Cocaine Induces changes in the expression of IncRNAs in T cells

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Cocaine is a commonly abused illicit drug, which poses a major hurdle in the prevention and treatment of HIV-1 infection by both fostering high risk behaviors and facilitating the pathobiology of the virus. Although studies have shown that cocaine significantly promotes HIV-1 replication in T cells, the mechanisms are still not clear. Long, non-coding RNAs (IncRNAs) are a major class of regulatory RNAs that mediate their functions strictly as RNA molecules. LncRNAs such as NEAT1 and NRON have been shown to modulate HIV-1 posttranscriptional expression and replication, respectively. Although cocaine-induced changes in expression of lncRNAs have been studied in brain cells, IncRNAs regulated by cocaine in T cells and their effect on HIV-1 replication are not known. We hypothesize that cocaine induces changes in the expression of lncRNAs in T cells that in turn facilitate HIV-1 replication. To test this hypothesis, we performed IncRNA microarray analysis on Jurkat T cells infected with HIV-1 with or without 1 μM cocaine treatment. We have looked at the expression of 40,173 IncRNAs and found that there is a significant 2-fold increase in 107 IncRNAs, and decrease in 362 IncRNAs with cocaine treatment compared to untreated control. We are in the process of characterizing several candidates for their functional role in HIV-1 replication. Understanding cocaine-induced IncRNA expression changes may provide new insights into development of novel targeted therapeutics to combat HIV-1 infection among the major at-risk demographic of drug users.