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[Molecular Biology] Biologists at Academia Sinica Develops a New Molecular Strategy to Suppress Hepatitis C Virus Replication

[Molecular Biology] Biologists at Academia Sinica Develops a New Molecular Strategy to Suppress Hepatitis C Virus Replication (Chinese Version)

Academia Sinica Newsletter (2012/09/20) A research team at Academia Sinica led by Drs. Michael M.C. LAI at the Institute of Molecular Biology and Tien-Hsien CHANG at the Genomics Research Center recently uncovered a key molecular player in hepatitis C virus replication. The team found that reducing the abundance of a cell component called the "40S ribosomal subunit" in a host cell could significantly cut down hepatitis C virus replication without negatively impacting host-cell health. This finding suggests a new strategy for combating hepatitis virus infection. The study was published in the June 28 issue of the scholarly journal PLoS Pathogens.

Hepatitis C is a blood-transmitted virus that causes chronic liver diseases that threaten roughly two percent of the world's population. So far, there is no hepatitis C vaccine and current therapies are only effective in a fraction of infected patients.

Viruses rely heavily on their host cells to replicate. Research Associate Jing-Ying HUANG, the first author of the article, and her colleagues in the Institute of Molecular Biology used RNAi technology to systematically search for the components of the host cell that the hepatitis C virus must borrow to successfully reproduce itself. She singled out the 40S ribosomal subunit. The 40S ribosomal subunit is normally found in sufficient abundance to satisfy the needs of both the host cell and the virus, but when the amount of the 40S ribosomal subunit is reduced below a certain threshold, the hepatitis C virus apparently becomes the weaker competitor and dwindles to its demise. Dr. CHANG made an interesting analogy of this finding: "It is like how, under favorable conditions, counterfeit cell phones may work nearly as well as the well-designed name brands in drawing signals. However, once the bandwidth of the signals falls below a certain threshold, those counterfeits fail to work, yet the name brands remain fully functional. Sooner or later, those counterfeits will fade away from the market, i.e., they will not be able to compete effectively with the name brands".

The finding provides a new strategy through which it becomes realizable to develop effective drugs to combat the hepatitis C virus. Conventionally, drugs have been designed to target the viral proteins, but mutations that accumulate through rapid cycles of viral replication often lead to emergence of drug-resistant viral strains. In contrast, the host cell's ribosomal 40S subunit has been perfected over millions of years of evolution, thus is extremely unlikely to morph or mutate as freely as viruses.

"Finding a good way to fine-tune 40S ribosomal subunit level as part of an hepatitis C virus therapy may not only be feasible, but also superior in terms of minimizing the drug-resistance problem" said Dr. LAI.

Related website:
http://www.natureasia.com/A-IMBN/

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