CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187

http://doi.org/10.5281/zenodo.4773952

Online at: http://www.iajps.com *Research Article*

NUMERICAL MODELING OF INTERNAL TRANSMISSION DYNAMICS HEPATITIS C VIRUS

[1]Sahar Noor, [2]Dr. Baseera Imran, [3]M Hassaan ul Haq

[1]MPhil In Mathematics, University of Lahore

[2]MBBS MPhil in Physiology,

[3]MS In Management in Sciences, University ofLahore.

Article Received: April 2021 **Accepted:** April 2021 **Published:** May 2021

Abstract:

In this paper, a mathematical model for internal transmission dynamics of HCV is formulated, based on the four-population immunized populace, an inoculated uncovered populace, recovered individual and immune response T cells populace. The model is analyzed by explicit numerical scheme i.e. Non Standard Finite Difference (NSFD) scheme preserve the monotonicity of solution irrespective of step size. Result are compared with conventional numerical scheme like Forward Euler's method and RK-4. Unlike Forward Euler's and RK-4 methods are failed for large time step; the proposed NSFD scheme is not only independent of time size but also conserve the stability of continuous dynamical behavior.

Keywords: Epidemic, cirrhosis, hepatocellular carcinoma, T cells, threshold parameter

Corresponding author:

Sahar Noor,

MPhil In Mathematics, University of Lahore

QR code

Please cite this article in press Sahar Noor et al., ***Numerical Modeling Of Internal Transmission Dynamics Hepatitis C Virus*** *, Indo Am. J. P. Sci, 2021; 08(05).*

INTRODUCTION:

Hepatitis C virus (HCV) is an contagious virus that causes acute hepatitis and chronic liver diseases leading to liver cirrhosis and hepatocellular carcinoma [1].It is a global epidemiology with over 170–200 million people have chronic hepatitis C virus (HCV) infection [2][3][4].

25% of hepatocellular carcinomas, 30% of all cases of cirrhosis and over 350000 mortality rate are attributable to this infection [5] [6]. .Hepatitis C Virus RNA is analyzed with the polymerase chain reaction (PCR) to examine the infectivity [7]. HCV genotype not only narrate the effects of treatment but also history of disease. Mathematical models are formulate to understand immunologic response to virus and effectiveness of the drug therapy. Appropriate mathematical models can not only define the dynamics of immune response and the effectiveness of drug therapy but also provide the biological answers withpathogenesis [8]. Our Epidemic mode possess four variables: Healthy liver cells H_S, infected liver cells H_i, Virusload V, and immune response T cells. The few assumptions are considered as β_S is a constant rate for the forming of healthy liver cells, H_S and μ_S is a constant rate for die out. H_S cells are infected at the rate proportional to the product of H_S *and* V with proportionality constant k; andonce infected die with the constant rate μ_i T cells eradicate infected cells H_i at the rate proportional to the product of H_i and T with proportionality constant δ.

Modified SIR Epidemic Model

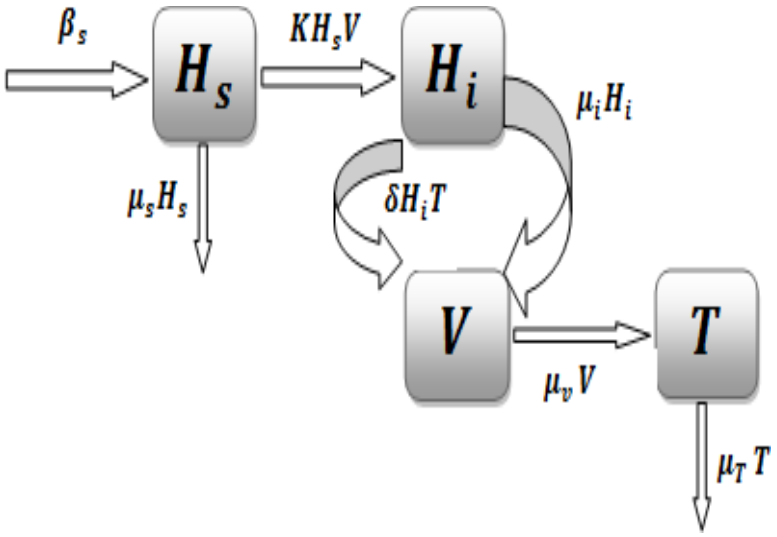

Figure 1: Compartmental model of internal dynamics of HCV Virus

Where,

Susceptible liver cells
Rate of growth of susceptible liver cellsRate of death of susceptible liver cells Infected liver cells
Rate of death of infected liver cellsVirus load
The response of cell on the dynamics of infectionGrowth rate of T cells
Maximum T cell population levelRate of death of T cells

System of Differential Equation
The following differential equations are obtained from the above model.
Where, then epidemic will exist

CONDITION FOR EPIDEMIC
is a basic reproductive number or threshold parameter for the proposed model.

