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# **Implementation of Mathematical Models for HCV Infection**

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# ABSTRACT

Hepatitis C Virus (HCV) is an infectious viral particle which transmits via blood contact. In this article the transmission dynamics have been simulated to visualize the propagation of HCV in given population which in turn would be helpful to develop the control strategies against disease. Therefore, differential equations have been used to evaluate the expression and MATLAB is used to visualize the obtained results. Furthermore, models for implementation on epidemic or viral diseases are more efficient to predict their progression pattern and transmission statistics that is more helpful in designing control strategies and prevention techniques. In case of viral diseases such as, HCV, SEIR model was found more applicable and helpful as it utilizes the population based data and provides the graphical result as per our designed tool for the study. Several parameters which are probability based are considered to manipulate the information regarding the transmission pattern of HCV infection.

KEYWORDS: HCV, SIR-model, transmission dynamics, simulation.

# **1. INTRODUCTION**

## **Hepatitis C:**

The root cause of HCV is Hepatitis virus which affects liver directly. During First four to six months of infection referred as acute form of Hepatitis C severe symptoms are observed in the patients. Whereas, there are mainly no symptoms of disease in early stages in chronic form which later on gets more severe and may be converted into liver carcinoma. [8] Major mode of disease is transfer is via blood contact and sexual intercourse with infected person or from infected pregnant woman to fetus, but the increasing use of drugs/narcotics results in increase of disease incidence day by day as sharing of needle can also result in disease, this shared needle in hospitals can also spread disease in health care units due to mishandling or carelessness of hospital staff. Use of unsterilized equipment during body piercing and tattoos encarving has also increased the disease incidence [1]

According to annual report of CDC, many people with Hepatitis C do not even know about their infection during a symptomatic early phase of disease. If symptoms occur, they may include: fever, feeling tired, appetite loss, bad stomach, abnormal colored excretion, joint pain, and yellowing of skin and eyes.[9] In acute stage of disease symptoms might appear in 3-4 weeks. In case of chronic infection (long asymptomatic phase) probability is that patient is diagnosed only when the infection has been developed to carcinoma of liver.

**SIR model:** In 1927, W. O. Kermack and A. G. McKendrick created a model with three fixed class consisting of three compartments i.e. susceptible S(t), infected I(t), and removed/recovered R(t). The individuals in a population is at susceptible class S at time t, which after acquiring infection is moved to infected class I, which afterwards is followed by removal either by immunity or by natural death.[5]

If  $\beta$  represents the spread rate in the class *I* and *r* the removal rate from the class *R* then the scheme can be modeled as the set of following differential equations:

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - rI$$
$$\frac{dR}{dt} = rI$$

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#### 2. SEIR Model and Simulation Process

This paper is an effort to simulate a well-known SEIR epidemic model, which considers each individual as being in one of four discrete states – susceptible S, exposed E, infected I, and resistant R. Initially an individual in class S can become a member of the class E after contact with an individual in class I. Exposed individuals cannot infect further at this stage but they eventually become infected and can infect others. Finally, after some time, infected individual becomes resistant, *i.e.* non-infectious and no longer susceptible to the disease.[7]

The SIR model discussed above takes into account only those diseases which cause an individual to be able to infect others immediately upon their infection. Many diseases have what is termed a latent or exposed phase, during which the individual is said to be infected but not infectious.

 $S \to E \to I \to R$ 

In this model the host population (*N*) is broken into four compartments: susceptible, exposed, infectious, and recovered, with the numbers of individuals in a compartment, or their densities denoted respectively by S(t), E(t), I(t), R(t), here *B* is the birth rate and  $\mu$  is the death rate while  $1/\varepsilon$  is termed as latent period of disease and  $1/\gamma$  is infectious time period, that is N = S(t) + E(t) + I(t) + R(t).[6]

$$\frac{dS}{dt} = B - \beta SI - \mu S$$
$$\frac{dE}{dt} = \beta SI - (\varepsilon + \mu)E$$
$$\frac{dI}{dt} = \varepsilon E - (\gamma + \mu)I$$
$$\frac{dR}{dt} = \gamma I - \mu R$$

#### METHODOLOGY

# Figure 1. Flow diagram indicating working of SIR model for disease transmission.



Our methodology was simple and based on the probabilistic values and we were considering population divided in sub groups of susceptible, infected and recovered individuals. The common thing was disease contact rate among these individuals if the effective contact rate (a threshold value) is less than zero then the population will be considered as disease free population but if the contact rate is greater than zero than population is diseased population and according to the predicted results control strategies will designed.

