C)}{x_1 x_2}, \tag{40}$$

$$I_C < \frac{\alpha(H)I_A}{x_3}. \tag{41}$$

Inequalities (40) and (41) together imply that

$$1 < \frac{\omega(H)\delta(H)(x_3 + \rho\alpha(H))}{x_1 x_2 x_3} = \mathcal{R}_{0H}.$$

Thus a unique endemic equilibrium point $\mathcal{E}^*$ exists when $\mathcal{R}_{0H} > 1$. Specifically, $\mathcal{E}_1^*$ is unique when there is minimum alcohol intake, that is

$$\mathcal{R}_{01} = \frac{\omega_0\delta_0(x_3 + \rho\alpha_0)}{x_1^* x_2^* x_3} > 1.$$

Additionally, $\mathcal{E}_2^*$ is unique when there is maximum alcohol intake, that is

$$\mathcal{R}_{02} = \frac{\omega(H_{\max})\delta(H_{\max})(x_3 + \rho\alpha(H_{\max}))}{x_1^{**} x_2^{**} x_3} > 1.$$

■

3.5. Local and global stability of disease-free equilibrium point of Hepatitis C virus model

This subsection looks into the local and global stability of the DFE. When the DFE is stable, then an outbreak of hepatitis C infection is not anticipated, however, if it is unstable, an outbreak is expected.

Theorem 3.4: *The DFE, $\mathcal{E}_0$ of hepatitis C virus model system (1) is locally asymptotically stable if $\mathcal{R}_{0H} < 1$ and unstable otherwise.*

Specifically, the equilibrium $\mathcal{E}_{01}$ is locally asymptotically stable if $\mathcal{R}_{01} < 1$, and $\mathcal{E}_{02}$ is locally asymptotically stable if $\mathcal{R}_{02} < 1$. The proof of Theorem 3.4 is provided in Appendix A.1.

Theorem 3.5: *The DFE point, $\mathcal{E}_0$ of hepatitis C virus model system (1) is globally asymptotically stable in Ω if $\mathcal{R}_{0H} \le 1$.*

Proof: Consider the infected compartments of hepatitis C virus model system (1). Let the Lyapunov function be defined as

$$L = \sum_{i \ge 2}(k_i x_i),$$

where x_i is the population of the i^{th} infected compartment for some $k_i > 0$ to be determined [26]. Thus we obtain

$$L(E, I_A, I_C) = k_1 E + k_2 I_A + k_3 I_C, \tag{42}$$

where $k_i(i = 1, 2, 3)$ are non-negative real numbers which will later be chosen. Differentiating Equation (42) with respect to time, t, we obtain

$$\frac{dL}{dt} = k_1\frac{dE}{dt} + k_2\frac{dI_A}{dt} + k_3\frac{dI_C}{dt}. \tag{43}$$

Substituting for $\frac{dE}{dt}, \frac{dI_A}{dt}, \frac{dI_C}{dt}$ from system (1), (43) becomes

$$\begin{aligned}\frac{dL}{dt} &= k_1\left(\frac{\omega(H)(I_A+\rho I_C)S}{N} - (\delta(H)+\mu)E\right)\\ &\quad + k_2(\delta(H)E - (\mu+\alpha(H)+\phi)I_A)\\ &\quad + k_3(\alpha(H)I_A - (\mu+\gamma_C+\psi)I_C).\end{aligned} \tag{44}$$

However, at DFE, $\mathcal{E}_0$, $\frac{S}{N} \approx 1$ and thus, on re-arranging and letting $x_1 = \delta(H)+\mu, x_2 = \mu+\alpha(H)+\phi, x_3 = \mu+\gamma_C+\psi$, it follows that

$$\begin{aligned}\frac{dL}{dt} &\le (k_1\omega(H) - k_2x_2 + k_3\alpha(H))\, I_A - (k_1x_1 - k_2\delta(H))\, E\\ &\quad + (k_1\omega(H)\rho - k_3x_3)\, I_C.\end{aligned} \tag{45}$$

Assuming that $k_1 = 1$, $k_2 = \frac{x_1}{\delta(H)}$, and $k_3 = \frac{\omega(H)\rho}{x_3}$, then simplifying, (45) yields

$$\frac{dL}{dt} \le \left(\omega(H)\left(1+\frac{\rho\alpha(H)}{x_3}\right) - \frac{x_1x_2}{\delta(H)}\right) I_A. \tag{46}$$

From

$$\begin{aligned}\mathcal{R}_{0H} = \frac{\omega(H)\delta(H)}{x_1x_2}\left(1+\frac{\rho\alpha(H)}{x_3}\right) &\Longrightarrow \left(1+\frac{\rho\alpha(H)}{x_3}\right)\\ &= \frac{\mathcal{R}_{0H}x_1x_2}{\omega(H)\delta(H)}.\end{aligned} \tag{47}$$

Substituting for $(1+\frac{\rho\alpha(H)}{x_3})$ in (46), we obtain

$$\frac{dL}{dt} \le \left(\frac{x_1x_2}{\delta(H)}(\mathcal{R}_{0H}-1)\right) I_A. \tag{48}$$

From inequality (48), it is realised that at minimum alcohol intake

$$\frac{dL}{dt} \le \left(\frac{x_1^*x_2^*}{\delta_0}(\mathcal{R}_{01}-1)\right) I_A,$$

where $x_1^* = \delta_0 + \mu$, and $x_2^* = \mu + \alpha_0 + \phi$. Similarly, at maximum alcohol intake,

$$\frac{dL}{dt} \le \left(\frac{x_1^{**}x_2^{**}}{\delta(H_{\max})}(\mathcal{R}_{02}-1)\right) I_A,$$

where $\delta(H_{\max}) = \delta_0 + B^*\tau$, $B^* = (\frac{H_{\max}-H_{\min}}{H_{\max}})$, $x_1^{**} = \delta(H_{\max}) + \mu$, $x_2^{**} = \mu + \alpha(H_{\max}) + \phi$.

