Hepatitis C virus channels efforts into cell survival

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Researchers at the University of Leeds have discovered a previously unknown mechanism that allows the hepatitis C virus (HCV) to remain in the body for decades.

A study published in the Proceedings of the National Academy of Sciences (*PNAS*) shows that the virus blocks the actions of a specific ion channel in the cell membrane that would usually trigger apoptosis - the cell's self-destruct programme - and in doing so, has evolved another way of protecting itself from being eliminated from the body.

Apoptosis occurs naturally in the body to allow the removal of unhealthy cells or the replacement of worn-out cells. One of the ways in which apoptosis can be triggered in a cell is to reduce its potassium levels. This can happen when the cell is exposed to oxidative stress that activates a specific ion channel (which acts as a pore in the cell membrane) causing it to open and allow out potassium ions.

However, the research team has discovered that a protein made by HCV, known as NS5A, is able to block the activation of this ion channel in liver cells, enabling these cells to resist cell death for longer.

"For a virus to persist in the body over a long time, it has to find a way of manipulating the host cell so that it becomes resistant to apoptosis," says lead researcher Professor Mark Harris of the University's Faculty of Biological Sciences. "We know of many ways that viruses have evolved to do this, but this is the first observation of a virus preventing cell death

by manipulating an ion channel."

HCV affects some 170 million people globally and only around half of these will respond to treatment. Many sufferers will be asymptomatic - some for twenty or even thirty years - but the virus remains in the liver, and its long-tem damage can ultimately cause cirrhosis or cancer.

"Cells in the liver are often exposed to high levels of oxidative, and other, stresses as they work to detoxify the blood of foreign compounds such as drugs and alcohol, and to remove chemicals produced by our own bodies," says Professor Harris. "In addition, the virus itself causes oxidative stress as it replicates in the cells. The research shows that the virus has evolved another way of protecting itself from this natural process, and to avoid elimination from the body for longer."

The research team believes that continued research may offer a potential target for drug development, perhaps through combination therapy.

"We need to find out exactly how the blocking action works, but it's possible that two drugs could be coupled together, one to prevent the virus from blocking the ion channel and another to induce stress to force apoptosis," says Professor Harris.

"It's a very exciting discovery, and ideally we'd like to expand our investigations to see whether other viruses that cause long term or chronic infections - such as HIV - have evolved the same ability."

Source: University of Leeds (news : web)

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