Study identifies how RNA viruses hijack a host cell to multiply

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(Phys.org) -- By discovering how certain viruses use their host cells to replicate, UC Irvine microbiologists have identified a new approach to the development of universal treatments for viral illnesses such as meningitis, encephalitis, hepatitis and possibly the common cold.

The UCI researchers, working with Dutch colleagues, found that certain RNA viruses hijack a key DNA repair activity of human cells to produce the genetic material necessary for them to multiply.

For many years, scientists have known that viruses rely on functions provided by their host cells to increase their numbers, but the UCI study – led by microbiology & molecular genetics professor Bert Semler – is the first to identify how the RNA-containing picornaviruses utilize a DNA repair enzyme to do so.

Study results appear in the early online edition of the *Proceedings of the National Academy of Sciences* the week of Aug. 20.

RNA viruses have ribonucleic acid as their genetic material (rather than deoxyribonucleic acid, or DNA). Notable human diseases caused by RNA viruses include SARS, influenza, hepatitis C, West Nile fever, the common cold and poliomyelitis.

The UCI and Dutch researchers examined one group of RNA viruses, called picornaviruses, using biochemical purification methods and confocal microscopy to see how they co-opt the functions of a cellular

DNA repair enzyme called TDP2 to advance their replication process.

"These findings are significant because all known picornaviruses harbor the target for this DNA repair enzyme, despite the fact that their genetic material is made up of RNA rather than DNA. Thus, identifying drugs or small molecules that interfere with the interaction between the virus and TDP2 could result in a broad-spectrum treatment for picornaviruses," said Semler, who also directs UCI's Center for Virus Research.

By targeting a host cell function required for viral replication and not the virus itself, he added, the primary challenge of antiviral drug resistance may be sidestepped.

As part of their survival mechanism, RNA viruses mutate often, and drugs intended for them usually become ineffective over time. HIV, for example, rapidly mutates, necessitating a combination therapy employing a number of antiviral agents.

A drug that blocks RNA viruses from hijacking DNA repair enzymes may avoid these resistance issues. Semler's lab plans to screen mixtures of drug candidates to find ones that inhibit this process in cells infected by the human rhinovirus, the predominant cause of the common cold.

More information: www.pnas.org/content/early/201 … /1208096109.abstract

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