If $\mathcal{R}_{0H} < 1$, then $\frac{dL}{dt} < 0$. Moreover, $\frac{dL}{dt} = 0$ only when $I_A = 0$ or $\mathcal{R}_{0H} = 1$. The largest compact invariant set within the region Ω for which $\{\frac{dL}{dt} = 0\}$ is the singleton set containing only the Hepatitis C-free equilibrium $\mathcal{E}_0$. Therefore, by LaSalle's Invariance Principle, the equilibrium point $\mathcal{E}_0$ is globally asymptotically stable in Ω whenever $\mathcal{R}_{0H} < 1$.

Specifically, the equilibrium $\mathcal{E}_{01}$ is globally asymptotically stable if $\mathcal{R}_{01} < 1$, and $\mathcal{E}_{02}$ is globally asymptotically stable if $\mathcal{R}_{02} < 1$. ■

This result shows that if the basic reproduction number is below 1, Hepatitis C will be eliminated from the population regardless of the initial infection level.

3.6. Local and global stabilities of endemic equilibrium point of Hepatitis C virus model

In this section, we look at the local and global stabilities of the EE point. If the EE point is stable, then it implies that HCV infection is being managed with minimal death occurrences.

3.6.1. Bifurcation analysis of the model

Using the Center Manifold Theory method presented by Castillo Chavez and Song [27], we investigate the local asymptotic stability of EE point. We need to first show that the model system (1) has six eigenvalues one of which is zero to determine whether bifurcation phenomena for the system (1) exists.

Let us denote $S(t) = r_1$, $E(t) = r_2$, $I_A(t) = r_3$, $I_C(t) = r_4$, $R(t) = r_5$, and $H(t) = r_6$. Using new state variables, model (1) takes the form

$$\begin{aligned}\frac{dr_1}{dt} &= \Lambda - \frac{\omega(H^*)(r_3+\rho r_4)r_1}{N} + \theta r_5 - \mu r_1 = f_1,\\ \frac{dr_2}{dt} &= \frac{\omega(H^*)(r_3+\rho r_4)r_1}{N} - x_1r_2 = f_2,\\ \frac{dr_3}{dt} &= \delta(H^*)r_2 - x_2r_3 = f_3,\\ \frac{dr_4}{dt} &= \alpha(H^*)r_3 - x_3r_4 = f_4,\\ \frac{dr_5}{dt} &= \psi r_4 + \phi r_3 - x_4r_5 = f_5,\\ \frac{dr_6}{dt} &= \epsilon(r_6 - H_{\min})\left(1 - \frac{r_6}{H_{\max}}\right) = f_6,\end{aligned} \tag{49}$$

where $x_1 = \mu + \delta(H)$, $x_3 = \mu + \gamma_C + \psi$, and $x_4 = \mu + \theta$. Let $\omega(H^*)$ be the bifurcation parameter and let $\mathcal{R}_{0H} = 1$, it follows that

$$1 = \left[\frac{\omega(H^*)\delta(H^*)}{x_1x_2x_3}\right]\left(x_3 + \rho\alpha(H^*)\right).$$

This implies that

$$\omega(H^*) = \frac{x_1x_2x_3}{\delta(H^*)\,(x_3 + \rho\alpha(H^*))} = \omega^*(H^*).$$

The Jacobian matrix $J^*(\mathcal{E}_0)$ evaluated at disease-free equilibrium, $\mathcal{E}_0$ and bifurcation parameter, $\omega^*(H^*)$ is constructed. Hence, we have

$$J^*_{\mathcal{E}_0} = \begin{pmatrix} -\mu & 0 & -\omega^*(H^*) & -\omega^*(H^*)\rho & \theta & 0 \\ 0 & -x_1 & \omega^*(H^*) & \omega^*(H^*)\rho & 0 & 0 \\ 0 & \delta(H^*) & -x_2 & 0 & 0 & 0 \\ 0 & 0 & \alpha(H^*) & -x_3 & 0 & 0 \\ 0 & 0 & \phi & \psi & -x_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & -x_5 \end{pmatrix}, \tag{50}$$

where $x_5 = \frac{\epsilon(2H^*-(H_{\min}+H_{\max}))}{H_{\max}}$.

Thus, at $\mathcal{R}_{0H} = 1$, the characteristic equation, $\mathcal{P}(\lambda)$ of (50) is obtained as

$$\lambda\left(\lambda^2 + (x_1+x_2+x_3)\lambda + x_1x_2\left(1-\frac{1}{b}\right) + x_1x_3 + x_2x_3\right) (-\mu-\lambda)(-x_4-\lambda)(-x_5-\lambda) = 0, \tag{51}$$

where

$$b = \left(1 + \frac{\rho\alpha(H)}{x_3}\right) > 1 \implies \frac{1}{b} < 1 \implies \left(1-\frac{1}{b}\right) > 0.$$

Since $x_1+x_2+x_3 > 0$ and $x_1x_2(1-\frac{1}{b}) + x_1x_3 + x_2x_3 > 0$, then $\mathcal{P}(\lambda)$ has five eigenvalues with negative real parts and one simple zero eigenvalue which determines that the bifurcation phenomena for the model system (49) exists.

It is also noted that since one of the eigenvalues is zero, then we can determine the existence of bifurcation phenomena for the model system (49).

The center manifold theory [27] is then applied to determine whether the system exhibits forward or backward bifurcation. Thus we need to calculate coefficients a and b (see Appendix A.2) to determine the nature of bifurcation and the local stability of the endemic equilibrium.

