Aims
HIV-1 infection correlates with the progression of immunological compromise, physiological changes as well as the neuropsychological dysfunctions identified as NeuroAIDS. Despite tremendous progress in understanding the host-virus interactions occurring after infection, relatively little is known about the biological mechanisms linking HIV-related neuropathology to neural dysfunction and the progression of behavioral abnormalities in vivo. Recent evidence suggests that HIV-accessory proteins may spread throughout brain from HIV-infected cells, inducing neuronal dysfunction that could account for neuropathology and behavioral changes.

Among the viral proteins, Tat is of particular interest. Tat transactivates HIV-1 gene expression, greatly increasing viral expression. However, intact and functional Tat protein exits infected macrophages/microglia and astrocytes, transactivate cellular genes, stimulating signal transduction cascades, and exhibiting chemokine activity through molecular mimicry. Tat causes excitotoxicity and inflammation, inducing oxidative stress and astrogliosis associated with neurotoxicity and brain dysfunction. Accordingly, I propose a themed issue of Current HIV Research focusing on the contribution of one HIV-regulatory protein, Tat, to HIV-sequelae.

Keywords:
HIV-Tat protein, HIV-sequelae, NeuroAIDS, HIV-infected cells, neuropathology

Contents
(Tentative Outline)

(1) Authors: Jay P. McLaughlin*, Jason Paris, Michelle Ganno-Sherwood, Shainnel Eans.
Title of article: "Exposure to HIV-1 Tat protein potentiates ethanol-conditioned place preference and produces reinstatement in mice."
E-mail: jmclaughlin@tpims.org

(2) Authors: Jason J. Paris*. Jay P. McLaughlin
Title of article: sex differences promote differential response to cocaine-CPP in Tat-induced mice.
E-mail: jparis@tpims.org

(3) Authors: Jonathan Geiger*
Title of article: Original research from ongoing Tat studies.
E-mail: jonathan.geiger@med.und.edu

(4) Authors: T. Celeste Napier, Amanda Parsons
Title of article: Review on reward circuitry, or original report pending ongoing experiments
E-mail: Celeste_Napier@rush.edu

(5) Authors: Dianne Langford*
E-mail: tdl@temple.edu

(6) Authors: Avindra Nath
E-mail: avindra.nath@nih.gov

(7) Authors: Marc Kaufman, Dionyssius, Jason Paris, Jay McLaughlin
E-mail: marc.kaufman.mclean@gmail.com

(8) Authors: Stan Thayer
E-mail: sathayer@umn.edu

(9) Authors: Susana Valente
E-mail: Svalente@scripps.edu

(10) Authors: Johnny J. He
E-mail: Johnny.He@unthsc.edu

(11) Authors: Eliezer Masliah
E-mail: emasliah@ucsd.edu

(12) Authors: Rosemarie Booze, Charles Mactutus, Sylvia Fitting
E-mail: BOOZE@mailbox.sc.edu

(13) Authors: Shilpa Buch
E-mail: sbuch@unmc.edu

(14) Authors: Pamela Knapp
E-mail: peknapp@vcu.edu

(15) Authors: Samikkannu Thangavel
E-mail: sthangav@fiu.edu

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