

HIV study identifies key cellular defence mechanism

November 7 2011

(Medical Xpress) -- Scientists have moved a step closer to understanding how one of our body's own proteins helps stop the human immunodeficiency virus (HIV-1) in its tracks.

The study, carried out by researchers at The University of Manchester and the Medical Research Council's National Institute for Medical Research and published in *Nature*, provides a blueprint for the design of new drugs to treat <u>HIV</u> infection, say the researchers.

Scientists in the United States and France recently discovered that a protein named SAMHD1 was able to prevent HIV replicating in a group of white blood cells called myeloid cells.

Now, crucially, the teams from Manchester and the MRC have shown how SAMHD1 prevents the virus from replicating itself within these cells, opening up the possibility of creating drugs that imitate this biological process to prevent HIV replicating in the sentinel cells of the immune system.

"HIV is one of the most common chronic infectious diseases on the planet, so understanding its biology is critical to the development of novel antiviral compounds," said Dr Michelle Webb, who led the study in Manchester's School of Biomedicine.

"SAMHD1 has been shown to prevent the HIV virus replicating in certain cells but precisely how it does this wasn't known. Our research



has found that SAMHD1 is able to degrade deoxynucleotides, which are the building blocks required for replication of the virus.

"If we can stop the virus from replicating within these cells we can prevent it from spreading to other cells and halt the progress of the infection."

Co-author Dr Ian Taylor, from the MRC's National Institute for Medical Research, added: "We now wish to define more precisely, at a molecular level, how SAMHD1 functions. This will pave the way for new therapeutic approaches to HIV-1 and even vaccine development."

More information: 'HIV-1 restriction factor SAMHD1 is a deoxynucleoside triphosphate triphosphohydrolase,' published in *Nature*

Provided by University of Manchester

Citation: HIV study identifies key cellular defence mechanism (2011, November 7) retrieved 7 May 2024 from <u>https://medicalxpress.com/news/2011-11-hiv-key-cellular-defence-</u> mechanism.html

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