



POSTER PRESENTATION

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# Cooperation between the human T-cell leukemia virus type-1 p30(II) protein and host cellular factors during oncogenic transformation and retroviral carcinogenesis

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The human T-cell leukemia virus type-1 (HTLV-1) pX-encoded accessory factors, p30II and Hbz, suppress proviral gene expression and help to maintain latency as a prerequisite for the development of adult T-cell leukemia/lymphoma (ATLL). Our laboratory is studying how these viral proteins influence host cellular gene expression and signaling which may contribute to hematological disease progression. There are five clinically-defined stages of HTLV-1-associated disease (pre-ATLL, smoldering T-cell leukemia, chronic T-cell leukemia, acute T-cell leukemia, and non-Hodgkin's T-cell lymphoma) and the transitional molecular events which lead to acute/lymphoma-stage disease are not well understood. We have previously shown that p30<sup>II</sup> interacts with the c-MYC oncoprotein and enhances c-MYC-dependent transactivation and oncogenic potential by stabilizing recruitment of the TIP60 acetyltransferase to p30<sup>II</sup>/c-MYC/TIP60 transcription complexes. We now extend these findings by demonstrating that p30<sup>II</sup> induces acetylation of the c-MYC protein. Acetylation-defective Lys → Arg substitution mutants of c-MYC (R5, K323R/K417R) are impaired for oncogenic foci-formation by p30<sup>II</sup>/c-MYC. Acute/lymphoma-stage ATLL clinical isolates frequently exhibit overexpression of c-MYC, due to 8q24 chromosomal translocations and/or c-myc gene amplification, and the HTLV-1-transformed T-cell-lines HuT-102 and MJG11 display significant acetylation of c-MYC. The p53 tumor suppressor is a downstream target of c-MYC

and, coincidentally, most ATLL leukemic lymphocytes contain high intracellular levels of wildtype p53. Our recent studies demonstrate that p30<sup>II</sup> activates p53 and induces the expression of p53-dependent anti-apoptotic genes which could promote oncogene-activation and contribute to ATLL tumorigenesis. These findings as well as a novel role for p30<sup>II</sup> in the long-term proliferation of lentiviral-p30<sup>II</sup>-transduced primary human T-lymphocytes will be discussed.

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