Let $\vec{w} = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ be the right eigenvector corresponding to a zero eigenvalue that satisfies the condition $J^*(\mathcal{E}_0)\vec{w} = 0$. This yields

$$\begin{aligned} w_1 &= \left(\frac{\delta(H^*)\theta(x_3\phi + \psi\alpha(H^*)) - x_1x_2x_3x_4}{x_2x_3x_4\mu}\right) w_2, w_2 > 0, \\ w_3 &= \left(\frac{\delta(H^*)}{x_2}\right) w_2, \\ w_4 &= \left(\frac{\alpha(H^*)\delta(H^*)}{x_2x_3}\right) w_2, \\ w_5 &= \left(\frac{\delta(H^*)(x_3\phi + \psi\alpha(H^*))}{x_2x_3x_4}\right) w_2, w_6 = 0. \end{aligned} \tag{52}$$

Similarly, the left eigenvector $\vec{v} = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ corresponding to a zero eigenvalue that satisfies the condition $\vec{v}J^*(\mathcal{E}_0) = 0$ is computed and yields

$$\begin{aligned} v_2 > 0, v_3 &= \left(\frac{x_1}{\delta(H^*)}\right) v_2, \\ v_4 &= \left(\frac{x_1x_2\rho}{\delta(H^*)(x_3+\rho\alpha(H^*))}\right) v_2, \\ v_1 &= v_5 = v_6 = 0. \end{aligned} \tag{53}$$

Also, the condition $\vec{w}.\vec{v} = 1$ yields

$$\begin{aligned} & v_2w_2\left[1 + \frac{x_1}{x_2} + \frac{x_1\rho\alpha(H^*)}{x_3(x_3+\rho\alpha(H^*))}\right] = 1, \\ & v_2w_2 = \frac{x_2x_3(x_3+\rho\alpha(H^*)) + x_1x_3(x_3+\rho\alpha(H^*)) + (x_1x_2\rho\alpha(H^*))}{x_2x_3(x_3+\rho\alpha(H^*))}, \\ & v_2 = x_2x_3(x_3+\rho\alpha(H^*)), \\ & w_2 = \frac{1}{x_2x_3(x_3+\rho\alpha(H^*)) + x_1x_3(x_3+\rho\alpha(H^*)) + (x_1x_2\rho\alpha(H^*))}. \end{aligned} \tag{54}$$

We then represent the k^{th} component of $f_i (i = 1, \ldots, 6)$ in Equation (49) to be $f_k (k = 1, \ldots, 6)$ with

$$\begin{aligned} a &= \sum_{i,j,k=1}^{6} v_k w_i w_j \frac{\partial^2 f_k}{\partial r_i \partial r_j}(0,0), \\ b &= \sum_{j,k=1}^{6} v_k w_j \frac{\partial^2 f_k}{\partial r_j \partial \omega_0^*}(0,0). \end{aligned} \tag{55}$$

To complete the bifurcation process, the Center Manifold Theory is used to calculate the coefficients a and b in Equation (55) by differentiating system (49). All second-order partial derivatives of $f_k (k = 1, \ldots, 6)$ with respect to $r_{i,j} (i,j = 1, \ldots, 6)$ at disease-free equilibrium are zero with exception of the following:

$$\begin{aligned} \frac{\partial^2 f_2}{\partial r_2 \partial r_3} &= -\frac{\omega^*(H)}{N} = \frac{\partial^2 f_2}{\partial r_3 \partial r_2}, \frac{\partial^2 f_2}{\partial r_2 \partial r_4} = -\frac{\omega^*(H)\rho}{N} \\ &= \frac{\partial^2 f_2}{\partial r_4 \partial r_2}, \frac{\partial^2 f_2}{\partial r_3 \partial r_3} = -2\frac{\omega^*(H)}{N}, \\ \frac{\partial^2 f_2}{\partial r_3 \partial r_4} &= -\frac{\omega^*(H)(1+\rho)}{N} = \frac{\partial^2 f_2}{\partial r_4 \partial r_3}, \frac{\partial^2 f_2}{\partial r_3 \partial r_5} = -\frac{\omega^*(H)}{N} \\ &= \frac{\partial^2 f_2}{\partial r_5 \partial r_3}, \frac{\partial^2 f_2}{\partial r_4 \partial r_4} = -2\frac{\omega^*(H)\rho}{N}, \\ \frac{\partial^2 f_2}{\partial r_4 \partial r_5} &= -\frac{\omega^*(H)\rho}{N} = \frac{\partial^2 f_2}{\partial r_4 \partial r_5}. \end{aligned} \tag{56}$$

Similarly, the non-zero second-order partial derivatives of $f_k (k = 1, \ldots, 6)$ with respect to $r_j (j = 1, \ldots, 6)$ and $\omega^*(H)$

evaluated around the disease-free equilibrium are as follows:

$$\frac{\partial^2 f_2}{\partial r_3 \partial \omega^*(H)} = \frac{S}{N} \approx 1, \frac{\partial^2 f_2}{\partial r_4 \partial \omega^*(H)} = \frac{\rho S}{N} \approx \rho. \quad (57)$$

The coefficients a and b are calculated to give

$$a = -2v_2 w_2^2 \delta(H^*) \frac{\omega^*(H^*)}{N x_2} \left[2 + \frac{\alpha(H^*)}{x_2 x_3} (2x_2\rho + 1 + \rho) + \frac{\delta(H^*)(x_3\phi + \psi\alpha(H^*))}{x_2 x_3^2 x_4} (x_3 + \alpha(H^*)) \right] < 0. \quad (58)$$

$$b = \frac{x_1 v_2 w_2}{\omega(H^*)} \mathcal{R}_{0H} > 0. \quad (59)$$

Thus, from Equations (58) and (59) it is seen that, $a < 0$ and $b > 0$, which implies that the model system (49) exhibits forward bifurcation at $\mathcal{R}_{0H} = 1$. Therefore, the above result is summarized as follows.

