

# Activation of Tax protein by c-Jun-N-terminal kinase is not dependent on the presence or absence of the early growth response-1 gene product

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## Resumen

The Tax protein of human T cell leukemia virus type 1 plays a major role in the pathogenesis of adult T cell leukemia (ATL), an aggressive neoplasia of CD4(+) T cells. In the present study, we investigated whether the EGR-1 pathway is involved in the regulation of Tax-induced JNK expression in human Jurkat T cells transfected to express the Tax protein in the presence or absence of PMA or ionomycin. Overexpression of EGR-1 in Jurkat cells transfected to express Tax, promoted the activation of several genes, with the most potent being those that contained AP-1 (Jun/c-Fos), whereas knockdown of endogenous EGR-1 by small interfering RNA (siRNA) somewhat reduced Tax-mediated JNK-1 transcription. Additionally, luciferase-based AP-1 and NF-kappa B reporter gene assays demonstrated that inhibition of EGR-1 expression by an siRNA did not affect the transcriptional activity of a consensus sequence of either AP-1 or NF-kappa B. On the other hand, the apoptosis assay, using all-trans retinoic acid (ATRA) as an inducer of apoptosis, confirmed that siRNA against EGR-1 failed to suppress ATRA-induced apoptosis in Jurkat and Jurkat-Tax cells, as noted by the low levels of both DEVDase activity and DNA fragmentation, indicating that the induction of apoptosis by ATRA was Egr-1-independent. Finally, our data showed that activation of Tax by JNK-1 was not dependent on the EGR-1 cascade of events, suggesting that EGR-1 is important but not a determinant for the activity for Tax-induced proliferation of Jurkat cells.

## Palabras clave

**Palabras clave de autor:** [ATRA](#); [Tax](#); [c-Jun-N-terminal kinase](#); [Jurkat cells](#); [early growth response-1](#)

**KeyWords Plus:** [T-CELL LEUKEMIA](#); [HTLV-I TAX](#); [KAPPA-B](#); [INDUCED APOPTOSIS](#); [TRANSCRIPTIONAL REGULATION](#); [DOWN-REGULATION](#); [VIRUS](#); [EGR-1](#); [P38](#); [CARCINOMA](#)

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