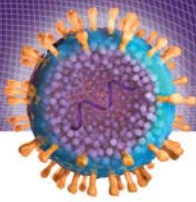


# Hepatitis C

A Media Handbook

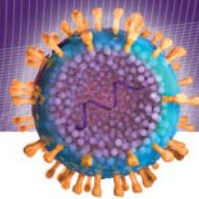




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## INTRODUCTION

Hepatitis C is a liver disease caused by infection with the hepatitis C virus (HCV). Chronic hepatitis C is a major public health problem that affects an estimated 170 million people worldwide and is a leading cause of liver failure, liver cancer and liver transplant.<sup>1</sup>

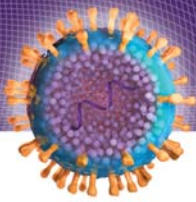
Although new cases of hepatitis C have declined in the United States over the past 20 years, new infections are only the tip of a very large iceberg.<sup>1</sup> Four million Americans – nearly four times the number of those with HIV – are believed to have hepatitis C, and three million are not aware they are infected.<sup>2-4</sup>

Who are the undiagnosed millions? An estimated two of every three people with chronic hepatitis C in this country are baby boomers born between 1946 and 1964 who were infected decades ago.<sup>2,4</sup> For these people, the consequences of not knowing they are infected can be deadly.

Hepatitis C is a silent killer that may produce no symptoms for decades. Often the first sign of illness occurs when a person's liver stops working or they develop liver cancer. Some physicians have compared the experience to suddenly "falling off a cliff." Since most people in the United States have had chronic hepatitis C for many years without knowing it, the number of people approaching the edge of this "cliff" is expected to skyrocket over the next 20 years.<sup>5</sup>

Unlike diseases such as hepatitis B and HIV/AIDS, hepatitis C can be cured.<sup>6</sup> But, available treatments for the most common form of hepatitis C in the United States (genotype 1) are given for 48 weeks and result in a cure for less than half of people treated.<sup>6-10</sup> New medicines for the treatment of hepatitis C are currently in development.

This handbook provides additional information on hepatitis C, current treatments and ongoing research for new medicines to treat this disease. It also includes information on external resources including organizations focused on hepatitis C and liver disease.



## WHAT IS HEPATITIS C?

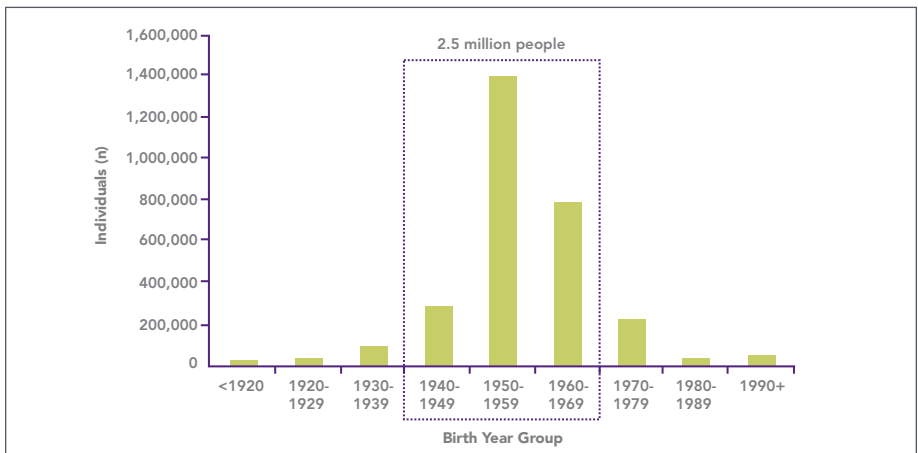
Hepatitis, which means “inflammation of the liver,” can be caused by infections, alcohol abuse, medication overdoses and abnormal immune system activity. In the case of hepatitis C, infection with the hepatitis C virus causes inflammation and liver damage over decades. Complications from hepatitis C cause an estimated 4,600 to 12,000 deaths each year in the United States, and hepatitis C-related deaths are expected to increase to nearly 40,000 per year by 2040.<sup>5, 11-13</sup>

## Who gets hepatitis C?

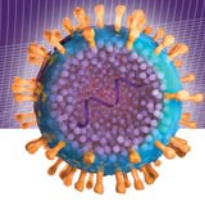
The hepatitis C virus is passed from person to person through infected blood. Many people were infected by blood transfusions prior to 1992, when blood screening for hepatitis C virus was introduced. Rates of new infection have since dropped by more than 80 percent in the United States because of more vigilant screening of the blood supply.<sup>1</sup> Today, most new infections are seen in people who engage in high-risk behavior.<sup>6</sup> However, since the majority of people were infected decades ago, two out of three people with chronic hepatitis C in the United States are baby boomers (Figure 1).<sup>4</sup> African-Americans are also disproportionately affected by hepatitis C. One of every seven African-American men between the ages of 50 and 59 in the United States is living with hepatitis C.<sup>2</sup>

