BioSpectrum the business of Bio & Health Sciences ASIA EDITION

Slash ribosome subunits to halt hep C virus spread

24 September 2012 | News | By BioSpectrum Bureau

Reducing ribosome subunits will halt hep C virus replication

Singapore: A research team led by Dr Michael M C Lai of the Institute of Molecular Biology and Dr Tien-Hsien Chang of the Genomics Research Center in Taiwan 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.

So far, there is no treatment for hepatitis C and current therapies are only effective in a fraction of infected patients. This finding suggests a new strategy for combating hepatitis virus infection. The study was published in the journal *PLoS Pathogens*.

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, they will not be able to compete effectively with the name brands."