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Common viruses have Achilles heel

10 September 2005

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A TREATMENT for SARS, pneumonia and the common cold may be in sight. By targeting a protein that all these so-called coronaviruses share, it might be possible to stop viral replication and prevent disease.

Most viral proteins mutate quickly, meaning vaccines can become ineffective in a matter of months. But in the wake of the SARS outbreak in 2003, researchers developed an antiviral that interfered with the SARS virus's main protease, a protein that controls and activates viral replication. Because the protease target is stable and slow to evolve, the treatment is likely to keep on working even as the viruses mutate.

Haitao Yang at Tsinghua University in Beijing and his colleagues screened viruses from each of the three distinct coronavirus groups, including those responsible for colds, bronchitis and pneumonia, and found that the main protease belonging to each virus had a very similar sequence and structure to the SARS protein.

They then created a synthetic molecule designed to bind to the protein, quashing its activity. Sure enough, the inhibitor damped down viral replication in infected cell cultures to one ten-thousandth of its original activity (*Public Library of Science Biology*, vol 3, p 324).

The inhibitor worked on all eight coronaviruses tested, and because it binds to a common sequence, it is also likely to work against other coronaviruses, says Yang.

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Printed on Mon Sep 26 11:56:34 BST 2005