### RISK FACTORS AND ROUTES OF HCV TRANSMISSION<sup>14,15</sup>

- Blood transfusion before 1992
- Birth date between 1946 and 1964
- Injection drug use (most common route for new infections)
- Organ transplantation from infected donors
- Exposure to infected blood by sharing contaminated razors, needles (i.e. tattoos) or piercing instruments, to name a few
- Unsafe medical practices
- High-risk sexual practices
- Birth to an infected mother (rare)
- Snorting cocaine (rare)



**Figure 1. Estimated Prevalence of Chronic Hepatitis C by Age Group.** Approximately 2.5 million people with hepatitis C in the United States are baby boomers.<sup>4</sup>



## How is hepatitis C diagnosed?

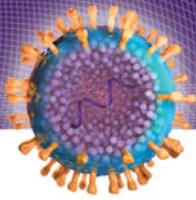
Hepatitis C is diagnosed by a simple blood test that looks for markers of hepatitis C virus (HCV) infection. If the initial test is positive, there are additional tests to learn more information about the infection and the virus. Rapid oral tests are also being developed to help diagnose hepatitis C.

Blood tests for hepatitis C include:<sup>6</sup>

- *Anti-HCV antibodies.* Produced by the immune system to fight the infection; they can be detected 8 to 12 weeks after initial infection.
- *Amount of HCV genetic material (ribonucleic acid, or RNA).* The amount of HCV RNA in the blood – or “viral load” – is related to how actively the virus is reproducing in the body.
- *HCV genetic makeup (genotype).* There are different forms, or “genotypes”, of the hepatitis C virus. Knowing the genotype is useful for making treatment decisions (for more information see *Treatment* section).

Once a person is diagnosed with hepatitis C, some doctors may choose to do additional blood tests to look at specific liver enzymes or examine small pieces of a person’s liver under a microscope (biopsy) to understand the amount of damage that may have occurred.

Hepatitis C may be silent for years and many people have been infected with the hepatitis C virus for decades without knowing it. Screening for hepatitis C is important to catch a disease that is initially without symptoms but can lead to major health complications in an aging population. Evidence suggests that effective screening should focus on at-risk populations other than recent injection drug users, such as baby boomers and African-Americans.<sup>2,16</sup> The Centers for Disease Control (CDC) is currently evaluating age-based testing, which may lead to updated hepatitis C testing recommendations.<sup>17</sup> Three out of four people with hepatitis C currently remain undiagnosed and unaware of their infection.<sup>3,4</sup>



## How does hepatitis C progress?

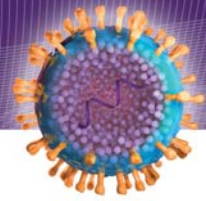
Hepatitis C starts with exposure to the virus and acute (short-term) infection. During this time, which lasts from four to 12 weeks, around 30 percent of people may experience some symptoms of the infection, including:<sup>18</sup>

- Fever
- Fatigue
- Stomach pain
- Loss of appetite
- Jaundice (yellow skin and/or eyes)

For many, these symptoms are mild enough that they do not visit a doctor. In 15 to 25 percent of cases, a person's immune system can remove the virus from the body without any treatment.<sup>18</sup> However, most (up to 85 percent) of people exposed to the hepatitis C virus develop a chronic infection, which means that the virus stays in the body for more than six months and will likely not go away without treatment.<sup>14,18</sup>

Like acute hepatitis C infection, chronic hepatitis C may not produce recognizable symptoms. A person could be infected for more than 20 or 30 years before signs of severe liver damage appear, such as:

- Build-up of fluid (ascites) in the abdomen
- Bleeding from the esophagus and stomach
- Mental problems (hepatic encephalopathy)
- Skin problems, such as spider veins

