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Hepatitis C virus blocks 'superinfection'

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There's infection and then there's superinfection – when a cell already infected by a virus gets a second viral infection. But some viruses don't like to share their cells. New research from Rockefeller University shows that the hepatitis C virus, which infects cells in the liver and can cause chronic liver disease, can block other hepatitis C variants from infecting the same cell.

Research from Charles Rice's laboratory at Rockefeller last year created the first hepatitis C virus that could be grown in cell culture. Using this virus, called HCVcc, the scientists, lead by graduate student Donna Tscherne, tried to infect cells previously infected with hepatitis C virus. But it didn't work; the cells couldn't be infected. The same was true when they tried to infect cells that contained hepatitis C virus of other genotypes than that of HCVcc. Only when they gave the cells a drug that could inhibit virus replication could they superinfect them with HCVcc. The first virus was stopping HCVcc from infecting the cells, a phenomenon called superinfection exclusion.

"A virus can interfere with a secondary infection in a variety of ways," says Rice, head of the Laboratory of Virology and Infectious Disease and the Maurice R. and Corinne P. Greenberg Professor. "It can interfere with how a virus attaches to the cell, its penetration, or its access to the cell's resources." If both viruses are competing for the same resources in the cell, then the first virus can confiscate them so none are available for the second virus.

Rice and Tscherne believe this phenomenon may explain the mechanism

of HCV superinfection exclusion. The scientists found that HCVcc is blocked at some point after it has entered the cell; most likely at a step, or steps, involved in replication. Future studies are being designed to try to identify what proteins are important for this step.

“Understanding superinfection exclusion has potentially important implications for understanding the biology of hepatitis C,” says Rice. The mechanism could, for instance, help the hepatitis C virus generate a large pool of variants that would be able to withstand attack from the immune system or from antiviral drugs. If the process of superinfection exclusion could be recreated therapeutically, it could also open up new treatment avenues.

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