

Cocaine reactivates latent HIV-1 by down regulating anti-HIV long-non coding RNAs

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Interplay between long-non coding RNAs (lncRNAs) and mRNAs is rapidly emerging as a key epigenetic mechanism in controlling various cell functions. HIV actively infects and/or persists latently for years by manipulating host epigenetics. Many mechanisms including microRNAs have been proposed to be responsible for the latency induction; but not many studies on CNS. We delineated the influence of HIV on global lncRNAs expression in monocytic cells lines and found that lncRNAs and miRNAs are differentially expressed in actively vs latently infected cells. Our analysis revealed the expression modulation of nearly 1060 lncRNAs, which are associated with differentially expressed mRNAs in active and latent infection, suggesting a greater role of lncRNAs in regulating transcriptional and post-transcriptional gene expression during HIV infection. Importantly, we also discovered that HIV induces expression reversal of more than 150 lncRNAs between its active and latent infection. Further we also found that cocaine reactivates latent HIV-1 by down regulating some of the differentially expressed anti-HIV lncRNAs. The differentially expressed mRNAs (between latent vs active infection) were involved in 155 different biological pathways where immunological networks were most enriched. The pathology specific “gene-expression reversal” and “on-and-off” switching of lncRNAs and associated mRNAs may lead to establish the relationship between active and latent HIV infection, and the role of cocaine in reactivation of latency.

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