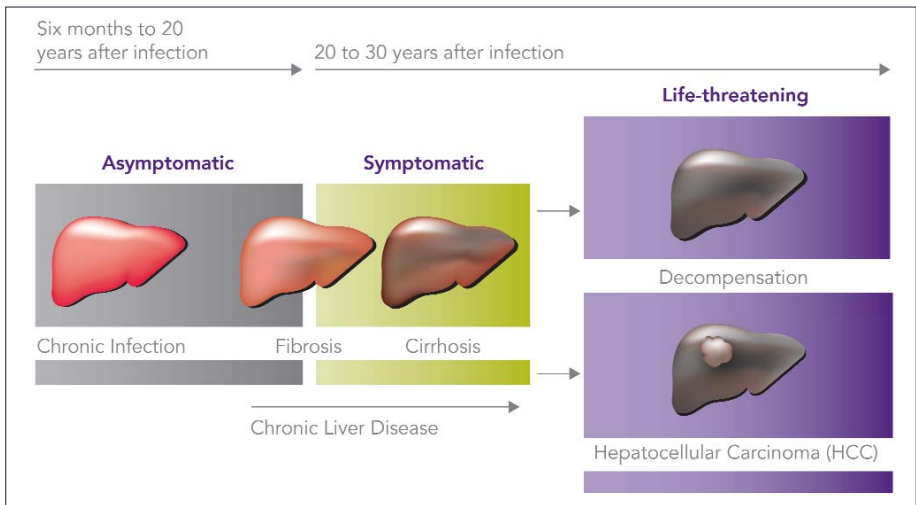


## What does hepatitis C do to the body?

In people with chronic hepatitis C, the immune system attacks infected liver cells and causes inflammation. Over the course of many years (Figure 2), this inflammation damages the liver, causing healthy tissue to be replaced with scar tissue (fibrosis). When scarring becomes very severe (cirrhosis), the liver can stop working (decompensate). People with cirrhosis due to hepatitis C develop liver cancer (hepatocellular carcinoma) at an annual rate of one to four percent.<sup>19</sup> Because liver damage gets worse over time and many people with chronic hepatitis C have been infected for decades, the number of people with liver failure (decompensation) is expected to more than double over the period from 2000 to 2030.<sup>5</sup> The number of people who die from hepatitis C-related liver problems is expected to increase by 207 percent in this period of time.<sup>5</sup>

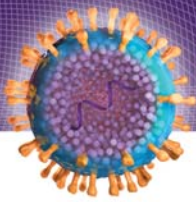
### HEPATITIS C, CIRRHOSIS AND LIVER CANCER

- 16 percent of people with hepatitis C may develop cirrhosis within 20 years.<sup>20</sup>
- 25 percent of people with hepatitis C are estimated to have cirrhosis today; this number is expected to rise to 45 percent by 2030.<sup>21</sup>
- People with cirrhosis have a high risk of developing liver cancer.<sup>22</sup>
- Worldwide, liver cancer is the fifth most common cancer and third leading cause of cancer death.<sup>23</sup>
- In the U.S., the most common cause of liver cancer is hepatitis C.<sup>24</sup>
- From 1995 to 2004, hepatitis C-related deaths rose 376 percent, from 1.76 to 8.01 per 100,000 in people aged 45 to 54.<sup>12</sup>



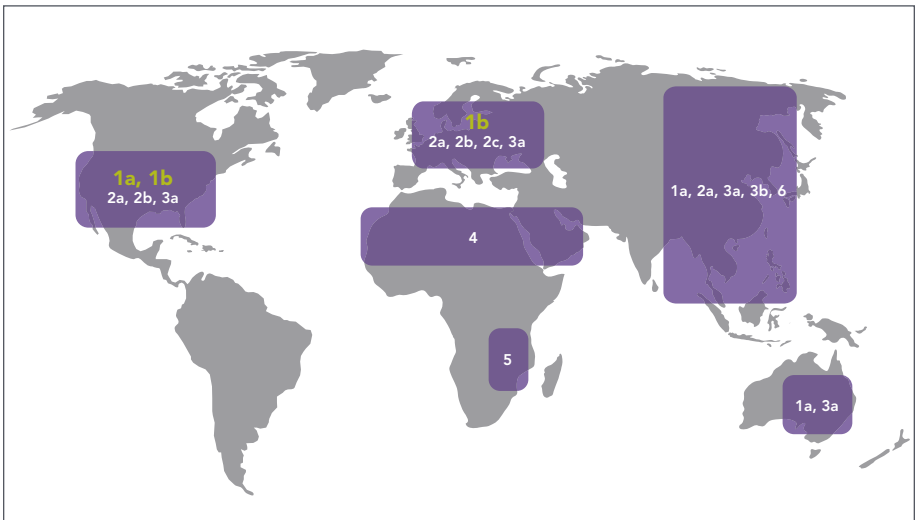
**Figure 2. Stages of Hepatitis C Liver Disease**

Stages of disease progression in a hepatitis C virus-infected liver are shown.



## A CLOSER LOOK AT THE HEPATITIS C VIRUS

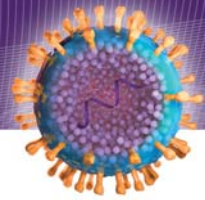
The hepatitis C virus (HCV) was discovered in 1989. It is distinct from hepatitis A and B. Although all hepatitis viruses infect the liver, they are spread in different ways, have different effects on the body and are common in different parts of the world.<sup>25</sup> There are six different forms, or genotypes, of HCV.<sup>26</sup> Each form is capable of causing chronic hepatitis C, but some forms – such as genotype 1 – are both more prevalent and less likely to respond to available treatments.<sup>1,6</sup> These genotypes can also be broken down into many subtypes, such as genotype 1a or 1b. As shown in Figure 3, genotype 1 is the most common form of HCV in the United States, accounting for around 70 percent of cases.<sup>7</sup> However, different forms are more common in other parts of the world.



