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[BioMedical] Academia Sinica's Scientists Discover How Hepatitis C Virus Represses Antiviral Immunity to Replicate in Cells

[BioMedical] Academia Sinica's Scientists Discover How Hepatitis C Virus Represses Antiviral Immunity to Replicate in Cells (Chinese Version)

Academia Sinica Newsletter (2011/01/28) Dr. Steve S.-L. CHEN and Dr. Po-Yuan KE, a Research Fellow and a Postdoctoral Research Fellow, respectively, at the Institute of Biomedical Sciences at Academia Sinica, recently unraveled a long standing question about how hepatitis C virus (HCV) interacts with host cell machinery to regulate its own replication. HCV causes hepatitis C in humans, a chronic, infectious disease affecting the liver. The study increases understanding of the disease mechanisms induced by HCV, and opens doors to investigation of new hepatitis therapies. The study was published in the Journal of Clinical Investigation on January 4, 2011.

HCV affects over 170 million individuals worldwide and often becomes chronic and results in liver-associated diseases. No vaccine against HCV is currently available. In their study, the scientists investigated the response of host cells to HCV infection and its consequence to HCV replication. They discovered that HCV infection induces an "unfolded protein response" (UPR), which then activates autophagy (cell digestion of itself) to complete autolysosome maturation. This activated UPR-autophagy represses antiviral immunity and promotes replication of the virus. This finding may aid in the design of anti-HCV therapies.

Furthermore, Dr. CHEN and Dr. KE found that disruption of the UPR-autophagy up-regulates antiviral innate responses. They also found that activation of UPR-autophagy by chemical inducers such as rapamycin (an immunosuppressant drug used to prevent rejection in organ transplantation), or by nutrient starvation, strikingly represses antiviral innate immunity. The results indicated, for the first time, that UPR-autophagy positively regulates HCV RNA replication through inhibiting innate immunity. In addition, they found that an autophagy inhibitor, chloroquine (a drug used for treatment of malaria), inhibits replication of HCV. Hence, the results also shed a new insight on the rational basis of anti-HCV drug design.

Related website:
http://www.jci.org/articles/view/41474
http://www.landesbioscience.com/journals/autophagy/KeAUTO7-5.pdf

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