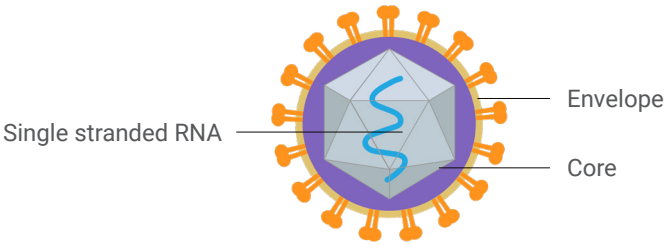
Hepatitis C virus infection

UNDERSTANDING THE PATHOGENESIS AND COURSE OF HCV INFECTION

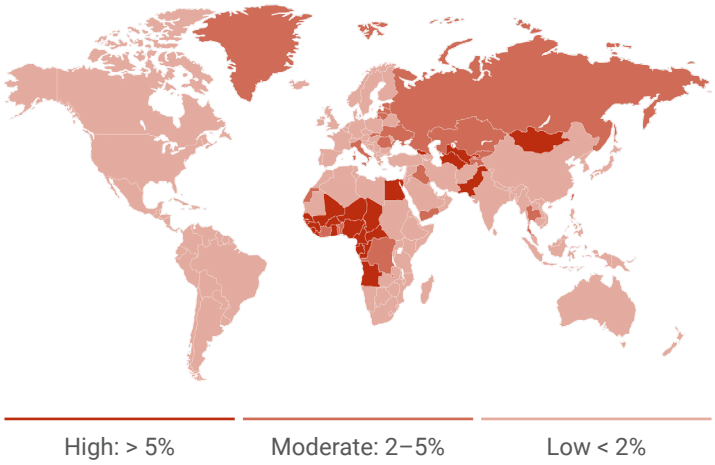
Profile

Hepatitis C virus (HCV) is an enveloped, single stranded RNA-virus.

Genus: Hepacivirus
Family: Flaviviridae
Genotypes: 1–6



Prevalence



Transmission

Intravenous drug use: sharing of contaminated needles, syringes, or other injection drug equipment
Occupational exposure: needlesticks or other sharp instrument injuries
Vertical: transmission from mother to child
Sexual contact: unprotected sexual intercourse
Blood transfusion: in countries without HCV screening

Pathogenesis

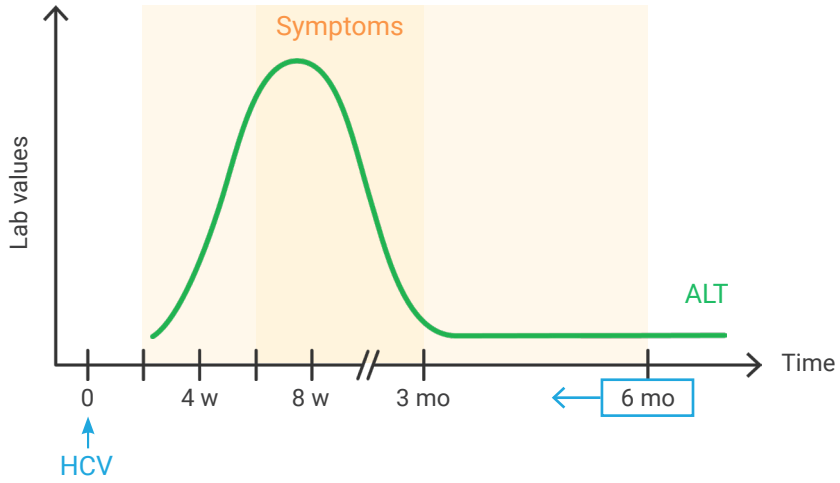
After infection, HCV enters hepatocytes and replicates in the cytoplasm. HCV itself is not cytopathic; cellular damage results from the immune response. Antibodies against HCV are produced: first immunoglobulin M (IgM) antibodies, followed by immunoglobulin G (IgG) antibodies. This immune activation leads to the damage and destruction of hepatocytes, which results in the clinical symptoms of HCV.

Clinical course

Incubation period: 2 weeks–6 months
Resolution: possible

Acute HCV infection

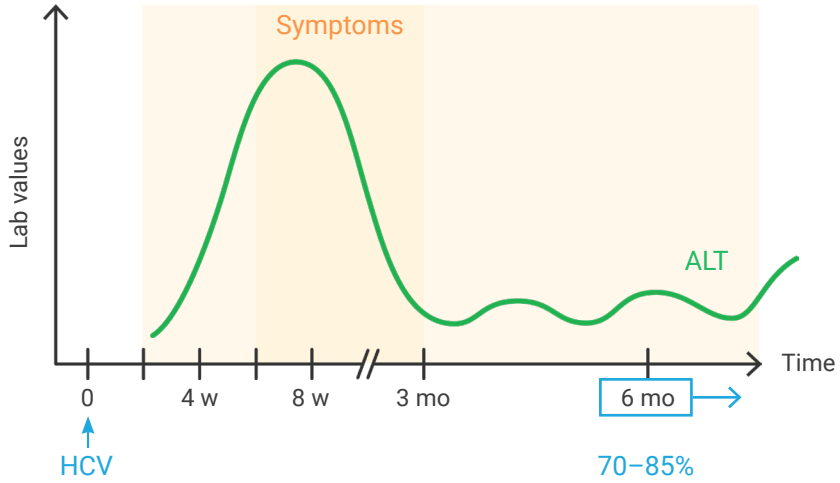
An acute infection occurs when viral RNA is detectable in blood for a maximum of six months. With the resolution of the infection, the levels of the liver enzyme alanine aminotransferase (ALT) decrease to within the normal range.



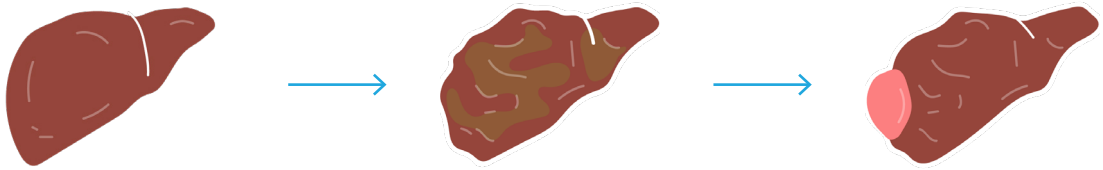
Chronic HBV infection

A chronic infection occurs when viral RNA is detectable in blood for longer than six months. According to the continuing liver damage, ALT levels fluctuate.

In 70–85% of adults, an acute HCV infection will become chronic.



After years, chronic HCV infection leads to the development of liver cirrhosis, which in turn can lead to hepatocellular carcinoma.



5–30% 1–2% per year

Predictors of disease progression

Predictors of disease progression can be organized into three categories: viral, host, and environmental factors.

Host factors of unfavorable outcomes:
- older age
- male gender
- African ancestry
- weakened immune status

Viral factors of unfavorable outcome:
- high viral load
- certain genotypes

E**nvironmental factors of unfavorable outcome**:
- chronic alcohol use
- coinfections such as hepatitis B or HIV infections
- metabolic diseases (e.g., obesity or diabetes)

Further Reading

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Dubuisson, J and Cosset, FL. 2014. Virology and cell biology of the hepatitis C virus life cycle: an update. *J Hepatol.* **61(1 Suppl):** S3–S13.

Petruzziello, A, Marigliano, S, Loquercio, et al. 2016. Global epidemiology of hepatitis C virus infection: An up-date of the distribution and circulation of hepatitis C virus genotypes. *World J Gastroenterol.* **22:**7824–7840.