Equilibrium points of the model
The two equilibrium points of the epidemic model are $E1$ = ($I0$) = Disease Free Equilibrium(DFE) if and $E2$ = (I_i) = *Endemic Equilibrium (EE) if*

Locally Stability of DFE Equilibrium
To analyze the local stability of the DFE point i.e. $E1$= ($I0$) =

$$J=\begin{bmatrix} -KV-\mu_S & 0 & -KH_S & 0 \\ KV & -\delta T-\mu_i & KH_S & -\delta H_i \\ O & \rho & -\mu_V & 0 \\ 0 & 0 & \beta_T\left(1-\frac{T}{T_{\max}}\right) & \frac{-\beta_T V}{T_{\max}}-\mu_T \end{bmatrix}$$

Clearly two Eigen values are . The two remaining two eigen values can be find out by
We know that if then, then the fixed point is stable.
Hence points are stable i.e.

NUMERICAL ANALYSIS
NSFD scheme was first introduced by R.E. Mickens in 1989. In different areas numerous discretization of non-linear equations have been established. According to Mickens the NSFD is defined as "A non-standard finite variance scheme is any discrete depiction of a system of differential equations that is based on the given rules

NSFD Method
Let H^n, H^n, V^n and T^n denote the value of H_S, H_i, V and T at t=n. Using non-standard finitedifference Modeling theory system (2) can be written as fellows

Convergence Analysis
The stability of the DFE point for the proposed NSFD scheme is discussed here, by consideringthe first equation of system (1)

Let

$$F^*=\begin{bmatrix} \frac{1}{1+\mu_s h}-\lambda & 0 & -\frac{\left(\frac{\beta_s}{\mu_s}+h\beta_s\right)(hK)}{(1+\mu_s h)^2} & 0 \\ 0 & \frac{1}{1+\mu_i h}-\lambda & \frac{hK\frac{\beta_s}{\mu_s}}{1+\mu_i h} & 0 \\ 0 & \frac{\rho h}{1+\mu_v h} & \frac{1}{1+\mu_v h}-\lambda & 0 \\ 0 & 0 & \frac{h\beta_T}{1+h\mu_T} & \frac{1}{1+h\mu_T}-\lambda \end{bmatrix}$$

Clearly

Lemma

Let and be the Eigen values of Jacobian matrix J and A and B be the trace and the determinantof the same Jacobean, Then the absolute values of and are less than unity if;

I. 1-A+B > 0
II. 1+A+B>0
III. B<1

(i) 1-A+B > 0
1 - +

(ii) 1 + A + B > 0

(iii) B < 1

Which is true

All values in nominator and denominator are positive as . As all the condition of above lemmaare satisfied Therefore, we can conclude that the DFE point is stable for all step sizes h whenever

Numerical Experiments

The following parameter values are used to perform numerical simulations,

Table 1: Parameter of the Values

Equilibrium Points		
Parameter	DFE	EE
$\beta s\ \mu s$	5000	5000
μs	0.2	0.2
μT	0.2	0.2
μi	0.5	0.5
μv	5	5
K	0.00003	0.00003
P	100	200
Δ	0.00001	0.00001
βT	0.00003	0.00003

Numerical Analysis of Schemes

All numerical experiments are performed by using MAT-Lab using parameter values of table2.1.

