Interplay between HIV-1 replication and RNAi effectors

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RNA silencing involving small non coding RNA is a mechanism for gene regulation as well as an innate host cell defence mechanism against viruses. miRNA genes are most often transcribed by RNApolII, and the resulting primary (pri)-miRNA is processed in the nucleus by the RNAse type III Drosha to produce precursor (pre)-miRNA. Pre-miRNAs are then exported to the cytoplasm by Exportin-5 and processed into miRNA/miRNA* (guide/passenger) duplexes through the action of the cytoplasmic type III RNAse Dicer. miRNA/miRNA* is incorporated into the RNA-Induced Silencing Complex (RISC) where miRNA* is degraded, with miRNA serving as a guide for its mRNA target. miRNA-armed RISC targets specific mRNA to inhibit its translation or induce its degradation. Accumulating evidence suggests that the miRNA pathway also controls the replication of both RNA and DNA viruses. We have recently provided evidence for a physiological role of the miRNA-silencing machinery in controlling HIV-1 replication and latency. Type III RNAses Dicer and Drosha, responsible for miRNA processing, inhibited virus replication in both PBMCs from HIV-1 infected donors and in latently infected cells. Additionally, cellular miRNAs can target HIV-1 mRNA to induce latency. Finally, HIV-1 actively regulates the expression of cellular miRNA which regulate virus replication. We will present further evidence and discuss the involvement of miRNA effectors and cellular miRNA in both activation and repression of HIV-1 replication.