**Figure 3. HCV Genotypes by region.**

*Green typeface indicates the most common genotypes in a particular region. For example, genotype 1 accounts for 70 percent of HCV in the United States.<sup>7</sup> Adapted from Fang JW, et al. <sup>27</sup>*

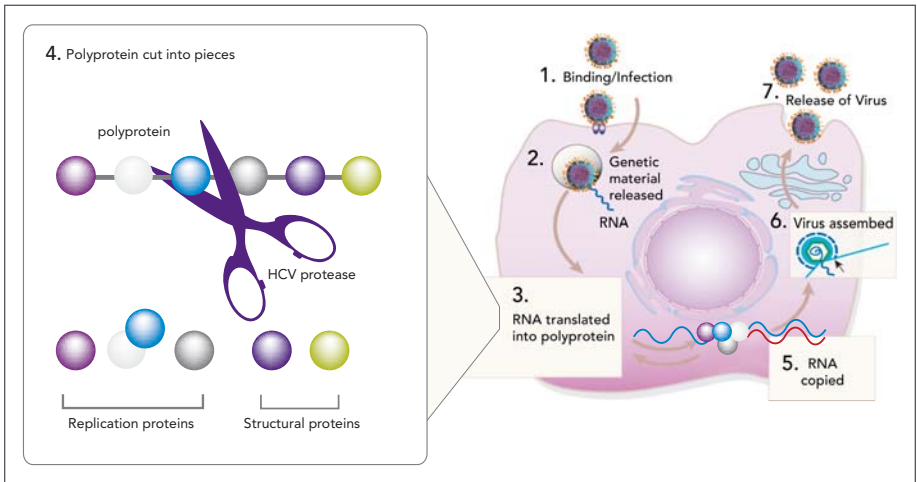
**Genotype 1 is the most common form of hepatitis C virus in the United States and is less responsive to current treatments than other genotypes.<sup>7-10</sup>**



## How does the hepatitis C virus reproduce?

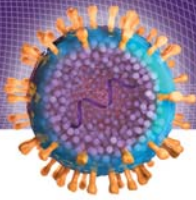
Since its discovery 20 years ago, much has been learned about HCV, how it infects and reproduces in liver cells, and the body's immune response to the infection.

HCV is made of an envelope of proteins and fats that cover a small structure called the nucleocapsid.<sup>28</sup> The nucleocapsid holds the virus' genetic material, which is a blueprint for reproducing the virus. HCV uses machinery inside a liver cell to read its RNA and produce proteins to build new viruses. Once assembled in the cell, these new viruses are released and infect other liver cells. HCV can produce up to 1 trillion new viruses every day.<sup>29</sup> More details about the life cycle of HCV are shown in Figure 4.



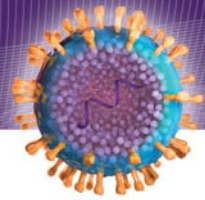
**Figure 4. How the hepatitis C virus reproduces itself in the body.**

The hepatitis C virus life cycle has several steps: 1) The virus binds to the liver cell and is absorbed; 2) Inside the cell, the virus releases its genetic material (RNA); 3) The virus uses the cell's machinery to translate its genetic code (shown in blue) into a large protein called a polyprotein; 4) A viral protein called protease cuts the polyprotein into pieces with each piece having a different role in creating new viruses; 5) Special viral proteins work together to make a new copy of the HCV RNA; 6) Other viral proteins (structural proteins) form capsules around RNA and create an envelope for the whole virus; and 7) New viruses are released from the cell. Adapted from Lindenbach BD and Rice CM.<sup>30</sup>



Understanding the proteins involved in the HCV life cycle has been critical for developing new approaches to treatment. Viral proteins necessary for producing more HCV and sustaining the infection include:<sup>28</sup>

- *Polyprotein*: Large protein created from HCV RNA after the virus enters the cell.
- *Protease*: Cuts up the polyprotein into smaller proteins that play various roles in producing more viruses.
- *Tether protein*: Ties together proteins that make copies of viral RNA.
- *Polymerase*: Makes new viral RNA copies from the original.
- *Nucleocapsid protein*: Encloses virus RNA and forms the framework for new viruses.
- *Envelope proteins*: Protect the virus and help it stick to and enter liver cells. Because they are on the outside of the virus, envelope proteins can be recognized by the body's immune system. Anti-HCV antibodies, measured in screening tests, often target the envelope proteins.