Theorem 3.6: *The disease endemic equilibrium point, $\mathcal{E}^*$, of the system* (1) *is locally asymptotically stable if $\mathcal{R}_{0H} > 1$ and unstable otherwise.*

To analyze the global stability of the endemic equilibrium point, a candidate Lyapunov function is assumed. If $L(x)$ is a Lyapunov function, and $\frac{dL}{dt} < 0$, it implies that the point at which the function exists is globally asymptotically stable.

Theorem 3.7: *The disease endemic equilibrium point, $\mathcal{E}^*$ of the system* (1) *is globally asymptotically stable in Ω if $\mathcal{R}_{0H} > 1$.*

Proof: Consider a Lyapunov function of the endemic equilibrium point when $\mathcal{R}_{0H} > 1$ to be of the form

$$L(x) = a_1 \left(x_1 - x_1^* + x_1^* \ln \frac{x_1^*}{x_1} \right) + a_2 \left(x_2 - x_2^* + x_2^* \ln \frac{x_2^*}{x_2} \right) + \cdots + a_n \left(x_n - x_n^* + x_n^* \ln \frac{x_n^*}{x_n} \right), \quad (60)$$

where x_i is the population of the i^{th} compartment and x_i^* is the endemic equilibrium point. Thus, letting $a_i = 1$, we obtain a Lyapunov function for the model system (1) as

$$L(x) = \left(S - S^* + S^* \ln \frac{S^*}{S} \right) + \left(E - E^* + E^* \ln \frac{E^*}{E} \right) + \left(I_A - I_A^* + I_A^* \ln \frac{I_A^*}{I_A} \right) + \left(I_C - I_C^* + I_C^* \ln \frac{I_C^*}{I_C} \right) + \left(R - R^* + R^* \ln \frac{R^*}{R} \right). \quad (61)$$

Thus, differentiating Equation (61) with respect to time, t, we obtain

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + \left(1 - \frac{I_A^*}{I_A}\right)\frac{dI_A}{dt} + \left(1 - \frac{I_C^*}{I_C}\right)\frac{dI_C}{dt} + \left(1 - \frac{R^*}{R}\right)\frac{dR}{dt}. \quad (62)$$

Substituting for $\frac{dS}{dt}, \frac{dE}{dt}, \frac{dI_A}{dt}, \frac{dI_C}{dt}$, and $\frac{dR}{dt}$ into (62) and simplification leads to

$$\begin{aligned} \frac{dL}{dt} &= \Lambda - \theta R \frac{S^*}{S} + \frac{\omega(H)(I_A + \rho I_C)S^*}{N} + \mu S^* \\ &+ (\mu + \delta(H))E^* + (\mu + \alpha(H) + \phi)I_A^* \\ &+ (\mu + \gamma_C + \psi)I_C^* + (\mu + \theta)R^* - \mu N - \gamma_C I_C \\ &- \Lambda \frac{S^*}{S} - \frac{\omega(H)(I_A + \rho I_C)S}{N}\frac{E^*}{E} \\ &- \delta(H)E\frac{I_A^*}{I_A} - \alpha(H)I_A\frac{I_C^*}{I_C} - (\psi I_C + \phi I_A)\frac{R^*}{R}. \end{aligned} \quad (63)$$

Re-arranging $\frac{dL}{dt}$ in terms of positives and negatives in the form

$$\frac{dL}{dt} = \mathcal{M} - \mathcal{N}.$$

It follows from (63) that

$$\begin{aligned} \mathcal{M} &= \Lambda + \frac{\omega(H)(I_A + \rho I_C)S^*}{N} + \mu S^* + (\mu + \delta(H))E^* \\ &+ (\mu + \alpha(H) + \phi)I_A^* \\ &+ (\mu + \gamma_C + \psi)I_C^* + (\mu + \theta)R^* \\ \mathcal{N} &= \mu N + \gamma_C I_C + \Lambda\frac{S^*}{S} + \theta R\frac{S^*}{S} + \frac{\omega(H)(I_A + \rho I_C)S}{N}\frac{E^*}{E} \\ &+ \delta(H)E\frac{I_A^*}{I_A} + \alpha(H)I_A\frac{I_C^*}{I_C} + (\psi I_C + \phi I_A)\frac{R^*}{R}. \end{aligned}$$

If $\mathcal{R}_{0H} > 1$, then $\mathcal{M} \leq \mathcal{N}$ and $\frac{dL}{dt} \leq 0$. Moreover, $\frac{dL}{dt} = 0$ if and only if $S - S^* = E - E^* = I_A - I_A^* = I_C - I_C^* = R - R^* = 0$. Therefore, the largest compact invariant set $\{(S, E, I_A, I_C, R) \in \Omega : \frac{dL}{dt} = 0\}$ is the singleton $\mathcal{E}^*$, where $\mathcal{E}^*$ denotes the endemic equilibrium point of system (1). It follows by LaSalle's Invariance Principle that $\mathcal{E}^*$ is globally asymptotically stable in Ω whenever $\mathcal{R}_{0H} > 1$.

Specifically, the equilibrium point $\mathcal{E}_1^*$ is globally asymptotically stable whenever $\mathcal{R}_{01} > 1$ and $\mathcal{E}_2^*$ is globally asymptotically stable whenever $\mathcal{R}_{02} > 1$. ■

3.7. Sensitivity analysis of the model

Local sensitivity analysis measures the relative contribution of each of the model parameters involved in HCV transmission, treatment and disease related mortality. Since the occurrence of early HCV infection is directly

Table 2. Sensitivity analysis of model parameters in $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$.

