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Hepatitis C virus genetic variability and evolution

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Abstract

1 Hepatitis C virus (HCV) has infected over 170 million people worldwide and creates a huge disease burden due to chronic, 15 progressive liver disease. HCV is a single-stranded, positive sense, RNA virus, member of the *Flaviviridae* family. The high error 21 rate of RNA-dependent RNA polymerase and the pressure exerted by the host immune system, has driven the evolution of HCV into 7 different genotypes and more than 67 subtypes. HCV evolves by means of different mechanisms of genetic variation. On the 50 one hand, its high mutation rates generate the production of a large number of different but closely related viral variants during 5 infection, usually referred to as a quasispecies. The great quasispecies variability of HCV has also therapeutic implications since the 4 continuous generation and selection of resistant or fitter variants within the quasispecies spectrum might allow viruses to escape control by antiviral drugs. On the other hand HCV exploits recombination to ensure its survival. This enormous viral diversity together with some host factors has made it difficult to control viral dispersal. Current treatment options involve pegylated 33 interferon-α (IFN-α) and ribavirin (RBV) as dual therapy or in combination with a direct-acting antiviral (DAA) drug, depending on

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