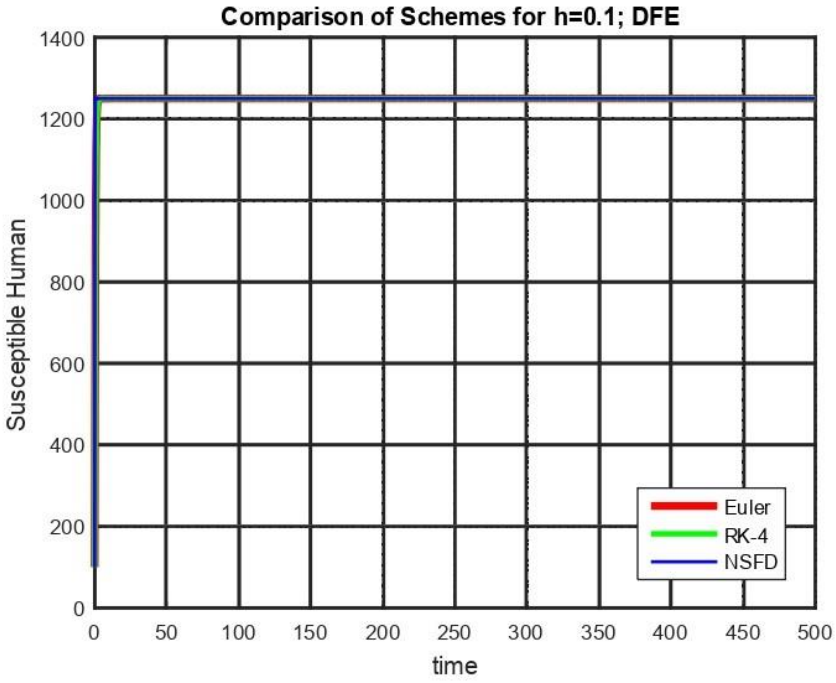

Fig 2: Comparison of Schemes of DFE (Susceptible Human)

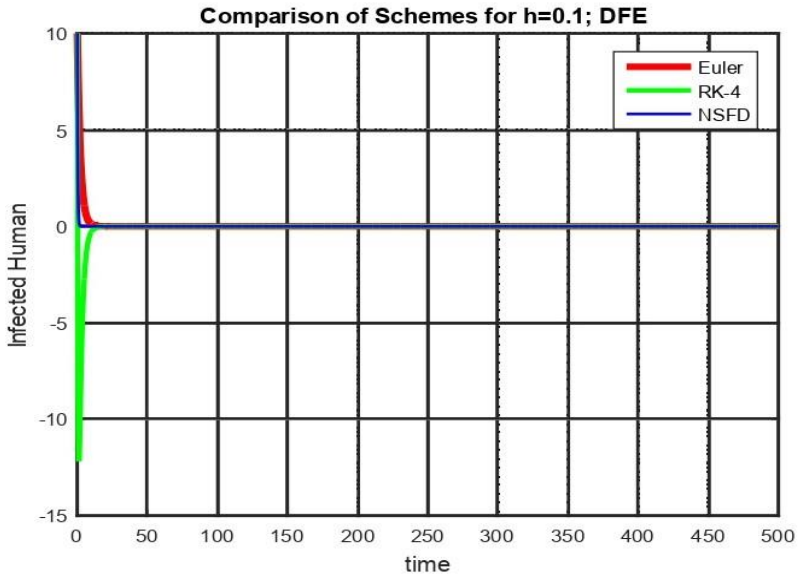

Fig 3: Comparison of Schemes of DFE (Infected Human)

