

Poster presentation

Open Access

Human miRNAs: an antiviral defense mechanism

Kartik Soni*¹, Jasmine K Ahluwalia¹, Sohrab Zafar Khan², Beena Pillai¹, Debashis Mitra¹ and Samir K Brahmachari¹

Address: ¹Institute of Genomics and Integrative Biology, New Delhi, India and ²National Centre For Cell Science, Pune, India

* Corresponding author

from *Frontiers of Retrovirology: Complex retroviruses, retroelements and their hosts* Montpellier, France. 21-23 September 2009

Published: 24 September 2009

Retrovirology 2009, **6**(Suppl 2):P83 doi:10.1186/1742-4690-6-S2-P83

This abstract is available from: <http://www.retrovirology.com/content/6/S2/P83>

© 2009 Soni et al; licensee BioMed Central Ltd.

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.

References

1. Scaria V, Hariharan M, Maiti S, Pillai B, Brahmachari SK: **Host-virus interaction: a new role for microRNAs.** *Retrovirology* 2006, **3**:68.
2. Deacon NJ, Tsykin A, Solomon A, Smith K, Ludford-Menting M, Hooker DJ, McPhee DA, Greenway AL, Ellett A, Chatfield C, et al.: **Genomic structure of an attenuated quasi species of HIV-1 from a blood transfusion donor and recipients.** *Science* 1995, **270**:988-991.
3. Hariharan M, Scaria V, Pillai B, Brahmachari SK: **Targets for human encoded microRNAs in HIV genes.** *Biochem Biophys Res Commun* 2005, **337**:1214-1218.
4. Ahluwalia JK, Khan SZ, Soni K, Rawat P, Gupta A, Hariharan M, Scaria V, Lalwani M, Pillai B, Mitra D, Brahmachari SK: **Human cellular microRNA *hsa-mir-29a* interferes with viral *nef* protein expression and HIV-1 replication.** *Retrovirology* 2008, **5**:117.