Host protein levels correlate with HIV-associated neurocognitive disorder

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Combination antiretroviral therapy has dramatically increased the life expectancy for HIV-infected patients. However, the prevalence of HIV-associated neurocognitive disorders, which may be triggered by inflammation in the central nervous system, has substantially risen.

A new study in the *Journal of Clinical Investigation* suggests that a host protein, heme oxygenase-1, is protective against HIV-associated inflammation and cognitive decline. Dennis Kolson and colleagues at the University of Pennsylvania determined that heme oxygenase-1 levels are low in brains of HIV infected patients.

In HIV infected individuals, heme oxygenase-1 deficiency correlated with cognitive decline and viral load in the brain. In a cell culture model, inhibition of heme oxygenase-1 increased viral load and promoted neurotoxicity. Increasing expression of heme oxygenase-1 in the same model decreased both viral burden and neurotoxic effects.

The results of this study suggest that strategies that increase heme oxygenase-1 may protect patients from HIV-associated neurocognitive disorders.

More information: Heme oxygenase-1 deficiency accompanies neuropathogenesis of HIV-associated neurocognitive disorders, *J Clin Invest*. DOI: 10.1172/JCI72279