Bubonic Plague–Driven Gene Tied to Less Fibrosis in Those With HIV and Hep C

Researchers believe this genetic mutation protected against the 14th century-bubonic plague.

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A genetic mutation that may have conferred protection against the 14th-century bubonic plague in Europe is associated with less severe liver fibrosis among those coinfected with HIV and hepatitis C virus (HCV).

Publishing their findings in Clinical Infectious Diseases, researchers studied blood samples from HIV/HCV–coinfected individuals who had contracted the viruses through hemophilia treatments during the 1980s. The scientists analyzed differences in liver fibrosis progression among them based on whether they had a genetic mutation known as the CCR5-delta 32 mutation, which yields a nonfunctioning CCR5 coreceptor on the surface of the immune cells that HIV targets. Most HIV latches onto that coreceptor to begin the process of infecting a cell.

The cohort members were followed for an average of four years.

Those with the genetic mutation had less fibrosis progression than those without it.

The researchers enrolled in a clinical trial a different cohort of HIV/HCV–positive people who had no liver disease and who were treated with the experimental HIV medication cenicriviroc, which functions by blocking the CCR5 receptor. The drug also blocks a protein on immune cells known as CCR2.

After one year of treatment, those given a higher dose of cenicriviroc had less liver fibrosis progression than those who received lower doses.

To read a press release about the study, click here.

To read the study abstract, click here.

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