

HIV-1 viral proteins gp120 and Tat induce oxidative stress in brain endothelial cells.

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The blood-brain barrier (BBB) has an important role in the development of AIDS dementia. The HIV-1 envelope glycoprotein (gp120) and transregulatory protein (Tat) of HIV-1 are neurotoxic and cytotoxic and have been implicated in the development of HIV dementia. They are known to cause oxidative stress and are associated with disruption of the BBB. Here, we used an immortalized endothelial cell line from rat brain capillaries, RBE4, to determine whether gp120 and Tat can induce oxidative stress in an in vitro model of the BBB. RBE4 cells were exposed to gp120 or Tat and the levels of reduced glutathione (GSH), oxidized glutathione (GSSG), catalase (CAT) activity, glutathione peroxidase (GPx) activity, and glutathione reductase (GR) activity, and malondialdehyde (MDA) used as measures of oxidative stress. Both gp120 and Tat significantly decreased the levels of intracellular GSH, GPx, and GR and increased the levels of MDA in RBE4 cells, showing that the cells were oxidatively challenged. The ratio of GSH/GSSG, a widely accepted indicator of oxidative stress, was also significantly decreased. These studies show that both of these viral proteins can induce oxidative stress in immortalized BBB endothelial cells.