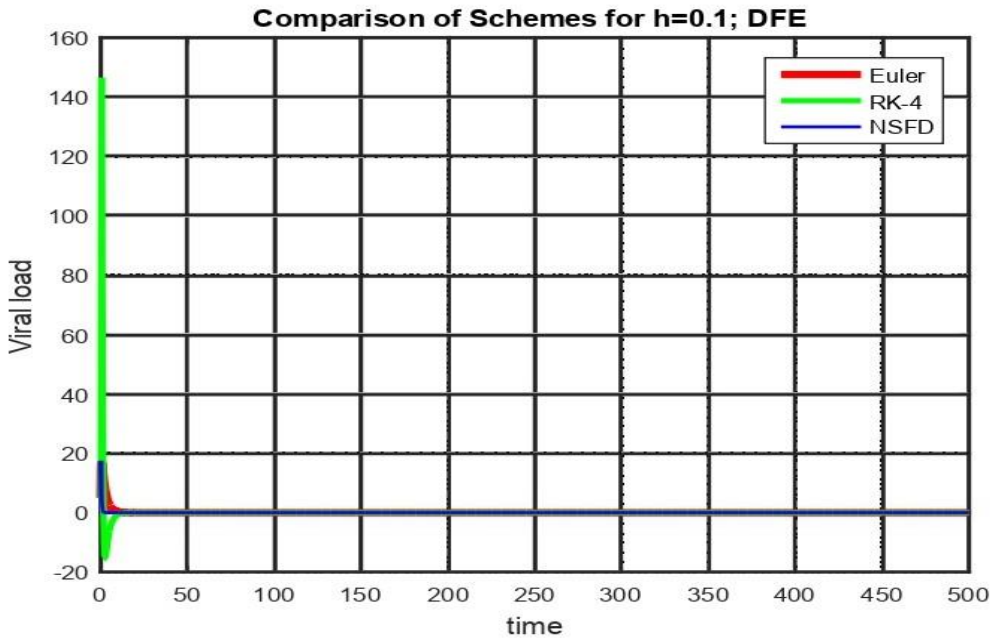

Fig 4: Comparison of Schemes of DFE (Viral Load)

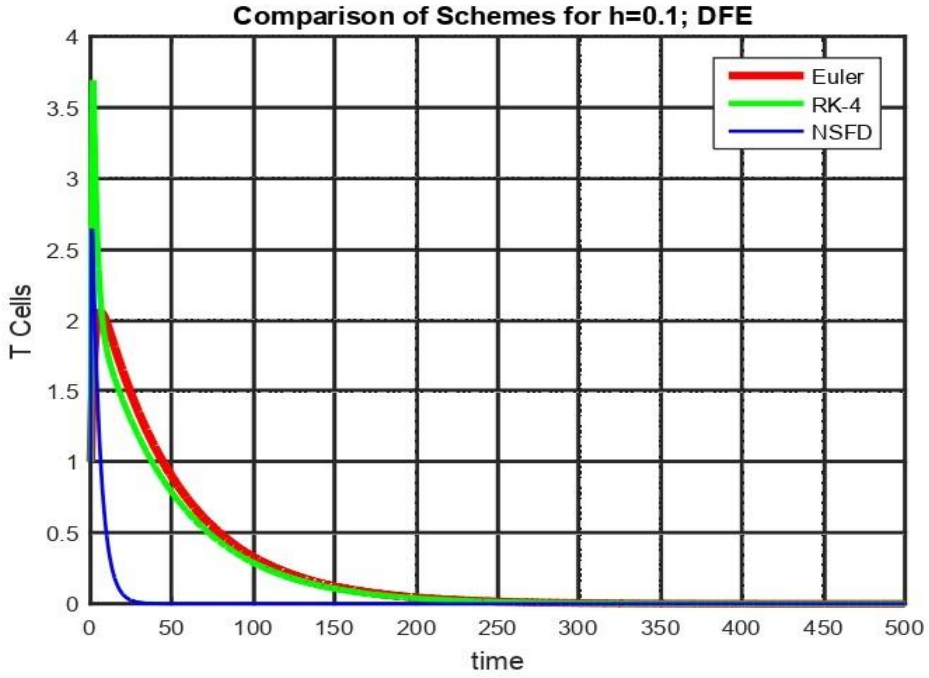

Fig 5: Comparison of Schemes of DFE (T Cells)

