



MEETING ABSTRACT

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Cloned HTLV-1+CD4+, but not CD8+, T-cells display an oncogenic miRNome

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HTLV-1 persistence *in vivo* relies on the persistent clonal expansion of its host cells. These are CD4+ and CD8+ T cells, yet ATL is regularly CD4+. Accordingly, untransformed HTLV-1+CD4+ but not CD8+ T cells cloned from carriers cumulate the features of preleukemic cells, including multinuclearity, chromatin bridges, increased cell cycling and inappropriate telomerase activity. MicroRNAs (miR) modify the maturation of a plethora of T-cells RNA and their deregulation would therefore constitute an appropriate explanation for the Tax-dependent or -independent pleiotropic changes in the phenotype of HTLV-1+CD4+ T cells. As the miRNome of naturally infected untransformed cells has not been investigated to date, we assessed the miR expression profiling of T cells cloned from carriers. Microarray results, confirmed by quantitative RTPCR, showed that, upon infection, CD4+ and CD8+ clones yielded aberrant expression of 15 distinct miRs including miR-34b and miR-494 that were respectively over- and underexpressed in both compartments. The more prominent effect of the infection consisted in the CD4+-restricted overexpression of the cancer-related miRs miR-21, -27b and -23b associated with the CD4+-restricted downregulation of the proapoptotic miR-15 and -16. Data were extended by the analysis of 40 additional CD4+ clones (20 infected). Crossing the miRNome against the whole transcriptome data identified putative miR-targeted genes. *In silico*, those targeted by miR-23b and -27b defined 2 hitherto unknown pathways involving the cell cycle and genetic disorders. Therefore HTLV-1 triggers a phenotype-specific miR signature consistent with the preleukemic HTLV-1+CD4+ phenotype.

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