

Poster presentation

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## Human miRNAs: an antiviral defense mechanism

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### Background

miRNAs are short 21-24 nt RNAs that mediate post transcriptional repression of target genes. Various reports have shown that miRNAs are capable of repressing the gene expression levels of different viruses, leading to the suggestion that miRNAs are key mediators of host-virus interaction [1]. HIV-1 is a retrovirus known to cause AIDS, one of the major diseases in humans. The *nef* gene of the HIV-1 has been shown to be important for virus repression of CD4+ cells and virus progression. It has also been shown earlier that patients infected with *nef* deleted HIV-1 do not progress from infected to diseased state for longer periods of time, resulting in the Long Term Non-Progressor phenotype [2].

### Materials and methods

We computationally predicted five endogenously expressed human miRNAs to target the *nef* gene of HIV-1 retrovirus. On applying other stringency parameters we could focus on two of the five miRNAs viz. *hsa-mir-29a* and *hsa-mir-29b* as they were predicted to target the *nef* gene, at sites highly conserved amongst other clades of HIV-1 [3].

We then created reporter carrying the *nef* gene inserted downstream of a luciferase reporter. miRNA expression vectors were also made which would express the pri-miRNA when processed and thereby lead to high levels of the miRNA inside the cells. We then identified various cell lines for validating *nef* as a target for *hsa-mir-29a* and *hsa-mir-29b*.

### Results and discussion

Gene reporter assays and ectopic over-expression of miRNAs conclusively showed that human cellular miRNAs *hsa-mir-29a* and *hsa-mir-29b* could bring down the *nef* protein levels and also affect viral replication [4]. These results would provide a better understanding of the mechanisms that could regulate the viral gene expression and human cellular antiviral defense mechanisms whereby miRNAs could serve as potential therapeutics to treat various viral diseases.

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