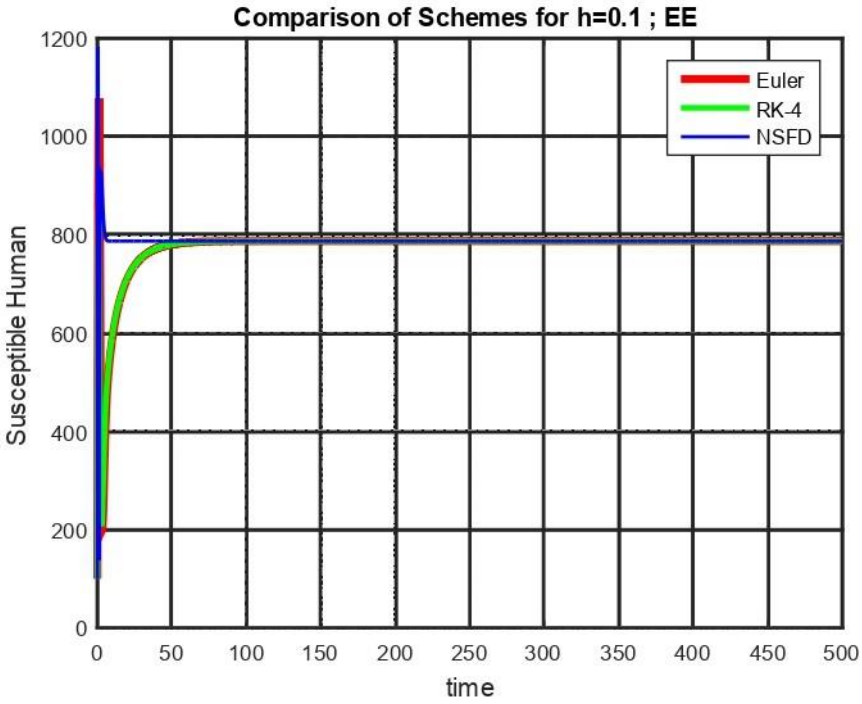

Fig 6: Comparison of Schemes of EE (Susceptible Human)

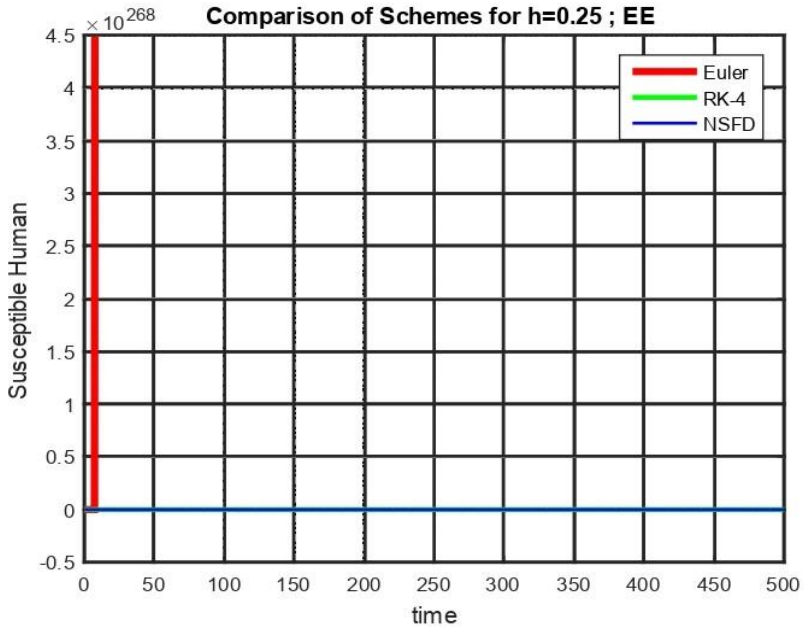

Fig 7: Comparison of Schemes of EE (Susceptible Human)

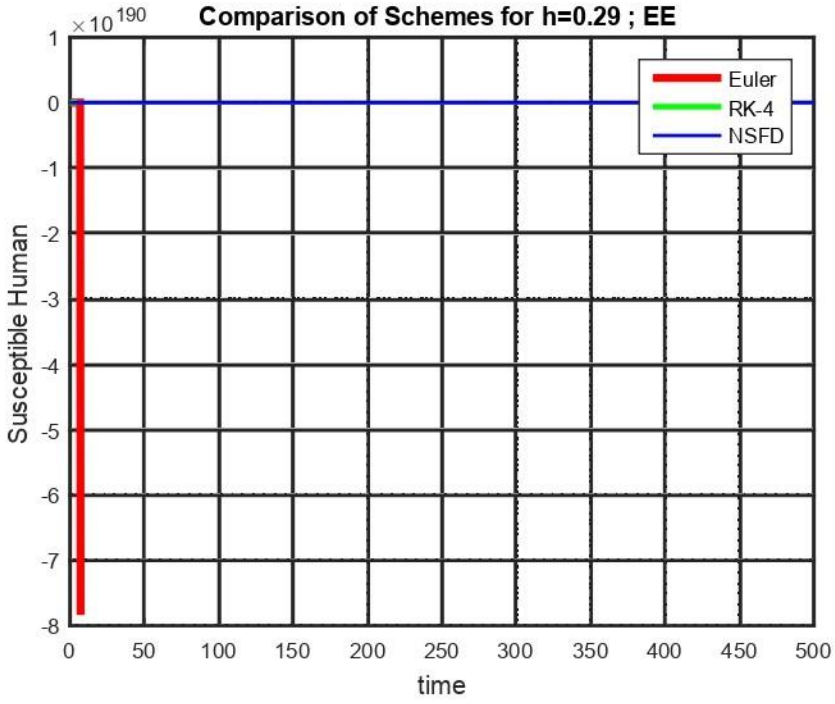

Fig 8: Comparison of Schemes of EE (SusceptibleHuman)

