The “Genetic Achilles’ Heel” That Makes Us Susceptible to Hepatitis C

Why does the world’s most common liver virus affect humans more than our most recent genetic ancestors?

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Why is it that humans are so much more susceptible to contracting hepatitis C virus (HCV) than our most recent genetic ancestors? That’s the subject of a recent research paper investigating a so-called genetic Achilles’ heel that humans appear to have in terms of our capacity to fight the world’s most common liver virus, ScienceDaily reports.

The study, which examined the genetic diversity of the human immune response against HCV, found that African hunter-gatherer Pygmies have a rare version of the antimicrobial signaling molecule (a kind of molecule critical in defending against infections, bacteria, fungi and viruses) called interferon lambda 4 (or IFNλ4) that appears better able to inhibit HCV infection than the most common human form. Interestingly, this version of IFNλ4 had similar properties to the IFNλ4 found in chimpanzees.

More specifically, the report, published in PLOS Pathogens, found this variation to be the result of a single amino acid substitution. This more active version of IFNλ4 was likely reacquired by African Pygmies after chimpanzees and humans diverged, according to PLOS. The findings, researchers say, suggest that the evolution of this gene has disadvantaged humans with regard to fighting the virus.

“We are astonished that humans were the only species to carry this mutation, and it remains a mystery as to why the human population has evolved an antiviral gene that is less able to control viral infections compared to our closest ancestors,” study authors John McLauchlan, PhD, and Connor Bamford, PhD commented.

According to the study, our genetic Achilles’ heel likely arose between six million and 360,000 years ago.