Parameter symbols	Sensitivity index of $\mathcal{R}_{01}$	Sensitivity index of $\mathcal{R}_{02}$
ω_0	$+1.0000 \times 10^{0}$	$+3.4210 \times 10^{-1}$
δ_0	$+3.1000 \times 10^{-3}$	$+2.7000 \times 10^{-3}$
ρ	$+7.0834 \times 10^{-4}$	$+7.5000 \times 10^{-3}$
α_0	-7.5470×10^{-2}	-4.3200×10^{-2}
ψ	-6.9474×10^{-4}	-7.4000×10^{-3}
ϕ	-8.8380×10^{-1}	-5.0890×10^{-1}
γ_C	-3.9601×10^{-6}	-3.8740×10^{-5}
B		$+2.4060 \times 10^{-1}$
τ		$+2.4070 \times 10^{-1}$

related to $\mathcal{R}_{0H}$, a sensitivity analysis of $\mathcal{R}_{0H}$ is performed to identify the most significant parameters that inhibit or accelerate the spread of HCV infection. A generalized forward sensitivity index is used to measure relative changes in model parameters for control of HCV. Therefore, the approach in [25, 28] is utilized to determine the proportional alteration of a state variable as the parameter changes and is defined as

$$\Gamma_g^{\mathcal{R}_{0H}} = \frac{\partial \mathcal{R}_{0H}}{\partial g} \times \frac{g}{\mathcal{R}_{0H}}. \tag{64}$$

Thus the sensitivity indices of $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ with respect model parameters in Table 3 are given in Table 2.

Table 2 provides a physical interpretation of each model parameter in $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$, respectively. It should be noted that $\mathcal{R}_{01}$ increases each time ω_0, δ_0, and ρ increase and $\mathcal{R}_{02}$ increases each time ω_0, δ_0, τ, B, and ρ increase. Thus these parameters should be targeted and efforts should aim at reducing their value if the HCV burden is to reduce.

Similarly, the parameters $\alpha_0, \psi, \phi, \gamma_C$ have a negative sign, which means that they have a negative effect on $\mathcal{R}_{01}$ and the same parameters negatively affect $\mathcal{R}_{02}$. Thus parameters ψ and ϕ should be increased in value since they are fundamental in HCV reduction.

Therefore, to control the spread and prevalence of HCV infection in the population, it is necessary to increase the treatment of chronically infected people and also influence the reduction of alcohol consumption to a minimum level so that the prevalence of HCV infection in the population can be reduced.

4. Numerical simulations

In this section, the numerical simulations of the model system (1) are provided. The model simulations are carried out by employing MATLAB programming tool with ODE45 in-built function to simulate the role of treatment and control in alcohol consumption to minimum levels on the dynamics of Hepatitis C virus infection. The parameter values used are obtained from the background literature

Table 3. Model parameter values.

Parameter symbols	Value(per year)	Source
Λ	8.000×10^{2}	Assumed
θ	3.650×10^{-10}	Assumed
ω_0	1.300×10^{-1}	Assumed
δ_0	4.420×10^{0}	Estimated
ρ	2.655×10^{-2}	Estimated
α_0	2.586×10^{-2}	Estimated
μ	1.360×10^{-2}	Estimated
ψ	9.500×10^{-1}	Estimated
ϕ	3.000×10^{-1}	Estimated
γ_C	5.000×10^{-3}	Estimated
ϵ	5.000×10^{-2}	Assumed
τ	5.000×10^{-1}	Assumed
$H_{\min}$	7.3 kg	[12]
$H_{\max}$	14.6 kg	[12]

and some are estimated in biologically feasible ways to obtain more realistic simulation results with initial conditions $S(0) = 10000, E(0) = 3500, I_A(0) = 3500, I_C(0) = 1500, R(0) = 10, H(0) = 10, H_{\min} = 7.3$, and $H_{\max} = 14.6$.

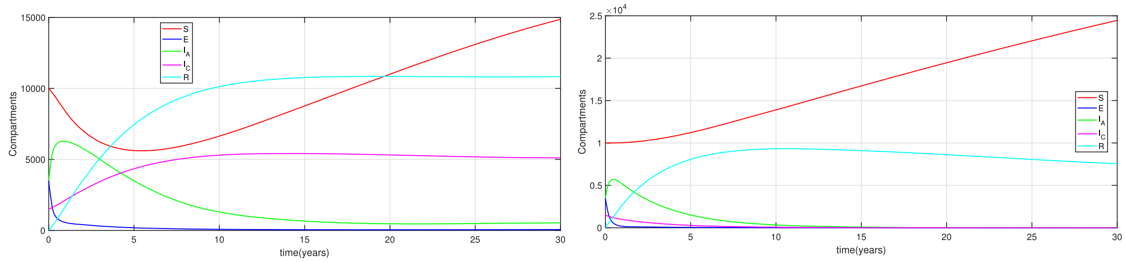
Considering parameter values in Table 3 and the above initial conditions, we simulate system (1) to obtain the following results.

In Figure 2(a), the number of acutely infected individuals initially rises before rapidly declining as cases progress to the chronic stage. Chronically infected individuals increase and eventually stabilize, indicating that without treatment, HCV persists in the population and the endemic equilibrium is locally stable. In contrast, Figure 2(b) shows a steady decline in both acute and chronic cases due to treatment, suggesting that HCV can be reduced as transmission rates drop. Hence, treatment lowers $\mathcal{R}_{0H}$ and leads to a disease-free state.

Figure 3 illustrates the variation in alcohol consumption under two scenarios: minimum consumption, corresponding to $\mathcal{R}_{0H} = \mathcal{R}_{01}$, and maximum consumption, corresponding to $\mathcal{R}_{0H} = \mathcal{R}_{02}$. At $\mathcal{R}_{01}$, the system reaches equilibrium at a minimum alcohol level, denoted by $H_{\min}$. In contrast, at $\mathcal{R}_{02}$, alcohol consumption increases progressively over the years.

