Viral reservoir disruption with Panobinostat and IFN-α: First results

Ciputra Adijaya Hartana\(^1\), Theresa Flynn\(^1\), Amy Sbrolla\(^2\), Carina Bannach\(^1\), Jane E. Blackmer\(^1\), Joshua M. Chevalier\(^1\), Pilar Garcia-Broncano\(^1\), Xu G. Yu\(^3\), Rajesh T. Gandhi\(^1\), Daniel Kuritzkes\(^2,3\), Mathias Lichterfeld\(^1,3,4\)

Affiliations: \(^1\)Ragon Institute of MGH, MIT and Harvard, Cambridge, MA, USA; \(^2\)Massachusetts General Hospital, Boston, MA, USA; \(^3\)Brigham and Women’s Hospital, Boston, MA, USA

Post# 0341

**METHODS**

Upon reactivation of viral transcription, viral reservoir cells are sensitized to immune-mediated killing resulting in the reduction of long-term persistence of virally-infected CD4\(^+\) T cells in ART-treated individuals.

The ACTIVATE study is an ongoing, prospective, randomized, dose-escalation clinical trial, which administered histone deacetylase inhibitor (HDACi) Panobinostat as a latency-reversing agent in combination with pegylated IFNα2a as an innate immune modulator.

**RESULTS**

Figure 3: HIV-1 RNA expression following panobinostat administration

**Figure 4:** The frequency of NK cell activation markers CD38 and NKp30

**Figure 5:** Polyfunctionality of HIV-1 specific CD4\(^+\) T cells

**Figure 6:** HIV-1 DNA levels during treatment

**REFERENCES**
