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## Plasma microRNAs as markers of cognitive decline in HIV/AIDS patients

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HIV Associated Neuro-cognitive Disorders (HAND) affect more than half of people living with HIV-1/AIDS. Similar to other neurological disorders the diagnosis of HAND is determined upon manifestation of symptoms. Identification of biomarkers representing the cognitive status of HIV-1/AIDS patients is a critical step for implementation of successful cognitive, behavioral and pharmacological strategies to prevent onset and progression of HAND. We have optimized a protocol to profile the plasma miRNAs using quantitative RT-PCR and it was found that a miRNA signature is capable to distinguish with good sensitivity and specificity HIV-1+/AIDS patients with cognitive impairment from those without cognitive impairment. Here, we have evaluated this miRNA signature in an independent number of patients from the LSU HIV outpatient clinic and confirmed their association with cognitive impairment. We have additionally tested and validated the biomarker miRNA signature in an independent cohort of patients using plasma samples from the CNS HIV Antiretroviral Therapy Effects Research (CHARTER) program. Interestingly, the miRNA biomarkers from the CHARTER cohort showed accuracy, specificity and sensitivity comparable to those obtained at LSU.

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## Primate specific adenosine to inosine RNA editing shapes the transcriptome

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The ADAR enzymes recognize double-stranded RNA and catalyze the deamination of adenosines to inosines (A-to-I) in all metazoan transcriptomes. Primates are particularly prone to A-to-I editing, largely due to the presence of inverted Alu repeats and their ability to form double-stranded structures. A surprisingly large fraction of the human transcriptome contains inverted Alu repeats, typically occurring in introns and UTRs of protein coding genes. Since inosine is structurally similar to guanosine (G), cellular processes such as splicing and translation recognize I as G. We show that inverted Alu repeats, expressed in the primate brain, induce site-selective editing in cis on sites located several hundred nucleotides from the Alu elements. Furthermore, our computational analysis, finds that site-selective editing often occurs close to edited Alu elements. These targets are poorly edited upon deletion of the editing inducers, as well as in homologous transcripts from organisms lacking Alus. We specifically show that a site specific editing event, with functional effects changing the coding sequence of the Glioma-associated oncogene 1 (GLI1), is unique to humans and perhaps closely related primates. We here propose a model whereby primate-specific editing is induced by adjacent Alu elements that function as recruitment elements for the ADAR editing enzymes. The enrichment of site-selective editing with potentially functional consequences on the expression of transcription factors indicates that editing may contribute more profoundly to primate specific transcriptomic regulation than previously anticipated.

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