

# AIDS may partly be the consequence of an evolutionary accident says scientist

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AIDS, a fatal disease in humans, may partly be the consequence of an evolutionary accident, scientists heard today at the Society for General Microbiology's 162nd meeting being held this week at the Edinburgh International Conference Centre.

“AIDS is a deadly disease in people that is caused by human immunodeficiency virus (HIV). But similar viruses such as simian immunodeficiency virus (SIV), which infects monkeys, usually don't cause disease in their natural monkey hosts,” says Professor Frank Kirchhoff from the University of Ulm in Germany.

Previous studies have established that one of the key differences between the way HIV-1 behaves in humans and closely related SIVs behave in monkeys is that when humans are infected with HIV-1 the immune system becomes highly stimulated. This means critical defence cells called helper T cells are continuously activated and die more quickly than usual.

The researchers found that the Nef protein of most SIVs removes a molecule from the cell surface that is critical to make T cells responsive to stimulation. This most likely limits the negative effects otherwise caused by the chronically strong immune response. However, Nef proteins in HIV-1 and its closest related SIVs lack this protective function, according to Professor Kirchhoff.

In natural SIV infections in monkeys, the ability of the Nef protein to

remove a specific receptor, named CD3, from the infected cell's surface may help the host animal to maintain a functional immune system, which means that it can still fight off other diseases. Only the Nef proteins of HIV-1 and its immediate SIV relatives do not perform this function.

“We suspect that this evolutionary loss of a protective function of Nef may contribute to the high virulence of HIV-1 in humans” says Prof Kirchhoff. “Well adapted viruses don't kill their hosts.”

The team will examine whether SIVs carrying Nef genes artificially made incapable of limiting T cell activation might become more pathogenic in their natural monkey hosts. The group will also examine whether Nef variation among HIV-2 strains might explain differences in the rate of progression to disease in infected humans.

Source: Society for General Microbiology

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