## TREATMENT OF CHRONIC HEPATITIS C

Unlike people with other diseases such as HIV/AIDS, people with hepatitis C can be cured.<sup>6</sup> This means that drug therapy can remove hepatitis C from a person's blood so that it cannot be detected even when using very sensitive tests several months or years after treatment. Sustained virological response, or SVR – also known as “viral cure” – occurs when a person's hepatitis C remains undetectable six months after treatment ends.<sup>6</sup> Achieving a viral cure is important because it may reduce the risk a person has of developing liver failure and liver cancer, as well as other problems related to hepatitis C.<sup>31</sup>

Approved treatments for hepatitis C include:

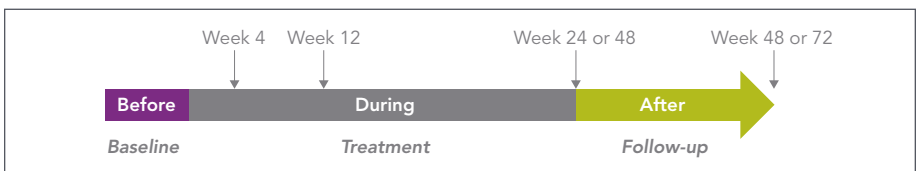
- Pegylated interferon alpha (Peg-IFN), which stimulates the immune response against HCV.
- Ribavirin (RBV), which has general antiviral activity and improves the response to Peg-IFN.<sup>32</sup>

Current treatment for hepatitis C is usually a combination of Peg-IFN and RBV for up to 48 weeks. Several factors are used to determine treatment time, including hepatitis C virus genotype, initial amount of virus in the blood (viral load), and early response to treatment (see below).<sup>6</sup> For people with genotype 1 hepatitis C, a 48-week course of treatment is recommended.<sup>33-35</sup> For other genotypes (2 and 3), which are less common in the U.S., a 24-week course is recommended.<sup>33-35</sup>

The majority of people in the United States diagnosed with hepatitis C – 69 to 88 percent – never receive treatment.<sup>36</sup> There are various reasons for this, including a “watch and wait” approach to therapy, and the nearly year-long (48 weeks) treatment time required for most people. In addition, for people with genotype 1 hepatitis C, less than half of those who are treated with available medicines achieve a viral cure.<sup>8-10</sup>

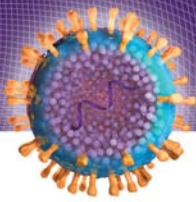
## How is response to hepatitis C treatment measured?

Progress toward the goal of viral cure is assessed by measuring the amount of hepatitis C virus in a person's blood (also called HCV RNA or viral load) at specific times before, during and after treatment, as shown in Figure 5.



**Figure 5. Treatment response milestones.**

A person's viral load is measured before treatment begins (at baseline) and at several key time points after treatment begins, including week 4, week 12, at the end of treatment (week 24 or 48), and 24 weeks after the end of treatment (week 48 or 72).



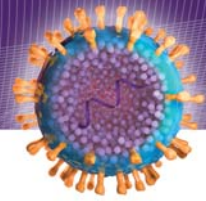
**Table 1. Key Response Definitions<sup>6</sup>**

BASELINE VIRAL LOAD	Amount of HCV RNA in the blood before treatment starts. High baseline viral load means that viral cure/SVR may be less likely to occur. <sup>9</sup>
LOG <sub>10</sub> REDUCTION	Used when recording changes in hepatitis C viral load. Each log <sub>10</sub> corresponds to a factor of 10. A 1 log <sub>10</sub> reduction means that the starting (baseline) viral load dropped by 10 times. A 2 log <sub>10</sub> reduction equals a 100-times drop; a 3 log <sub>10</sub> reduction equals a 1,000-times drop; and so forth.
RAPID VIROLOGICAL RESPONSE (RVR) – WEEK 4 RESPONSE	HCV RNA has dropped to an undetectable amount in the blood at week 4 of treatment; predicts a higher likelihood of achieving viral cure/SVR. <sup>37</sup>
EARLY VIROLOGICAL RESPONSE (EVR) – WEEK 12 RESPONSE	HCV RNA cannot be detected in the blood at week 12 of treatment (complete EVR); or HCV RNA drops by more than 2 log <sub>10</sub> (100 times) from the baseline level by week 12 (partial EVR). <sup>6</sup> Not reaching EVR predicts a lower likelihood of achieving viral cure. <sup>37,38</sup>
SUSTAINED VIROLOGICAL RESPONSE (SVR OR VIRAL CURE)	HCV RNA is not detected six months after treatment ends – also called viral cure.

Measuring early response has become an increasingly important part of treatment for people with hepatitis C because “on-treatment” response can be used to identify people likely to achieve SVR. For example, RVR is associated with higher SVR rates and lower relapse rates.<sup>37</sup> However, fewer than 20 percent of people with genotype 1 hepatitis C achieve RVR with current therapies.<sup>39</sup> Additional early response measurements, such as undetectable virus both at week 4 and week 12 (also called “extended RVR” or eRVR), are being studied as ways to predict SVR (viral cure) and may help to decide on length of treatment.

