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Late HIV infection modulates the expression and activity of Cathepsin B, and its inhibitors in macrophages: implications in neuropathogenesis

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Background

To determine the mechanism by which HIV infection alters the expression and activity of CATB and its inhibitor, cystatin B (CSTB).

Methods

Peripheral blood derived macrophages (MDM) were infected with HIV_{ADA} at a MOI of 0.1 and cultured for up to 12 days. Intracellular and extracellular expression of CATB, CST B and CSTC in uninfected and HIV-infected cells were analyzed by Western blots and ELISA at 6, 9 and 12 days post infection (days p.i.). Activity of CAT B after HIV infection was determined by fluorescence and confocal microscopy.

Results

Expression of CATB protein and its intracellular inhibitor, CSTB, was increased in HIV infected cells after 12 days p.i. compared to uninfected controls ($p =< 0.05$). However CSTC increase was not significant in HIV infected cells. CATB was secreted to similar (>400 ng/ml) levels in both HIV-infected and uninfected cells at higher levels than those proved by others to promote cell damage (100 ng/ml or more). Importantly, secreted CATB from HIV-infected MDMs was significantly ($p= 0.008$) more active than that secreted from control cells throughout the extent of the infection.

Discussion

HIV infection increases the levels of active CATB in supernatants 4 times higher than those previously reported by other groups to be toxic to neuronal cells. Although CSTB increased in HIV-infected cultures, no effective inhibition of CATB was seen at 12 days pot-infection. Our results suggest that HIV infection is capable of altering the interactions between CATB and its inhibitors promoting, an increase in active CATB secretion, which may contribute to neuronal damage.

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