Retrovirology

Oral presentation

yesHIV Impairs Reverse Cholesterol Transport from Macrophages: A Possible Mechanism of Atherogenic Effect of HIV-1 Infection

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from 2005 International Meeting of The Institute of Human Virology
Baltimore, USA, 29 August – 2 September 2005

Published: 8 December 2005

Retrovirology 2005, **2**(Suppl 1):S151 doi:10.1186/1742-4690-2-S1-S151

Both asymptomatic HIV-1 infection and AIDS are consistently associated with increased risk of coronary artery disease (CAD). The accumulation of cholesterol-loaded 'foam cells' (macrophages) in the walls of arteries is a characteristic feature of atherosclerosis. Here we demonstrate that HIV-1 infection of macrophages leads to impairment of apoA-I-dependent cholesterol efflux, accumulation of cholesterol and formation of foam cells. This effect is mediated by the HIV-1 protein Nef. Transfection of RAW cells with the Nef-expressing plasmid resulted in reduction of efflux and cholesterol accumulation. Nef impaired activity of ABCA1, the main transporter of cholesterol to apoA-I. The role of HIV-infected macrophages in atherosclerosis was supported by the presence of HIV-positive foam cells in atherosclerotic plaques of HIV-infected patients. These results suggest a mechanism by which HIV-infected macrophages may contribute to atherosclerotic plaque formation.