We have summarized all the variables and parameters the analysis,

Table 1. Variables and parameters summary used in differential equations. [2]

Following are the variables used in our analysis,	$\gamma$ : Recovery rate of isolations
N (t): Total population	<i>f</i> : Fraction of isolated that becomes susceptible
S (t): Population of susceptible individuals	$\xi$ : Progression rate from acute to chronic
A (t): Population of individuals with acute Hep C	$\alpha$ : Isolation rate of chronic individuals
C(t): Population of individuals with chronic Hep C	$\kappa$ : Natural recovery rate for acute individuals
Q(t): Population of isolated individuals	$\psi$ : Recovery rate for chronic individuals
R (t): Population of recovered individuals	$\omega$ : Progression rate of recovered individuals to susceptible
Following are the parameters used in our analysis,	individuals
Π: Recruitment rate	$\beta$ : Effective contact rate
$\mu$ : Natural death rate	$\eta$ : Modification parameter for reduction in infectiousness of acute
$\delta_a$ : Disease-induced death rate for individuals with acute Hep C	individuals
$\delta_c$ : Disease-induced death rate for individuals with chronic Hep C	$\zeta$ : Modification parameter for reduction in infectiousness of
$\delta_q$ : Disease-induced death rate for isolated individuals	quarantined individuals

Using the above variables and parameters the disease transmission scheme can be modeled with the following set of differential equations,

$$\begin{aligned} \frac{dS}{dt} &= \Pi + \omega R - \lambda S - \mu S, \\ \frac{dA}{dt} &= \lambda S + \gamma (1 - f)Q - (\xi + \kappa + \mu + \delta_a)A, \\ \frac{dC}{dt} &= \xi A - (a - \psi + \mu + \delta_c)C, \\ \frac{dQ}{dt} &= aC - (\gamma + \mu + \delta_q)Q, \\ \frac{dR}{dt} &= \gamma fQ + \kappa A + \psi C - (\omega + \mu)R, \end{aligned}$$

Where,

# 3. RESULTS AND DISCUSSIONS

 $\lambda = \frac{\beta(\eta A + C + \zeta Q)}{N}$ 

This tool is helpful in the prediction of progression rate of disease in population and further the very advantage is that it can help researchers to predict the diagnosis strategy against HCV infection. In this project we were more leaned towards the development of a platform one can input the required parameters according to the population based study.

# **Parameter and Results Interface:**



Figure 2. Parameter intake GUI and results showing panel.



Figure 3. Results based on the parameters passed to tool.

Using MATLAB 2009Ra we have design a GUI based tool which can simulate the transmission of population from susceptible to infected and from infected to recovered. For the project the parameter values used are according to Elbasha et al.2013.[7]

Figure 3 depicts results indicating high susceptibility rate towards HCV, where acute infected individuals are more than chronic individuals and the recovery rate is higher in quarentined individuals.

The values of A(t), C(t), Q(t), and R(t) does not show any major change in the values and not even minor changes. On the other hand, Population of susceptible individuals can have major impact.

The susceptibility rate highly affects the population transmission from one compartment to other and once an

SIR		
Total Dopulation	0.12	SIR Model
Ponulation of suscentible individuals	0.654	140
Population of individuals with acute HepC	0.2654	Acute
Population of individuals with chronic Hep C	0.6454	120 Chronic
Population of isolated individuals	0.2654	Isolated
Population of recovered individuals	0.2646	100 Recovered
Effective contact rate (beta)	0.55	
Reduction in infectiousness of acute individuals (E)	0.664	Q 80
Progression rate of recovered individuals to	0.6116	
susceptible individuals	0.64613	0 00
Natural Death rate (meu) Bacovory rate of isolations (namma)	0.646	40
Fraction of isolated that becomes suscentibles (f)	0.464	
Natural recovery rate for acute individuals (K)	0.2464	20
Death rate for individuals with acute Hep C (sigmaa)	0.2654	
Death rate for individuals with chronic Hep C (Sigmac)	0.6541321	
Death rate for isolated individuals (sigmaq)	0.1154	0 10 20 30 40 50 60 Time in Davs
Isolation rate of chronic individuals (alpha)	0.4236	
Recovery rate for chronic individuals (psi)	0.693234	Back Close Results
	0.147777	

individual is moved from susceptible to exposed/infected compartment it will affect the rest of susceptible population.

FIGURE 4. Parameter variation in heterogeneous population along with varied parameters. The green line shows increased acute infection (A), red line shows higher chronic infection probabilities (C), while the susceptibility (S) far lesser than recovery rate (R).

# 4. Conclusion.

The results produced in the form of graphs are in accordance to the literature under study. The objectives mentioned are all achieved and a tool is working properly. A deterministic epidemic model for the spread of Hep C, which incorporates the possibility of an isolation state, is formulated. Global analysis of the equilibrium solution is performed. The existence of a disease-free equilibrium and an endemic equilibrium will be shown in graphs.

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