Retrovirology

Poster presentation

A monoclonal antibody against HIV Tat attenuates neurotoxicity via glutamate receptors

Jeffrey Rumbaugh*[1], Muznabanu Bachani[1], Ilia Tikhonov[2], David Pauza[2] and Avindra Nath[1]

Address: [1]Department of Neurology, Johns Hopkins University, Baltimore, Maryland, USA and [2]Institute of Human Virology, University of Maryland, Baltimore, Maryland, USA

Email: Jeffrey Rumbaugh* - jrumbaug@jhmi.edu

* Corresponding author

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HIV dementia is a neurodegenerative disease for which there is no available neuroprotective therapy. Viral proteins, such as Tat, have been implicated as agents of neurotoxicity via multiple mechanisms, including effects on glutamate receptors.

We coincubated HIV-1 Tat protein with a monoclonal antibody against the N-terminal of Tat, in human neuronal cultures. The antibody significantly ($P < 0.005$) attenuated the neurotoxicity caused by Tat alone, as measured by mitochondrial membrane potential. This protection occurred despite previous observations that peptides from the N-terminal of Tat are non-toxic. We hypothesized that the antibodies may have a more indirect neuroprotective function, preventing excitotoxicity at glutamate receptors. Thus, we coincubated Tat and anti-Tat antibody with NMDA and kainate, in our cultures. The Tat-antibody combination attenuated the toxicity seen with NMDA ($p < 0.05$). There was some protection against kainate (AMPA) excitotoxicity, but it was not statistically significant ($p < 0.2$).

The Tat-antiTat antibody complex appears to prevent excitotoxic effects at glutamate receptors, especially NMDA receptors. Host immune responses may influence host susceptibility to the effects of viral proteins, altering HIV complications, such as onset of HIV dementia.