Figure 4(a) shows that the number of chronically infected individuals decrease as the treatment rate increases, indicating an inverse relationship. Figure 4(b) illustrates that an increase in the treatment rate leads to a corresponding rise in the number of recovered individuals.

Figure 5 demonstrates that acutely infected individuals who consume minimal amounts of alcohol take longer to progress to the chronic stage compared to those who consume higher amounts. Figure 6(a) shows that, with treatment, chronically infected individuals who consume minimal alcohol recover more quickly than those with high alcohol intake. Figure 6(b) illustrates that, in the absence of treatment, the number of chronically infected



(a) (b)

Figure 2. Simulating human population when $\mathcal{R}_{0H} > 1$ and when $\mathcal{R}_{0H} < 1$: (a) when $\mathcal{R}_{0H} > 1$ and (b) when $\mathcal{R}_{0H} < 1$.

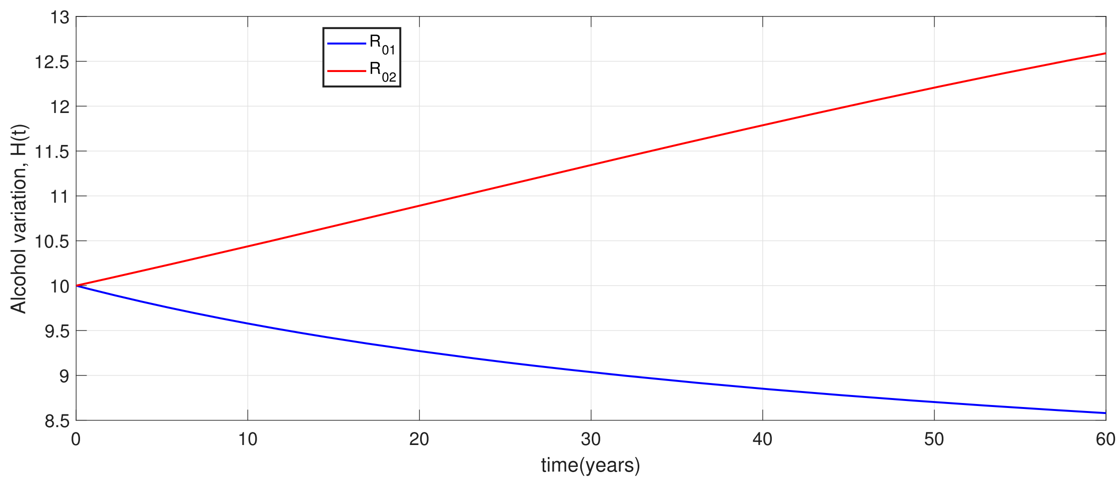


Figure 3. Simulating alcohol variation at $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$.

individuals with high alcohol consumption is greater than those with low alcohol consumption. These observations highlight the positive impact of reduced alcohol intake on recovery rates.

We further examine the combined effects of treatment and alcohol control by analyzing the following strategies:

(a) No treatment with low alcohol consumption: $\psi = 0$ and $H = H_{\min}$, denoted as strategy S_1.
(b) No treatment with high alcohol consumption: $\psi = 0$ and $H = H_{\max}$, denoted as strategy S_2.
(c) Treatment with low alcohol consumption: $\psi = 0.95$ and $H = H_{\min}$, denoted as strategy S_3.
(d) Treatment with high alcohol consumption: $\psi = 0.95$ and $H = H_{\max}$, denoted as strategy S_4.

Figure 7(a) illustrates that strategies S_1 and S_3 are more effective in reducing the number of acutely infected individuals who progress to the chronic stage over time, compared to strategies S_2 and S_4. In Figure 7(b), both strategies S_3 and S_4 contribute to a decline in the number of chronically infected individuals. However, strategy S_3 yields the most significant reduction in chronic infections. This highlights the importance of enhancing treatment efforts and minimizing alcohol intake as key measures in controlling Hepatitis C infection.

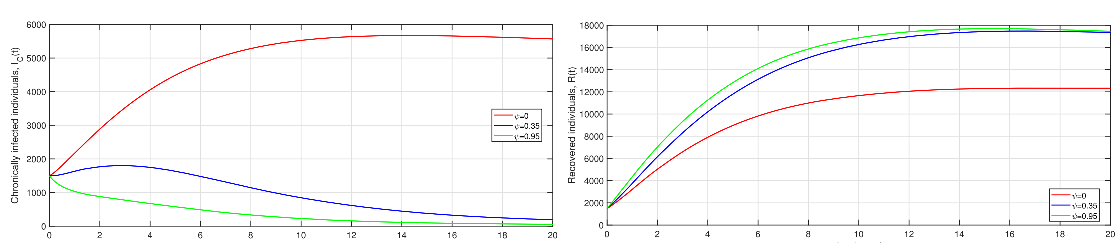


Figure 4. Simulating the effect of treatment on acutely and chronically infected population: (a) effect of treatment on chronically infected population and (b) effect of treatment on recovered population.

