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HTLV-I Tax inhibits innate antiviral signaling via NF-κB-dependent induction of SOCS1

Soratree Charoenthongtrakul*, Qinjie Zhou, Noula Shembade, Nicole S Harhaj, Glen N Barber, Edward W Harhaj

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The human T cell leukemia virus type I (HTLV-I) inhibits host antiviral signaling pathways although the underlying mechanisms are unclear. Here we found that the HTLV-I Tax oncoprotein induced the expression of SOCS1, an inhibitor of interferon signaling. Tax required NF-κB, but not CREB, to induce the expression of SOCS1 in T cells. Furthermore, Tax interacted with SOCS1 in both transfected cells and in HTLV-I transformed cell lines. Although SOCS1 is normally a short-lived protein, in the presence of Tax, the stability of SOCS1 was greatly increased. Accordingly, Tax enhanced the replication of a heterologous virus, vesicular stomatitis virus (VSV), in a SOCS1-dependent manner. Surprisingly, Tax required SOCS1 to inhibit RIG-dependent antiviral signaling, but not the interferon-induced JAK/STAT pathway. Inhibition of SOCS1 by RNA-mediated interference in the HTLV-I transformed cell line MT-2 reduced HTLV-I replication and p19Gag levels. Taken together, our results reveal that Tax inhibits antiviral signaling, in part, by hijacking an interferon regulatory protein.

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* Correspondence: scharoenthongtrakul@med.miami.edu
Department of Microbiology and Immunology, The University of Miami, Miller School of Medicine, Miami, FL, 33136, USA