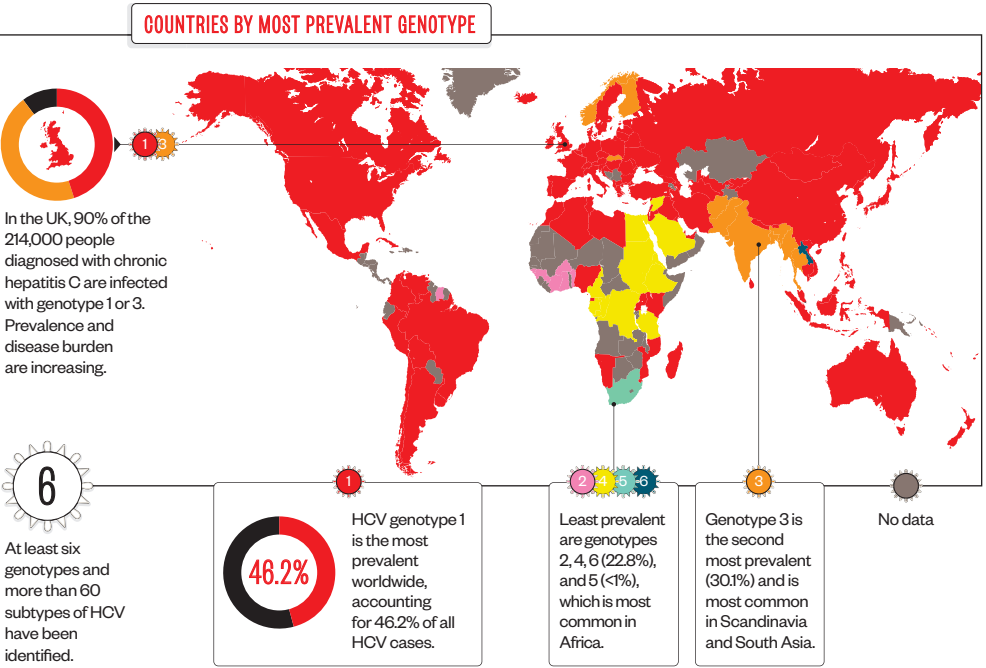


TARGETING HEPATITIS C: HOW NEW TREATMENTS WORK

There are >185 million people with Hepatitis C infection worldwide but new treatments mean that most people with access to therapy can now be cured. By Dawn Connelly.

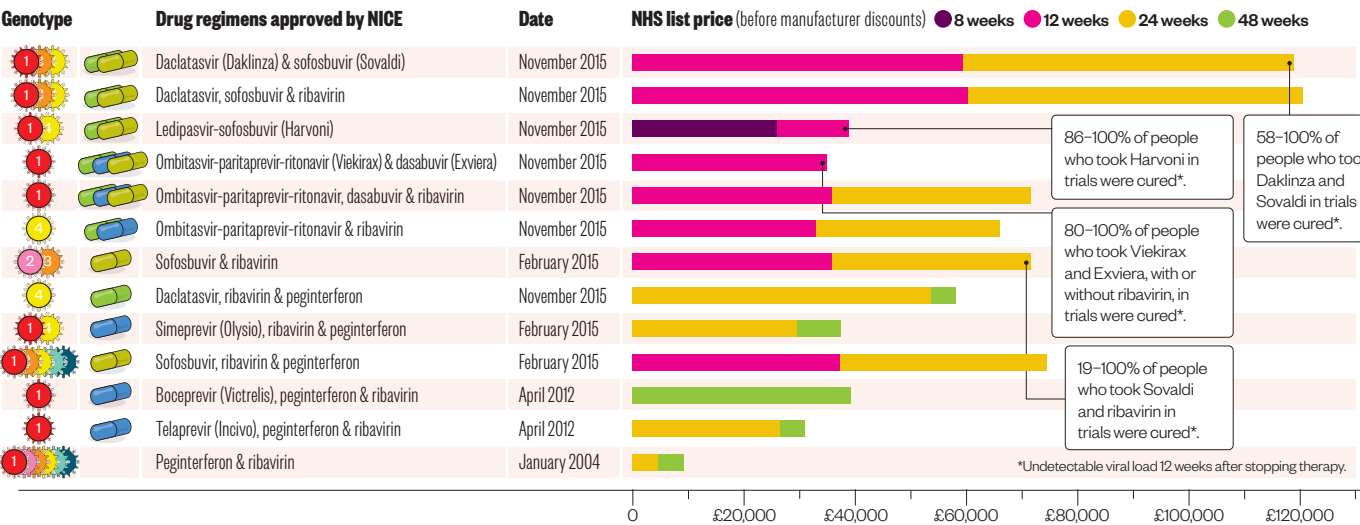
TRAVELLING THE WORLD

The hepatitis C virus (HCV) is bloodborne and in developed countries is most commonly transmitted by sharing needles or other drug-injecting equipment. Acute infection is usually asymptomatic, but around 75–85% of people go on to develop chronic infection and, if left untreated or if treatment fails, 5–20% develop cirrhosis within 20–30 years and 1–5% die from cirrhosis or liver cancer.



BALANCING COSTS WITH BENEFITS

For more than a decade, standard therapy was pegylated interferon and ribavirin, which had severe side effects and was only effective in around 50% of genotype 1 and 75% of genotype 2/3 patients. Boceprevir and telaprevir, which target HCV specific proteins, improved cure rates to about 70% in genotype 1 patients, but worsened side effects. Simpler and shorter interferon-free regimens have now been approved by the National Institute for Health and Clinical Excellence (NICE), with higher cure rates (most >90%) and fewer side effects. Regimen choice and length depends on several factors, including genotype, presence of cirrhosis, treatment history, potential drug interactions, HIV or hepatitis B co-infection, NICE guidance, and commissioning arrangements.



VIRAL REPLICATION AND DRUG TARGETS

HCV is a single stranded RNA flavivirus, which constantly mutates, enabling it to escape attack by the immune system. The HCV genome is translated to produce a polyprotein, which is further processed by host and viral proteases to produce three structural and seven non-structural proteins. A greater understanding of the HCV genome and proteins has enabled the development of direct-acting antivirals.