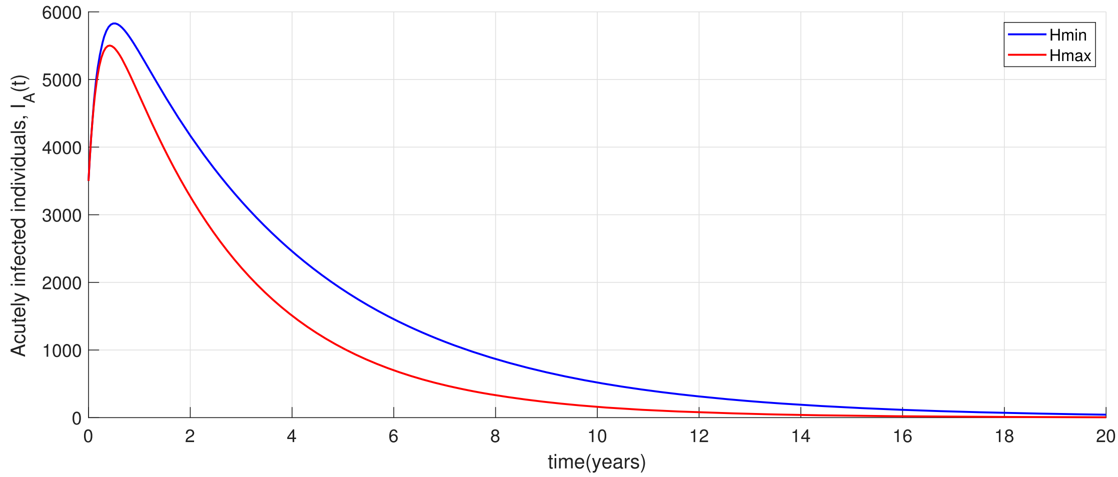


Figure 5. Effect of alcohol consumption on acute population.

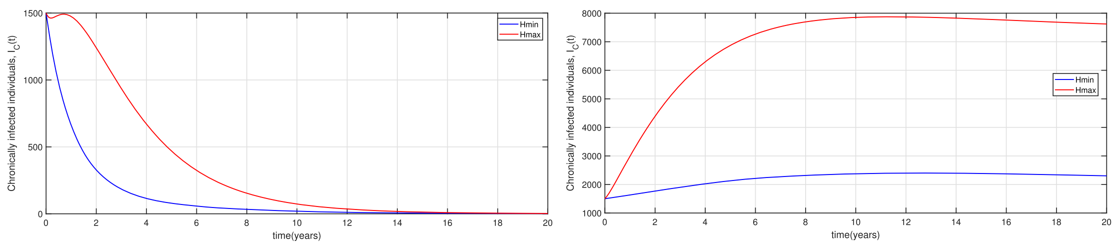


Figure 6. Simulating the effect of alcohol consumption on chronically infected population: (a) effect of alcohol consumption on chronic infection with treatment and (b) effect of alcohol consumption on chronic infection without treatment.

4.1. Conclusion

Hepatitis C remains a significant global public health threat. This study presents a deterministic mathematical model, incorporating a logistic alcohol intake model, to investigate the impact of treatment and alcohol consumption on the progression of HCV infection. The model system (1) was shown to be well-posed and biologically meaningful.

Through analysis, we established the existence of both disease-free and endemic equilibrium points for cases where $H(t) = H_{\min}$ and $H(t) = H_{\max}$. The basic reproduction number, $\mathcal{R}_{0H}$, was determined using the next-generation matrix approach. The disease-free equilibrium (DFE) point was proven to be locally and globally stable when $\mathcal{R}_{0H} < 1$, using the Routh–Hurwitz criterion and Lyapunov stability. The unique endemic equilibrium was shown to be locally and globally stable when $\mathcal{R}_{0H} > 1$, using the center manifold theorem and Lyapunov's approach.

Numerical simulations showed that increasing treatment for chronically infected individuals, along with reducing alcohol consumption to minimal levels, significantly decreased the number of chronically infected individuals. Xu et al. [29] found that HCV-infected individuals consuming more than 40 g of ethanol per day experienced faster progression of liver diseases compared to those with minimal alcohol intake. Additionally, alcohol abstinence was recommended to protect the liver,

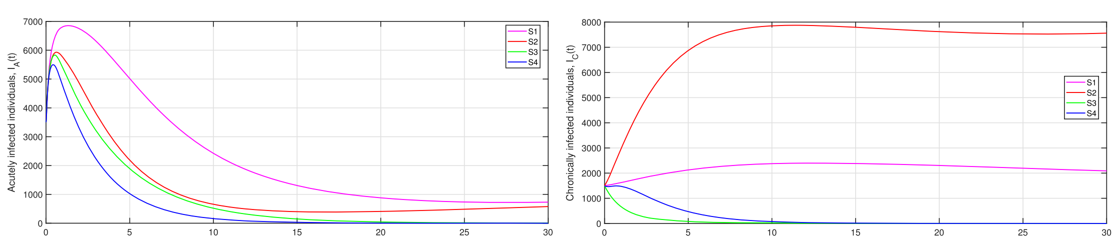


Figure 7. Simulating the combined effect of treatment and alcohol variation on infected population: (a) effect of strategies on acutely infected population and (b) effect of strategies on chronically infected population.

enhance the effectiveness of antiviral treatments, and slow the progression to chronic HCV infection, aligning with the findings of this study.

Thus, to effectively control HCV transmission and prevalence, a combination of reducing alcohol consumption to minimal levels, along with increasing treatment rates for chronically infected individuals, is essential.

To effectively control the transmission and prevalence of HCV, it is recommended to increase treatment rates for chronically infected individuals, promote alcohol abstinence or reduce alcohol consumption to minimal levels, and implement targeted public health campaigns that raise awareness about the risks of alcohol on HCV progression, combining these strategies for maximum impact.

Further research should focus on the effectiveness of public health interventions and the expansion of mathematical models to include co-infections and comorbidities in HCV transmission dynamics.