## Failure to achieve a viral cure

Less than half of people with the most common type of chronic hepatitis C in the United States (genotype 1) achieve viral cure with current treatments, which are given for 48 weeks.<sup>7-10</sup> The rate of viral cure is even lower for some groups of people, including Hispanics and African-Americans.<sup>40,41</sup>

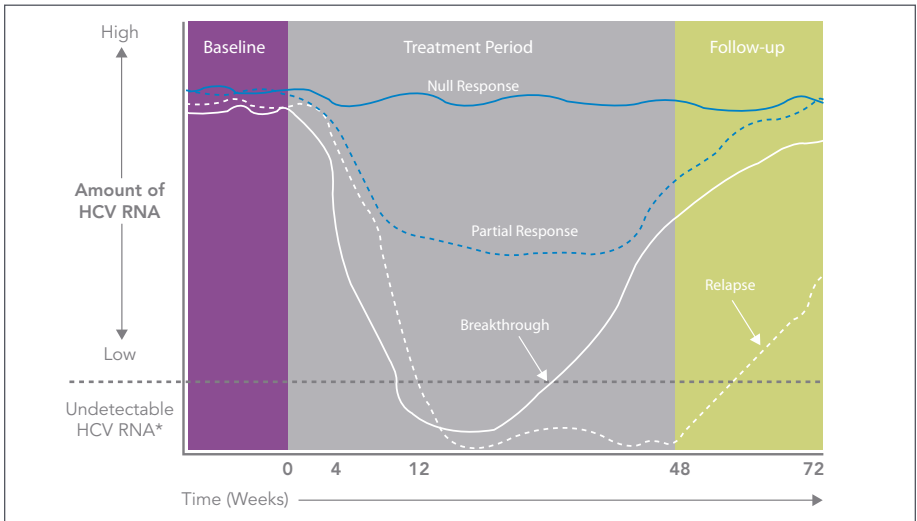


*Less than half of people in the United States with genotype 1 hepatitis C achieve SVR (viral cure) with current treatments.<sup>8-10</sup>*

Other predictors of poor response to current therapies include:<sup>6</sup>

- Gender (males are less likely to respond to treatment)
- Age (more than 40 years old)
- Higher body weight (overweight/obese)
- Severe liver damage at the start of treatment (cirrhosis or advanced liver disease)

Failure to achieve viral cure is described in various ways, as shown in Figure 6.

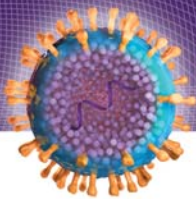


\* Illustrative representation of the lower limit of detection.

**Figure 6. Response in Patients Who Fail to Achieve a Viral Cure.** This chart shows four possible response “patterns” for people who fail to achieve a viral cure:<sup>6</sup>

- **Null response (solid blue line):** Failure to reduce HCV RNA by at least  $2 \log_{10}$  (100 times) after 12 weeks of prior treatment, which is the currently recommended U.S. Food and Drug Administration (FDA) definition for clinical trials of investigational hepatitis C treatments.<sup>42</sup>
- **Partial response (dashed blue line):** At least a  $2 \log_{10}$  (100 times) decrease in HCV RNA, but continued presence of the virus in the blood by week 24.
- **Breakthrough (solid white line):** After dropping to undetectable levels, HCV RNA is detected again in blood during treatment.
- **Relapse (dashed white line):** After dropping to undetectable levels, HCV RNA is detected again in blood after treatment ends.

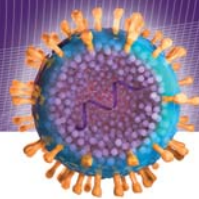
Adapted from Ghany MG, et al.<sup>6</sup>



People who don't achieve a viral cure following their initial treatment course can later choose to try therapy again with current treatments (retreatment). However, only one in five people achieve a viral cure after retreatment, and the rate of success is even lower for people who never reach undetectable levels of virus during their initial treatment (null responders and partial responders).<sup>43-46</sup> People who fail to achieve a viral cure remain at risk for serious liver damage.

## New medicines in development

Within the next 20 years, many of the nearly 4 million Americans with chronic hepatitis C will be at risk for developing advanced liver disease.<sup>2,5</sup> Cases of liver failure and liver-related death are expected to more than double in this time.<sup>5</sup> Thus, developing new medicines has become a major health priority. Dozens of experimental drugs targeting the hepatitis C virus, including polymerase and protease inhibitors, are in various stages of research and development. The availability of new drugs may have the potential to provide new combination treatment options for those people who fail to achieve an SVR with available therapies, and may affect current treatment protocols. As research progresses, more people may be able to reach viral cure.



## GLOSSARY OF TERMS

### **Acute Hepatitis C**

A short-term illness that usually occurs within the first six months after someone is exposed to the hepatitis C virus (HCV).<sup>47</sup>

### **Antibodies**

Proteins produced as part of the body's immune response to a foreign substance, such as a virus in the body.<sup>48</sup> The presence of antibodies against HCV in a person's blood is a sign of exposure to the virus.

### **Blood Transfusion**

The transfer of blood from one person to another (donor to recipient). Risk factors for HCV infection include having received a blood transfusion prior to July 1992.<sup>15</sup> After 1992, better practices for the screening of blood donors became available.

