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Recombinant Tax1 and Tax2 proteins inhibit HIV-1 replication in peripheral blood mononuclear cells

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Patients with HIV-1 and HTLV-2 coinfections often exhibit a clinical course similar to HIV-1 infected long-term nonprogressors. This observation has been attributed in part to the ability of the HTLV Tax2 protein to activate production of antiviral chemokines and to down-regulate the CCR5 co receptor on lymphocytes. Therefore we investigated the possibility that a recombinant Tax2 protein could suppress HIV-1 viral replication *in vitro*. R5-tropic HIV-1 (NLAD8)-infected-PBMCs were treated daily with recombinant Tax1 and Tax2 proteins (dosage range 1-100 pM). Culture supernatants were collected at intervals for 22 days post-infection and assayed for levels of HIV-1 p24 antigen. Treatment of PBMCs with Tax2 protein resulted in significant reduction in HIV-1 p24 antigen levels ($p<0.05$) at days 10, 14, and 18 post-infection compared to HIV-1-infected or mock-treated PBMCs. This was preceded by the detection of increased levels of CC-chemokines MIP-1α/CCL3, MIP-1β/CCL4, and RANTES/CCL5, on days 1-7 of infection ($p<0.05$). Similar, but less robust inhibition was determined in Tax1 treated PBMCs. Addition of Tax2 proteins starting 48 hours previous to infection resulted in a significant inhibition of HIV-1 p24 levels ($p<0.05$ at days 7 to 14 compared to the untreated, R5 infected PBMCs. In contrast, when addition of Tax2 began two days after R5 infection, significant HIV-1 p24 levels were only determined at days 7 and 10 for 1 pM and at day 7 for 10 pM ($p<0.05$). These results support the contention that Tax1 and Tax2 play a role in generating antiviral responses against HIV-1 *in vivo* and *in vitro*.

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