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Naturally C-Terminally truncated STAT5 (STAT5Δ): a novel negative controller of HIV-1 transcription and expression

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We have previously observed that signal transducers and activator of transcription (STAT) proteins, namely STAT1 and STAT5, are often constitutively activated in the PBMC of most of HIV-1$^+$ individuals; furthermore, most patients are characterized by the dominant expression of a C-terminally truncated isoform of STAT5 (STAT5Δ) [1]. STAT5Δ is also the prevalent isoform of STAT5 found in the chronically HIV-1 infected promonocytic cell line U1, characterized by a constitutive state of viral latency and inducibility of virus expression by PMA or several cytokines. We recently reported that activated STAT5Δ can act as a negative regulator of HIV-1 expression in GM-CSF stimulated U1 cells and IL-2-stimulated PBMCs. Indeed, in U1 cells we have shown that activated STAT5Δ can directly *in vivo* bind to STAT consensus sequences in the HIV-LTR promoter with an impaired recruitment of RNAPol II. GM-CSF also triggered the late activation of an ERK/AP-1 dependent pathway inducing HIV-1 expression in U1 cells. Selective inhibition of this pathway turned off, while inhibitors of STAT5 enhanced viral expression in GM-CSF stimulated U1 cells [2]. We are currently investigating whether the reduced recruitment of RNA Pol II and the consequent decreased viral transcription and delayed kinetics of HIV expression that follow GM-CSF stimulation could be entirely attributed to the negative role of STAT5Δ alone or whether other proteins participate to the negative control of HIV transcription in U1 cells.

References

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