### **Breakthrough (of HCV viral load)**

After dropping to undetectable levels, HCV RNA becomes detectable again in a person's blood during treatment.<sup>6</sup>

### **Chronic Hepatitis C**

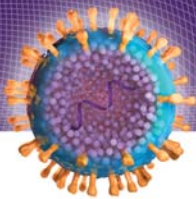
A long-term illness that occurs when HCV infection lasts longer than six months.<sup>14</sup> Approximately 75 to 85 percent of people who become infected with HCV develop chronic infection.<sup>18</sup> Chronic hepatitis C is a silent disease that damages the liver over the course of decades. It is a leading cause of liver failure, liver cancer and liver transplant.<sup>1</sup>

### **Cirrhosis**

A process by which liver cells are either damaged or killed and replaced by scar tissue.<sup>48</sup> Cirrhosis is caused by many conditions that damage the liver, including alcohol abuse and the hepatitis viruses. Cirrhosis is the end stage of chronic liver disease and has three key features: presence of fibrosis, or "scar tissue," changes in the normal liver structure, and nodules created by liver cell regeneration. Cirrhosis is a cause of death in patients with liver disease.<sup>49</sup>

### **Early Virological Response (EVR)**

Measurement of response to hepatitis C treatment at week 12. Occurs when HCV RNA cannot be detected in a patient's blood or decreases by more than 2 log<sub>10</sub> (100 times) from the starting level by week 12.<sup>6</sup> Not reaching EVR predicts a lower likelihood of achieving cure.<sup>38</sup>



## **Extended Rapid Virological Response (eRVR)**

Occurs when a patient has undetectable HCV RNA at weeks 4 and 12. Achieving eRVR is associated with a higher chance of viral cure. This measurement has been used in studies of some experimental agents to decide on length of treatment (also known as response-guided treatment).

## **Fibrosis**

Scarring caused by repair of tissue damage.<sup>49</sup> Fibrosis of the liver can lead to cirrhosis, which is a potential long-term consequence of chronic infection with HCV.

## **Genotype**

The genetic make-up of an organism or a virus.<sup>48</sup> HCV has six genotypes and many subtypes.<sup>28</sup> A person's HCV genotype may be important in determining the severity of the disease and predicting response to treatment.<sup>6</sup>

## **Genotype 1**

Most common form of HCV in the United States and Europe.<sup>1</sup> More than 70 percent of people in the U.S. have genotype 1 HCV.<sup>7</sup> People with genotype 1 HCV infections typically require longer treatment and are less likely to respond to available therapies.<sup>6,8-10</sup>

## **HCV Antibody (Anti-HCV)**

The antibody specific to HCV. Its presence in the blood tells that a person was infected with HCV; however, it does not tell whether the infection is new (acute), long-term (chronic), or no longer present.<sup>6,18</sup>

## **HCV RNA**

Genetic material found in people with hepatitis C. HCV RNA in the blood indicates a patient is currently infected with the hepatitis C virus.<sup>18</sup>

## **Hepatitis**

Inflammation of the liver.<sup>48</sup>

## **Interferon**

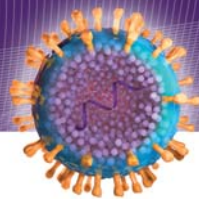
Naturally occurring protein made by the immune system in response to viral infection and inflammatory diseases. Interferon alpha, a synthetic (man-made) form, is given by injection to treat HCV infection, usually in combination with the drug ribavirin.<sup>50</sup>

## **Jaundice**

A yellowish pigmentation of the skin, tissues, and certain bodily fluids.<sup>48</sup> Jaundice can result when normal functioning of the liver is disrupted by liver disease, and it is one of the signs of infection with the hepatitis C virus.<sup>18</sup>

## **Limit of Detection**

The level at which a test cannot detect any trace of virus in the blood.



## **Liver Biopsy**

A procedure used to monitor the progression of certain forms of liver disease, including hepatitis C, after diagnosis. During a liver biopsy, a small sample of liver tissue is collected and then examined under a microscope to evaluate suspected or known liver disease. A liver biopsy can reveal inflammation, scar tissue formation (cirrhosis), or cancer.<sup>6</sup>

## **Null response**

Failure to reduce HCV RNA by at least 2  $\log_{10}$  (100 times) after 12 weeks of prior treatment, which is the currently recommended Food and Drug Administration (FDA) definition for clinical trials of investigational hepatitis C treatments.<sup>42</sup>

## **Partial Response**

At least a 2  $\log_{10}$  (100 times) decrease in HCV RNA during treatment for hepatitis C, but inability to fully remove the virus from the blood by week 24.<sup>6</sup>

## **Pegylated Interferon (Peg-IFN)**

A synthetic (man-made) version of interferon alpha that is often used in combination with the drug ribavirin to treat HCV infection; also called Peg-IFN. Pegylated interferon lasts in the body longer than standard interferon alpha. PegIntron® and Pegasys® are both brand-name pegylated interferons.<sup>33,35</sup>

## **Polymerase Inhibitor**

A class of compounds that inhibits the polymerase enzyme, which is necessary for hepatitis C virus replication.<sup>51</sup> New compounds are in clinical development for the treatment of hepatitis C.

