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HIV-1 Tat Upregulates IFN-γ mRNA in Normal PBMCs In Vitro

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IFN-γ, a cytokine produced mainly by T lymphocytes, interferes with viral replication by acting as a powerful immunomodulator, and has a negative effect on HIV-1 Tat-mediated viral transactivation *in vitro*. The production of this cytokine, which is upregulated during acute infection with HIV-1, seems to decrease during progressive HIV-1 infection, perhaps in part by de novo methylation of its promoter, yet the full mechanism for this decrease is still unclear. The HIV-1 Tat protein has been shown to induce the production of several cytokines and interferon-inducible proteins in high quantities, triggering a toxic cascade of events on surrounding cells, yet the role of Tat in the stimulation of these gene products is not fully known. We have assessed the effect of HIV-1 Tat on the production of IFN-γ, since it may provide an explanation for the modulation of these genes and perhaps the subsequent downregulation of IFN-γ itself. Our model system consists of normal PBMCs transfected with plasmids encoding either one-exon or two-exon Tat. We measured changes in IFN-γ mRNA by real time RT-PCR as well as intracellular cytokine levels by flow cytometry. Our results indicate a small consistent increase in IFN-γ mRNA in cells treated with either form of Tat compared to control cells. However, levels of IFN-γ protein assessed by flow cytometry do not yield a consistent pattern, leaving open the possibility that Tat regulates IFN-γ expression at multiple levels.