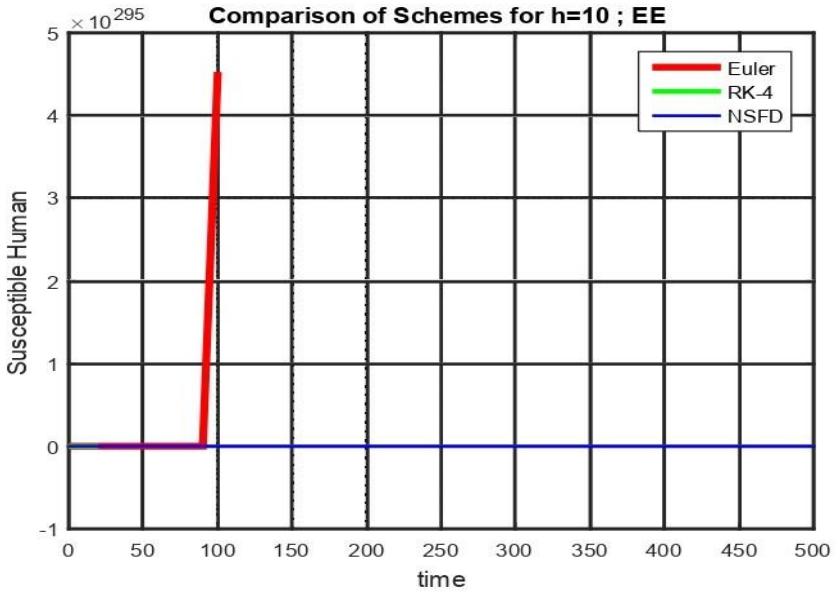

Fig 9: Comparison of Schemes of EE(Susceptible Human)

REFERENCES:

1. H. B. El-Serag, "Hepatocellular carcinoma: recent trends in the united states,"*Gastroenterology*, vol. 127, no. 5, pp. S27–S34, 2004.
2. W. H. Organization *et al.*, "Hepatitis c—global prevalence (update)," *Weekly Epidemiological Record= Releve´e´pide´miologique hebdomadaire*, vol. 75, no. 03, pp. 18–19, 2000.
3. K. M. Hanafiah, J. Groeger, A. D. Flaxman, and S. T. Wiersma, "Global epidemiology of hepatitis c virus infection: new estimates of age-specific antibody to hcv seroprevalence," *Hepatology*, vol. 57, no. 4, pp. 1333–1342, 2013.
4. G. Cooke, M. Lemoine, M. Thursz, C. Gore, T. Swan, A. Kamarulzaman, P. DuCros, and N. Ford, "Viral hepatitis and the g lobal b urden of d isease: a need to regroup," *Journal of viral hepatitis*, vol. 20, no. 9, pp. 600–601, 2013.
5. R. Lozano, M. Naghavi, K. Foreman, S. Lim, K. Shibuya, V. Aboyans, J. Abraham, T. Adair, R. Aggarwal,
6. S. Y. Ahn *et al.*, "Global and regional mortality from 235 causes of death for 20 agegroups in 1990 and 2010: a systematic analysis for the global burden of disease study 2010," *The lancet*, vol. 380, no. 9859, pp. 2095–2128, 2012.
7. M. G. Ghany and N. Gara, "Quest for a cure for hepatitis c virus: the end is in sight," *The Lancet*, vol. 384, no. 9941, pp. 381–383, 2014.
8. D. R. Gretch, "Diagnostic tests for hepatitis c," *Hepatology*, vol. 26, no. S3, pp. 43S–47S, 1997.
9. R. Avendano, L. Esteva, J. Flores, J. Allen, G. Go´mez, and J. Lo´pez- Estrada, "A mathematical model for the dynamics of hepatitis c," *Computational and Mathematical Methods in Medicine*, vol. 4, no. 2, pp. 109–118, 2002.