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Appendix

A.1 Proof of Theorem 3.4

Proof: The Jacobian matrix of system (1) should be evaluated at DFE, $\mathcal{E}_0$ to give

$$J_{\mathcal{E}_0} = \begin{pmatrix} -\mu & 0 & -\omega(H) & -\omega(H)\rho & \theta & 0 \\ 0 & -x_1 & \omega(H) & \omega(H)\rho & 0 & 0 \\ 0 & \delta(H) & -x_2 & 0 & 0 & 0 \\ 0 & 0 & \alpha(H) & -x_3 & 0 & 0 \\ 0 & 0 & \phi & \psi & -x_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & -x_5 \end{pmatrix} \quad \text{(A1)}$$

where $x_1 = \mu + \delta(H)$, $x_2 = \mu + \alpha(H) + \phi$, $x_3 = \mu + \gamma_C + \psi$, $x_4 = \mu + \theta$, and $x_5 = \frac{\epsilon(2H-(H_{\min}+H_{\max}))}{H_{\max}}$. The characteristic equation of (A1) is obtained from $|\lambda I - J_{\mathcal{E}_0}| = 0$ such that

$$\mathcal{P}(\lambda) = |\lambda I - J_{\mathcal{E}_0}| = \begin{vmatrix} \lambda+\mu & 0 & \omega(H) & \omega(H)\rho & -\theta & 0 \\ 0 & \lambda+x_1 & -\omega(H) & -\omega(H)\rho & 0 & 0 \\ 0 & -\delta(H) & \lambda+x_2 & 0 & 0 & 0 \\ 0 & 0 & -\alpha(H) & \lambda+x_3 & 0 & 0 \\ 0 & 0 & -\phi & -\psi & \lambda+x_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \lambda+x_5 \end{vmatrix} = 0. \quad \text{(A2)}$$

Clearly from (A2), the eigenvalues of the characteristic equation are six and the first three are $\lambda = -\mu, \lambda = -x_4, \lambda = -x_5$ which are all negative.

The other three eigenvalues can be obtained from the reduced matrix M given as

$$M = \begin{pmatrix} \lambda+x_1 & -\omega(H) & -\omega(H)\rho \\ -\delta(H) & \lambda+x_2 & 0 \\ 0 & -\alpha(H) & \lambda+x_3 \end{pmatrix}. \quad \text{(A3)}$$

The remaining eigenvalues are determined using the Routh–Hurwitz criterion [30]. The characteristic equation of (A3) is of the form $\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0$ such that $C_1 = x_1 + x_2 + x_3 > 0, C_2 = x_1x_2(1 - \frac{\mathcal{R}_{0H}}{b}) + x_1x_3 + x_2x_3$, and $C_3 = x_1x_2x_3(1 - \mathcal{R}_{0H})$. Since $b = 1 + \frac{\rho\alpha(H)}{x_3} > 1$ then $\frac{\mathcal{R}_{0H}}{b} < 1$ if $\mathcal{R}_{0H} < 1$ which implies that $C_2 > 0$, and if $\mathcal{R}_{0H} < 1$ then $C_3 > 0$.

By Routh–Hurwitz criterion for a polynomial of degree 3, the roots of $\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0$ have negative real parts $\Rightarrow C_1, C_2, C_3 > 0$ and $C_1C_2 > C_3$. It has already been established that C_1, C_2, and C_3 are positive, the task now is to show that $C_1C_2 > C_3$. Thus

$$C_1C_2 - C_3 = x_1x_2\left(1 - \frac{\mathcal{R}_{0H}}{b}\right)(x_1 + x_2 + x_3) + 2x_1^2x_3 + x_2^2x_3 + x_2x_3^2 + x_1x_2x_3\mathcal{R}_{0H} > 0, \quad \text{(A4)}$$

if $\mathcal{R}_{0H} < 1$. This implies that the necessary conditions for Routh–Hurwitz criteria for third-degree polynomials are satisfied. Hence, DFE, $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_{0H} < 1$. ■

A.2 Bifurcation analysis

Theorem A.1: *Consider the following general system of ODES with a parameter υ.*

$$\frac{dr}{dt} = f(r, \upsilon), \quad \text{(A5)}$$

$f : \mathbb{R}^n \times R \longrightarrow \mathbb{R}^n$, $f \in C^2(\mathbb{R}^n \times R)$ where zero is an equilibrium point of the system that is $f(0, \upsilon) \equiv 0$ for all υ.

Assume the following:

A1 *$\mathcal{A} = Drf(0,0) = (\frac{\partial f_i}{\partial r_j}(0,0))$ is the linearization matrix of the system (A5) around equilibrium zero with υ evaluated at 0. Zero is a simple eigenvalue of $\mathcal{A}$ and other eigenvalues have negative real parts.*

A2 *Matrix $\mathcal{A}$ has a non negative right eigenvector w and a left eigenvector v each corresponding to the zero eigenvalue.*

Let f_k be the k^{th} component of f and

$$a = \sum_{i,j,k=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial r_i \partial r_j}(0,0)$$
$$b = \sum_{j,k=1}^{n} v_k w_j \frac{\partial^2 f_k}{\partial r_j \partial \upsilon^*}(0,0). \quad \text{(A6)}$$

The local dynamics of the system around 0 is completely determined by the sign of a and b.

(1) *$a > 0, b > 0$, when $\upsilon < 0$ with $|\upsilon| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \upsilon \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.*
(2) *$a < 0, b < 0$, when $\upsilon < 0$ with $|\upsilon| \ll 1$, 0 is unstable; when $0 < \upsilon \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium.*
(3) *$a > 0, b < 0$, when $\upsilon < 0$ with $\upsilon \ll 1$, 0 is unstable and there exists a locally asymptotically stable negative equilibrium; when $0 < \upsilon \ll 1$, 0 is stable and a positive unstable equilibrium appears.*
(4) *$a < 0, b > 0$ when υ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable. Particularly if $a > 0, b > 0$, then a backward bifurcation occurs at $\upsilon = 0$.*