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Involvement of miRNAs in the development of HIV-1-associated neurocognitive disorders

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Over the last decade, small non-coding RNA molecules such as microRNAs (miRNAs) have emerged as critical regulators in the expression and function of eukaryotic genomes. It has been suggested that viral infections and neurological disease outcome may also be shaped by the influence of small RNAs. This has prompted us to suggest that HIV infection alters the endogenous miRNA expression patterns, thereby contributing to neuronal deregulation and AIDS dementia. Therefore, using primary cultures and neuronal cell lines, we examined the impact of a viral protein (HIV-1 Tat and Vpr) on the expression of miRNAs due to its characteristic features such as release from the infected and taken up by non-infected cells. Using microRNA array assay, we demonstrated that Tat and Vpr deregulate the levels of several miRNAs. Interestingly, miR-34a was among the most highly induced miRNAs in Tat- or Vpr-treated neurons. Tat and Vpr also decrease the levels of miR-34a target genes such as CREB protein as shown by real-time PCR. The effect of Tat and/or Vpr was neutralized in the presence of anti-miR-34a. Using *in situ* hybridization assay, we found that the levels of miR-34a increases in Tat- and in Vpr-transgenic mice when compared to the parental mice. Therefore, we conclude that deregulation of neuronal functions by HIV-1 Tat or Vpr protein is miRNAs-dependent.