## **Protease**

An enzyme that cuts proteins into smaller pieces.<sup>48</sup> The hepatitis C virus protease plays an essential role in the replication of the virus.<sup>52</sup>

## **Protease Inhibitor**

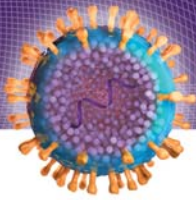
A class of compounds that inhibits the HCV protease enzyme, a protein necessary for making new viruses. Specifically, protease inhibitors prevent HCV protease from cutting a large protein (polyprotein) into smaller pieces that are used to build new viruses.<sup>51</sup> New compounds are in clinical development for the treatment of hepatitis C.

## **Rapid Virological Response (RVR)**

Measure of treatment response at week 4. Indicated by undetectable HCV RNA (confirmed by HCV PCR) after 4 weeks of treatment.<sup>6</sup> Reaching RVR predicts a higher likelihood of SVR (viral cure).<sup>37</sup>

## **Relapse (Hepatitis C)**

After dropping to undetectable levels, HCV RNA is detected again in blood after treatment ends.<sup>6</sup>

**Ribavirin (RBV)**

An antiviral medication called a nucleoside analogue. Ribavirin is usually used in combination with Peg-IFN, and is not generally effective in the treatment of HCV when used alone.<sup>48</sup> Copegus® and Rebetol® are brand-name ribavirin.<sup>34,35</sup>

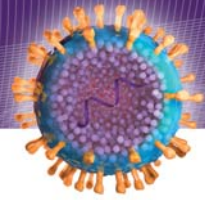
**Sustained Virological Response (SVR) - also known as viral cure**

A term used when a patient's HCV RNA remains undetectable six months after stopping treatment.<sup>6</sup> SVR is also known as viral cure.

**Viral Load**

The amount of HCV RNA in the blood.

PEGASYS® and Copegus® are trademarks of Hoffman-La Roche. PEGINTRON® and Rebetol® are registered trademarks of Schering Corporation.



## APPENDIX: HEPATITIS C RESOURCES

### American Association for the Study of Liver Diseases (AASLD)

<http://www.aasld.org>  
(703) 299-9766  
aasld@aasld.org

### Centers for Disease Control and Prevention, Hepatitis Division

<http://www.cdc.gov/hepatitis>

### Infectious Diseases Society of America

<http://www.idsociety.org>  
(703) 299-0200

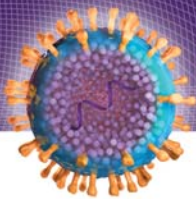
### World Health Organization

<http://www.who.int/topics/hepatitis/en>

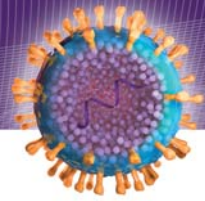
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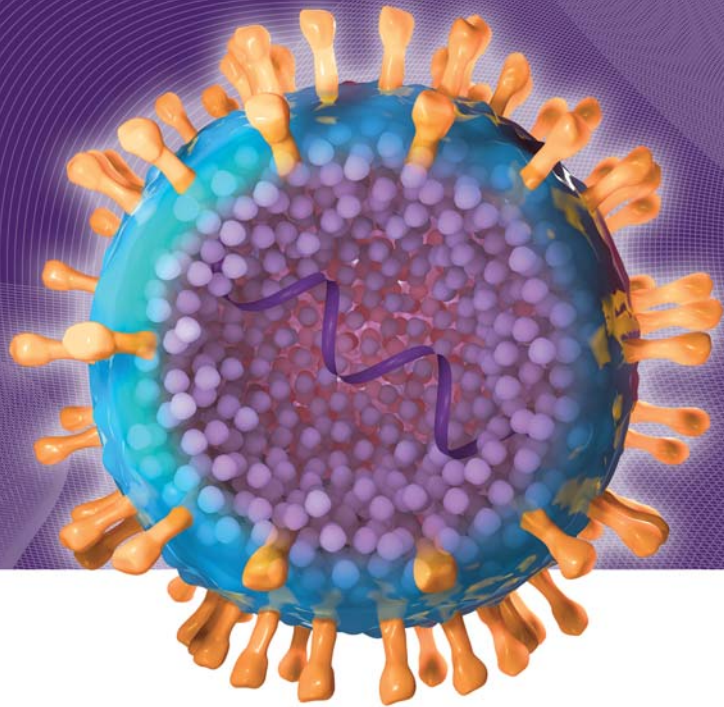
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