Retrovirology

Poster presentation

GBV-C acute infected cells reveal IL-16 cell mediated downregulation of critical hosts proteins involved in HIV-1 replication

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Background

GB virus C (GBV-C) a member of the Flaviviridae, is a non-pathogenic virus, which replicates in lymphocytes and shown to slow viral replication in HIV-1 patients thereby decreasing progression to AIDS. The mechanism by which GBV-C acts remains elusive.

Th1 cytokines are strongly correlated with GBV-C/HIV-1 co-infection, thus allowing for latent seroconversation and longer longevity. IL-16 a potent anti-HIV-1 Th1 cytokine could explain GBV-C role in co-infection. Hijacking host proteins is critical in the replication, infection and expansion of HIV-1 in the human host.

Methods/results

Jurkat T-cells were transfected with (+) strand GBV-C genome and viral load was measured with specific 5'-UTR quantitative digital PCR assay. The GBV-C positive cells were then assayed for IL-16 expression via digital PCR and protein array. The RNA was then hybridized to high density oligonucleotide arrays for total human transcriptome analysis. Over 3000 genes showed differential expression in GBV-C monoinfection however only 20 were critical in HIV-1 replication with significant p-values under (α = 0.05) via two tailed pair wise t-test. The average upregulation of IL-16 in Jurkat T-cells was over 19-fold, p-value 0.001 on the transcription level and secreted protein were similarly upregulated. IL-16 is a known HIV-1 Tat/Rev gene inhibiting cytokine and high density oligonucleotide arrays showed host factors that are 5-fold downregulated that are involved in Tat mediated elongation and Rev mediated importation/exportation. The TAR specific binding protein 1 and the Rev binding protein were both downregulated under GBV-C infection as well as a VPU related binding protein.

Conclusion

GBV-C mediated IL-16 upregulation allows for HIV-1 suppression in co-infected patients. The effects of IL-16 on HIV-1 suppression of Rev/Tat are relativity unknown except that down-regulation of host HIV-1 related proteins give a more conclusive clue to IL-16 suppressive effects. High density oligomicroarrays give new incites to the transcriptome of GBV-C infected patients and their longer longevity when co-infected with HIV-1.